Report of the New York State Task Force on Flame Retardant Safety

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Text Abbreviations

€ /kg	euro per kilogram
ATO	antimony trioxide
ATSDR	Agency for Toxic Substances and Disease Registry
BDE-47	2,2‘,4,4‘- tetrabromodiphenyl ether (congener)
BDE-85	2,2‘,3,4,4‘- pentabromodiphenyl ether (congener)
BDE-99	2,2‘,4,4‘,5 - pentabromodiphenyl ether (congener)
BDE-100	2,2‘,4,4‘,6 - pentabromodiphenyl ether (congener)
BDE-153	2,2‘,4,4‘,5,5‘- hexabromodiphenyl ether (congener)
BDE-154	2,2‘,4,4‘,5,6‘- hexabromodiphenyl ether (congener)
BDE-171	2,2‘,3,3‘,4,4‘,6 - heptabromodiphenyl ether (congener)
BDE-183	2,2‘,3, 4,4‘,5‘,6 - heptabromodiphenyl ether (congener)
BDE-196	2,2‘,3,3‘,4,4‘,5‘,6 - octabromodiphenyl ether (congener)
BDE-197	2,2‘,3,3‘,4,4‘,6,6‘ - octabromodiphenyl ether (congener)
BDE-201	2,2‘,3,3‘,4,5‘,6,6‘- octabromodiphenyl ether (congener)
BDE-202	2,2‘,3,3‘,5,5‘,6,6‘- octabromodiphenyl ether (congener)
BDE-203	2,2‘,3,4,4‘,5,5‘,6 - octabromodiphenyl ether (congener)
BDE-206	2,2‘,3,3‘,4,4‘,5,5‘,6 - nonabromodiphenyl ether (congener)
BDE-207	2,2‘,3,3‘,4,4‘,5,6,6‘- nonabromodiphenyl ether (congener)
BDE-208	2,2‘,3,3‘,4,5,5‘,6,6‘ - nonabromodiphenyl ether (congener)
BDE-209	2,2‘,3,3‘,4,4‘,5,5‘,6,6‘- decabromodiphenyl ether (congener)
BFRIP	Brominated Flame Retardants Industry Panel
BMF	biomagnification factor
Br_4-DE	polybrominated diphenyl ether group containing four bromines
Br_5-DE	polybrominated diphenyl ether group containing five bromines
Br_6-DE	polybrominated diphenyl ether group containing six bromines
Br_7-DE	polybrominated diphenyl ether group containing seven bromines
Br_8-DE	polybrominated diphenyl ether group containing eight bromines
Br_9-DE	polybrominated diphenyl ether group containing nine bromines
Br_{10}-DE	polybrominated diphenyl ether group containing ten bromines
CA EPA	California Environmental Protection Agency
CAS	Chemical Abstract Service
CPSC	Consumer Product Safety Commission
DecaBDE	commercial decabromodiphenyl ether
EC_{50}	concentration causing an effect in 50 percent of the test subjects
ECA	enforceable consent agreement
GC-MS	gas chromatography mass spectrometry
GD	gestation day
IEPA	Illinois Environmental Protection Agency
IRIS	Integrated Risk Information System
K_{ow}	octanol/water partition coefficient
LC_{50}	concentration causing lethality in 50 percent of the test subjects
LCSP	Lowell Center for Sustainable Production
LOEL	lowest-observed effect level
mcg/L	microgram per liter
Mg^{2+}	magnesium ion
mg/kg bw	milligram per kilogram body weight
mg/kg dw	milligram per kilogram dry weight
mg/kg/day	milligram per kilogram per day

mg/L	milligram per liter
MRL	minimal risk level
NCEA	National Center for Environmental Assessment
ng/g	nanogram per gram
ng/g bw/day	nanogram per gram body weight per day
ng/g lwt	nanogram per gram lipid weight
ng/g ww	nanogram per gram wet weight
NHANES	National Health and Nutrition Examination Survey
NOEL	no-observed effect level
NRC	National Research Council
NTP	National Toxicology Program
OctaBDE	commercial octabromodiphenyl ether
OECD	Organization for Economic Cooperation and Development
PA 6 GF	glass-fiber-reinforced polyamide
PBDE	polybrominated diphenyl ether
PBDF	polybrominated dibenzofuran
PCB	polychlorinated biphenyl
PentaBDE	commercial pentabromodiphenyl ether
PND	postnatal day
RDP	resorcinol bis(diphenylphosphate)
RoHS	Restriction of Hazardous Substances
SCHER	Scientific Committee on Health and Environmental Risks
SCTEE	Scientific Committee on Toxicity, Ecotoxicity and the Environment
SIM	selected ion monitoring
SNUR	significant new use rule
TSCA	Toxic Substances Control Act
TRI	Toxics Release Inventory
TURI	Massachusetts Toxic Use Reduction Institute
UL	Underwriters Laboratories
US	United States
US EPA	United States Environmental Protection Agency

Glossary of Terms

Anaerobic: The ability, usually for microorganisms, to be able to live and grow without oxygen.

Appurtenance: Something that is less important than something else. An appurtenance often applies to buildings or property, for example, an annex connected to a larger structure.

Aquatic: Living or growing in water.

Bioaccumulation: A process by which chemicals are taken up by a plant or animal to levels higher than the surrounding environment. Bioaccumulation occurs when a plant or animal is exposed to soil/dust, sediment, water, air or food containing the chemical, and the chemical builds up in living tissues.

Bioaccumulation factor: The concentration of a chemical in tissue of a plant or animal divided by its concentration in the diet or other medium containing the chemical.

Bioavailability: The amount of the original applied dose (exposure) of a chemical that is absorbed and reaches the systemic circulation.

Biochemical debromination: The removal of a bromine atom from a chemical compound by an organism.

Bioconcentration: A process by which chemicals are taken up by an animal or plant to levels higher than in the surrounding water. Bioconcentration differs from bioaccumulation because it refers to the uptake of substances into the organism from water alone. Bioaccumulation is the more general term because it includes all means of uptake into the organism.

Bioconcentration factor: The concentration of a chemical in a tissue divided by its concentration of the chemical in the surrounding water.

Biomagnification: A process by which chemical levels in plants or animals increase from transfer through the food web (e.g., predators have greater concentrations of a particular chemical than their prey).

Biomagnification factor: A number relating the concentration of a chemical in the lipids of an animal to the concentration in the lipids of its prey.

Biomonitoring: Analyzing chemicals, hormone levels or other substances in biological materials (blood, urine, breath, etc.) as a measure of chemical exposure, health status, etc. in humans or animals. A blood test for lead is an example of biomonitoring.

Biota: The animal or plant life of a particular region.

Bolus: The administration of a substance in the form of a single, large dose.

Bromination: A chemical reaction that incorporates a bromine atom into a molecule. The level of bromination can refer to the number of bromine atoms in a molecule.

Borate: A salt of boric acid. A salt is any ionic compound formed by the replacement of one or more hydrogen atoms of an acid with other elements. Examples of boric acid salts are sodium borate and zinc borate.

Cancer potency factor: An estimate (95 percent confidence limit) of the increased cancer risk from a lifetime exposure to 1 milligram per kilogram per day (mg/kg/day) of a chemical.

Carcinogenicity: The ability of a chemical to cause cancer (i.e., a benign or malignant tumor).

Centrilobular hypertrophy (of the liver): Abnormal or excessive enlargement or growth affecting the center of a liver lobule (small divisions of the liver).

Chronic: Occurring over a long period of time, several weeks, months or years.

Congeners (polybrominated diphenyl ether): Any of 209 possible compounds containing diphenyl ether and 1 to 10 bromine atom substitutions on the diphenyl ether ring (see Section 2.1).

Copolymer: A natural or synthetic organic compound of usually high molecular weight consisting of many repeated linked units, each of which is one of two relatively light and simple molecules.

Corpora lutea: Yellow, progesterone-secreting masses of cells that form from an ovarian follicle after the release of a mature egg.

Debromo: The state of not having bromine present at a specific carbon atom of a molecule.

Debromination: A reaction that removes a bromine atom from a chemical compound.

Degradation: The breakdown of a chemical compound into other chemicals.

Deposition: The process by which airborne substances can fall to the ground in precipitation, in dust, or by gravity.

Depurated: The state of an animal‘s digestive tract having been cleared of any accumulated material. This is sometimes done before the animal is analyzed for chemicals.

Detection limit: The smallest amount of substance that a specific laboratory test can reliably and accurately measure in a sample of air, water, soil or other medium.

Diffusion: The movement of atoms or molecules from an area of higher concentration to an area of lower concentration, particularly across cell membranes.

Dimer: A molecule composed of two identical, simpler molecules.

Dose: The amount of substance to which an organism is exposed, typically expressed in milligrams of substance per kilogram of body weight per day.

Ecotoxicity: The identification of chemical hazards to the environment. Ecotoxicity studies measure the effects of chemicals to fish, wildlife, plants, and other wild organisms, as opposed to the effects of chemicals to humans or animal surrogates for humans.

Epidemiology: The study of the study of the causes, distribution, and control of disease in human populations.

Environmental breakdown products: The chemicals formed when a chemical degrades in the environment.

Environmental fate: The processes by which chemicals move and are transformed in the environment.

Environmental media: Parts of a surrounding environment, usually soil, water, or air. The singular of ~~"m~~media" is ~~"m~~medium."

Environmental persistence: The length of time a chemical remains in the environment once it is introduced. Highly persistent chemicals (such as DDT and PCBs) are not easily broken down in the environment.

Epididymis: An elongated structure connected to the back side of the testis that transports, stores, and matures sperms between testis and vas deferens.

Exposure: Contact with a chemical by swallowing, by breathing or by direct contact (such as through the skin or eyes). Exposure may be either short term (acute), or long term (chronic).

Exposure pathways: The way in which a person can come into contact with an environmental chemical, consisting of a source of contamination, an environmental media/transport mechanism, a point of exposure, a route of exposure (eating, drinking, breathing, or touching), and a receptor population.

Gavage: A technique of exposing a laboratory animal (usually a rodent) to a substance by using a tube inserted through the mouth and into the stomach.

Gas chromatography-mass spectrometry: An analytical technique for identifying and measuring concentrations of chemicals (often in mixtures).

Gas phase photolysis: The chemical decomposition of chemicals under the influence of light in the gaseous phase.

Gestation: The period of development in the uterus from conception until birth.

Gravid: Carrying developing young or eggs.

Habituation: A decrease in spontaneous motor activity in response to the diminished novelty of the test chamber. In animal experiments, normal habituation is characterized by a gradual decline in activity as animals become acclimated to a new environment.

Half-life (environmental): The time required for a chemical to be reduced in the environment by one-half of its original concentration. The half-life of a chemical may vary depending on the environmental medium (air, water, soil, etc.).

Hazard: A potential source of harm. Hazard identification (assessment) for human health is the process of determining whether exposure to a chemical can cause health effects (e.g., cancer, birth defects, liver effects, etc.), characterizing the strength of the evidence of causation and the associated exposures (e.g., dose, duration and frequency) at which the health effects are caused. Other hazards can also be assessed such as ecological hazards.

Hepatocellular adenoma: A benign liver tumor.

Hepatocellular carcinoma: An invasive malignant liver tumor that tends to metastasize to other areas of the body.

Histological: Having to do with small structures of animal and plant tissues as discernible with the microscope.

Histopathological: Having to do with microscopic tissue changes characteristic of abnormalities and disease.

Homeostasis: The maintenance of relatively stable internal physiological conditions, such as body temperature or the pH of blood.

Homogenized: Being made uniform in consistency.

Homolog (polybrominated diphenyl ether): A subcategory of polybrominated diphenyl ethers whose compounds (congeners) have the same number of bromines, but at different positions on the diphenyl ether rings (see Section 2.1).

Hyperactivity: A state or condition of being excessively or pathologically active, often characterized by a general restlessness or excess of movement.

Hypoactivity: A state or condition of having abnormally decreased motor and cognitive activity.

Intake: The amount of substance to which an organism is exposed. Intake is similar to dose, but does not consider the body weight of the organism, and is typically expressed in milligrams of substance per day.

Intumescent: A substance that swells as a result of exposure to heat or fire.

Lactate: A by-product of energy metabolism produced during exercise. Accumulation of lactate in the blood is an indicator of oxygen use and/or deprivation in muscles.

Lecithin (egg): Any of a group of phospholipids (the main component of cell membranes) found in egg yolks.

Light absorbance spectrum: The absorption of light as a function of wavelength.

Limit of detection: The smallest amount of substance that a specific laboratory test can reliably and accurately measure in a sample of air, water, soil or other medium.

Lipophilicity: The ability to be attracted to or be soluble in fats or oils.

Liver microsomes: A small vesicle-like structure that is formed when liver cells are broken up in the laboratory. Microsomes are important tools for investigating metabolism of chemicals and drug interactions.

Liver thrombosis: The formation of a clot in the blood that either blocks, or partially blocks a blood vessel in the liver.

Mattress ticking: A strong, tightly woven fabric of cotton or linen used to make mattress coverings.

Meta-analysis: A statistical analysis that combines the results of several studies that address a common hypothesis or subject.

Metabolism: All the chemical reactions that enable the body to work. For example, food is metabolized (chemically changed) to supply the body with energy. Chemicals can be metabolized by the body and made either more or less harmful.

Metabolite: A substance produced by metabolism.

Micropipette: A laboratory device consisting of a very fine-pointed tube used to measure and transfer very small volumes of liquid.

Neonatal: Pertaining to the newborn, typically in the first 28 days after birth.

Neoplastic nodule: A small, abnormally growing mass of tissue or aggregation of cells. Neoplastic nodules can be benign, pre-cancerous or cancerous.

Neurotoxic: Poisonous or damaging to nerve cells or nerve tissue.

Octanol/water partition coefficient (Kow): The ratio of the concentration of a chemical in octanol and in water at equilibrium and at a specified temperature. The octanol/water partition coefficient is a measure of the differential solubility of the chemical between these two solvents.

Oligomer: A molecule that contains a small number of repeating, identical chemical structural units (see Section 3.3.1 and Table 3.3.1.1-1).

PBT Profiler: A modeling approach based on chemical structure that is used as a screening tool to predict a chemical‘s potential to be persistent and bioaccumulative in the absence of measured data.

Phenyl rings: An aromatic functional group in organic chemistry that contains six carbon atoms arranged in a cyclic ring structure, five of which are bonded to hydrogen atoms (see Section 2.1 and Figure 2.1-1).

Photochemical debromination (also called photodebromination): A reaction catalyzed by light that removes a bromine atom from a chemical compound.

Photolysis: Chemical decomposition induced by light.

Polymer: Any of numerous natural or synthetic organic compounds of usually high molecular weight consisting of many repeated linked units, each of which is a relatively light and simple molecule (e.g., polyethylene).

Postnatal: Of or occurring after birth, especially during the period immediately after birth.

Reference dose: An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of adverse noncancer health effects during a lifetime.

Resin: The viscous, hydrocarbon secretion of many plants (particularly coniferous trees), or synthetic substances of similar properties. Resins are useful in the production of varnishes, adhesives and various types of coatings.

Risk: The chance or probability of harm or loss. A risk assessment for humans evaluates scientific information on the hazardous properties of chemicals (hazard identification), the dose-response relationship (dose-response assessment), and the extent of human exposure to those chemicals (exposure assessment) for the purpose of drawing conclusions about the probability that populations or individuals so exposed will be harmed and to what degree (risk characterization). Risks can also be assessed for animals, plants, etc.

Seminal vesicle: Either of two small saclike glands, located on each side of the bladder in males that add nutrient fluid to semen during ejaculation.

Sensitization: The state of being sensitive to a chemical, usually involving an immune response, as a result of previous exposure or exposures to the chemical.

Solar radiation spectrum: The intensity of sunlight as a function of wavelength.

Subchronic: Repeated exposure for more than 30 days, up to approximately 10 percent of the life span in humans. Subchronic exposure is typically more than 30 days up to approximately 90 days in laboratory experiments involving rats or mice.

Terrestrial: Of or pertaining to land, as distinct from water.

Terrestrial temperate zone: Land in either of two regions of the earth of intermediate latitude, the north temperate zone, between the Arctic Circle and the Tropic of Cancer the south temperate zone, between the Antarctic Circle and the Tropic of Capricorn.

Thyroxine: The major hormone secreted by the follicular cells of the thyroid gland. Thyroxine is also called T4.

Ticking: The cloth case of a mattress.

Triiodothyroxine: A thyroid hormone more potent than thyroxine and triiodothyroxine but present in smaller quantities. Triidothyroxine is also called T3.

Tocolytic: Of or being an agent that inhibits uterine contractions in labor.

Troposphere: The lowest layer of the atmosphere, 6 miles high in some areas and as much as 12 miles high in others, within which there is a steady drop in temperature with increasing altitude and within which nearly all cloud formations occur and weather conditions manifest themselves.

Ubiquitous: Existing or being everywhere, especially at the same time; omnipresent.

Undissociated: The state of not being separated into simpler groups of atoms, single atoms, or ions.

Uptake: Absorption of substances by tissues or body organ systems.

V-0: A designation for flammability in the Underwriters Laboratories 94 Test for Flammability of Plastic Materials. Plastics that show the highest degree of flame retardancy (i.e., resistance to combustion) are rated V-0, which means that burning stops within 10 seconds after two applications (10 seconds each) of a flame to the test material, with no flaming material dripping from the surface of the tested plastic.

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Executive Summary

Chapter 387 of the Laws of New York, 2004 established the New York State Task Force on Flame Retardant Safety (hereafter Task Force). The charge of the Task Force is to "review and report on relevant studies, risk assessments, findings or rulings in connection with the flame retardant decabrominated diphenyl ether [decaBDE], including but not limited to any issued by the United States Environmental Protection Agency and the European Union," and to "evaluate the availability of safer alternatives to the flame retardant decabrominated diphenyl ether, including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes."

Fires are a major cause of property damage, injuries and death in the United States and in New York State and pose a risk to firefighter health and safety. To reduce risks associated with fires, state and federal laws and regulations aim to improve the fire safety of building materials, furnishings and transportation-related materials. In addition, voluntary industry standards seek to improve fire performance characteristics of commercial materials to reduce fire risk. Materials and substances that retard flame are one mechanism to help reduce the risk and dangers associated with fires.

To fulfill its charge, the Task Force summarized the more recent risk assessments, findings or rulings by the United States Environmental Protection Agency (US EPA) and the European Union on decaBDE. The Task Force also identified examples of alternatives to decaBDE in its major applications that manufacturers have developed through chemical substitution, material substitution, and combinations of chemical and material substitution and briefly outlined product redesign possibilities. For each decaBDE alternative involving another chemical, a comparative summary for toxicity, persistence, bioaccumulation, ecotoxicity, and breakdown products is provided for the alternative and BDE-209. Information on the alternatives' effectiveness as a flame retardant, availability and cost-effectiveness is also summarized.

Decabromodiphenyl Ether

DecaBDE is a commercial flame retardant used in the electronic, transportation, building and textile industries. DecaBDE is a mixture of several chemicals; the chemical that makes up about 97 percent of the mixture is called BDE-209 (chemical name: 2,2'3,3',4,4',5,5',6,6'-

decabromodiphenyl ether). In addition to decaBDE, pentaBDE and octaBDE (commercial mixtures containing a number of PBDEs) have been used in commerce with production beginning in the 1970s. In 2004, pentaBDE and octaBDE were phased out of domestic production. In New York State, the 2004 law that created the Task Force also prohibited the manufacture, processing and distribution of a flame-retardant part of a product containing more than 0.1 percent penta or octaBDE.

BDE-209 can break down to less brominated PBDEs (e.g., containing fewer bromine atoms – including chemicals found in penta and octaBDEs) following exposure to sunlight or through biochemical or chemical processes. How fast this occurs in the environment isn‘t clear. Some of the less-brominated PBDEs are considered to be persistent, bioaccumulative, and toxic chemicals and are more of a concern than BDE-209 for some of these properties.

BDE-209 has been detected in the environment, animals and people. The concentration of BDE-209 in air (both vapor and attached to particulate matter) is low, although it is distributed widely in the atmosphere. BDE-209 is found in sediment in rivers and lakes and in sewage sludge. Its presence in these media can come from sources such as wastewater that gets into water bodies or from airborne particles containing BDE-209. The levels of BDE-209 in fish and wildlife are generally low, indicating that it has not extensively built up (bioaccumulated) in animal tissues. BDE-209 has been detected in food and house dust which can be sources of human exposure. It has been detected in human blood and breast milk.

Short term (acute) and long term (chronic) studies have examined the effects of BDE-209 on a number of different aquatic (water) species, including algae, fish, and aquatic invertebrates. Studies have also examined the effects of BDE-209 in terrestrial (land) species. In general, these studies rarely report adverse effects from BDE-209. Levels of BDE-209 in the environment (despite its widespread occurrence) suggest it is unlikely to cause measurable adverse effects to fish and wildlife. Studies on the health effects of BDE-209 in humans are not available, but studies in laboratory animals have looked at the potential for BDE-209 to cause health effects. Studies (by Viberg and coworkers, Rice et al.) of young rats and mice report that BDE-209 causes adverse effects on the developing nervous system. A recent study (Biesemeier, et al.) was undertaken because of concerns about the adverse effects reported in the earlier studies and because of the methods used in the earlier studies. The recent study reported no nervous system effects in young animals. These studies have different study designs so that interpreting the

significance of the results is difficult. Other animal studies of BDE-209 report adverse effects on the liver and thyroid, and the endocrine and reproductive systems. These used higher exposure levels than the studies reporting effects on the nervous system. National Toxicology Program studies exposed rodents to high levels of BDE-209 for their lifetimes and concluded that BDE-209 showed some evidence of carcinogenicity in male and female rats, with equivocal evidence of carcinogenicity in male mice and no evidence in female mice. Whether or not exposure to BDE-209 increases the risk for cancer in humans is not known.

Estimated BDE-209 exposures can be compared to reference doses, the dose that is estimated to be without appreciable risk of noncancer health effects over a lifetime. The estimated BDE-209 adult exposure is about 3500 times lower than the lowest available BDE-209 reference dose (US EPA). Assuming that estimated PBDE exposure of infants is all BDE-209, the infant dose is about 50 times lower than the lowest BDE-209 reference dose.

In March 2006, the US EPA issued a PBDE project plan. For decaBDE, the US EPA has completed an Integrated Risk Information System assessment (2008), an exposure and environmental fate assessment (2010) and a draft alternatives assessment (2012). The US EPA has given a high hazard ranking to BDE-209 based on developmental neurotoxic effects in rats and concluded that decaBDE can degrade to less brominated PBDEs under certain conditions.

In 2009, the US EPA announced that the two major United States manufacturers (Chemtura Corporation and Albemarle Corporation) of decaBDE and the major United States importer (Israeli Chemicals Limited) agreed to phase out production, importation, and sales for most uses of decaBDE by December 31, 2012, and to end all such domestic production or importation by December 31, 2013. The phase out does not currently affect the resale, reuse, disposal, or continued use of finished products that contain pre-phase out decaBDE, the importation of products manufactured elsewhere containing decaBDE, the sale of products made from recycled materials that contain decaBDE, or minor importers of decaBDE. The US EPA proposed Significant New Use Rule and Test Rule, if finalized in their current form, could affect certain of these products.

The European Union (EU) has also initiated actions related to decaBDE. In 2002, the European Union published a human health and environmental risk assessment for BDE-209. The risk assessment recommended an additional study in mice or rats on the developing nervous system

to assist in evaluating the BDE-209 risks for people. The study has been completed and reported that BDE-209 caused no adverse effects on the developing nervous system of young rats. A 2009 update of the environmental data in the European Union risk assessment concluded that although risks from exposure to BDE-209 have not been identified, newer data suggest the potential for its breakdown in the environment to form less brominated PBDEs. As of 2008, decaBDE is restricted in electronic and electrical equipment produced in the European Union due to its Restriction of Hazardous Substances directive.[1]

The most recent actions by the EU occurred late in 2012. In September 2012, the EU proposed to identify BDE-209 as a substance of very high concern. The draft conclusion is that decaBDE or BDE-209 is considered to be a persistent, bioaccumulative and toxic (PBT)/very persistent very bioaccumulative) (vPvB) forming substance because of the high probability that it forms substances in the environment that have PBT/vPvB properties. In December 2012, after reviewing public comments, the EU finalized its decision to include BDE-209 on its list of substances of very high concern.

Decabromodiphenyl Ether Alternatives

Manufacturers have developed alternatives to using decaBDE that can still meet flammability standards by chemical substitution (replacing decaBDE with an alternative flame retardant), material substitution (replacing a material that requires decaBDE with a material that does not), and combinations of chemical and material substitution (using an alternative flame retardant in a different host material). The Task Force identified specific examples of these alternative strategies that cover the major uses of decaBDE. Magnesium hydroxide in coatings of wires and cables is an example of a chemical that can be substituted for decaBDE. Aluminum alloy casings (rather than plastic) in computer products is an example of a material substitution that eliminates the need for a chemical flame retardant. Resorcinol bis(diphenylphosphate) (RDP) in electronic enclosures and boric acid in fire barriers of mattresses are examples of chemical and material substitution combinations that can be alternatives for decaBDE.

For this report, an alternative is deemed to be safer if the characteristics of persistence, bioaccumulation and toxicity considered together of the alternative and its environmental

[1] A 2005 exemption to this restriction was annulled on procedural grounds by the European Court of Justice in 2007. The ruling did not address any scientific or technical issues related to decaBDE.

breakdown products generally appear to be less than the characteristics of persistence, bioaccumulation and toxicity considered together of BDE-209 and its breakdown products. Making such a conclusion required some judgment because the alternatives and BDE-209 were compared across a number of characteristics and their databases and their strengths and weaknesses varied. Based on the information reviewed, the Task Force concluded that magnesium hydroxide, one of the three alternatives to decaBDE considered, generally appears to be safer than BDE-209 and is available, cost effective and offers reliable flame retardant properties. The majority of the Task Force members concluded that RDP and boric acid, the other two alternatives considered, also generally appear to be safer than BDE-209 and are available, cost effective and offer reliable flame retardant properties. Concerns over data gaps and interpretation of studies meant that representatives from the brominated flame retardant manufacturing industry did not agree with the conclusion for RDP and boric acid.

In this report, stating that an alternative is safer than BDE-209 should not be equated with stating that the alternative is safe, that is presenting no hazard or risk (all chemicals may present some hazards or risks, depending on the situation). This report compares the characteristics of BDE-209 and its breakdown products with the characteristics of possible alternatives and their breakdown products and makes a statement about the relative hazard of the two chemicals. As more information becomes available, the results of the comparisons could change. In other cases an alternative chemical or product design may be preferable to or safer than the particular alternatives considered in this report.

Overall Conclusion

The historical use of decaBDE has led to the widespread presence of BDE-209 in the environment that can result in exposure of fish, wildlife and humans. BDE-209 can also form less brominated PBDEs under some environmental conditions. Certain PBDEs can be more of a concern than BDE-209 because of their persistence, bioaccumulative properties and toxicity. How quickly and how much debromination occurs in the environment is unclear. Thus, BDE-209 is also a concern because of risks posed by BDE-209 breakdown products. The phase out of production, importation, and sales for most decaBDE uses by December 31, 2012, and ending all uses by December 31, 2013 will limit the amount of decaBDE and any debromination products that can be released to the environment over time. The phase out does not currently affect the resale, reuse, disposal, or continued use of finished products that contain pre-phase out

decaBDE, importation of products manufactured elsewhere containing decaBDE, or the sale of products made from recycled materials that contain decaBDE, and will not affect minor importers of decaBDE. The US EPA proposed Significant New Use Rule and Test Rule, if finalized in their current form, could affect certain of these products. The availability, reliability and cost effectiveness of safer alternatives (as defined earlier) for decaBDE in many applications have made the phase out practical, while meeting the need for commercial products that maintain sufficient flame retardant properties.

1 Introduction

1.1 New York State Task Force on Flame Retardant Safety

Chapter 387 of the Laws of New York, 2004 (see Appendix 1)[1] established the New York State Task Force on Flame Retardant Safety (hereafter Task Force). The legislation establishing the Task Force states:

> "There is hereby created the 'state task force on flame retardant safety,' referred to hereafter as the task force…The task force shall, at a minimum:
>
> a. review and report on relevant studies, risk assessments, findings or rulings in connection with the flame retardant decabrominated diphenyl ether, including but not limited to any issued by the United States Environmental Protection Agency and the European Union; and
>
> b. evaluate the availability of safer alternatives to the flame retardant decabrominated diphenyl ether, including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes."

The legislation also states that the Task Force is to have thirteen members, including the Commissioner of Health, the Commissioner of Environmental Conservation and the Secretary of State.[2] The ten at large members of the Task Force are to include "two representatives of organizations whose prime function is the enhancement of the environmental quality of the state; two representatives from the brominated flame retardant manufacturing industry; two representatives with expertise in the area of environmental health from academic institutions; two representatives from industries that manufacture products that use flame retardants and two health care professionals with expertise in the area of environmental health." The Commissioner of Health or his or her designee is to serve as the chair of the Task Force.

[1] The same 2004 New York State law that created the Task Force also prohibited the manufacture, processing and distribution of a flame-retardant part of a product containing more than 0.1 percent pentaBDE or octaBDE.

[2] The enacting language for the Task Force was created in 2004 when the Department of State included the Office of Fire Prevention and Control (OFPC). The Department's participant on the Task Force was John Mueller of OFPC. Effective June 1, 2010, the OFPC's duties and accompanying staff were transferred to the State Division of Homeland Security and Emergency Services.

The Task Force members and their affiliations are in Appendix 2. Task Force meeting and conference call agendas are in Appendix 3.

A draft version of this report was released for public comment on April 30, 2012, and written public comments were accepted on the draft through July 31, 2012. The comments dealt primarily with 1) the report's characterization of developmental neurotoxicity studies on BDE-209, 2) the level of emphasis the report placed on the debrominated breakdown products of BDE-209 and how these factor into the overall health and environmental concerns for BDE-209, and 3) the need to come to definitive conclusions about the relative health and environmental risks for BDE-209 and its alternatives. The task force revised the report in response to the comments. The public comments on the draft version of the report and responses are included in Appendix 4.

1.2 Background

Fires are a major cause of property damage, injuries and death in the United States. According to statistics compiled by the United States Fire Administration, fires resulted in $14.6 billion in property damage, caused 17,675 civilian injuries, and killed 3,430 civilians in 2007 (USFA/NFDC, 2009). In New York State from 2004 to 2008, there were over 265,000 structure fires that resulted in 902 civilian deaths. The incidence of fires, the number of fire-related injuries and fatalities, and the amount of fire-related property loss have declined over the last 25 years (Birnbaum and Staskal, 2004; USFA/NFDC, 2007) at least in part because of the introduction of flame retardants into consumer and industrial products.

Brominated flame retardants represent the largest commercially marketed group of chemical flame retardants because of their low cost and high efficiency (Birnbaum and Staskal, 2004). Polybrominated diphenyl ethers (PBDEs) are a major class of brominated flame retardants. The extensive production and use of PBDEs has resulted in their detection in several environmental media (e.g., air, indoor dust, and sediments) in fish and wildlife, and in human serum, breast milk and other tissues. Two commercial PBDEs, pentabromodiphenyl ether (pentaBDE) and octabromodiphenyl ether (octaBDE) have been phased out of production, leaving decabromodiphenyl ether (decaBDE) as the only PBDE flame retardant currently used in commerce. In recent years decaBDE has been the subject of numerous scientific studies evaluating its toxicity, levels in environmental media and humans, and environmental fate.

These studies have led to concern that the continued use of decaBDE as a flame retardant may pose risks to human health and the environment. The use of decaBDE has been restricted in Europe and in some states. In 2009, the United States Environmental Protection Agency announced that the major United States producers and importer of decaBDE will phase out production, importation, and sales of the flame retardant for most uses by December 31, 2012, and will end all uses by December 31, 2013.

1.3 Strategies for Reporting on Decabromodiphenyl Ether and Identifying Alternatives

The Task Force charge requires reporting on relevant studies, risk assessments, findings or rulings for decaBDE, including those of the US EPA and the European Union. The chemical 2,2‘,3,3‘,4,4‘5,5‘,6,6‘-decabromodiphenyl ether (BDE-209) is the main component of commercial decaBDE. The Task Force report has information on commercial decaBDE use and production (Sections 2.2 and 2.3), BDE-209 analysis (Section 2.4), and scientific studies (e.g., environmental fate, debromination, ecotoxicity, human exposure and toxicity) and risk assessments on BDE-209 (Sections 2.5, 2.6, 2.7, 2.8 and 2.9). Actions of the US EPA and European Union are also summarized (Sections 2.10 and 2.11). The Task Force relied on peer-reviewed scientific literature and published reports from governmental agencies to briefly summarize recent and/or important information on decaBDE. Other, non-peer reviewed literature exists for decBDE or BDE-209, but was not used in this report.

The Task Force charge also requires an evaluation of the availability of safer alternatives to decaBDE, including an assessment of the reliability, ready availability and cost-effectiveness of such alternatives. To evaluate alternatives, information is needed on how decaBDE is used and what possible alternatives are available. DecaBDE has been used primarily (about 80 percent) in high-impact polystyrene enclosures of electronic products, particularly televisions, and in cables and wiring. It has also been used in textiles (about 20 percent) for mattress ticking, drapes and certain institutional upholstered furniture.

The Massachusetts Toxic Use Reduction Institute (TURI, 2005) and the Lowell Center for Sustainable Production (LCSP, 2005) have identified alternative flame retarding methods that could be used to eliminate decaBDE in products. Also, manufacturers have identified methods to eliminate the use of decaBDE and still meet flammability standards.

Chemical Substitution: Chemical substitution is replacing decaBDE with alternative chemical flame retardants. A current example of a chemical substitution for decaBDE is the use of magnesium hydroxide as a flame retardant in the coatings of wires and cables.

Material Substitution: Materials are the physical constituents of a product, such as metal, wood or plastic. Material substitution is replacing a material that uses decaBDE for flame retardancy with a material that does not require decaBDE to retard flame. A current example is substituting aluminum for plastic treated with decaBDE in laptop computer enclosures.

Combinations of Chemical and Material Substitution: In some cases, a manufacturer will reformulate the host material so that an alternative flame retardant can be used. An example of using a combination of chemical and material substitution is the use of an alternative plastic with RDP for electronic enclosures rather than high impact polystyrene containing decaBDE. Another example is introducing mattress fire barriers treated with boric acid as a flame retardant rather than using decaBDE as a flame retardant in the backcoatings and resin binders of the fabric covering of the mattress.

Product Redesign: Product redesign is changing the organization, structure or form of a product such that a chemical flame retardant is not needed. An example of product redesign is increasing the distance or placing a metal barrier between ignitable plastic and current-carrying or heat-generating parts in electronics.

The Task Force‘s approach to its charge was to determine if at least one alternative was available for each of the primary uses of decaBDE. The alternative for each use had to have characteristics that made it likely to be safer than decaBDE and likely to be reliable, readily available and cost effective. For this report, an alternative is deemed to be safer if the characteristics of persistence, bioaccumulation and toxicity considered together of the alternative and its environmental breakdown products generally appear to be less than the characteristics of persistence, bioaccumulation and toxicity considered together of BDE-209 and its breakdown products. Making such a conclusion requires judgment in that the alternatives and BDE-209 are being compared for a number of characteristics and their databases and their strengths and weaknesses will vary.

For this report, an alternative is deemed to be safer if the characteristics of persistence, bioaccumulation and toxicity considered together of the alternative and its environmental breakdown products generally appear to be less than the characteristics of persistence, bioaccumulation and toxicity considered together of BDE-209 and its breakdown products. To make a conclusion, four different comparisons are made.

- The persistence of BDE-209 is compared with the persistence of the alternative.
- The potential for bioacummulation of BDE-209 is compared with the bioaccumulation of the alternative.
- The toxicity of BDE-209 and the alternative are compared considering both noncancer and cancer effects. For noncancer effects, the primary focus of the comparison is on the doses of the effect that occurs at the lowest dose level for each substance. This corresponds to a certain extent in comparing reference doses.
- The breakdown products of BDE-209 are compared to the breakdown products of the alternative. The characteristics of persistence, bioaccumulation, and toxicity are used in the comparison.

No one comparison was considered more important than the others and a weight of evidence approach was generally used. When considered together, all the characteristics of the alternative did not have to be less than all the characteristics of BDE-209 for the alternative to be considered safer. For example, if three comparisons were less for the alternative and one was somewhat more for the alternative, the overall conclusion considering all four comparisons could result in the alternative being considered safer.

In making a conclusion, a judgment had to be made about the importance of the lack of data. In general, the lack of data was considered an uncertainty, rather than resulting in no conclusion. For example, BDE-209 has data that provide some evidence of carcinogenicity. One of the alternatives has not been studied for carcinogenicity although its mutagenicity data are negative. In this case, the positive carcinogenicity results were considered important in the comparison, even though the alternative lacks a cancer bioassay. In other words finding a fact was generally weighed more than not having any data.

In this report, stating that an alternative is safer than BDE-209 should not be equated with stating that the alternative is safe, that is without hazard or risk. All chemicals have some hazards and therefore risks associated with them. Hazard refers to an adverse effect that a chemical can cause. For toxicity, hazard is the chemical's innate ability to cause health effects (can cause cancer, birth defects, nervous system effects, etc.). Hazard assessment considers fewer variables than risk assessment. Risk takes into account how likely it is that a hazardous effect will occur. How much risk there is in any given situation is therefore determined by how exposure occurs, how much, how often and how long a person is exposed, and other factors affecting disease, such as age, gender, health, medication and nutrition. For example, some of the health effects (hazards) that alcohol can cause are dizziness, nausea, liver effects, reproductive effects and even death. Which effect or whether or not a specific effect will occur (e.g., the risk of an effect occurring) depends on how much a person drinks over what period of time, their age, medications, weight, whether they are exposed in utero, etc. In addition, people are exposed to a number of chemicals which can influence risk (risks from more than one chemical could be additive, greater than additive, lower, or not affected by a mixture).

This report generally does not estimate risk from using a particular alternate chemical flame retardant. Estimating risks, especially for new uses of chemicals, is difficult for a number of reasons (e.g., the lack of data on potential exposures to people and the ecosystem) and was beyond the resources of the Task Force. Other organizations (e.g., European Union, US EPA, other states) that evaluated or compared chemical flame retardants primarily compared hazards and not risks.

The strengths and limitations of the conclusions are given for each alternative. The more complete the data for each characteristic of BDE-209 and the alternative, the more reliable the conclusion and the less likely the conclusion will change over time. For example, one of the uncertainties associated with RDP (one of the alternative flame retardants evaluated) is the lack of information on chronic toxicity and carcinogenicity. If information became available showing that RDP caused health effects at much lower doses and if the concentration of one of its breakdown products (phenol) was fairly high in aquatic systems, the conclusion that RDP is safer than BDE-209 might change.

The Task Force also acknowledges the limitations of science in assessing chemicals and recognizes that new tools are being used and developed. Hazard and/or risk assessment is one tool used to help understand the hazards or risk posed by a chemical(s). More recently, life-cycle analysis and sustainability principles have been introduced and used. Research (US EPA, 2013) is being carried out to improve the data and the methods used for assessing chemicals including

- improving risk assessments for mixtures,
- using automated technology to assess chemicals for endocrine disruption,
- integrating molecular biology, chemistry and computer science to assess risks from chemicals, and
- determining if current chemical testing strategies are or are not capturing adverse effects.

The following excerpt gives an interesting perspective (NRC, 2009). It can also be applied to the assessment contained in this report.

> ―Decisions regarding risks and risk changes expected under various risk management options are informed by the availability of risk assessments. The goal of achieving accurate, highly quantitative estimates of risk, however, is hampered by limitations in scientific understanding and the availability of relevant data, which can be overcome only by the advance of relevant research. Decisions to protect public health and the environment cannot await ‗perfection' in scientific knowledge (an unachievable goal in any case); in the absence of the understanding that risk assessments, however imperfect, can bring, it will not be possible to know the public-health or environmental value of whatever decisions are ultimately made. It is therefore important that risk assessments incorporate the best available scientific information in scientifically rigorous ways and that they capture and describe the uncertainties in the information in ways that are useful for decision-makers. Moreover, the goal of timeliness is as important as (sometimes more important than) the goal of a precise risk estimate."

After reviewing possible alternatives (LCSP, 2005; SCI, 2005; WDE/WDOH, 2006; DEPA, 2006, 2007), the alternatives for decaBDE in this report were selected using the concepts listed below.

- Alternatives for all major uses of decaBDE are covered.
- Test data are available to show that the product using an alternative method meets flammability requirements.
- Information is sufficient to show that the alternative method is available, is in use, and (if possible) is cost effective. In the absence of specific information on the costs associated with use of the alternative, an alternative that is commercially available and currently in use is assumed to be cost effective.
- For chemical alternatives, sufficient information is available to evaluate its toxicity and its potential for bioaccumulation and persistence in the environment and to show that it is likely to be safer than decaBDE.

Magnesium hydroxide, resorcinol bis(diphenyl phosphate) (RDP) and boric acid are chemical alternatives discussed in the report. Other alternative chemical flame retardants have been used, including other brominated or chlorinated chemicals, but are not discussed. According to a 2005 report published by the Swedish Chemicals Inspectorate (SCI, 2005) about 370 different chemical flame retardants have been described in the scientific literature and about 70 of these are commercially available. Recently, the US EPA also issued a draft decaBDE alternatives assessment (US EPA, 2012a). Whether or not and which chemicals are being considered or used as flame retardants is likely to change over time as is the assessment of the hazards, risks and benefits. In any specific application or product, a chemical may be used either alone or with other chemical(s) (with or without reformulation of the host material) as an alternative to decaBDE.[3]

The Task Force responded to the mandates of the legislation in completing its report. It discussed other, somewhat related issues, but felt they were outside the mandate of the legislation or that the Task Force did not necessarily have the appropriate expertise for evaluating them. Thus, the Task Force did not include them in its report. These issues included:

[3] Recently, a number of scientists published a statement about brominated and chlorinated flame retardants and alternatives (Birnbaum and Bergman, 2010; DiGangi et al., 2010). The task force did not agree on the role of these references in its report.

- Discussing environmental problems with e-wastes (an issue in other countries - Leung, et al., 2007; Qu, et al., 2007, Gao et al., 2011),
- Completing life cycle analyses for decaBDE or alternative flame retardants,
- Discussing chemicals formed when products containing flame retardants were burned (mentioned briefly in section 1.4.1 for decaBDE, but not for alternative flame retardants), and
- Evaluating the effectiveness of flammability requirements (e.g., CPSC requirements) or flame retardants in saving property or reducing morbidity and mortality.

1.4 Fire Safety in New York State

The following sections provide general background information on several aspects of fire safety in New York State, including health impacts to fire fighters, as well as state and federal laws and regulations, and voluntary guidance.

1.4.1 The Incidence of Fire in New York

From 2004 to 2008 fire departments in New York State reported more than 265,000 structure fires that resulted in over 900 deaths and more than $1.1 billion in property loss (not including New York City) (Table 1.4.1-1).

Table 1.4.1-1: Fires Reported in New York State, 2004–2008

Structure Fires	Vehicle Fires	Other Fires	Property Loss	Civilian Deaths	Firefighter Deaths
267,692	50,701	147,784	$1,102,461,464[a]	902	30

[a]Does not include property loss for New York City.

1.4.2 Health Impacts to Fire Fighters

In addition to injuries and death, firefighters are also exposed to products of combustion and other chemicals at fire scenes during flaming combustion, overhaul, and fire investigation and salvage activities. A recent meta-analysis that reviewed 32 epidemiology studies on cancer risk among firefighters reported significant associations between firefighting and 10 types of cancer

(Lemasters et al., 2006). Four cancers (multiple myeloma, non-Hodgkin‘s lymphoma, prostate and testicular cancers) were designated as a –probable risk" based on the authors‘ assessment of the strength of the collective epidemiological evidence from the reviewed studies.

Firefighters responding to fires involving materials containing brominated flame retardants or other flame retardants are exposed to their combustion byproducts in addition to other combustion products. Burning materials that contain brominated flame retardants, including polybrominated diphenyl ethers such as decaBDE, can form brominated compounds such as hydrogen bromide (a corrosive, irritating gas) and brominated dibenzo-*p*-dioxins and dibenzofurans (similar to chlorinated dioxins and furans – Sakai, et al., 2001; Ebert and Bahadir, 2003; Weber and Kuch, 2003).

1.4.3 Fire Safety Efforts

Hostile fires (i.e., those not confined to their normal habitat) occur when a source of heat and a source of fuel come together, usually in an unintentional manner. Fire safety efforts are directed at preventing fires from occurring and minimizing the effects of fires on people and property when prevention efforts fail.

Fire safety efforts can include 1) preventing ignition, 2) limiting the combustibility of structures and their contents, 3) suppressing fires through the use of fixed extinguishing systems, and 4) providing for rapid evacuation of occupants in the event of a fire. Fire safe design of buildings, their contents, consumer products and vehicles depends on a balanced combination of these fire safety efforts.

Preventing ignition typically involves using engineering design and controls, such as the proper design and installation of a gas furnace or safe design of a home‘s electrical distribution system. Preventing ignition can also be accomplished by physically preventing the spread of fire using structural components that resist the passage of flames and smoke, such as corridor smoke barrier doors and exit stairway enclosures.

Suppressing fires with fixed extinguishing systems involves the rapid application of an extinguishing agent through a system engineered to control a fire and prevent it from spreading.

Examples include sprinkler systems that use water or extinguishing systems that use chemical fire suppressants, such as those used for commercial cooking operations.

Providing for rapid means of egress involves notifying occupants about a fire and having adequate ways for people to escape. Notification is accomplished through smoke alarms and fire alarm systems. Proper design and maintenance of corridors and exit stairs help ensure quick egress.

1.4.4 New York State Requirements

In New York, requirements for fire safety are established in the New York State Uniform Fire Prevention and Building Code (Uniform Code), which has eight specific codes on general building construction, residential building construction, fire prevention, existing buildings, plumbing, mechanical systems, property maintenance and fuel gas systems. The Uniform Code provides minimum requirements designed to safeguard public safety, health and general welfare through requirements for structural strength, means of egress, sanitation, adequate light and ventilation, and energy conservation. The Uniform Code also contains stringent building construction and maintenance standards that are consistent with nationally recognized good practices that are intended to provide a reasonable level of life safety and property protection from the hazards of fire, explosion or dangerous conditions in new and existing buildings, structures and premises. The Uniform Code is applicable throughout the State with the exception of New York City, which promulgates its own code that contains equivalent or more stringent requirements. The Uniform Code is administered and enforced by local governments.

The Residential Code of New York State (part of the Uniform Code) contains fire performance requirements for wall and ceiling finishes, foam plastic trim and insulation products in one and two family dwellings and multiple single-family dwellings (i.e., townhouses) (Appendix 5, Table 2).

The Building Code of New York State and the Fire Code of New York State (both are parts of the Uniform Code) contain fire performance requirements for interior wall, ceiling and floor finishes, curtains, draperies, hanging and decorative materials; foam plastic materials and insulation; certain plastics; motion picture screens; upholstered furniture, mattresses, artificial vegetation and tents in buildings not covered by the Residential Code (Appendix 5, Table 3).

Some of these requirements are specific to certain occupancies (e.g., assembly and institutional occupancies).

1.4.5 Federal Requirements

The federal Consumer Products Safety Act and the Flammable Fabrics Act direct the United States Consumer Product Safety Commission (CPSC) to establish fire performance requirements for textiles used in clothing (including vinyl plastic film) and children‘s sleepwear. The regulations under these statutes establish classes of fabric flammability and minimum standards for flammability of textiles and vinyl film used in clothing. They also require that children‘s sleepwear be either flame resistant or snugfitting. Other regulations under these statutes cover carpets and rugs for resistance to small ignition sources, mattresses and mattress pads for resistance to cigarette and open flame ignition, and cellulose insulation for flame resistance.

Other federal statutes establish fire performance requirements. The National Traffic and Motor Vehicle Safety Act directs the National Highway Traffic Safety Administration to establish fire performance requirements for the interiors of motor vehicles with respect to burn resistance. The Federal Railway Administration establishes fire performance requirements for passenger rail cars and for railroad locomotive cab interiors under the Federal Transportation Act. The Federal Aviation Act directs the Federal Aviation Administration to establish fire performance requirements for materials used in aircraft compartments for passengers, baggage and cargo and for thermal and acoustic materials. The federal requirements generally supersede state requirements and preclude states from promulgating regulations in these areas. A summary of selected federal regulations that establish fire performance requirements is found in Appendix 5, Table 4.

1.4.6 Voluntary Industry Standards

There are many voluntary standards pertaining to fire performance requirements of materials and consumer products that are not required by state or federal governments. These are generally developed by a standards organization (e.g., Underwriters Laboratories [UL]). The existence of a voluntary industry standard may result in the state or federal government not establishing a mandatory requirement through regulation. Selected voluntary industry standards are summarized in Appendix 5, Table 5.

1.4.7 Conclusion

In conclusion, fires have caused property damage, injuries and death in New York State, and fire fighters experience increased risks to their health and safety. These risks include those for lung and heart disease, and cancer. To reduce the risks associated with fires, numerous state and federal laws and regulations have been enacted to improve the safety, including fire safety, of building materials, furnishings and transportation-related materials.

2 Decabromodiphenyl Ether

DecaBDE has been studied for its presence in environmental media, environmental fate, debromination to lower PBDE congeners, ecotoxicity, potential human exposure pathways, and toxicity. Several major reviews on decaBDE and its potential alternatives have been published (see Appendix 6) and several US states have pursued legislative initiatives to limit its use (see Appendix 7). The following sections are not a comprehensive review, but briefly summarize the more recent and/or important studies on decaBDE. Most of the studies are on BDE-209, the primary congener component of commercial decaBDE.

2.1 PBDE Nomenclature

PBDEs are a group of organic chemicals that have been widely used as flame retardants. Their chemical structures are variations on the chemical diphenyl ether, which is shown in Figure 2.1-1:

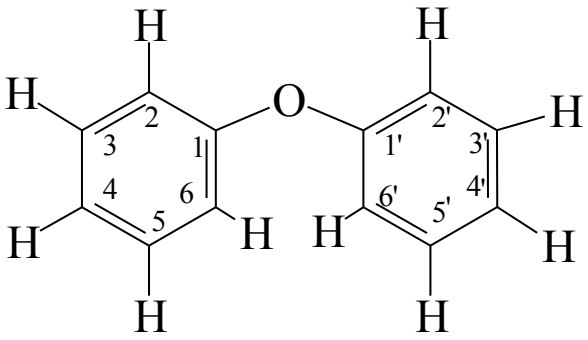


Figure 2.1-1: Diphenyl Ether

Diphenyl ether has hydrogen atoms (H) attached at the carbons numbered 2 to 6 and 2‘ to 6‘ in Figure 2.1-1. The fully brominated compound, 2,2‘,3,3‘,4,4‘,5,5‘,6,6‘-decabromodiphenyl ether (BDE-209, Chemical Abstracts Service (CAS) Registry number 1163-19-5), contains a total of ten bromine atoms in place of the hydrogen atoms, as shown in Figure 2.1-2:

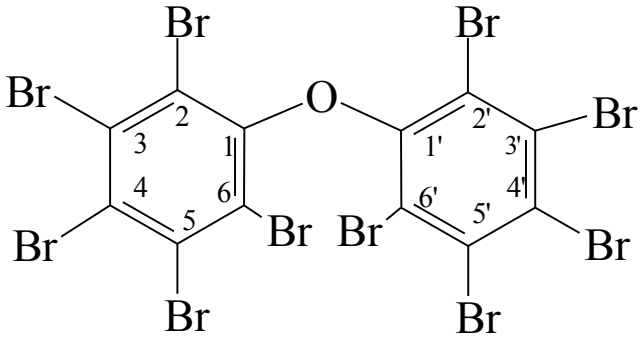


Figure 2.1-2: 2,2',3,3',4,4',5,5',6,6'-Decabromodiphenyl Ether (BDE-209)
CAS Number 1163-19-5
Empirical Formula: $C_{12}Br_{10}O$
Molecular Weight 959.2

Other PBDEs contain one to nine bromine atoms at various positions on the phenyl rings. The number of possible chemical structures depends on the number of bromine atoms. For example, 46 possible compounds contain five bromines, three possible compounds contain nine bromines and only one contains ten bromines. Considering all the ways that hydrogen and bromine can be positioned, 209 unique PBDE chemicals are possible, containing one to ten bromines. These individual chemicals are called congeners.

2.2 Congener Composition of Commercial Mixtures

Commercial PBDE flame retardant products contain mixtures of congeners formulated to contain a specific average number of bromines per congener. In this report we refer to the commercial mixtures containing an average of five, eight or ten bromines per molecule as pentaBDE, octaBDE and decaBDE, respectively. We refer to groups containing a specific number of bromines as homolog groups and write them as Br_x-DE, where *x* is the number of bromines per molecule. For example, Br_5-DE refers to the congeners containing five bromines. Specific congeners will be identified as BDE-xxx, where xxx follows the numbering system developed by Ballschmiter et al. (1992). For example, the congener shown in Figure 2.1-2 is BDE-209, it belongs to the congener group Br_{10}-DE (in fact, it is the only member of that group) and it is the major component (97 percent) of the commercial flame retardant decaBDE.

While many congeners are possible, most are not formed in large quantities during manufacturing. Therefore, the commercial PBDE products are relatively simple mixtures of a

few major congeners. Table 2.2-1 summarizes information on the commercial products by group and individual congeners. The congeners listed make up more than 90 percent of the total amount of the respective mixtures that have been sold in the United States (La Guardia et al., 2006).

Table 2.2-1: Commercial PBDE Flame Retardant Mixtures by Homolog Group and Individual Congeners

Commercial Mixture	Homolog Group*	Major Congeners*
PentaBDE	Br_4-DE	BDE-47
	Br_5-DE	BDE-99, BDE-100
	Br_6-DE	BDE-153, BDE-154
OctaBDE	Br_6-DE	BDE-153
	Br_7-DE	BDE-183
	Br_8-DE	BDE-196, BDE-197
	Br_9-DE	BDE-207
DecaBDE	Br_{10}-DE	BDE-209

*See Text Abbreviations on page ix of this document.

There are some relatively minor differences between United States and European products. Examples for some, but not all, products are described. PentaBDE (formulated as DE-71, among others, in the United States; Bromkal 70-5DE in Europe) contains 3 congeners (BDE-99 >47 >100) that account for over 85 percent of its mass, with most of the remaining mass from 3 additional congeners (BDE-85, 154, 153). OctaBDE formulated in the United States as DE-79, among others, is dominated (greater than 90 percent by mass) by 5 congeners (BDE-183 >197 >207 ≈196 ≈153) with small contributions from 6 additional congeners (BDE-154, -180, -171, -203, -206, -209). The European octaBDE product (Bromkal 79-8DE) contains less of the lighter congeners and more of the heavier congeners (BDE-209 >>183 ≈207 ≈197 ≈203 ≈206 >196). DecaBDE has been sold in the United States as Saytex 102E and as DE-83R and in Europe as Bromkal 82-0DE. BDE-209, the fully-brominated decabromodiphenyl ether congener, comprises greater than 90 percent of the mass of both products. Saytex 102E has a small amount (2 percent) of BDE-206, the Br_9-DE congener unsubstituted at the 2-position, and Bromkal 82-0DE has small amounts (4–5 percent each) of BDE-206 and BDE-207, the Br_9-DE group unsubstituted at the 3 position.

2.3 Background, Production and Use

Five major classes of brominated flame retardants (PBDEs, and brominated bisphenols, cyclododecanes, phenols and phthalic acids) have been used in consumer products (Birnbaum and Staskal, 2004). In addition to decaBDE, pentaBDE and octaBDE have been used in commerce with production beginning in the 1970s (ATSDR, 2004a). In 2004, pentaBDE and octaBDE were phased out of production by the Great Lakes Chemical Corporation (now Chemtura Corporation), which was the sole United States manufacturer of these PBDE commercial formulations (GLCC, 2003). In 2009, the US EPA announced that the major United States producers (Albemarle Corporation and Chemtura Corporation) and an importer (ICL Industrial Products, Inc.) of decaBDE will phase out production, importation, and sales for most uses of decaBDE by December 31, 2012, and will end all uses by the end of 2013 (see Sections 2.10.2).

Historically, worldwide demand for decaBDE ranked second (behind tetrabromo-bisphenol A) among all brominated flame retardants. The annual demand worldwide was estimated as 56,100 tons in 2001, of which the Americas accounted for 24,500 tons, Asia 23,000 tons, and Europe 7,600 tons (BSEF, 2003). However, this estimate did not include the quantity that appears in finished products containing decaBDE. Worldwide, there have been four major producers of decaBDE, including Albemarle Corporation (Arkansas), the Dead Sea Bromine Corporation (Israel), Chemtura (Arkansas) and US Tosoh Corporation (Japan) (LCSP, 2005).

The major use of decaBDE is as a flame retardant with the largest amount used in electronic products. DecaBDE is used primarily in the high-impact polystyrene enclosures of electronic products, particularly televisions. In televisions, the back panel and sometimes the front panel is made from high-impact polystyrene containing up to 12 percent by weight decaBDE in combination with antimony trioxide (ATO). DecaBDE is also used in a variety of applications as a flame retardant in some products using upholstery textiles, particularly in mattress ticking, drapes and some institutional upholstered furniture.

In the United States, about 80 percent of the decaBDE use has occurred in the enclosures of electronic products (Hardy, 2003). A 2003 study by the American Chemistry Council confirmed this estimate finding that 10 to 20 percent of the decaBDE used in the United States occurs in textiles (ACC, 2003).

The European Commission in its 2002 risk assessment of decaBDE found that it was no longer produced in the European Union (ECB, 2002). However, large quantities were taken into the European Union by at least three importers. The actual European Union consumption during the 1990s was estimated at 8,210 tons per year. In 2001, the European market for decaBDE was estimated at 7,600 tons per year out of a total global market of 56,100 tons (DEPA, 2006). The European Commission further estimated that an additional 1,300 tons per year of decaBDE were imported into the European Union in finished goods. Of this total, some 400 tons per year were imported as constituents of televisions, 500 tons per year were included in other non-television products, and 400 tons per year enter as flame retardants in polystyrene products (ECB, 2002).

A 2006 study of decaBDE emissions in Japan notes that the 2002 demand was 2,200 tons per year and the stock level was about 60,000 tons. The decaBDE flow into the waste stream was estimated to be about 6,000 tons per year and the flow out through second-hand product exports was more than 700 tons per year (Shin-ichi Sakai et al., 2006).

Aggregate production and use data for decaBDE in the United States are difficult to locate. In 2008, the Stanford Research Institute in its Flame Retardants Report provided the following numbers for the consumption of decaBDE in the United States (SRI, 2008):

Table 2.3-1: United States Consumption Estimates for DecaBDE

Year	United States Consumption Estimate (metric tons)
1998	21,600
2001	18,000
2004	19,400
2007	18,000

DecaBDE is an additive flame retardant, meaning that it is mixed into but not chemically reacted and bound to the host material. It can therefore migrate from the host material and be released into the environment. The commercial formulation of decaBDE is typically 97 percent pure BDE-209, and contains less than 3 percent nonabromodiphenyl ether congeners and small amounts of octabromodiphenyl ether congeners (Alaee et al., 2003; La Guardia et al., 2006).

2.4 Analytical Approaches and Issues

Identifying (i.e., which chemical) or quantifying (i.e., how much of the chemical) an individual PBDE or specific homolog group in a sample can be difficult, and studies must be examined carefully to determine their validity. First, because some PBDEs are very similar, determining which PBDE or how much of that PBDE is present is not always possible, even with very sophisticated equipment. Secondly, some PBDEs may be difficult to collect with some sampling equipment. In some cases they may be present in air or other media, but may not be collected by the equipment. Thirdly, PBDEs are present in the environment. If care is not taken, a sample can be unknowingly contaminated. Analyzing field and laboratory blank samples is necessary to determine if accidental contamination may have occurred. Fourthly, a scientist may not look for all PBDEs or PBDE homolog groups. If a paper does not report that a PBDE or homolog group is present, it does not necessarily mean that it is absent. It could be present, but the sample was not analyzed for that particular PBDE or PBDE homolog group.

The gas chromatographic properties of 133 of the possible 209 PBDE congeners, including many not reported in the commercial mixtures, and the detailed composition of some commercial mixtures have been determined (Korytár et al., 2005; La Guardia et al., 2006). Since commercial products of PBDEs are mixtures of congeners, recognition of the congener pattern in a sample aids in identification of source products, and deviation from these patterns suggests action of physical, chemical or biological processes on the source products.

The analytical approach used can influence the interpretation of studies of PBDEs including BDE-209. The typical approach is some variation of gas chromatography-mass spectrometry (GC-MS). Generally, in field studies the MS is operated in the selected ion monitoring (SIM) mode to detect only selected masses in order to achieve lower detection limits. One SIM approach is to operate the MS to only respond to ions with the atomic masses of bromine isotopes. Using this approach only the presence of brominated compounds is determined. Identification of PBDEs requires a retention time match to a specific PBDE congener, with the possibility of interference from a brominated compound that is not a PBDE but has the same retention time. A more definitive SIM approach is to operate the MS to respond only to characteristic molecular or fragment masses of PBDE congeners. This allows detection and classification of PBDE congeners in terms of the number of bromines in a molecule. While a

retention time match to a standard is necessary to identify a specific congener, PBDE congeners can still be identified at the group level without a standard. This is generally the approach used in studies to detect and classify products for which congener-specific standards are not available.

Not all possible PBDE congeners have commercial standards available, particularly, for the congeners unbrominated at the 4-position. Some pairs or groups of congeners are not separated by GC (Korytár et al., 2005; La Guardia et al., 2006). Therefore, even in the best cases it has not possible to identify all PBDEs. Some field and laboratory studies use or report only the major congeners found in commercial PBDE products so that information on other congeners that might be present is not available for those studies.

A recent survey of research on PBDEs in the marine environment (Shaw and Kannan, 2009) illustrates that many studies do not include or report BDE-209 or other heavy congeners in the results. Earlier work may have assumed that decaPBDE congeners would not be present because of low volatility, solubility and bioavailability. Others have encountered analytical difficulties with BDE-209 in particular, either because of high blank values (e.g., Litten et al., 2003) or because of thermal stability of this congener in some GC systems (e.g., Kimbrough et al., 2009). In addition, sampling techniques such as diffusion-based passive air sampling will not capture particle-bound compounds such as BDE-209 (Gouin et al., 2005).

2.5 Debromination

All the major congeners in commercial mixtures discussed above (Table 2.2-1) share the 2, 4-substitution pattern of the smallest congener reported, 2,2‘,4,4‘-tetrabromodiphenylether (BDE-47). The occurrence of congeners that are unsubstituted at the 2, 2‘, 4, or 4‘-position at proportions comparable to or greater than those in commercial mixtures could suggest chemical or biochemical changes to the original source of commercial PBDEs. An exception to this is BDE-208, which is unsubstituted at the 4' position but has been reported in commercial deca-BDE products (Korytár et al., 2005; La Guardia et al., 2006) as well as in many environmental samples.

Laboratory studies of BDE-209 demonstrate that it can be debrominated by photochemical and biochemical processes to form less-brominated PBDEs. Both the debromination products and less-brominated congeners in commercial mixtures can be debrominated as well. The potential

for debromination should be considered when evaluating exposures to decaBDE. Formation of less-brominated products is a concern because these may be more mobile in the environment, more persistent, more bioaccumulative and/or more toxic than BDE-209 itself (US EPA, 2009a). Laboratory studies of debromination are reviewed in Section 2.5. Field evidence of debromination is considered in Section 2.6.

2.5.1 Photolysis Studies

This section gives information about how BDE-209 may react when exposed to sunlight. Laboratory studies of BDE-209 in water and on natural particles show that BDE-209 can form lower brominated PBDEs and other compounds when exposed to sunlight.

The light absorbance spectrum of BDE-209 overlaps the solar radiation spectrum at the earth's surface (Raff and Hites, 2007; Soderstrom et al., 2004). This is true for lesser-brominated congeners as well, with (in general) decreasing absorbance and overlap with decreasing bromination. Photolysis studies at sunlight wavelengths show debromination of BDE-209 in organic solvents (Eriksson et al., 2004; Bezares-Cruz et al., 2004; Fang et al., 2008; Raff and Hites, 2007; Soderstrom et al., 2004), quartz-water, solvent-water and humic acid-water solutions (Hua et al., 2003; Eriksson et al., 2004) and several particle phases (Soderstrom et al., 2004). In all cases, the initial PBDE products are all 3 possible nonabromo congeners (BDE-206, -207, -208). Debromination continues sequentially to at least Br_4-DEs. High quantum yield in solution (10 to 20 percent) combined with large overlap with the solar spectrum give BDE-209 to Br_9-DEs the most rapid reaction rate. As compounds become less brominated, they become less reactive, at least partly because of lower absorbances in the solar spectrum.

The PBDE products formed from BDE-209 are similar in all studies, although proportions depend on matrix and extent of reaction. The products and product ratios differ from commercial congener mixtures. For example, BDE-208, the Br_9-DE that is unsubstituted at the 4-position, formed at similar rates as BDE-206 and BDE-207 (the other two Br_9-DE compounds), although it is present only at very low levels relative to BDE-206 and BDE-207 in some commercial mixtures. At least 5 Br_8-DE products are formed (Eriksson et al., 2004; Soderstrom et al., 2004) and only 3 of these comprise greater than 98 percent of the Br_8-DE congeners reported in commercial mixtures (BDE-196, -197, -203). At high extent of reaction,

polybrominated dibenzofurans (PBDFs) are also formed. This appears to require a 2 or 6-brominated position on a ring ((Raff and Hites, 2007) and possibly an unbrominated 2 or 6-position on the other ring.

Most of the PBDE and PBDF photoproducts of BDE-209 have not been identified at the congener level because of a lack of analytical standards, particularly a lack of 4-unsubstituted standards (La Guardia et al., 2006). Formation of BDE-208 indicates that further formation of other 4-unsubstituted PBDEs is likely. Recent studies of sunlight photolysis of specific congeners in solvents (Davis and Stapleton, 2009) and in dry house dust (Stapleton and Dodder, 2008) find that BDE-208 forms BDE-202 (4,4' debromo), and BDE-207 forms BDE-197 and BDE-201 (4-debromo) in approximately equal amounts. Since BDE-202 is not reported in commercial mixtures and BDE-201 is present only in very small amounts, the authors suggest that these congeners are markers of photochemical debromination of BDE-209 and its products.

Gas phase photolysis of PBDEs is difficult to study because of the very low vapor pressure of all but the least brominated congeners. Experiments with mono and dibromo BDEs (Raff and Hites, 2007) indicate that quantum yields in the gas phase are higher than in solution and may approach one. Dibenzofurans were the only observed products formed in a helium atmosphere. Products that would form in the troposphere are uncertain, but formation of less-brominated PBDEs is unlikely because of a lack of a hydrogen source in the gas phase and competition from oxygen to form a peroxy radical from the primary photoproduct. Gas phase photolysis of BDE-209 in the atmosphere is unlikely because it is essentially entirely bound to particles.

The conclusion given in the first paragraph of 2.5.1 is consistent with that of the European Union‘s report (EA, 2009a) for this issue.

–Overall the new data provide evidence that decaBDE can undergo photolytic debromination when adsorbed onto particulates forming nona- to heptaBDEs (and possibly hexaBDEs) as products. The formation of polybrominated dibenzofurans (tri- to octabromo) also appears to be possible. It is also likely that debromination could occur in other systems where natural reductants are present (e.g., sediments). It is not, however, possible to reliably quantify the rate of degradation in the environment or the yield of debrominated products in the environment.”

2.5.2 Degradation and Metabolism Studies

The degradation or metabolism of BDE-209 has been studied in sludge, fish, birds and mammals. Some of the experiments indicate that BDE-209 can be metabolized to other PBDEs although the amount metabolized is usually small. In many of these experiments, the most abundant PBDE observed in the animals and waste products was BDE-209. Small amounts of lower brominated BDEs could be detected and other oxygenated brominated products could be observed, but not necessarily identified.

Some anaerobic microorganisms are able to debrominate BDE-209. Commercial decaBDE (mostly BDE-209) or specific congeners (BDE-207 or BDE-206) were incubated with sewage sludge under methanogenic conditions for up to 8 months (Gerecke et al., 2005). With primers added, BDE-209 decreased by a factor of 30 percent. Without primers, the rate of decomposition was about half of that with primers. Products included BDE-207 and BDE-208, but not necessarily BDE-206, suggesting that bromines at the 2 or 6 positions were more difficult to remove or were not removed. Br_9-DE and Br_8-DE products observed accounted for 17 percent of the BDE-209 lost. All three of the Br_9-DE congeners could be debrominated to BR_8-DE congeners, some of which are not present or not major components of commercial mixtures that have been characterized. Reaction rates were enhanced by addition of brominated primers (4-bromobenzoic acid, 2,6-dibromobiphenyl, tetrabromobisphenol A, hexabromocyclododecane, decabromobiphenyl). Cultures of specific anaerobic microorganisms are also able to debrominate BDE-209 (He et al., 2006) over 2–12 months. *Sulfospirillum multivorans* formed unidentified Br_7- and Br_8-DEs after incubation for 2 months. Cultures containing *Dehalococcoides* species could not dehalogenate BDE-209, but they could dehalogenate a mixture of Br_8-DEs to produce Br_7-, Br_6-, Br_5- and Br_4-DE congeners.

The uptake and metabolism of BDE-209 in fish fed BDE-209 has been observed (Kierkegaard et al., 1999; Stapleton et al., 2004; Stapleton et al., 2006). Rainbow trout and common carp fed food amended with BDE-209 contained products that included Br_9- to Br_5-DE congeners. In a study of rainbow trout the authors estimated the uptake of BDE-209 to be 3.2 percent (Stapleton et al., 2006). Rainbow trout body burden of PBDEs was about 25 percent BDE-209 and 75 percent identifiable Br_9-, Br_8- and Br_7-DE congeners (Stapleton et al., 2006). In studies with trout and carp with somewhat different doses and dosing regimens, carp contained more Br_8 -,

Br_7 - and Br_6 -DE congeners relative to BDE-209 and Br_9-DE congeners (Stapleton et al., 2004, 2006). In fact, BDE-209 and the Br_9-DE congeners were not detected in carp (Stapleton et al., 2004), but were observed in trout (Stapleton et al., 2006). Metabolism by carp liver microsomes was faster than by trout liver microsomes (Stapleton et al., 2006). Congeners and congener patterns were different from those reported in commercial mixtures. BDE-202, -201, -188 and unidentified congeners were found in both species (Stapleton et al., 2006). Much more BDE-154 was found relative to BDE-153 (Kierkegaard et al., 1999). Major congeners in commercial penta-PBDE mixtures (BDE-47, -99, -100) were not detected.

BDE-209 surgically implanted in European starlings (*Sturnus vulgaris*) redistributed to blood, muscle and liver in the birds (Van den Steen et al., 2007). Some metabolism to Br_9-, Br_8- and Br_7-DE congeners occurred. The most abundant identified product was BDE-207, followed by BDE-208 and BDE-197. With the exception of BDE-208, only major congeners found in commercial mixtures were determined, so the extent to which unusual congeners were formed could not be discerned. Unmetabolized BDE-209 accounted for about 75 percent of the identified PBDEs in muscle tissue.

Morck et al., 2003 studied the metabolism of a single dose of ^{14}C-labelled BDE-209 in Sprague-Dawley rats. This study was designed to help maximize the potential for detecting metabolites by increasing the amount of BDE-209 absorbed by the body. The dose was administered by gavage in a vehicle that was formulated to enhance solubility and optimize absorption. Using this method helps to detect the potential for debromination, but may not give the same results as studies using other dosing methods. The most abundant PBDE observed in the animals and waste products was BDE-209 itself. Small amounts of all three Br9-DEs were the only other PBDEs detected. Fractionation of extracts of tissues and feces led to detection of ^{14}C activity that could not be identified as the original ^{14}C-labelled BDE-209. In other studies BDE-209 was predominantly excreted in the feces as unabsorbed parent molecule (ATSDR, 2004a).

A feeding experiment with Sprague-Dawley rats dosed daily in food with commercial decaBDE (Huwe and Smith, 2007) indicated metabolism of BDE-209. About 50 percent of the administered dose of BDE-209 was recovered in the feces and about 6 percent from other tissues. The most abundant PBDE in tissues and feces was BDE-209. A small fraction was metabolized to observable PBDE congeners. As with sparrows, the greatest PBDE product was BDE-207,

followed by BDE-206, -208 and -197. However, these accounted for less than 5 percent of the missing BDE-209.

The conclusion in the first paragraph of this section is consistent with the European Union's (EA, 2009a). That conclusion follows.

> "Overall there is now strong evidence from both the available laboratory and field data…that metabolism of decaBDE to lower brominated congeners (down to nonaBDEs, octaBDEs and probably heptaBDEs) occurs in a number of species. The yield of these metabolites is generally low (typically <5 per cent of the dosed decaBDE in the various studies), but several studies indicate that other, as yet unidentified metabolites, may also be formed."

2.6 Environmental Patterns

2.6.1 Atmosphere

BDE-209 in air is almost entirely attached to particulate matter. The concentration of BDE-209 in air is fairly low although it is distributed widely in the atmosphere.

DecaBDE is widespread in air across North America (Gouin et al., 2006; Strandberg et al., 2001; Venier and Hites, 2008). It is found in most samples collected from the Gulf of Mexico (Hoh and Hites, 2005) to the Canadian Arctic (Su et al., 2007). The major decaBDE congeners occur almost entirely in the particulate phase (Gouin et al., 2006; Hoh and Hites, 2005; Strandberg et al., 2001; Venier and Hites, 2008) and Gouin et al., report that BDE-209 is the major PBDE congener on air borne particles in their study. BDE-209 is also the major BDE congener on tree bark in the United States and Canada, presumably deposited from the atmosphere (Qiu and Hites, 2008; Zhu and Hites, 2006). Air concentrations are typically higher at urban areas than at rural or remote sites (Strandberg et al., 2001; Venier and Hites, 2008). At a rural site in Canada, concentrations are higher in air coming from populated areas (generally south) than from unpopulated areas (generally north) (Gouin et al., 2006). Concentrations of BDE-209 increased by at least a factor of 10 at sites around the Great Lakes between the periods 1997–1999 (Strandberg et al., 2001) and 2005–2006 (Venier and Hites, 2008). Over the same times, pentaBDE generally decreased. Consequently, BDE-209 went from being a minor component of

the total PBDEs in air to a major component. Similarly, BDE-209 appeared to have increased in arctic air over the period 2002-2004, with a doubling time of 6 years (Su et al., 2007).

Laboratory studies (Section 2.5.1) indicate that BDE-209 can be debrominated to other PBDEs by sunlight. However, field evidence for debromination in the atmosphere is not strong. While none of the field studies cited above included congeners produced only by photodebromination of BDE-209, many studies did include BDE-209 and the three Br_9-DEs. All show that BDE-209 is present in much greater amounts than the Br_9-DEs, which would be the first debromination products. Varying ratios of BDE-209 to the Br_9-DEs might be explained by varying ratios of the decaBDE and octaBDE commercial products. While not conclusive, these results do not point to photodebromination as a major loss mechanism for decaBDE in the atmosphere, nor do they indicate that debromination is an important source of less-brominated congeners in air. However, these studies do not rule out the possibility of photolysis of PBDEs to nonPBDE products.

In spite of strong association with particles and susceptibility to washout, BDE-209 is widely distributed in North American air and tree bark. Simple radial dilution from a point source, without any removal mechanism, explained about 73 percent of the variation in PBDEs in tree bark across the United States and Canada (Zhu and Hites, 2006). The field data extended to greater than 3000 kilometers from the apparent point sources, PBDE manufacturing plants in Arkansas.

Breivik et al. (2006) found that the half-distance for travel of BDE-209 in the atmosphere is about 600 km, based on deposition rates in lakes on a latitudinal transect in Canada. The half-distance is analogous to half-life, in that it is the distance over which the substance travels before half is removed from the atmosphere. Lake Ontario, the southernmost lake used in this study, is approximately 1,700 km from the point source that is the center of the radial diffusion model of Zhu and Hites (Zhu and Hites, 2006). At that distance, the Zhu and Hites model predicts a half-distance of approximately 840 km. The agreement between these two very different approaches suggests that the sources in Arkansas might be responsible for much of the PBDEs, including decaBDE, at rural and remote sites in the United States and Canada. However, there is much more scatter between predicted and observed concentrations at greater distances from the point source, leaving open the possibility of important atmospheric sources and sinks other than the

manufacturing facilities. In fact, generally higher concentrations in the air at urban areas compared to rural and remote locations indicate that local sources are important, especially for human exposure.

There have been some process-based models developed for BDE-209 behavior in the atmosphere. An atmospheric transport model of BDE-209 over the Great Lakes (Raff and Hites, 2007) concluded that this congener should have a net lifetime of 0.8 days in the atmosphere in the vicinity of Lake Superior. Predicted removal was dominated by wet deposition of particle-bound PBDE. The model assumed no photolysis of BDE-209. The short atmospheric lifetime predicted by this model is not consistent with field observations of long-range movement of BDE-209 in the atmosphere. Another model of the atmosphere in the terrestrial temperate zone (Schenker et al., 2008) assumed some particle-phase photolysis and concluded that photolysis accounted for 45 percent of BDE-209 loss from atmosphere in temperate zone. Deposition accounted for 30 percent of the loss, and the remainder was transported out. The net lifetime in the atmosphere was calculated to be 183 days. Model results were very sensitive to photolysis rates in the particle phase and gas-particle partitioning. The authors concluded that debromination could account for the presence of some of the lighter PBDEs found in the atmosphere.

The conclusion in the first paragraph of this section is consistent with that of the European Union's (EA, 2009a) which is given below.

> "The available new data show that decaBDE occurs widely in…outdoor air...In air samples, decaBDE appears to be associated mainly with the particulate phase rather than the vapour phase, and the concentrations of decaBDE in outdoor air are generally <1 ng/m^3 in Europe. Higher levels have been measured in outdoor air from China (up to 50 ng/m^3) and from close to electronic equipment recycling facilities in the United States (up to 11 ng/m^3)…"
>
> "Another interesting point is that decaBDE has been determined in outdoor air at monitoring stations in remote regions (e.g., Alert, Canada and the Waliguan Baseline Observatory in China). Although the levels found are generally very low (up to about 10 pg/m^3 and decaBDE was not detected in all samples analysed)

the results do suggest that long-range transportation of decaBDE can occur. However, given the widespread use of articles containing decaBDE, local sources (e.g., the research stations themselves) cannot be totally ruled out. In addition, the data from Alert, Canada, suggest that the concentration in air at this location is increasing with time."

2.6.2 Aquatic Systems

BDE-209 is found in sediment in rivers and lakes and in sewage sludge. BDE-209 can come from a number of places including point sources such as wastewater discharges or runoff into water bodies or diffuse sources such as atmospheric deposition.

Accumulation of BDE-209 in remote lakes with no obvious local sources indicates that the atmosphere is a source of decaBDE to surface water in the United States and Canada (Breivik et al., 2006; Raff and Hites, 2007). DecaBDE is also found in sewage sludges across the United States, including New York (Hale et al., 2001; La Guardia et al., 2007; Litten et al., 2003; North, 2004) and in sewage discharges (deBruyn et al., 2009; La Guardia et al., 2007; North, 2004). BDE-209 tends to be the major PBDE congener in sludges but is not always the major congener in discharged water. Point sources such as wastewater discharges or diffuse sources such as atmospheric deposition may provide the dominant supply of decaBDE and other PBDEs to surface water, depending on location.

BDE-209 occurs widely in sediments in North America. It is the dominant congener in Great Lakes sediments, generally making up over 90 percent of the total PBDEs (Breivik et al., 2006; Qiu et al., 2007; Raff and Hites, 2007; Song et al., 2004; Song et al., 2005a,b; Zhu and Hites, 2005). Dated sediment core data show that concentrations have been increasing rapidly in the lakes. Fluxes to Lake Erie were estimated to be 1.6 metric tons per year in 2002 (Song et al., 2005a) and 1.3 metric tons per year to Lake Ontario in 1998 (Breivik et al., 2006) and in 2002 (Song et al., 2005a). Loading rates per unit lake surface area are 5–50 times higher to Lakes Erie and Ontario than they are to the upper Great Lakes (Song et al., 2004; Song et al., 2005b). Loading rates were also much lower to remote lakes in New York, Ontario and Quebec than to the lower Great Lakes (Breivik et al., 2006). Nearshore sediments from the Hudson River and East River around lower Manhattan had concentrations of BDE-209 that were 5–10 times less

than reported in Lake Ontario, although these were relatively deep sediments that probably did not represent recent activity (Litten et al., 2003). Sediment-laden runoff from the World Trade Center attack site had concentrations of BDE-209 approaching 1 milligram per liter (mg/L) in the water days after the attack (Litten et al., 2003). Although the types of samples aren't exactly the same, taking into account suspended solids concentrations, this represents a concentration of BDE-209 on particles that is approximately 300 times greater than in Lake Ontario sediment. As with the Great Lakes, BDE-209 is by far the major PBDE congener in the two runoff samples for the Manhattan area.

As with atmospheric samples, sediment samples have not typically been analyzed for PBDE congeners characteristic of photochemical or biochemical debromination. One study (Kohler et al., 2008) found that the pattern of Br_8-DE congeners in Swiss lake sediments was different from that found in commercial mixtures and included congeners (BDE-201, BDE-202) that are not important components of commercial mixtures, suggesting octaBDE in house dust and photodegradation products of decaBDE were contributing to sediment concentrations. A study summarized in the Environmental Agency's update of the European Union BDE-209 risk assessment (EA, 2009a) found a Br_5-DE congener (BDE-126) in some sediments and sewage sludges in Europe. This congener is a known product of debromination of decaBDE and is not found in commercial mixtures, indicating that less-brominated congeners are being formed. Since BDE-209 remains the major congener in most sediments, *in-situ* debromination must be very slow. However, formation of less-brominated congeners may still be environmentally significant because these congeners may be more mobile in the environment.

There are far fewer reports of PBDE concentrations in surface waters than in sediments. BDE-209 is the major PBDE congener on suspended particles in the Niagara River (Marvin et al., 2007). Concentrations approximately doubled from the late 1980s to the early 2000s. Concentrations consistently increased from Lake Erie to Lake Ontario, indicating the effect of the urban/industrial activity along the river. Whole water concentrations were not determined, but (Oros et al., 2005) found that PBDEs were almost entirely in the suspended particle phase in San Francisco Bay waters.

The information in this section is consistent with that found in the European Union assessment. That report (EA, 2009a) notes that BDE-209 has been found in sediment from water bodies and in sewage sludge at a variety of locations.

2.7 Ecotoxicity

This section reviews the available toxicological data for decaBDE in fish and wildlife (summarized in Table 2.7-1), and examines the potential for exposure to decaBDE by fish and wildlife through food chain bioaccumulation. Toxicity testing has shown that decaBDE (or BDE-209, its principal congener) demonstrates little or no acute toxicity; that is, animals are unlikely to die from exposure to a single dose received during a short period of time when exposed to concentrations likely to be encountered in the environment. However, a limited number of studies suggest that decaBDE could have the potential to cause some harmful effects to organisms exposed continuously over long periods of time, or when a sensitive animal is exposed to a sufficiently large dose during a critical period of development. Possible adverse effects include effects on liver metabolic enzymes, and weight loss, and reproductive, neurological, and behavioral effects.

Table 2.7-1: Summary of Ecotoxicity Data for DecaBDE/BDE-209

Species	Test Material	Test	Result/Effect
Algae species *Skeletonema costatum* *Thalassiosira pseudonana* *Chlorella spp.*	decaBDE	72 hr EC_{50} for growth (cell numbers) 72 hr EC_{50} for growth (cell numbers) 96 hr EC_{50} for growth (cell numbers)	At the highest test concentration, growth for all three species was reduced < 50%. $EC_{50} \leq 1$ mg/L
Daphnia magna	octaBDE*	21 day test for growth, reproduction, and survival	NOEC = 0.002 mg/L
Lake trout (*Salvelinus namaycush)*	BDE-209	168 day bioaccumulation study (56 day exposure followed by 112 days for depuration) when exposed to 3.4 and 27.5 ng/g decaBDE in food	No effects to body weight or liver somatic index; % lipid in exposed fish decreased slightly. Biomagnification factor (BMF) = 0.3
Rainbow trout (*Oncorhynchus mykiss*)	decaBDE	120 day feeding study	Liver weight and lactate levels in blood increased after 120 days. LOEC = 7.5–10 mg/kg/day
Orange-red killifish	decaBDE	48 hr LC_{50}	$LC_{50} > 500$ mg/L
Lumbriculus variegatus	decaBDE	28 day study of survival, growth, and reproduction in sediment	NOEC (2.4% TOC) = 4,536 mg/kg dw NOEC (5.6% TOC) = 3,841 mg/kg dw
Earthworm *Eisenia fetida/andrei*	decaBDE	56 day survival, growth, and reproduction study	NOEC ≥ 4,910 mg/kg dw in soil
European starling (*Sturnus vulgaris)*	BDE-209	76 day exposure via internal implant	Birds receiving a total dose of 22.6 mcg BDE-209 via the implant showed a statistically significant loss of body mass beginning on about day 10 of the 76 day exposure
Neonatal mice	BDE-209	Developmental neurotoxicity test; single dose gavage exposure	Neonatal mice exposed on post natal day 3 exhibited deranged spontaneous behavior, learning and memory defects, and derangements of the cholinergic system that worsened with age. NOEL = 1.34 mg/kg bw; LOEL = 2.22 mg/kg bw

* Similar results for decaBDE are inferred from these results.

EC_{50} = concentration causing an effect in 50 percent of the test subjects; ng/g = nanograms per gram; NOEC = no-observed effect concentration; TOC = total organic carbon; LOEC = lowest-observed effect concentration; NOEL = no-observed effects level; LOEL = lowest-observed effect level; LC_{50} = concentration causing lethality in 50 percent of the test subjects; mg/kg dw = milligrams per kilogram dry weight; mg/kg/day = milligrams per kilogram per day; mg/kg bw = milligrams per kilogram body weight.

2.7.1 Aquatic Toxicity

Both short term (i.e., acute) and long term (i.e., chronic) toxicity tests have been conducted with a number of different aquatic organisms, including algae, various fish species, and aquatic invertebrates. Although one fish species showed some signs of stress, adverse effects generally did not occur.

The results of toxicity tests suggest that decaBDE does not appear to be harmful to aquatic life. This is consistent with the conclusions of the European Union's risk assessment of decaBDE (ECB, 2002) which stated (although there is a measure of uncertainty), there is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already. These include a European Commission directive that requires member states to set up collection strategies to ensure the proper treatment, recovery and disposal of waste electrical and electronic equipment, and an ongoing monitoring program in sewage sludge, sediment and air to establish contaminant level trends and levels of any toxic and bioaccumulative degradation products (ECB, 2002). In addition, the use of decaBDE in electrical and electronic equipment has been restricted in the European Union since 2008 (see Section 2.11.2).

Three different species of marine algae were exposed to 1 mg/L of decaBDE in seawater for 72–96 hours. The algae survived the exposure but growth was reduced about 30 percent (Walsh et al., 1987, cited from ECB, 2002). These studies were done above the solubility limit for BDE-209 (less than 0.1 microgram per liter [mcg/L] [ECB, 2002]), which reduces the confidence in the experimental results.

ECB (2002) reported that octaBDE had no observable effects to *Daphnia magna* exposed to concentrations up to 2 mcg/L over 21 days, and therefore it would be unlikely for decaBDE to have adverse effects at this concentration because the toxicity of PBDEs decreases with the level of bromination. When rainbow trout (*Oncorhynchus mykiss*) were exposed to decaBDE at a dose of 7.5–10 milligram per kilogram body weight (mg/kg bw) through their diet for more than 120 days, survival and growth were unaffected, but they showed signs of increased stress (Kierkegaard et al., 1999). The liver size was increased, which is a typical response by fish exposed to environmental contaminants for a long period of time (Heath, 1995). Also, the level

of lactate in the blood increased. This is a response that is normally observed when fish are exposed to very low levels of dissolved oxygen (Virani and Rees, 2000), suggesting that long term exposure to decaBDE could cause metabolic stress in trout similar to that triggered by exposure to low levels of dissolved oxygen. Lake trout (*Salvelinus namaycush*) were given food with concentrations of 3.4 and 27.5 nanograms per gram (ng/g) of BDE-209 for 56 days, and no harmful effects were observed (Tomy et al., 2004). ECB (2002) similarly reported that no harmful effects were observed when orange-red killifish (*Oryzias latipes*) were exposed to concentrations of 500 mg/L of decaBDE for 48 hours. *Lumbriculus variegatus* is a small, aquatic worm very similar in appearance to a common earthworm. In a sediment toxicity study, *Lumbriculus* was exposed to decaBDE for 28 days in sediment, and no harmful effects were observed at concentrations as high as 3,841 milligrams per kilogram dry weight (mg/kg dw) (ECB, 2002 citing Krueger et al., 2001a,b).

2.7.2 Terrestrial Toxicity

Terrestrial organisms have also been the subject of toxicity testing with decaBDE. Like aquatic organisms, very few adverse effects were observed when different terrestrial species were exposed to decaBDE.

ECB (2002) reported a 56-day earthworm reproduction study. Adult worms were exposed to concentrations of decaBDE in soil ranging from 300–5,000 mg/kg dw. The worms exposed to decaBDE actually showed better growth and reproduction than the control worms, and no harmful effects were observed. Van den Steen et al. (2007) injected BDE-209 into European starlings (*Sturnus vulgaris*) using small tubes implanted under the skin that allowed BDE-209 to slowly diffuse into the bird. The average dose was about 3.3 nanograms per gram body weight per day (ng/g bw/day) over the 80 day test. Birds in the treatment group showed a statistically significant loss of body mass about 10 days after the exposure began. This was the only harmful effect of the BDE-209 that was observed.

Toxicity tests of BDE-209 with mammals, primarily mice, rats, and rabbits, are used to indirectly assess the risks to humans. Those same studies can be used to directly describe the toxicity of BDE-209 to wildlife. The available mammalian toxicity studies for BDE-209 are described and referenced in a separate section of the report (Section 2.9 Health Effects), and are not repeated

here. The lowest reported effect level for BDE-209 is 2.22 mg/kg in newly born mice given single oral doses of BDE-209 on postnatal day (PND) 3. These mice were reported to have changes in spontaneous motor activity tests compared to control animals, which continued in the exposed animals as they aged from 2 to 6 months (Viberg et al., 2003b; Johansson et al., 2008). However, study quality limitations for these investigations of BDE-209 developmental neurotoxicity reduce confidence in this effect level (See Section 2.9.1; Goodman, 2009; Hardy et al., 2009). Also, for wild mammals at the post-natal stage of development, the major source of BDE-209 would be their mother‘s milk. The transfer of BDE-209 from diet to milk as observed in lactating cows, is low; on the order of 0.2 to less than 1 percent of that absorbed (Kierkegaard et al., 2007). The small transfer percentage suggests that in the environment, mother‘s milk may be an unlikely source of a sufficient amount of BDE-209 to adversely affect the neurological development of post-natal mammal species. However, the dose of BDE-209 potentially present in mother‘s milk of mammalian wildlife species needs to be more thoroughly investigated.

With respect to environmental toxicity, there is little information currently available to suggest that the concentrations of decaBDE present in the environment are likely to cause measurable adverse effects on wildlife populations. This is consistent with the conclusions of the European Union‘s risk assessment of decaBDE (ECB, 2002) which stated (although there is a measure of uncertainty), there is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already, which include monitoring of birds and environmental media to establish contaminant level trends and levels of any toxic and bioaccumulative degradation products (see Sections 2.7.1 and 2.11 for further discussion). The European Union risk assessment suggested that additional toxicity testing be considered on birds, such as an avian reproduction test.

2.7.3 Bioavailability, Bioaccumulation, and Biomagnification

Few direct effects from decaBDE were observed as the results of toxicity tests with fish and wildlife. However, other important issues are, how available is decaBDE in the environment to fish and wildlife, whether or not they can accumulate decaBDE from the environment into their bodies, and if it can be accumulated to higher doses that are potentially harmful. Bioavailability is the measure of the ability of organisms to transfer contaminants into their bodies from the surrounding environment. Bioaccumulation is a measure of the amount of a contaminant in the

organism compared to the amount of the contaminant in the environment around it. Biomagnification occurs when there is an increase of bioaccumulation by animals at a higher trophic level, that is, animals higher in the food chain. For example, many toxins are bioaccumulated directly from contaminated sediment or soil by various invertebrates such as insects and worms. For a contaminant that can biomagnify, animals that eat the worms and insects would have higher body burdens of the contaminant than the worms and insects that they ate; and animals that eat animals that ate the worms and insects would have even higher concentrations of the contaminant. The very large octanol-water partition coefficient of BDE-209 suggests that it should both bioaccumulate and biomagnify; however, numerous studies suggest that decaBDE does not biomagnify; nor does BDE-209 appear to bioaccumulate to any significant degree.

BDE-209 is a very large molecule, and does not easily pass through most of the pores in cell membranes, meaning that is it not readily bioavailable to biota. BDE-209 also adsorbs, or –sticks" strongly to organic carbon present in soil, sediment and even dust particles, and is not easily displaced. When an animal ingests soil or sediment particles containing BDE-209, it is not easily removed from the sediment or soil particle, and even when it is released, the large molecule does not easily pass through the membranes of the gills, stomach or intestine. Thus, BDE-209 is not readily bioavailable. Despite its low bioavailability, BDE-209 is routinely detected in tissue from both aquatic and terrestrial biota, suggesting that it does bioaccumulate, if only to a limited extent. Burreau et al. (1997) investigated the uptake of larger organic molecules such as BDE-209, and found that uptake may depend upon cotransport with lipids and/or proteins through a mediated, possibly active transport mechanism. The amount of BDE-209 that can be absorbed in this manner is generally low.

2.7.3.1 Aquatic Food Chain Bioaccumulation

DecaBDE appears to be bioaccumulated by aquatic organisms, but not to a very large degree. There is very little evidence that decaBDE biomagnifies. Researchers have observed that BDE-209 is abundantly present in the aquatic environment. For example, De Boer et al. (2003) found BDE-209 was the most abundant PBDE measured in suspended particulate matter, sediment, water flowing out of sewage treatment plants, and industrial wastewater discharges in the Netherlands. Eljarrat et al. (2005) found BDE-209 to be the most abundant PBDE detected in

sediments from two of three sample sites on the Cinca River in Spain. Despite being abundantly present, neither Eljarrat et al. (2005) nor de Boer et al. (2003) detected BDE-209 in fish. De Boer et al. (2003) did detect low concentrations (~5 ng/g) of BDE-209 in both freshwater and saltwater mussels, but the mussels were not depurated[1] before analysis, so the BDE-209 detected could well have been associated with particulate matter and not actually absorbed by the mussel.

La Guardia et al. (2007) found high concentrations of BDE-209 in stream sediments in a watershed downstream of a wastewater discharge from a plastic goods manufacturer in eastern North Carolina as well as in pumpkinseed sunfish (*Lepomis gibbosus*) and crayfish (*Cambarus puncticambarus*). The concentration of BDE-209 in crayfish (21,600 nanograms per gram lipid weight [ng/g lwt]) was about an order of magnitude higher than that found in sunfish (2,880 ng/g lwt). Here in New York, BDE-209 was measured in a variety of fish collected from the Buffalo River. The highest BDE-209 concentrations observed were: bluntnose minnow (*Pimephales notatus*) – 8.15 ng/g; brown bullhead (*Ameiurus nebulosus*) – 0.67 ng/g (carcass); carp (*Cyprinus carpio*) – 0.304 ng/g (edible tissue); and pumpkinseed sunfish – 0.28 ng/g (edible tissue). Interestingly, BDE-209 was not detected in largemouth bass (*Micropterus salmoides*) (Skinner et al., 2009). In Lake Ontario, Ismail et al. (2009) consistently detected low levels of BDE-209 in lake trout (*Salvelinus namaycush*) in samples collected between 1979 and 2004. Concentrations ranged from 0.27–1.3 nanograms per gram wet weight (ng/g ww) or 2.3–12 ng/g lwt from individual homogenized whole fish. Verslycke et al. (2005) found concentrations of BDE-209 in the sediments and in mysid shrimp (*Neomysis integer*) (269–600 ng/g) from the Scheldt estuary in the Netherlands. Like the mussels analyzed by de Boer et al. (2003), the mysids were not depurated before analysis, so the BDE-209 present may have been adsorbed to ingested particles. Booij et al. (2002) examined blue mussels (*Mytilus edulis*) collected along the Dutch coast. They found that after 24 hours depuration, the whole body concentration of BDE-209 dropped from 3,350 ng/g to 50 ng/g. These results seem to confirm that BDE-209 is not readily bioavailable or easily assimilated in aquatic organisms.

Feeding studies with rainbow trout (*Oncorhynchus mykiss*) and carp have shown that, in fact, the uptake of decaBDE is low, and the concentration of decaBDE in the test animals resulting from the feeding was generally lower than the concentration of decaBDE in the food (Kierkegaard et

[1] Depuration means clearing an animal's digestive tract of any accumulated material before analyzing it for contaminants. When animals are not depurated, any contaminant measured might not have been absorbed into the animal's body, but might have been present in or on particles still within the digestive system.

al., 1999; Stapleton et al, 2004; 2006). Concentrations ranged from 0.27–1.3 ng/g ww (2.3–12 ng/g lwt) from individual homogenized whole fish.

BDE-209 does not appear to biomagnify in aquatic organisms, meaning that the concentration of BDE-209 in organisms does not increase with increasing trophic or feeding level. For example, Shaw et al. (2009) found that the accumulation of BDE-209 in harbor seal blubber and their fish prey was about the same. They indicated that because BDE-209 is strongly associated with sediment, marine fishes most likely accumulated BDE-209 from sediment-associated prey such as zooplankton, benthic invertebrates, echinoderms, marine worms, and flatfishes. Their data indicate that although BDE-209 may be accumulated from fish to seals, the biomagnification potential for aquatic organisms was low, with biomagnification factors (BMFs) ranging only from less than 1 to 1.3.

2.7.3.2 Avian Bioaccumulation

Birds are also capable of bioaccumulating decaBDE, and much of the decaBDE that is bioaccumulated is transferred to the eggs. Lam et al. (2007) measured the accumulation of different PBDE congeners in the eggs of four species of bitterns, egrets, and herons from three different industrialized locations in southeastern China. BDE-209 concentrations were quite low, particularly when compared to concentrations of other PBDEs, such as BDE-47 and BDE-154. The exception was the eggs of the Chinese pond heron (*Ardeola bacchus*), where BDE-209 was the dominant PBDE present, comprising 38 percent of the total of PBDEs measured (99 of 220 ng/g lwt) in the eggs. In cattle egret eggs (*Bubulcus ibus*), BDE-209 was the third highest PBDE measured (14 percent, or 28 of 200 ng/g), behind BDE-153 (26 percent) and BDE-183 (20 percent). BDE-209 has also been detected in the eggs of a wide variety of birds of prey in China (Chen et al., 2007a), Norway (Herzke et al., 2005), Greenland (Vorkamp et al., 2005) and Sweden (Lindberg, et al., 2004; Johansson et al., 2009). In particular, the eggs of peregrine falcons have generally been found to contain high concentrations of BDE-209.

Here in New York, BDE-209 has been detected in peregrine falcon eggs from the Albany/Troy area (6.013 ng/g ww) and from the Catskill area (6.740 ng/g ww) (Hudson River Natural Resource Trustees, 2004). BDE-209 has also been reported in herring gull (*Larus argentatus*) eggs from colonies around the Great Lakes at concentrations of 6–20 ng/g in 2006 (Gauthier et

al., 2008). Gauthier, et al. (2008) goes on to speculate that the congener patterns in Great Lakes herring gull eggs suggest that some PBDE congeners may have formed from the metabolic debromination of BDE-209 in birds or possibly even in prey fish. Alternatively, it is also possible that the congeners believed to be present as a result of the metabolic debromination of BDE-209 were, in fact, undocumented impurities from the commercial decaBDE product, or the results of historical environmental contamination from other PBDE products.

While it is clear that BDE-209 can be accumulated by birds, the question remains as to whether or not these concentrations are harmful. Johansson et al. (2009) reported that in Sweden, peregrine falcons with a higher concentration of total PBDEs (as measured in eggs) on average produced a smaller number of young. However, BDE-209 was not the dominant PBDE present and the elevated concentration cannot be attributed to a single congener.

2.7.3.3 Terrestrial Food Chain Bioaccumulation

Like aquatic species and birds, mammals can bioaccumulate decaBDE, but it similarly does not appear to biomagnify. Voorspoels et al. (2006) reported detecting BDE-209 in red fox. They observed that whenever BDE-209 was detected in red fox, it was the dominant congener present. The concentrations of BDE-209 ranged between less than 3–760 ng/g lwt in adipose tissue, liver, and muscle. They conclude that in general PBDE levels in fox were lower than those found in various food sources. In a related study, Voorspoels et al. (2007) suggested that BDE-209 was accumulated as a result of ingesting food externally contaminated with soil or atmospheric particulate matter. Of 33 fox carcasses examined, only five had detectable levels of BDE-209 in more than one tissue, and that detectable amounts of BDE-209 were only found in 4 out of 27, 12 out of 40, and 6 out of 21 samples of adipose, liver, and muscle respectively. The relatively low number of detections of BDE-209 in red fox tissue does not conflict with Voorspoels, et al. (2006) observation that whenever BDE-209 was detected in red fox, it was the dominant congener present and suggests that not all foxes are consistently exposed to environmental sources of BDE-209.

Christensen et al. (2005) examined the concentrations of a variety of persistent organic pollutants in Grizzly Bears in British Columbia including PBDEs. They were able to compare the contaminant loading between a population of bears that regularly consumed salmon (maritime

population) with that of a more interior bear population that rarely consumed salmon. BDE-209 was present in both populations, but the total PBDE content for the maritime population was dominated by BDE-47, while the total PBDE content for the interior population was dominated by BDE-209. Like Voorspoels, Christensen suggests that the source of BDE-209 might be the consumption of terrestrial vegetation contaminated via atmospheric deposition.

The European Union risk assessment for decaBDE (ECB, 2002) suggests that more widespread monitoring should be considered to determine whether the finding of decaBDE in top predators (including birds‘ eggs) is a widespread or localized phenomenon, and trends (if possible).

2.7.4 Conclusion

Mammals, birds, and aquatic species, both freshwater and marine, appear to be capable of bioaccumulating some decaBDE in various tissues. However, most of the decaBDE that is ingested is excreted in feces. A small fraction is absorbed, but most of that is removed by the liver before it can enter systemic circulation. Some studies have suggested that of the unknown fraction that is assimilated, some might possibly be metabolically debrominated to other PBDE congeners (see Section 2.5 Debromination). Despite the widespread occurrence of decaBDE in the environment and its presence in various animal tissues, there is little information available at present to suggest that decaBDE at current environmental levels is likely to cause measurable adverse effects on fish and wildlife populations. This is consistent with the conclusions of the European Union risk assessment for decaBDE (ECB, 2002).

Table 2.7.3.3-1: Summary of the Residue Values of DecaBDE Reported in Biota in Studies Referenced in This Section

Species	BDE-209 Residue Value	Source
Fresh and saltwater mussels	5 ng/g, wet weight	de Boer et al., 2003
Crayfish (*Cambarus puncticambarus*)	21,600 ng/g lwt	LaGuardia et al., 2007
Pumpkinseed sunfish (*Lepomis gibbosus*)	2,880 ng/g lwt	LaGuardia et al., 2007
Pumpkinseed sunfish	0.28 ng/g ww (edible tissue)	Skinner et al., 2009
Bluntnose minnow (*Pimephales notatus*)	8.15 ng/g ww (whole body)	Skinner et al., 2009
Brown bullhead (*Ameiurus nebulosus*)	0.67 ng/g ww (carcass)	Skinner et al., 2009
Common carp (*Cyprinus carpio*)	0.304 ng/g ww (edible tissue)	Skinner et al., 2009
Lake trout (*Salvelinus namaycush)*	0.27–1.3 ng/g ww 2.3–12 ng/g lwt (homogenized whole fish)	Ismail et al., 2009
Mysid shrimp (*Neomysis integer)*	269–600 ng/g lwt	Verslycke et al., 2005
Blue mussels (*Mytilus edulis*)	3,350 ng/g ww before depuration; 50 ng/g ww after depuration	Booij et al., 2002
Harbor seal (*Phoca vitulina*) blubber: adult male adult female yearling pup	 1.1 ng/g lwt 0.5 ng/g lwt 1.9 ng/g lwt 0.8 ng/g lwt	Shaw et al., 2008
American plaice (*Hippoglossides platessoides*)	1.8 ng/g lwt (homogenized whole fish)	Shaw et al., 2009
White hake (*Urophycis tenuis*)	0.91 ng/g lwt (homogenized whole fish)	
Atlantic mackerel (*Scomber scombrus*)	1.6 ng/g lwt (homogenized whole fish)	
Harbor seal blubber	1.2 ng/g lwt	
Eggs of the Little egret (*Egretta garzetta*)	0.89–2.6 ng/g lwt	Lam et al., 2007
Black-crowned night heron (*Nycticorax nycticorax*) eggs (China)	< 0.5–14 ng/g lwt	
Chinese pond heron (*Ardeola bacchus*) eggs (China)	99 ng/g lwt	
Cattle egret (*Bubulcus ibis*) eggs (China)	28 ng/g lwt	
eggs of the White-tailed sea eagle (*Haliaeetus albicilla*); Peregrine falcon (*Falco peregrinus*); Merlin (*Falco columbarius*) Golden eagle (*Aquila chrysaetos*); Osprey (*Pandion haliaetus*); Goshawk (*Accipiter gentilis*), (Norway)	< limit of detection (LOD) to 4 ng/g wet weight, < LOD to 80 ng/g lwt	Herzke et al., 2005
Peregrine falcon eggs (Greenland)	3.8–250 ng g lipid, with a median of 11 ng/g lwt	Vorkamp et al., 2005
Peregrine falcon eggs (Sweden)	45 and 67 ng/g lwt (means of two separate populations)	Johansson et al., 2009
Peregrine falcon eggs (Sweden)	< 20–430 ng/g lwt (wild birds) < 7–9 ng/g lwt captive birds	Lindberg et al., 2004
Peregrine falcon eggs (New York)	6.013 ng/g ww (Albany/Troy) 6.740 ng/g ww (Catskills)	Hudson River Natural Resource Trustees, 2004
Herring gull (*Larus argentatus*) eggs, Great Lakes Region	6–20 ng/g ww, 51–192 ng/g lwt	Gauthier et al., 2008
Red fox (*Vulpes vulpes*)	3–760 ng/g lwt in adipose tissue, liver, and muscle	Voorspoels et al., 2006

ng/g lwt = nanograms per gram lipid weight; ng/g ww = nanograms per gram wet weight; LOD = limit of detection.

2.8 Human Exposure

2.8.1 BDE-209 in Food and House Dust

BDE-209 has been detected in foods and house dust. Tables 2.8.1-1 and 2.8.1-2 summarize the major United States studies of BDE-209 in food and dust, respectively.

Table 2.8.1-1: United States Studies Reporting Detection of BDE-209 in Food

Reference	Description	BDE-209 Levels (ng/g ww*)	Notes
Huwe et al., 2002	Samples of chicken fat (n = 13) from southern US	0.44–3.35 (range) 1.1 (mean)	BDE-209 detected in all 13 samples. Contribution of BDE-209 to total PBDEs ranged from 2 to 41%
Schecter et al., 2006a	Samples of meat, fish, and dairy products (n = 18, 24 and 15, respectively) from market basket survey of foods from three Dallas, TX supermarket chains.	Meat: ND (0.01–0.17) – 0.24 (range) 0.05 (mean)	BDE-209 detected in 8 of 18 samples. When detected, the contribution of BDE-209 to total PBDEs ranged from 4 to 62%
		Fish: ND (0.001–0.05) – 1.27 (range) 0.09 (mean)	BDE-209 detected in 10 of 24 samples. When detected, the contribution of BDE-209 to total PBDEs ranged from 1 to 57%
		Dairy-based food: ND (0.002–0.04) – 0.48 (range) 0.04 (mean)	BDE-209 detected in 7 of 15 samples. When detected, the contribution of BDE-209 to total PBDEs ranged from 2 to 70%
Schecter et al., 2009	Composite samples (n = 31) of various meat, fish, dairy products and vegetables from market basket survey of foods from five Dallas, TX supermarkets.	Meat: ND (0.005–0.01) – 0.039 (range)	BDE-209 detected in 5 of 8 composite samples of various meats.
		Fish: ND (0.007–0.08) – 0.05 (range)	BDE-209 detected in 2 of 7 composite samples of various meats.
		Dairy-based food: ND (0.007–0.01) – 5.2 (range)	BDE-209 detected in 7 of 9 composite samples of various dairy products.
		Vegetable-based food: ND (0.003–0.08)	BDE-209 was not detected in any of 7 composite samples of various vegetable-based foods.
Schecter et al., 2010	Samples (n = 90) collected from each of three cities (Los Angeles, CA; Dallas, TX; and Albany, NY) in a market basket survey of an assortment of foods (i.e., fish, meat, dairy and vegetables).	Fish: ND (0.01–0.1) - 0.76 (range) 0.13 (mean) 0.063 (geometric mean)	BDE-209 was detected in 11 of 24 fish samples.
		Meat: ND (0.06–0.1) – 3.0 (range) 0.37 (mean) 0.18 (geometric mean)	BDE-209 was detected in 17 of 24 meat samples.
		Dairy-based food: ND (0.02–0.1) – 0.55 (range) 0.13 (mean)	BDE-209 was detected in 6 of 18 dairy-based food samples.
		Vegetable-based food: ND (0.02–0.1) – 0.81 (range) 0.14 (mean)	BDE-209 was detected in 9 of 18 vegetable-based food samples.

*nanograms per gram wet weight.
ND = not detected, numbers in parenthesis are the reported detection limits.

Table 2.8.1-2: United States Studies Reporting Detection of BDE-209 in House Dust

Reference	Description	BDE-209 Levels (ng/g dry weight*)	Notes
Schecter et al., 2005a	Vacuum dust samples (n = 9) collected by eight individual residents in their home in Dallas, TX in 2004	143–65,777 (range) 8,567 (mean) 665 (median)	BDE-209 was the predominant congener in the dust samples, constituting over 60% of the total PBDEs in six of the nine samples. The method detection limit was not reported.
Stapleton et al., 2005	Vacuum dust (n=17) and clothes dryer lint (n=5) samples collected in 2004 from homes in the Washington, D.C. metropolitan area	Dust: 162–8,750 (range) 2,090 (mean) 1,350 (median) Dryer lint: 58–2,890 (range) 816 (mean) 230 (median)	BDE-209 was detected in all samples. The method detection limit was 6 ng/g dry weight. BDE-209 ranged from 8 to 88% of the total PBDEs in individual samples and made up 41% of the PBDE total in a composite dust sample.
Wu et al., 2007	Vacuum dust samples taken in 2006 from floor and furniture of commonly-used rooms in 11 homes in greater Boston, MA area	ND–9,600 (range)	The detection limit was 500 ng/g. BDE-209 was detected in 45% of the samples.
Allen et al., 2008	Vacuum dust samples (n = 60) collected from 20 urban residences (main living area, bedroom and home vacuum cleaner bags) in greater Boston, MA area in 2006	Main living area: 791.1–184,600 (range) 4,502 (geometric mean) Bedroom: 24.0–36,130 (range) 1,703 (geometric mean) Vacuum bags: 227.8–263,000 (range) 1,811 (geometric mean)	BDE-209 was the predominant congener is dust samples, with the geometric means levels being higher than any other congeners. BDE-209 was detected in 95% of bedroom samples and in 100% of living area and vacuum bag samples.
Harrad et al., 2008	Vacuum dust samples collected from homes in Amarillo/Austin, TX in 2006 (n = 17)	530–3,000 (range) 1,600 (average) 1,300 (median) 1,300 (geometric mean)	Method detection limits for individual PBDE congeners were typically 0.03 ng/g. BDE-209 was the predominant congener in dust samples.
Sjodin et al., 2008	Vacuum dust samples collected from disposable vacuum cleaner bags from private households (n = 10) in Atlanta, GA	120–21,000 (range) 2,000 (median)	The authors concluded that BDE-209 was the predominant PBDE congener in the dust samples. The method detection limit was not reported.
Johnson-Restrepo and Kannan, et al., 2009	Vacuum dust samples collected from 12 houses in Albany, NY	327–9,210 (range) 2,810 (average) 903 (median)	BDE-209 was the predominant congener is the dust samples, and accounted for 26–99% of the total PBDEs in dust.

*nanograms per gram dry weight.
ND = not detected.

The presence of BDE-209 in foods and house dust can lead to human exposure. In its recent exposure assessment of PBDEs, the US EPA identified some general trends from studies that evaluated BDE-209 and PBDE levels in food and indoor dust (US EPA, 2010a). The level of BDE-209 in food measured in market basket surveys varies considerably, depending on the type of food product being sampled. In general, PBDEs (including BDE-209) were found at higher levels in terrestrial animal food products (e.g., meat and dairy products) compared to foods of vegetative origin (e.g., cereals, fruits and vegetables), which is consistent with the tendency of hydrophobic halogenated organic compounds to accumulate in animal fat. In house dust, BDE-209 is generally the predominant PBDE congener, followed by BDE-47 and BDE-99. Based on the ratio of congeners, some researchers have concluded that the dust results reflect the presence of the commercial pentaBDE and decaBDE formulations.

2.8.2 Presence of BDE-209 in Human Blood, Sera and Breast Milk

The most recent (2003–2004) National Health and Nutrition Examination Survey (NHANES), an ongoing survey that samples participants from the United States population every two years, provides information on human exposure to several PBDE congeners (CDC, 2009), but does not include BDE-209. However, Sjodin et al. (2008) reported that the mean serum concentration of pooled samples from the NHANES 2001–2002 survey was about 2 ng/g lwt for participants over 12 years of age. Evidence of BDE-209 exposure in people is also provided by several studies that report its detection in human blood, sera and breast milk. Tables 2.8.2-1 and 2.8.2-2 summarize United States studies that report on levels of BDE-209 in these body fluids.

Table 2.8.2-1: United States Studies Reporting Detection of BDE-209 in Human Blood and Sera

Reference	Description	BDE-209 Levels (ng/g lwt*)	Notes
Sjödin et al., 2001	Twelve serum samples collected in 1988 from blood donors in Illinois	ND (1.0) – 33.6 (range) < 1.0 (mean)	BDE-209 was detected in 5 of 12 samples at a detection limit of 1 ng/g lwt weight.
Schecter et al., 2005b	One pooled whole blood sample (n = 100) from Texas, 2003	1.4	
	One archived pooled serum sample (n = 100) from Texas, 1973	ND (1.0)	
	Individual whole blood samples from New York (n = 10), 2003	ND (2.0–6.0)	BDE-209 was not detected in any New York samples.
	Individual whole blood samples from Mississippi (n = 27), 2003	ND (1.0–15) – 6.1 (range) 2.7 (mean) 2.3 (median)	BDE-209 was detected in 9 of 27 Mississippi samples
Fischer et al., 2006	Case study of California family of four; sera sampled in September and December of 2004	Father: 23 (Sept); 2.5 (Dec) Mother: 14 (Sept); 4 (Dec) Child: 143 (Sept); 11 (Dec) Toddler: 233 (Sept); 22 (Dec)	Child and toddler were 5 years and 18 months old, respectively. Reason for decrease in levels from September to December is not known.
Schecter et al., 2006b	Whole blood sampled from 8 adult vegetarians	ND (2–7)	
Schecter et al., 2006c	Whole blood sampled from 11 nursing mothers	ND (1–22) – 13 (range) 5.2 (mean)**	BDE-209 detected in 6 of the 11 samples.
Lunder et al., 2010	Sera sampled concurrently from 9 mothers and 13 children	Mothers: ND (1.9) – 3.2 1.7 (mean) Children: ND (1.8) – 19 3.5 (mean)	On average, results for BDE-209 in children were twice as high as those for mothers

*nanograms per gram lipid weight.
**Value calculated from published data using one-half the detection limit for non-detects.
ND = not detected, numbers in parenthesis are the reported detection limits.

Table 2.8.2-2: United States Studies Reporting Detection of BDE-209 in Breast Milk

Reference	Description	BDE-209 Levels (ng/g lwt*)	Notes
Schecter et al., 2003	Milk samples from 47 nursing mothers aged 20–41 in Austin, TX.	ND** – 8.2 (range) 0.92 (mean)	BDE-209 was detected in 7 of 23 samples.
Schecter et al., 2006c	Milk samples from 11 nursing mothers	ND (0.04–0.2) – 2.5 (range) 0.45 (mean)***	BDE-209 was detected in 4 of 11 samples.
Johnson-Resptrepo et al., 2007	Milk samples donated by 38 nursing mothers across MA	ND (204)	Detection limit higher than in most studies.
She et al., 2007	Milk samples from 40 first time mothers from Pacific Northwest†	0.048–4.26 (range)** 0.80 (mean)	BDE-209 detected in all samples, detection limits not reported.
Wu et al., 2007	Milk samples from 46 first time mothers 2–8 weeks after birth in greater Boston, MA	ND (0.05–1) – 10.9 (range) 0.48 (mean)***	BDE-209 detected in 11 of 46 samples, detection limits not reported.

*nanograms per gram lipid weight
**Detection limits not reported.
***Value calculated from published data using one-half the detection limit for non-detects.
†Study includes 10 samples from Vancouver, British Columbia
ND = not detected, numbers in parenthesis are the reported detection limits.

General trends from studies of BDE-209 and PBDE levels in blood, sera and breast milk are as follows (US EPA, 2010a):

- BDE-209 is generally present at lower levels in blood and breast milk than other PBDE congeners such as BDE-47, BDE-99, and BDE-153. In breast milk, BDE-209 typically makes up less than 10 percent of the total PBDE congeners (Schecter et al., 2003; She et al., 2007; Wu et al., 2007).
- In two studies that measured BDE-209 levels in the sera of both adults and children (Fischer et al., 2006; Lunder et al., 2010) the levels in children are reported to be higher.

2.8.3 Human Exposure Estimates

The US EPA recently published an extensive exposure assessment for total PBDEs that estimated daily intakes across exposure pathways and age groups (US EPA, 2010a). The agency used exposure factors and approaches developed in the US EPA's Dioxin Reassessment (US EPA, 2003) which relied on US EPA's Exposure Factors Handbook (US EPA, 1997) and data from the US Department of Agriculture (USDA, 1995). The pathways included dust ingestion, dermal contact with dust, inhalation, water ingestion, and the diet (milk, dairy products, eggs, meats and fish). The estimated exposure dose for each age group is shown in Table 2.8.3-1.

Table 2.8.3-1: US EPA Estimates of Total Daily PBDE Exposures for Various Age Groups[a]

Age	Total PBDE Exposure (ng/kg/day)
> 20 (adults)	7.1
12–19	8.3
6–11	13
1–5	47.2
< 1 (infants)	141

[a]Data from US EPA (2010a)
ng/kg/day = nanograms per kilogram body weight per day

Dust comprises the majority of the estimated exposure for adults and children, while for infants, the predominant estimated exposure pathway is through breast milk ingestion. The US EPA estimates that the total PBDE intake for adults is about 500 ng/day, of which about 28 percent (142 ng/day) is BDE-209. This corresponds to a daily estimated exposure dose of about 2 ng/kg/day assuming an adult body weight of 70 kg (US EPA, 2010a). These exposure estimates are generally consistent with many literature studies (reviewed in US EPA, 2010a) that estimate daily exposure to PBDEs using a variety of methods.

As part of its Risk Assessment Report on BDE-209, the European Union estimated exposure to BDE-209 for workers who are exposed dermally and by inhalation, and for people who are exposed via the environment (ECB, 2002). The estimated body burdens for workers were 120,000 ng/kg/day for the dermal pathway and 700,000 ng/kg/day for the inhalation pathway. For people exposed via the environment, the European Union Risk Assessment Report estimated BDE-209 concentrations in fish, plant leaves and roots, drinking water, meat, milk and air

resulting from several processes involving the chemical (production, polymer processing, textile compounding and applications). The overall human exposure from environmental sources was estimated to be 8,000–12,000 ng/kg/day. This estimate is higher than the daily exposure recently estimated by the US EPA (about 2 ng/kg/day [US EPA, 2010a]), and likely overestimates human exposure. Most of the exposure (greater than 90 percent) in the European Union value comes from estimated uptake of BDE-209 in the root crops. This root crop estimate is based on a modeling approach that assumes a soil pore water concentration at the limits of solubility for BDE-209 (0.1 mcg/L). The model also estimates the uptake from water for BDE-209 from its octanol/water partition coefficient (K_{ow}) value, assuming that uptake increases across chemicals as K_{ow} increases. The European Union document states that there is considerable uncertainty in this approach for chemicals such as BDE-209 that have high K_{ow} values. Based on the K_{ow} value, the model predicts a significant level of bioaccumulation, but experimental data both in fish (CITI, 1992) and more recently in plants (Vrkoslavova et al., 2010; Huang et al., 2011) indicate lower bioaccumulation than would be predicted by the K_{ow}. Based on these considerations, the European Union estimate for BDE-209 is not used in this document to represent exposure.

2.8.4 Human Exposure to PBDE Mixtures

Humans exposed to BDE-209 are also exposed to lower brominated PBDE congeners because of the widespread presence of commercial PBDEs in the environment. The lower brominated congeners, particularly BDE-47, BDE-99, and BDE-153, are present with BDE-209 in food (Huwe et al., 2002; Schecter et al., 2006a, 2009, 2010) and house dust (Schecter et al., 2005a; Stapleton et al., 2005; Wu et al., 2007; Allen et al., 2008; Harrad et al., 2008; Sjodin et al., 2008; Johnson-Restrepo and Kannan et al., 2009). Human biomonitoring data show that BDE-209 and lower brominated PBDEs are found in human blood and milk (Sjödin et al., 2001; Schecter et al., 2003; Schecter et al., 2005b; Fischer et al., 2006; Schecter et al., 2006b,c; Johnson-Resptrepo et al., 2007; She et al., 2007; Wu et al., 2007). There is evidence that BDE-209, -47, -99, and -153 cause adverse effects on the developing nervous system of young animals (Eriksson et al., 2001, 2002; Viberg et al., 2003a, 2004, 2006; Viberg et al., 2003b; Rice et al., 2007; Viberg et al., 2007; Johansson et al., 2008), and these effects are reported to occur at lower levels of exposure than effects on other organs or body systems. In general, additive interactions between chemicals are most likely to occur when the chemicals cause the same effect on the same organ

by the same toxicological mode of action (ATSDR, 2004b; US EPA, 2000). PBDEs share common effects on the nervous system as well as other body systems. Since exposures to contaminant mixtures having similar toxic endpoints may pose different risks than exposures to individual contaminants, the possibility of additive toxicity of the lower brominated congeners and BDE-209 should be considered in evaluating human environmental exposures to PBDE mixtures.

2.8.5 Conclusion

BDE-209, the PBDE congener that makes up more than 97 percent of decaBDE, has been detected in food and house dust, and its presence in these media can lead to human exposure, as demonstrated by its detection in human sera, blood and breast milk. Human exposure estimates for BDE-209 made by the US EPA and the European Union differ significantly. The European Union estimate is much higher than that of the US EPA, and likely overestimates human exposure because of assumptions made about BDE-209 water concentrations and its uptake from water into plants. BDE-209 exposure in dust, food and breast milk is likely to also include exposure to other PBDEs, and therefore the possibility for additive toxicity should be considered when evaluating exposures in these media.

2.9 Health Effects

Human studies on the health effects of BDE-209 are not available (US EPA, 2008a). The following sections discuss some of the important toxicological studies on the effects of BDE-209 in animals, as well as risk assessments for BDE-209 that have been published in the peer-reviewed scientific literature or by government agencies. Some of the studies provide the basis for the derivation of toxicity values such as reference doses and cancer potency factors for BDE-209.

The reference dose is defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious noncancer health effects during a lifetime. Scientists derive reference doses from points of departure (a no-observed effect level [NOEL], a lowest-observed effect level [LOEL], or a benchmark dose) after reviewing the available studies on the health effects of the chemical in animals or humans. The point of

departure is divided by uncertainty factors, which reflect the quantity, quality and limitations of the data. Reference doses are used in risk assessments to evaluate the risk for health effects for a given exposure to a chemical.

The cancer potency factor is an upper bound estimate (i.e., the upper 95 percent confidence limit) of the increased cancer risk from lifetime exposure to 1 milligram per kilogram per day (mg/kg/day) of a chemical (US EPA, 2009b). Cancer potency factors are based on dose-response information from studies on the carcinogenic effects of the chemical in animals or humans, and are used in risk assessments to estimate the lifetime cancer risk associated with a given exposure to a chemical (US EPA, 2005).

In animals, studies on the effects of BDE-209 on the nervous system of young rodents suggest that developmental neurotoxicity may be the most sensitive BDE-209 toxicological endpoint. Since BDE-209 is reported to be present in breast milk, the studies reporting effects on the developing nervous system have raised concern among scientists about potential risks to nursing infants. However, there are uncertainties about the validity and the biological significance of the results of these investigations due to study design limitations. In addition, a recently completed study designed to meet accepted standards for conducting developmental neurotoxicity studies showed no effects in young animals who were exposed during gestation and through lactation. Long-term investigations report liver effects in animals, but these were done at higher exposure levels and did not evaluate developmental neurotoxicity endpoints (see Sections 2.9.1 and 2.9.2 for additional details). The results of the developmental neurotoxicity studies and the liver toxicity studies have been interpreted differently by scientists, and there is no clear consensus as to which dataset should be the basis for risk assessment or derivation of a BDE-209 reference dose.

The US EPA maintains testing requirements for pesticides and industrial chemicals, which include test guidelines for conducting toxicity studies (US EPA, 2010b). These studies are used for generating information about pesticides and industrial chemicals regulated under the Federal Insecticide, Fungicide, and Rodenticide Act and the Toxic Substances Control Act (TSCA). The US EPA uses this information to evaluate whether chemicals meet federal safety standards to protect human health and the environment. The health effects test guidelines, developed by the US EPA‘s Office of Chemical Safety and Pollution Prevention, include those for studies on acute

toxicity, subchronic toxicity, chronic toxicity, genetic toxicity and neurotoxicity (including developmental neurotoxicity testing). The guidelines provide specifications for many aspects of experimental design, including animal selection (species and strain), age, sex and number of animals, types of parameters to be evaluated, dose levels and administration methods, dosing periods, selection of animals for evaluation, and data collection, recording and evaluation. One of the primary controversies surrounding the toxicity data for BDE-209 is that some of the studies that report effects on the developing nervous system of young animals at low exposure levels do not conform to the US EPA's established testing guidelines.

The following sections summarize important studies on BDE-209 that evaluate liver, thyroid, endocrine, and reproductive toxicity, developmental neurotoxicity and carcinogenicity. Toxicity values (reference doses and cancer potency factors) based on some of these studies have been derived by health agencies and others. The strengths and limitations of the studies and toxicity value derivations are discussed.

2.9.1 Developmental Neurotoxicity

Viberg and coworkers conducted a series of studies on the effects of BDE-209 exposure on neurobehavioral development in neonatal mice (Viberg et al., 2003b, 2007; Johansson et al., 2008). In the first of these studies, male mice were given a single oral dose of BDE-209 (0, 2.22 or 20.1 mg/kg on PNDs 3 or 19; 1.34, 13.4 or 20.1 mg/kg on PND 10), and evaluated in adulthood for effects on habituation capability. The animals were administered BDE-209 in a mixture of water, peanut oil and egg lecithin by gavage (i.e., a tube inserted through the mouth and into the stomach). Animals treated with the highest dose (20.1 mg/kg BDE-209) on PND 3 showed decreased habituation during spontaneous motor activity tests (manifested by either hyperactivity or hypoactivity) compared to control animals. Habituation continued to decrease in the exposed animals as they aged from 2 to 6 months. These effects did not occur in animals exposed to BDE-209 on PNDs 10 or 19.

Two more recent studies from the same laboratory using the same study methods reported effects on spontaneous behavior and habituation in mice and rats given single oral BDE-209 doses of 2.22 mg/kg (Johansson et al., 2008) and 6.7 mg/kg (Viberg et al., 2007). As in the Viberg et al. (2003b) study, the animals were exposed to BDE-209 as neonates by gavage in a water/peanut

oil/egg lecithin mixture. Similar effects on neurobehavioral development have also been reported for other PBDE congeners (i.e., BDE-47, -99 and -153) in studies from the same laboratory (Eriksson et al., 2001, 2002; Viberg et al., 2003a, 2004, 2006). These findings provide evidence for a critical window of vulnerability for the developing nervous system to the effects of BDE-209 and other PBDEs.

The US EPA selected the NOEL of 2.22 mg/kg from the Viberg et al. (2003b) study as the primary basis for deriving a reference dose for BDE-209 (US EPA, 2008a,b). This study was chosen over the Viberg et al., 2007 study (which reported a lower effect level) because it identified both a NOEL and a LOEL. A later study (Johansson et al., 2008) which reported effects at an exposure equal to the Viberg et al. (2003b) NOEL was not published at the time of the US EPA derivation. A total uncertainty factor of 300 (10 for interspecies differences, 10 for intraspecies differences, and 3 as a dosing duration adjustment to account for the animals having been dosed only once during the hypothesized critical window) was applied to the NOEL to obtain a reference dose of 0.007 mg/kg/day. The US EPA noted that the Viberg et al. (2003b) used a unique study protocol that does not conform to test guidelines for neurotoxicity screening battery or developmental neurotoxicity studies (US EPA, 1998a,b,c, 2010b). Specific aspects of the studies that did not conform to the guidelines included:

- use of single pups rather than the litter as the basic analytical unit;
- use of a single dose which precluded evaluation of exposure during gestation and lactation;
- evaluation of only one sex and a limited number of neurobehavioral parameters (locomotion, rearing, and total activity); and
- choosing more than one pup per litter for behavioral testing, which could bias the analysis toward false positives.

The overall confidence level assigned by the US EPA to the reference dose for BDE-209 is low, based on low confidence in the primary study (Viberg et al., 2003b) and medium confidence in the adequacy of the toxicological database. Several health agencies and investigators (see following sections) have chosen other studies as the basis for deriving a reference dose.

In a study reported by Rice et al. (2007), male and female neonatal mice (grouped with littermates) were orally exposed to 0, 6, or 20 mg/kg BDE-209 from PND 2 through 15. Rice et al. (2007) administered BDE-209 in the same water/peanut oil/egg lecithin mixture as did Viberg et al. (2003b), but delivered the dose to the animal's mouth using a micropipette. The study was more consistent with established guidelines for developmental neurotoxicity than the Viberg et al (2003b) study, since the litter was used as the statistical unit, the animals were exposed to BDE-209 several times during the postnatal period, and both sexes of mice were evaluated for effects on a broad range of developmental and behavioral endpoints at different life stages (from PND 2 through 1 year of age). The authors reported that BDE-209 caused statistically significant effects on eyelid reflex, forelimb grip and struggling behavior in immature animals, and effects on the rate of change in locomotor activity in adults. The effects on eyelid reflex (closing of the eyelid when touched) occurred only on PND 14, and on that day, the authors noted "some evidence of eyelid droop or closure in both sexes." Since this test is valid only when the eyes of the neonate (which are closed at birth) are fully open, the effects on eyelid reflex on PND 14 may not be due to BDE-209 exposure. The effects on locomotor activity on PND 70 were not altogether consistent with those reported by Viberg et al. (2003b), since in the Rice et al. (2007) study, animals had higher initial locomotor activity, while in the Viberg et al. (2003b) study they had lower initial locomotor activity. The US EPA cited the Rice et al. (2007) study as mitigating some of the design limitation concerns of the Viberg et al. (2003b) study, since the authors based the results on the litters rather than single animals, and designed a functional observational battery to evaluate numerous parameters for neurobehavioral development.

The European Union evaluated the Viberg et al. (2003b) study in its update of their 2002 Risk Assessment Report on BDE-209 (EA, 2004). They concluded that it provides some indication that BDE-209 has potential to cause developmental effects on mammalian systems at exposure levels lower than those of other studies showing BDE-209 health effects, but also that the results cannot be used to draw a final conclusion for human health. The update, as well as the final conclusions of the risk assessment published in the Official Journal of the European Union (OJ 29.5.2008 C131/9), further recommended that a new developmental neurotoxicity study could address some of the issues raised by the studies of Viberg and coworkers. The study was completed as required under the European Union's existing substances regulation and carried out by WIL Research Laboratories with funding from the Bromine Science and Environmental Forum, an international organization of bromine-producing industries. The methods used in the

study were designed to meet guidelines for developmental neurotoxicity studies established by the US EPA (1998c) and the Organization for Economic Cooperation and Development (OECD, 2007). Groups of 35 mated female rats were treated once daily with decaBDE in corn oil gavage to 0, 1, 10, 100 or 1000 mg/kg/day from gestation day 6 through lactation day 21 (Biesemeier et al., 2011). A preliminary study by the same researchers (Biesemeier et al., 2010) which evaluated how much BDE-209 is absorbed into the animals using this exposure method reported that the amount of BDE-209 in the blood plasma and milk of the mothers was relatively consistent when the dose was increased from 100 to 1000 mg/kg/day, and was similar in the blood plasma of the offspring when the dose was increased from 300 to 1000 mg/kg/day. Several indicators of neurological development were evaluated in the offspring, with the litter as the statistical unit (Biesemeier et al., 2011). Evaluations included assessments of motor activity at PNDs 13, 17, 21 and at 2, 4 and 6 months of age, as well as tests for learning, memory, and auditory startle response. Brain tissue and development were also evaluated. In contrast to the Viberg et al. (2003b) and Rice et al. (2007) studies, the authors reported that there were no decaBDE treatment-related effects on motor activity. The authors also reported that there were no treatment-related changes in tests for learning, memory or startle response, and no treatment-related alterations were found in brain tissue or in measures of brain development. A notable difference between the Biesemeier et al. (2011) study and the earlier studies is that the offspring were exposed through the parent animal during gestation and through lactation, while in the Viberg et al. (2003b) and Rice et al. (2007) studies, the neonates were exposed on PND 3 (by gavage) and PNDs 2 through 15 (by micropipette), respectively. The Biesemeier et al. (2011) study also used corn oil to deliver decaBDE to the parent animals, and the offspring were exposed to BDE-209 in mother‘s milk, while the Viberg et al. (2003b) and Rice et al. (2007) studies used a water/peanut oil/egg lecithin mixture for the neonates. The amount of BDE-209 absorbed into the animal is known to be influenced by the exposure vehicle (Costa and Giordano, 2011; Biesemeier et al., 2010). The Biesemeier et al. (2011) study included information on the amount of BDE-209 absorbed (Biesemeier et al., 2010). This information is not available for the Viberg et al. (2003b) and Rice et al. (2007) studies. Consequently, there is uncertainty about whether the BDE-209 exposures in the studies represent similar or different amounts of the chemical absorbed into the animals (i.e., the absorbed or internal dose).

The Environmental Agency of the United Kingdom (one of the two Member States responsible for the European Union risk assessment of BDE-209) summarized the Biesemeier et al. (2011)

study prior to its publication as follows in its update of the European Union risk assessment (EA, 2009a):

> –As a result of EU Member State concerns about a potential neurotoxic effect of decaBDE, a study has recently been carried out by industry following the OECD 426 Test Guideline…The study was carried out using rats exposed to doses of 1, 10, 100 and 1,000 mg/kg body weight/day from gestation day 6 to lactation day 21. The study summary indicates that exposure to decaBDE caused no maternal toxicity, no effects on offspring survival and growth, and no effects on any of the neurobehavioural endpoints studied compared with the control groups at all dosage levels. The study is currently being reviewed by the French competent authority to decide what, if any, further action is necessary.
>
> It should be noted that there are a number of other recently published studies that suggest that decaBDE does have the potential to cause developmental neurotoxic effects. Several important concerns about some of these studies have been identified in a critical review submitted to the Environment Agency by industry. An initial review has also been prepared by the Institute of Environment and Health (Cranfield University) for the Environment Agency as an unpublished internal report. They concluded that many of industry's reservations are valid, highlighting the need to take due consideration of study limitations when considering the various reported findings. Therefore it is appropriate to wait until the whole data set has been fully evaluated by appropriate experts before the implications for setting a no-observed adverse effect level (NOAEL) for decaBDE are considered."

The CPSC in its assessment of potential health effects from fire retardant chemicals in mattresses (CPSC, 2006b) noted several limitations of the Viberg et al. (2003b) study, including the small number of animals per treatment group, dosing with a fat emulsion, conducting the behavioral tests only once, evaluating only one neurobehavioral endpoint, and the use of only one species. The CPSC stated that the relevance of the results of the study to human health is uncertain, and based its reference dose on liver toxicity (see next section).

Another federal agency, the Agency for Toxic Substances and Disease Registry (ATSDR), selected an alternative study and toxicity endpoint (i.e., reproductive toxicity based on Hardy et al., 2002) as the basis for deriving an intermediate minimal risk level (MRL) (a value similar in concept to a reference dose, but derived for exposure durations of more than two weeks to less than one year rather than a lifetime). The ATSDR noted that Viberg et al (2003b) and similar studies with other PBDEs collectively provide increasing evidence that the developing nervous system is a sensitive target of some PBDEs, including BDE-209. The agency further stated that additional studies using comprehensive neurobehavioral test batteries are needed to better evaluate if PBDEs increase the risk for neurotoxicity in humans (ATSDR, 2004a).

Hardy et al. (2009) and Goodman et al. (2009) offered in-depth analyses of the Viberg et al. (2003b) and Rice et al. (2007) studies, and the US EPA reference dose derivation. Both sets of authors concluded that due to the same limitations noted by the US EPA in its derivation, as well as uncertainties about the purity of the test material, the Viberg et al. (2003b) study was unsuitable for derivation of a reference dose. Goodman et al. (2009) also concluded that the effects noted in the functional observational battery in Rice et al. (2007) (which the US EPA used to mitigate some of the limitations of the Viberg et al. [2003b] study) were unlikely to be treatment related because the changes in the parameters were of marginal statistical significance and few in number compared to the large number of endpoints and time points tested. In addition, Goodman et al. (2009) concluded that the test for changes in eyelid reflex was invalid because the neonates‘ eyes (closed at birth) were not fully open by PND 14, when the difference between control and exposed animals was noted. On this day, Rice et al. (2007) noted ―evidence of eyelid droop or closure in both sexes.” Finally, both sets of authors noted that the effects on locomotor activity in the Rice et al. (2007) study are not consistent with those in the Viberg et al. (2003b) study because the direction of the trend differed. In the Rice et al. (2007) study the locomotor activity of exposed animals decreased across the observation period compared to controls, while in Viberg et al. (2003b) study, the activity increased across the observation period.

Three primary studies have investigated the effects of BDE-209 on the nervous system of young laboratory animals (Viberg et al., 2003b; Rice et al., 2007; Biesemeier et al., 2011), and are summarized in Table 2.9.1-1.

Table 2.9.1-1: Developmental Neurotoxicity Studies on Decabromodiphenyl Ether

Study	Species	Exposure	Result
Viberg et al. (2003b)	mice	One time gavage (stomach tube) of neonates to BDE-209 in a water/peanut oil/egg lecithin mixture on PND 3	Effects on habituation as measured by tests showing increases in locomotion, rearing and total activity during 1 hour test periods at 2, 4 and 6 months of age
Rice et al. (2007)	mice	Once a day of neonates by micropipette to BDE-209 in a water/peanut oil/egg lecithin mixture on PNDs 2 through 15	Effects on eyelid closure (PND 14), forelimb grip strength (PND 16), struggling during handling (PND 20). Decreased rate of decline in locomotor activity during 2 hour test periods (PND 70)
Biesemeier et al. (2011)	rats	Through mother animal from gestation day 6 and through nursing until three weeks old; Mothers exposed to decaBDE in corn oil by gavage (stomach tube)	No effects reported on locomotor activity at PNDs 13, 17, 21 and at 2, 4 and 6 months of age, and on tests for learning and memory (PND 22, 62), and auditory startle response (PND 20, 60)

PND = postnatal day, or days immediately following birth.

The three studies are markedly different with respect to their experimental methods and the reported results. The animals were exposed in different ways. Viberg et al. (2003b) and Rice et al. (2007) exposed the neonates (through gavage or micropipette), while Biesemeier et al. (2011) exposed the young animals through their mothers during gestation and through lactation. The Viberg et al. (2003b) study exposed the animals only once, while the Rice et al. (2007) and Biesemeier et al. (2011) studies exposed the animals over multiple days. The exposures were also not always on the same days of the postnatal period. Further, the Viberg et al. (2003b) and Rice et al. (2007) used a water/peanut oil/egg lecithin mixture to deliver the chemical to the young animals, while Biesemeier et al. (2011) used corn oil to bring decaBDE to the parent animals, and the offspring were exposed to BDE-209 in mother‘s milk. The use of different exposure vehicles could result in different amounts of BDE-209 being absorbed by the animals (Costa and Giordano, 2011; Biesemeier et al., 2010). The evaluation of the animals was not the same among the studies, as Viberg et al. (2003b) tested individual animals for developmental neurotoxicity, which does not take into account the possibility of litter effects, or the tendency of two animals from the same litter to respond to the tests in the similar ways. Rice et al. (2007)

and Biesemeier et al. (2011) evaluated litters according to OECD and US EPA guidelines (US EPA, 1998c; OECD, 2007), and thereby accounted for possible litter effects. Finally, the results of the studies are dissimilar. No effects were reported by Biesemeier et al. (2011), while in the studies reporting effects, the changes in locomotion are in opposite directions, with Viberg et al. (2003b) reporting that activity increased and Rice et al. (2007) reporting a decrease in activity during the one and two hour evaluation periods, respectively. Overall, the differences among the three studies do not allow for a definitive conclusion to be made on whether BDE-209 is a developmental neurotoxicant. The Biesemeier et al. (2011) study, which reported no effects on the developing nervous system, has only recently been published in its entirety in the open scientific literature, and is expected to be reviewed and evaluated by experts in both the United States and the European Union.

2.9.2 Liver Toxicity

Short and long term oral studies report that exposure to high levels of BDE-209 causes liver toxicity in rodents. In a study conducted by the National Toxicology Program (NTP, 1986), rats and mice were exposed to BDE-209 in the diet for 103 weeks. The dose levels for rats were 0, 1,120 or 2,240 mg/kg/day in males and 0, 1,200, or 2,550 mg/kg/day in females). The dose levels for mice were 0, 3,200 or 6,650 mg/kg/day in males and 0, 3,760 or 7,780 mg/kg/day in females. The study design was generally consistent with guidelines for chronic toxicity studies established by the US EPA (1998). It exposed two rodent species, a sufficient number of animals (50 animals/sex/exposure group) and included clinical evaluations, microscopic evaluations of many organ tissues, and detailed reporting of study results and statistical analyses. A limitation of the study is the use of only two exposure groups rather than the three specified by the guidelines. Several effects on the liver of the animals were reported. Liver thrombosis was reported in male rats in the highest dose group. Males also had dose-related increases in liver degeneration, but these were not statistically significant. In the same study, mice exposed to BDE-209 had increased incidences of liver granuloma. However, these effects were only statistically significant in males in the lowest dose group, and a clear dose-related trend was not evident. Increased incidences of centrilobular hypertrophy in the liver were reported in the male mice at both dose levels.

In shorter-term studies, the offspring of female mice orally exposed to BDE-209 (10, 100, 500 or 1,500 mg/kg/day) from gestational day 0 to 17 had dose-related histopathological changes (e.g., swelling of hepatocytes) in the liver (Tseng et al., 2008). In a 28-day oral study in rats, Van der Ven et al. (2008) reported slight histopathological liver changes (i.e., centrilobular hypertrophy) in male rats exposed to 60 mg/kg/day commercial decaBDE, which was the highest dose tested. No effects on the liver were observed in rats or mice exposed to BDE-209 in the diet for 13 weeks at levels up to 11,566 mg/kg/day (NTP, 1986).

The European Union Risk Assessment Report on BDE-209 (ECB, 2002) used the NOEL of 1,120 mg/kg/day for liver toxicity and other noncarcinogenic effects in male rats from the 103 week NTP study (1986) to calculate margins of safety for workers and for people who may be exposed to BDE-209 in the environment. In this evaluation, the margin of safety is the numerical difference between an exposure of interest to a chemical (in this case, workplace and environmental exposure to BDE-209) and a NOEL or LOEL for the chemical. The margins of safety for BDE-209 were 96 and 560 for inhalation and dermal exposure in workers, respectively, meaning that the estimated exposures were 96 and 560 times lower than the NOEL. For people who may be exposed to BDE-209 from environmental sources, the margin of safety was 93,000 (i.e., the estimated environmental exposure was about 93,000 times lower than the NOEL of 1,120 mg/kg/day). Based on these margins of safety, which were considered sufficient (ECB, 2003), the risk assessment concluded (ECB, 2002) that for BDE-209 noncancer endpoints in workers and humans exposed via the environment, "there is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already." This evaluation predated publication of studies (Viberg et al., 2003b; Viberg et al., 2007; Johansson et al., 2008) reporting effects on the developing nervous system at lower exposure levels compared to those causing liver toxicity.

The National Academy of Sciences (NRC, 2000) chose the 103 week NTP study (1986) as the basis for their BDE-209 provisional reference dose. The NAS derivation used the NOEL of 1,120 mg/kg/day for liver thrombosis and degeneration in male rats as the point of departure, and applied a total uncertainty factor of 300 (10 for interspecies extrapolation, 10 for intraspecies variability and 3 for database uncertainties) to obtain a provisional reference dose of 4 mg/kg/day (NRC, 2000). The NAS noted that confidence in the provisional reference dose is medium to low based on high confidence in the NTP study and low confidence in the toxicological database

for BDE-209. As with the European Union evaluation, derivation of the provisional reference dose predated publication of studies on BDE-209 developmental neurotoxicity by Viberg et al. (2003b; 2007) and Johansson et al. (2008).

The CPSC also chose the 103 week NTP (1986) study as the basis for its reference dose (CPSC, 2006b), but used the LOEL of 3,200 mg/kg/day for increased incidence of liver granuloma in male mice as the point of departure. A total uncertainty factor of 1,000 (10 for interspecies extrapolation, 10 for intraspecies variability and 10 for use of a LOEL) was applied to the LOEL to obtain a reference dose of 3.2 mg/kg/day.

Hardy et al. (2009) modeled the dose response information for liver degeneration in male rats from the 103 week NTP study (1986) to obtain a lower bound on the dose associated with a 10 percent response. The authors applied a total uncertainty factor of 30 (3 for interspecies extrapolation, and 10 for intraspecies variability) to a point of departure of 113 mg/kg/day to obtain a reference dose of 4 mg/kg/day (Hardy et al., 2009).

2.9.3 Thyroid and Endocrine Toxicity

Several studies have evaluated the effects of BDE-209 on the endocrine system of animals. Rice et al. (2007) reported a dose-related reduction in thyroxine (T4) in 21-day old male mice orally exposed to 6 or 20 mg/kg BDE-209 on PNDs 2 through 15, although no statistically significant changes were noted between treated and control groups. Tseng et al. (2008) administered pregnant mice oral doses of 0, 10, 500, or 1,500 mg/kg/day BDE-209 on gestation days (GD) 0 through 17 (Tseng et al., 2008). Adult male offspring in the highest and lowest dose groups had significantly decreased serum levels of triiodothyronine (T3) compared to controls, but the middle dose group showed no change, and therefore the effects were not dose dependent. Animals in the high dose group were also reported to have mild histological changes of the thyroid glands. Hardy et al. (2009) stated that the results on T3 were of questionable biological relevance, particularly because of the magnitude of the highest dose level (1,500 mg/kg/day), which was higher than limits recommended by guidance documents (US EPA, 1998). In the 103 week NTP carcinogenicity study, male mice exposed to 3,200 or 6,650 mg/kg/day BDE-209 had an increased incidence of follicular cell hyperplasia of the thyroid gland, but these effects were not observed in female mice or rats in the same study (NTP, 1986). In rats, a 28-day oral study

reported dose related trends for a decrease in adrenal enzyme activity and an increase in thyroid (T3) hormone levels) in females orally exposed to commercial decaBDE doses ranging from 1.87 to 60 mg/kg/day (Van der Ven et al., 2008).

2.9.4 Reproductive Toxicity

Available information on the reproductive toxicity of BDE-209 appears mixed. Hardy et al. (2002) reported no treatment related effects on number of dams with viable fetuses, mean number of corpora lutea, number of implantation sites, percent preimplantation loss per dam, number of viable fetuses, and gravid uterine weights in Sprague-Dawley rats exposed by corn oil gavage to commercial decaBDE at doses up to 1,000 mg/kg/day during GDs 0 through 19. More recent studies report reproductive toxicity in animals exposed to BDE-209. Tseng et al. (2006) reported that mice orally exposed to 500 and 1,500 mg/kg/day BDE-209 from PND 21 through 70 had effects on sperm, including reduced mitochondrial membrane potential (a predictor of sperm fertility potential), reduced sperm motion velocity, and increased generation of hydrogen peroxide (Tseng et al., 2006). Van der Ven et al. (2008) reported dose-dependent effects on epididymis and seminal vesicle weights in male rats orally exposed to commercial decaBDE at doses ranging from 1.87 to 60 mg/kg/day for 28 days. However, these findings have been questioned by Hardy et al. (2008) who did an independent statistical analysis of the seminal vesicle weight data from the Van der Ven et al. (2008) study and found no significant difference in response between dose groups. They also noted that ~~"t~~though seminal vesicle weight was reported to increase significantly with dose, no dose response relationship is obvious from the organ weight data." Hardy et al. (2009) also questioned the biological significance of the effects reported in the Tseng et al. (2006) study, noting that the authors did not control for litter effects, and that no effects on the testes of rats or mice were observed at higher dose levels used in the NTP study (1986).

The ATSDR selected the NOEL of 1,000 mg/kg/day (the highest dose tested) from the Hardy et al. (2002) study as a point of departure to develop an intermediate MRL for BDE-209 (ATSDR, 2004a). The intermediate MRL is derived for exposure durations of more than two weeks to less than one year. A total uncertainty factor of 100 (10 for interspecies extrapolation and 10 for intraspecies variability) was applied to the NOEL to obtain the intermediate MRL of 10 mg/kg/day.

2.9.5 Carcinogenicity

Dietary studies of BDE-209 were conducted by the NTP (1986) to evaluate carcinogenic effects in rodents. Male and female rats exposed to BDE-209 in their diets (0, 1,120 or 2,240 mg/kg/day in males and 0, 1,200 or 2,550 mg/kg/day in females) for 103 weeks had dose-related increased incidences of liver neoplastic nodules (NTP, 1986). In males, these effects were statistically significant in both the high and low dose groups. The effects in females showed a dose-related pattern, but were only statistically significant in the highest dose group. Based on the increased incidences of liver neoplastic nodules in the low dose males and high dose groups of each sex, the NTP concluded there was some evidence of carcinogenicity for male and female rats. Male and female mice in the same study were exposed to BDE-209 in their diets over their lifetimes (3,200 and 6,650 mg/kg/day in males and 3,760 and 7,780 mg/kg/day in females). Male mice had statistically significant increases in hepatocellular adenomas or carcinomas (combined) at the lowest dose, and marginally increased incidences (not statistically significant) at the high dose. Increased incidences of hepatocellular adenomas and carcinomas were also reported in female mice, but these effects were not statistically significant. Male mice (but not female mice) also had an increase incidence of thyroid gland follicular cell hyperplasia (which is often considered to be a precursor to thyroid tumors [US EPA, 2008b]). The combined incidence of thyroid follicular cell adenomas and carcinomas was increased in both sexes of mice, but the increases were not statistically significant. Based on the combined increased incidences of hepatocellular adenomas or carcinomas in the low dose group and combined increased incidence of thyroid gland follicular cell adenomas or carcinomas in both dosed groups, the NTP concluded that there was equivocal evidence of carcinogenicity for male mice. The NTP concluded there was no evidence of carcinogenicity in female mice. A summary of the NTP conclusions is found in Table 2.9.5-1:

Table 2.9.5-1: Summary of NTP Conclusions for103 Week Carcinogenicity Studies of BDE-209 in Rats and Mice (NTP, 1986)

Species	Strain	Sex	NTP Conclusion	Carcinogenic Response
Rat	F344/N	Male	Some evidence of carcinogenicity	Increased incidences of liver neoplastic nodules in both dosed groups
Rat	F344/N	Female	Some evidence of carcinogenicity	Increased incidence of liver neoplastic nodules in high dose group
Mouse	$B6C3F_1$	Male	Equivocal evidence of carcinogenicity	Combined increased incidences of hepatocellular adenomas or carcinomas in low dose group; Combined increased incidence of thyroid gland follicular cell adenomas or carcinomas in both dosed groups
Mouse	$B6C3F_1$	Female	No evidence of carcinogenicity	Not applicable

The US EPA (2008a,b) concluded that there is "suggestive evidence of carcinogenic potential" (US EPA, 2008a) for BDE-209 and derived an oral cancer potency factor (7×10^{-4} per mg/kg/day) for BDE-209 based on the combined incidences of liver neoplastic nodules and carcinomas in male rats reported in the NTP (1986) study. Based on this estimate of cancer potency, the exposure dose associated with an increased lifetime cancer risk of one in one million is 1.4×10^{-3} mg/kg/day.

The European Union Risk Assessment Report did not classify BDE-209 for carcinogenicity based on the results of the 103 week NTP (1986) study, but still considered the lowest exposure dose in rats (1,120 mg/kg/day) a LOEL for carcinogenic effects that could be used to characterize the cancer risk for exposure to BDE-209 in the workplace and from environmental sources (ECB, 2002). Identical margins of safety were calculated using the same exposure estimates and methods used in the European Union's risk characterization of noncancer endpoints (see summary in Section 2.9.2, Liver Toxicity). The margins of safety were 96,560 and 93,000 for workers exposed by inhalation, workers exposed dermally, and people exposed from environmental sources, respectively. Based on these margins of safety, which were considered sufficient (ECB, 2003), the risk assessment concluded (ECB, 2002) that for BDE-209 carcinogenic endpoints in workers and humans exposed via the environment, "there is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already."

The European Union's method for evaluating carcinogenic risks differs from the default approach used by the US EPA (2005), which assumes in the absence of information on the cancer-causing mechanism of a chemical, there is no threshold for carcinogenic effects (i.e., exposure to even very small amounts of BDE-209 carries with it an incremental increase in cancer risk). The National Academy of Sciences (NRC, 2000) and the California Environmental Protection Agency (CA EPA, 2006) also made this assumption when evaluating cancer risks for BDE-209 (see following paragraphs).

The CA EPA Workgroup on PBDEs derived cancer potency factors for BDE-209 based on the same NTP study in rats used by the US EPA in their cancer potency factor derivation (CA EPA, 2006). Using the data for male and female rats, the cancer potency values were 1.08×10^{-3} per mg/kg/day and 5.9×10^{-4} per mg/kg/day, respectively. Based on these estimates of potency, the exposure doses associated with an increased lifetime cancer risk of one in one million are 9.3×10^{-4} mg/kg/day and 1.7×10^{-3} mg/kg/day for males and females, respectively. The cancer potency factors were derived using older methods for transspecies extrapolation that are no longer recommended in the current Guidelines for Carcinogen Risk Assessment (US EPA, 2005).

The National Academy of Sciences (NRC, 2000) used the data for liver neoplastic nodules in male rats to derive two cancer potency factors for BDE-209. The potency factors were 7×10^{-4} per mg/kg/day and 9×10^{-4} per mg/kg/day. The additional potency factor attempted to adjust for the differential mortality between the high and low dose male rats during the course of the study. Based on the National Academy of Science estimates of potency, the exposures associated with an increased lifetime cancer risk of one in one million are 1.4×10^{-3} mg/kg/day and 1.1×10^{-3} mg/kg/day.

Hardy et al. (2009) evaluated studies on the carcinogenicity of BDE-209, including the NTP (1986) study. They note that the liver and thyroid tumors in the male mice are likely the result of reduced survival in the control group, and the animals not living long enough to develop tumors typically found in older mice. Hardy et al. (2009) also suggested that there is uncertainty related to the description of the liver tumors as ~~"n~~eoplastic nodules," which, under the terminology used at the time could have pertained to liver hyperplasia (a lesion that does not necessarily become a

tumor) or liver adenomas (a benign tumor). Overall, they concluded that the high doses used in the study may produce some proliferative lesions in certain tissues (e.g., follicular hyperplasia of the thyroid) which may result in cancer effects by mechanisms that are not relevant to humans, and that the study results suggest low dose exposures will not induce non-neoplastic or neoplastic changes in cells.

2.9.6 Summary of Reference Doses for BDE-209

Animal studies have evaluated BDE-209 for its ability to cause developmental neurotoxicity, liver toxicity, thyroid and endocrine toxicity and reproductive toxicity. Table 2.9.6-1 summarizes the reference doses derived for BDE-209 which have been derived by health agencies and other investigators.

Table 2.9.6-1: Summary of Reference Doses for BDE-209

Agency/ Investigator	Reference Dose[1] (mg/kg/day)	Point of Departure		UF	Endpoint/Critical Study
		Dose (mg/kg/day)	Basis		
ATSDR, 2004a	10*	1,000	NOEL	100	Reproductive toxicity/Hardy et al. (2002)
CPSC, 2006	3.2	3,200	LOEL	1,000	Liver toxicity/NTP (1986)
Hardy et al., 2009	4	113	$BMDL_{10}$	30	Liver toxicity/NTP (1986)
NRC, 2000	4**	1,120	NOEL	300	Liver toxicity/NTP (1986)
US EPA, 2008a,b	0.007	2.22	NOEL	300	Neurodevelopmental toxicity/Viberg et al. (2003b)

[1]Agencies use different terms for the reference dose, including acceptable daily intake and minimal risk level (MRL).
*The ATSDR toxicity value is intermediate-MRL, which is derived for less than lifetime exposure durations.
**Provisional.
Intermediate MRLs are derived for exposure durations of more than two weeks to less than one year.
NOEL = no-observed effect level
LOEL = lowest-observed effect level
$BMDL_{10}$ = the lower bound estimate on the dose associated with a 10 percent response
UF = uncertainty factor
ATSDR = Agency for Toxic Substances and Disease Registry
CPSC = Consumer Products Safety Commission
NRC = National Research Council/National Academy of Sciences

Of the four available reference doses derived for long-term exposure, three are based on the 1986 NTP study and have produced similar values. Of these three reference doses, the derivation of Hardy et al. (2009) is preferred because it uses more current methods for determining a point of

departure (e.g., benchmark dose modeling). This reference dose is over 500 times higher (meaning the chemical is considered less toxic) than the US EPA reference dose (US EPA, 2008a,b) based on the developmental neurotoxicity study of Viberg et al. (2003b). The NTP is generally considered the best long-term toxicity study of BDE-209. The results of the Viberg et al. (2003b) study (and consequently the US EPA reference dose) should be considered in conjunction with the Biesemeier et al. (2011) study.

2.9.7 Summary of Cancer Potency Factors for BDE-209

Table 2.9.7-1 summarizes the cancer toxicity values derived for BDE-209 which have been derived by health agencies and other investigators, all of which were based on the NTP (1986) carcinogenicity study.

Table 2.9.7-1: Summary of Cancer Potency Factors for BDE-209 Based on NTP Carcinogenicity Study (1986)*

Agency	Cancer Potency Factor $(mg/kg/day)^{-1}$	Dose Response Information
NRC, 2000	7×10^{-4} 9×10^{-4} **	Liver neoplastic nodules in male rats
US EPA, 2008a,b	7×10^{-4}	Combined incidences of liver neoplastic nodules and carcinomas in male rats

*The cancer potency factor developed by the CA EPA was derived using a method for transspecies extrapolation no longer recommended in the current Guidelines for Carcinogenic Risk Assessment (US EPA, 2005), and is therefore not included.

**Derived to adjust for the differential mortality between the high and low dose male rats during the course of the study.

The cancer potency factor of 9×10^{-4} per mg/kg/day derived by the National Academy of Sciences (NRC, 2000) is the preferred value. In this derivation, animals in all the exposed groups that died before the 87th week of the study (before the first neoplastic nodule occurred) were removed and not considered in the calculation of tumor incidences. These animals may not have been exposed long enough to develop the tumors. This is considered a more refined treatment of the data and avoids the cancer potency from being reduced on the basis of animals that were at lower risk for cancer than animals that survived for the duration of the study.

2.9.8 Comparison of BDE-209 Exposure Estimates to BDE-209 Reference Doses

The US EPA estimated daily human intakes for PBDEs across exposure pathways and age groups (US EPA, 2010a). The pathways included dust ingestion, dermal contact with dust, inhalation, water ingestion, and the diet (milk, dairy products, eggs, meats and fish). The total estimated PBDE intake for adults is about 500 ng/day, of which about 28 percent (142 ng/day) is BDE-209. This corresponds to a daily estimated exposure dose of about 2 ng/kg/day assuming an adult body weight of 70 kg (US EPA, 2010a). Similar BDE-209 exposure estimates were not made for infants or other age groups (US EPA, 2010a).

The estimated BDE-209 exposure of 2 ng/kg/day for adults can be compared to the available reference doses derived by health agencies and other investigators, which ranged from 7000 to 10,000,000 ng/kg/day (Table 2.9.6-1). The reference dose represents an estimated exposure to humans (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime, and exposures below the reference dose are considered unlikely to result in adverse health effects. The lowest available reference dose, or the one that suggests the greatest degree of toxicity for BDE-209 is the US EPA value of 7000 ng/kg/day (0.007 mg/kg/day). The estimated BDE-209 exposure for adults of 2 ng/kg/day is 3,500 times lower than the US EPA reference dose. Although a BDE-209 exposure for infants was not estimated, the US EPA (2010a) estimated that infants are exposed to 141 ng/kg/day total PBDEs. If all of the PBDE exposure is assumed to be BDE-209, the infant exposure estimate is about 50 times lower than the US EPA reference dose. Since the estimated BDE-209 exposures are below the reference dose, they are unlikely to result in adverse health effects.

2.9.9 Conclusion

Human studies on the health effects of BDE-209 are not available. Therefore information on the health effects of BDE-209 comes primarily from toxicity studies in laboratory animals. These studies suggest that developmental neurotoxicity may be the most sensitive BDE-209 toxicological endpoint. However, the primary studies that evaluate the developmental neurotoxicity of BDE-209 in animals are markedly different with respect to their experimental methods and reported results, and are interpreted differently by scientists on this basis. The differences among the studies do not allow for a definitive conclusion to be made on whether

BDE-209 is a developmental neurotoxicant. Several health agencies and researchers have derived different reference doses for BDE-209, which represents an estimated human exposure (including sensitive subgroups) that is likely to be without an appreciable risk of noncancer adverse health effects during a lifetime. There is no scientific consensus on which reference dose is preferred for risk assessment of BDE-209. Current BDE-209 exposure estimates are lower than the available reference doses for BDE-209, which suggests they are unlikely to result in health effects.

2.10 Actions by the US EPA

The following section summarizes recent actions and activities of the US EPA related to decaBDE.

2.10.1 US EPA 2009 Action Plan

On December 30, 2009, the US EPA released an Action Plan for PBDEs (US EPA, 2009a,c). The following actions pertained to decaBDE:

- The US EPA supports the plan announced on December 17, 2009, to reduce the manufacturing, import and sales of decaBDE (see below). This 3-year phase out negotiated by the US EPA was by several of the major US manufactures of decaBDE. The US EPA intends to encourage other importers of decaBDE to also join this effort. The action plan noted that the phase out does not address articles made with BDE-209, nor was the phase out designed to address the recycling of materials containing decaBDE.

- In 2009, the US EPA announced its intention to propose a Significant New Use Rule (SNUR). The SNUR was proposed in 2012 and would, if finalized in its current form, designate the manufacturing and/or importation of decaBDE or new articles to which decaBDE has been added as significant new uses and would require that the agency be notified of such uses. The SNUR would go into effect after the 3-year phase out of decaBDE has been completed. The US EPA also proposed in 2012 a Test Rule under Section 4 of the Toxic Substances Control Act (TSCA) which, if finalized in its current

form would require information to determine the effects that manufacturing, and use of decaBDE has on human health or the environment.

- In 2009, the US EPA announced it intends to propose rulemaking under Section 5(b)(4) of TSCA that would add decaBDE commercial mixes to the list of chemicals that ~~p~~present or may present an unreasonable risk to health or the environment."

- The US EPA is developing an analysis of alternatives for decaBDE that will evaluate the alternatives' efficacy, availability, cost, hazard and risks (US EPA, 2011).

- In 2009, the US EPA announced it intends to evaluate the impact of decaBDE use on children and other subpopulations (subpopulations not specified).

2.10.2 US EPA Announcement of DecaBDE Industry Phase-Out Initiative

On December 17, 2009, the US EPA announced that the major US producers and importers of decaBDE will phase out production, importation, and sales for most uses by December 31, 2012, and will end all uses by the end of 2013 (US EPA, 2009d). The timeframe allows for phasing in alternatives, and provides time to evaluate and test new materials, especially for more complex uses in transportation and by the military. The phase out was a result of the US EPA's negotiations with the major United States manufacturers of decaBDE.

Company commitment letters to this phase-out initiative from Albermarle Corporation, Chemtura Corporation (formerly Great Lakes Chemical Corporation and Crompton Corporation), and ICL Industrial Products have been posted on the US EPA web site (US EPA, 2009d). Albermarle and Chemtura are the two major manufacturers of decaBDE, while ICL Industrial Products is a major importer of decaBDE. The corporations have agreed to submit annual progress reports that will include United States production, import, and sales figures as compared to their 2008 baseline figures. Companies have agreed not to stockpile decaBDE, and it is anticipated that any unsold inventory of decaBDE will be liquidated (sold) within six months of ending production.

The phase-out, however, will not affect the resale, reuse, disposal, or continued use of finished products that contain pre-phase out decaBDE, or the sale of products made from recycled materials that contain decaBDE, and will not affect minor importers of decaBDE or products containing decaBDE. When production has ceased, letters from the manufacturers confirming an end to decaBDE production will be posted on the US EPA's web site. As mentioned above in the 2009 Action Plan, it is anticipated that after the phase-out is complete, decaBDE would be regulated under a proposed SNUR, which if finalized in its current form, will designate the manufacturing and/or importation of decaBDE or new articles to which decaBDE has been added as significant new uses.

2.10.3 US EPA PBDE Project Plan

The US EPA's PBDE Project Plan, issued March 2006, outlines the agency's activities regarding PBDEs. Of the four major objectives in this plan, objective two was devoted specifically to "Assess and evaluate decabromodiphenyl ether" (US EPA, 2006). In December 2008, the US EPA issued a 14-page follow-up report, entitled "Tracking Progress on US EPA's PBDEs Project Plan: Status Report on Key Activities" (US EPA, 2008c). For the decaBDE objective, activities included:

- Completion of the agency's final Integrated Risk Information System (IRIS) assessment of BDE-209 in 2008 (US EPA, 2008b). A 126-page toxicological review was issued in support of the IRIS assessment in June 2008 (US EPA, 2008a).

- Monitoring the availability of risk assessments and toxicology findings on BDE-209, including the European Union developmental neurotoxicology study and additional studies published in the peer-reviewed literature. The developmental toxicity study (Biesemeier et al., 2011) has recently been published (see Section 2.9.1).

- Working with industry to investigate and identify data needs documenting the environmental fate of decaBDE. In September 2008, the Brominated Flame Retardants Industry Panel agreed to work with the US EPA to develop a TSCA Section 4 enforceable consent agreement as a means to ensure development of identified data

needs." The US EPA announced a proposed test rule in the December 30, 2009 Action Plan (see second bullet under "US EPA 2009 Action Plan").

- Peer review of the US EPA's National Center for Environmental Assessment (NCEA) chapter in its draft report on exposure and environmental fate of decaBDE, its debromination, and other fate pathways. The public comment period for the draft report ended on February 2, 2009. The final peer-reviewed report entitled "An Exposure Assessment of PBDEs" was posted on the NCEA homepage on May 14, 2010 (US EPA, 2010a).

- Continuing work toward the agency's goal of conducting an interim review of all available scientific information on BDE-209.

- Continuing to monitor a draft CPSC standard for the flammability of residential upholstered furniture. The 3-year phase out of decaBDE (see previous section) is anticipated to cover all uses for decaBDE, including those for residential upholstered furniture.

- In October 2010 under its Design for the Environment Program, the US EPA established the Flame-Retardant Alternatives for DecaBDE Partnership to "explore the human health and environmental profiles of functional and viable alternatives to decabromodiphenyl ether...to help inform the process of substituting to safer alternatives, with reduced health and environmental concerns..." (US EPA, 2011). In July 2012, the US EPA released a draft alternatives assessment report "An Alternatives Assessment for the Flame Retardant Decabromodiphenyl Ether (DecaBDE)" (US EPA, 2012a). Using measured and computer model-generated parameters, the assessment profiles the environmental and human health hazards of 30 alternatives to decaBDE that are already on the market. The report gave decaBDE a hazard ranking of "high" for developmental toxicity. This conclusion is consistent with the agency's earlier assessment of decaBDE (US EPA 2008a,b). The report also states that "under certain conditions, decaBDE can degrade to less brominated congeners, which are potentially more toxic." The public comment period on the draft report closed on September 30, 2012, and the agency's current plans are to issue a final report in early 2013.

2.10.4 US EPA Toxics Release Inventory (TRI)

The TRI is a publicly available US EPA database that includes toxic chemical release information on approximately 650 chemicals (US EPA, 2009e). BDE-209 is one of the chemicals tracked in this system. The TRI provides information on the quantity of chemicals released to air, surface water and land, and chemicals managed through recycling, energy recovery, treatment and disposal. The data reflect annual emissions and do not reflect the frequency or duration of emissions. The TRI does not cover all facilities, and does not include information about public exposure to chemicals. TRI data for 1987 through 2011, including data for BDE-209 and other chemicals are available on line (US EPA, 2012b). Information on the 2011 releases and on tools that can be used to view the data is also available (US EPA, 2012b,c).

2.10.5 Conclusions

The actions and activities of the US EPA related to decaBDE include a 2009 Action Plan that supports the 3-year phase out of the manufacturing, import, and sales of decaBDE, the proposed Significant New Use Rule (SNUR) and Test Rule for articles to which decaBDE has been added, proposed rulemaking to add decaBDE commercial mixes to a list of chemicals that may pose a risk to health or the environment, and plans to evaluate decaBDE alternatives and the impact of decaBDE use on children and other vulnerable populations. The US EPA continues to work with the CPSC on issues pertaining to PBDEs and flame retardancy of furniture, and maintains the TRI database which tracks releases of BDE-209 under the Emergency Planning and Community Right-to-Know Act and the Pollution Prevention Act.

2.11 Actions by the European Union

The following section summarizes the recent and major actions of the European Union involving decaBDE. These include human health and environmental risk assessments for BDE-209 (the major congener in commercial decaBDE) and actions taken under the Restriction of Hazardous Substances (RoHS) Directive. Some of the technical conclusions of the risk assessment and its updates have been already been discussed in Sections 2.5 through 2.9.

2.11.1 European Union Risk Assessment

In 1993, under Council Regulation (EEC) No 793/93 (23 March 1993), the existing substances program was initiated to identify and control the risks posed by certain chemical substances in the European Union. The program outlines a general strategy for the evaluation and control of chemical substances in four steps, specifically, data collection, priority setting, risk assessment, and risk reduction (EC, 2010). BDE-209 was included in the first list of priority substances published in 1994, which was developed based on several criteria, including health effects, exposure, and the availability of data. France and the United Kingdom were identified as ―rapporteur" Member States for BDE-209, meaning they were responsible for evaluating its human health and environmental risks, respectively, and where appropriate, for overseeing proposed strategies on limiting these risks. The European Union's health and environmental risk assessment for BDE-209 was therefore prepared under the existing substances program in compliance with Council Regulation No 793/93 (23 March 1993). The risk assessment was released in 2002 (ECB, 2002), and an addendum was added two years later (EA, 2004).

The results of the 2002 risk assessment were published in the Official Journal of the European Union (OJ 29.5.2008 C131/9) in 2008, and earlier reports are available on the European Commission Joint Research Center website (ECJRC, 2008). Conclusions were based on the consensus opinion of experts from all European Union Member States, followed by review of the Scientific Committee on Toxicity, Ecotoxicity and the Environment (SCTEE) and the Scientific Committee on Health and Environmental Risks (SCHER). SCTEE and SCHER are independent scientific committees of experts that provide the European Commission with advice on the toxicity of chemicals to humans and the environment.

The human health risk assessment made recommendations and conclusions about data needs about the potential for health risks to workers, consumers, and people exposed to BDE-209 via environmental media (see Table 2.11.1-1).

Table 2.11.1-1: Human Health Recommendations and Conclusions of the European Union Risk Assessment on BDE-209 (ECB, 2002)

Category	Conclusion	Information Needed
Workers	Further information and/or testing needed	Developmental neurotoxicity study in mice or rats*
Consumers	No need for further information, testing or risk reduction measures	---
Humans Exposed via the Environment	Further information and/or testing needed	Developmental neurotoxicity study in mice or rats*
Risks from Physico-Chemical Properties	No need for further information, testing or risk reduction measures	---

*Study completed (Biesemeier et al., 2011). See Section 2.9.1.

The environmental risk assessment (ECB, 2002) made recommendations and conclusions about aquatic and terrestrial ecosystems, the atmosphere, and microorganisms in the sewage treatment plant (see Table 2.11.1-2).

Table 2.11.1-2: Environmental Recommendations and Conclusions of the European Union Risk Assessment on BDE-209 (ECB, 2002)

Category	Conclusion	Information Needed
Aquatic and Terrestrial Ecosystem	Further information and/or testing needed	Ten year monitoring in birds, sewage sludge, sediment and air for contaminant levels of BDE-209 and some degradation products*
Atmosphere	No need for further information, testing or risk reduction measures	---
Microorganisms in the Sewage Treatment Plant	No need for further information, testing or risk reduction measures	---

*In progress.

In 2006, the European Union passed the Registration, Evaluation, Authorization and Restriction of Chemicals (REACH) regulation (EC 1907/2006) to address the production and use of

chemical substances and their potential effects on human health and the environment (EC, 2011a). Two purposes of the legislation were to control risks through better and earlier identification of the toxicological and environmental properties of chemicals, and to replace the existing patchwork of European Union regulations on chemicals which resulted in different rules for ~~"e~~existing" and ~~"n~~ew" substances (EC, 2007). In light of the adoption of REACH, the United Kingdom (one of the ~~"r~~apporteur" Member States responsible for the BDE-209 environmental risk assessment under the existing substances program) evaluated new scientific information that had become available since the publication of the original European Union risk assessment (EA, 2009a). This report concluded that although risks arising from the direct toxicity of BDE-209 have not been identified, there are still concerns related to its presence in food chains and the potential to debrominate to lower and more toxic PBDEs which can be persistent and bioaccumulative (EA, 2009a). The EA report further concluded that recent studies on the ability of BDE-209 to cause neurotoxic effects in mammals need to be fully evaluated by appropriate experts before conclusions can be drawn about any risk arising from toxicity (EA, 2009b).

2.11.2 The RoHS Directive

A second major action of the European Union involving decaBDE is the RoHS directive. The RoHS (2002/95/EC) directive was passed into law by the European Parliament and Council in 2003. The directive arose out of European Union strategies for reducing the content of hazardous substances in waste. The RoHS directive restricts the use of lead, cadmium, mercury, hexavalent chromium, polybrominated biphenyls and PBDEs in electronic and electrical equipment, and became effective on July 1, 2006.

In April 2005, the European Commission proposed that decaBDE be exempt from the RoHS directive. The European Council issued a decision in October 2005, exempting decaBDE from the regulation. In January, 2006, the European Parliament and Denmark initiated legal action to rescind decaBDE's exemption. In April, 2008 the European Court of Justice ruled that the exemption be annulled because the European Commission had not followed the procedures and requirements set out in the RoHS directive for the exempting substances. The Court did not rule on any scientific or technical aspects of the exemption. The annulment became effective on July 1, 2008.

2.11.3 Identification of BDE-209 as a Substance of Very High Concern

In September of 2012, the European Chemicals Agency, on request of the European Commission, proposed to identify BDE-209 as a substance of very high concern (SVHC). BDE-209 is considered to be a PBT/vPvB forming substance because of the high probability that it forms substances in the environment that have PBT/vPvB properties (UK, 2012). Inclusion on the list of SVHCs imposes new information requirements on manufacturers and importers of listed substances and on suppliers of preparations and articles containing the substances. The public comment period for the proposal closed on October 18, 2012. In December 2012, after reviewing public comments, the EU finalized its decision to include BDE-209 on its list of substances of very high concern.

The conclusion (UK, 2012) about decaBDE itself is, "DecaBDE is therefore not considered to meet the PBT or vPvB criteria on the basis of its intrinsic properties." DecaBDE is concluded to be very persistent; the bioaccumulation data are equivocal and the toxicity requirement is not met (the toxicity conclusion is tentative for now).

The PBT properties of the tetra through nona congeners groups are evaluated in the draft United Kingdom document (see Appendix 8 for more details). In summary, the draft evaluation is that the tetra congeners group meets the PBT and vPvB criteria with generally decreasing PBT concern going from the tetra through the nona congeners groups.

The overall conclusion and reasoning in the European document for decaBDE follows. "DecaBDE is very persistent and widely detected in many environmental compartments (including wildlife species). On the basis of available data it can be concluded that there is a high probability that decaBDE is transformed in the environment to form substances which themselves have PBT/vPvB properties, or act as precursors to such substances, in individual amounts greater than 0.1 percent w/w over timescales of a year.

DecaBDE is therefore considered to meet the definition of a PBT/vPvB forming substance in accordance with Annex XIII of the Reach Regulation, and thereby Article 57(d) and (e)."

2.12 Conclusions

DecaBDE has been used as a flame retardant in the electronics and textile industries. The widespread historical use of decaBDE has led to the presence of BDE-209 (the main congener in decaBDE) in the environment. BDE-209 can break down to less brominated PBDEs following exposure to sunlight or through biochemical or chemical processes. How fast and how much debromination occurs under environmentally relevant conditions isn‘t clear. Some of the less brominated PBDEs are considered to be persistent, bioaccumulative, and toxic chemicals and are more of a concern than BDE-209 for some of these properties.

BDE-209 has been detected in eggs and tissue from terrestrial and aquatic organisms, but the residues are generally low, suggesting that it does not strongly bioaccumulate. There is little information available at present to suggest that the concentrations of decaBDE present in the environment are likely to cause measurable adverse effects on fish and wildlife populations.

The detection of BDE-209 in human serum and breast milk indicates that the presence of BDE-209 in the environment can lead to human exposure. The US EPA estimated an average daily exposure of BDE-209 for adults of 2 ng/kg/day and an average daily exposure of total PBDEs for infants of 141 ng/kg/day.

Animal studies with BDE-209 have reported liver, thyroid, endocrine, and reproductive effects. Developmental neurotoxicity studies for BDE-209 have been carried out by several groups of scientists using different methods and obtaining different results (see Table 2.9.1-1). NTP concluded that BDE-209 exposure was associated with some evidence of carcinogenicity in rats. The evidence for carcinogenicity is considered equivocal in male mice, with no evidence of carcinogenicity in female mice. The data for some of these effects are stronger than for others.

Exposures are typically evaluated using risk assessment methods, which in turn depend on the results of studies in laboratory animals in the absence of human data. Estimated BDE-209 exposures can be compared to reference doses, the dose that is estimated to be without appreciable risk of noncancer health effects over a lifetime. The estimated BDE-209 adult exposure is about 3500 times lower than the lowest available reference dose (US EPA). Assuming that estimated PBDE exposure of infants is all BDE-209, the infant dose is about 50 times lower than the lowest reference dose.

In March 2006, the US EPA issued a PBDE project plan. For decaBDE, the US EPA has completed an Integrated Risk Information System assessment (2008), an exposure and environmental fate assessment (2010) and a draft alternatives assessment (2012). The US EPA has given a high hazard ranking to BDE-209 based on developmental neurotoxic effects in rats and concluded that decaBDE can degrade to less brominated PBDEs under certain conditions. It also supports the industry phase out plan for decaBDE.

In September of 2012, the EU proposed to identify BDE-209 as a substance of very high concern (ECA, 2012). BDE-209 is considered to be a PBT/vPvB forming substance because of the high probability that it forms substances in the environment that have PBT/vPvB properties (UK, 2012), but not because BDE-209 itself meets the PBT/vPvB criteria. In December 2012, after reviewing public comments, the EU finalized its decision to include BDE-209 on its list of substances of very high concern.

The phase out of production, importation, and sales on the part of the two main domestic manufacturers (Chemtura Corporation and Albemarle Corporation) and the main importer (Israeli Chemicals Limited) for most decaBDE uses by December 31, 2012, and ending all uses by December 31, 2013 will help limit the amount of decaBDE that can be released to the environment over time. The phase-out will not affect the resale, reuse, disposal, or continued use of finished products that contain pre-phase out decaBDE, importation of products manufactured elsewhere containing decaBDE, or the sale of products made from recycled materials that contain decaBDE, and will not affect minor importers of decaBDE.

The phase out should also help limit the amount of debromination products in the environment over time. Although the task force has not evaluated the toxicity of lower brominated PBDEs, several states, the EPA, and the EU have stated or suggested that some lower brominated PBDEs are more toxic and/or more bioaccumulative than BDE-209. Thus, BDE-209 can also be a concern because of risks posed by BDE-209 breakdown products.

3 Decabromodiphenyl Ether Alternatives

The Task Force charge requires an evaluation of alternatives to decaBDE. The method the Task Force used to identify possible alternatives for decaBDE is similar to strategies developed by the TURI (2005) and the LCSP (2005). Briefly, manufacturers have developed alternatives to using decaBDE that can meet flammability standards by chemical substitution (replacing decaBDE with an alternative flame retardant), material substitution (replacing a material that uses decaBDE with a material that does not), and combinations of chemical and material substitution (using an alternative flame retardant in a different host material). This section discusses examples of each of these substitution strategies. The specific alternatives include magnesium hydroxide in coatings of wires and cables as an example of chemical substitution, the use of aluminum casings in computers as an example of material substitution, and the use of resorcinol bis(diphenyl phosphate) in electronic enclosures and boric acid in mattress and upholstered furniture fire barriers as examples of chemical and material substitution combinations. Information on the toxicity, potential for bioaccumulation and potential breakdown products of magnesium hydroxide, RDP and boric acid is also presented.

An additional substitution strategy is product redesign, in which the organization, form or structure of a product is changed so that a chemical flame retardant such as decaBDE is no longer needed. We were unable to identify specific examples where redesign of a product replaced decaBDE.

Choosing a suitable alternative flame retardant depends on many considerations and is not a simple process. A manufacturer‘s choice of an alternative for a particular decaBDE application requires it to balance an evaluation of toxicity, environmental impacts, potential human exposure, overall data limitations, flame retardant effectiveness and cost. Parameters can be compared that describe a chemical‘s toxicity and ability to accumulate and/or persist in the environment. However, toxicity information on the alternative or its breakdown products may be limited or in some cases may not be available for some toxicological endpoints, and similar types of toxicity data (which would facilitate a comparison with decaBDE) may not be available for both chemicals. Estimates of potential human exposure associated with the use of the flame retardant should also be evaluated. However, these evaluations may also be limited by the availability and adequacy of data. Because of data limitations or differing properties among

chemicals (e.g., mammalian toxicity versus degradation data), being able to make definitive conclusions about one chemical being better than another is unlikely.

3.1 Chemical Substitution: Magnesium Hydroxide

3.1.1 Background

Magnesium hydroxide (CAS number 1309-42-8) is a white crystalline compound that can be manufactured by reacting magnesium salts with sodium hydroxide or by hydration of magnesium oxide (21 CFR Part 184.1428, 2009). It is also formed in nature as the mineral brucite (21 CFR Part 184.1428, 2009). Magnesium hydroxide can be used as an alternative flame retardant to decaBDE in several kinds of plastics and polymers, including polyolefins, polystyrene, polyvinyl chloride, polycarbonate, polyurethanes and polyamides. Examples of products for which magnesium hydroxide can be used as a flame retardant include wires and cables, electronic housing and parts (connectors, relays, switches and conduits), audio and video equipment, appliances, battery cases, and upholstery (Weil and Levchik, 2004; SCI, 2005; DEPA, 2006; MDEP/MCDCP, 2007; PPRC, 2005). The chemical structure of magnesium hydroxide is shown in Figure 3.1.1-1.

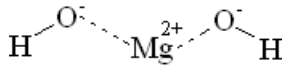


Figure 3.1.1-1: Magnesium Hydroxide
CAS Number: 1309-42-8
Empirical Formula: MgO_2H_2
Molecular Weight: 58.32

Some commercial magnesium hydroxide flame retardant products, such as MAGNIFIN®, are marketed specifically for use in wires and cables (Albermarle Corporation, 2013). Other magnesium hydroxide flame retardant products, such as FR-20, Kisuma® 5, and Magshield™, are commercially available and are marketed for general use (MMMS, 2008) or for use with polyolefins (Kisuma Chemicals, 2009).

Magnesium hydroxide is found in many commercial products such as toothpaste, frozen desserts, and over the counter medicines such as antacids and laxatives (e.g., Milk of Magnesia) (NRC, 2000). The U.S. Food and Drug Administration has designated magnesium hydroxide a "generally recognized as safe" direct human food substance for its use as a nutrient supplement, pH control agent, and processing aid (21 CFR Part 184.1428, 2009). Magnesium is an essential nutrient and is ubiquitous in food, water, biota, soil and dust. Typical levels of magnesium in soil in the eastern United States range from about 400 to 7,000 mg/kg with a geometric mean of 2,100 mg/kg for 528 samples (Shacklette and Boerngen, 1984). In a survey of rural surface soil throughout New York State, 118 samples had magnesium levels that ranged from 177 to 46,000 mg/kg, with a 98^{th} percentile of 7790 mg/kg (NYS DEC/NYS DOH, 2006). The range for magnesium in house dust in a survey of 48 Ottawa, Canada residences was 5,130 to 23,250 mg/kg, with a geometric mean of 9,442 mg/kg (Rasmussen et al., 2001).

The following sections summarize recent evaluations of magnesium hydroxide and the available toxicity, environmental fate and persistence information on magnesium hydroxide (including information on other divalent magnesium compounds). Information on the availability, efficacy and cost-effectiveness of magnesium hydroxide flame retardants is also summarized.

3.1.2 Previous Evaluations

Several reports from agencies and organizations have acknowledged the use and availability of magnesium hydroxide as an alternative flame retardant to decaBDE in electrical and electronic equipment such as wires and cables (DEPA, 2006, 2007; GFEA, 2000; IEPA, 2007; MDEP/MCDC, 2007; NRC, 2000; WDE/WDOH, 2004; ECB, 2007; US EPA, 2008d). The Illinois Environmental Protection Agency (IEPA, 2007) and the National Research Council (NRC, 2000) evaluated magnesium hydroxide as a flame retardant. These evaluations are summarized in Table 3.1.2-1.

Table 3.1.2-1: Evaluations of Magnesium Hydroxide as an Alternative to DecaBDE

Agency/Organization	Description	Conclusion
Illinois Environmental Protection Agency (IEPA, 2007)	Evaluation based on qualitative scoring (i.e., high, moderate, low, or of no concern) for human toxicity, ecotoxicity, persistence and bioaccumulation. Qualitative scoring formed the basis for designations of overall levels of concern (i.e., potentially unproblematic, potentially problematic, insufficient data, and not recommended).	Use of magnesium hydroxide meets the criteria for an overall scoring of potentially unproblematic. No cancer or reproductive/developmental data, but risk likely to be low based on professional judgment. Low concern for other effects based on existing data and professional judgment (human exposure data from food, medicinal, and cosmetic uses). Key data deficiencies include studies on reproductive and developmental toxicity, cancer and chronic aquatic toxicity.
National Research Council (NRC, 2000)	Based on an evaluation of toxicological and exposure data, potential health risks to consumers and the general population, and identification of data gaps associated with the use of magnesium hydroxide as a flame retardant in residential furniture. Evaluation included data on other divalent magnesium compounds.	The National Research Council concluded that despite the lack of a complete database, magnesium hydroxide can be used on residential furniture with minimal risk, even under worst-case assumptions.

The conclusions of these reports suggest that magnesium hydroxide is a suitable alternative flame retardant to decaBDE. A report from the IEPA (2007) also suggests that magnesium hydroxide poses less risk to human health and the environment than decaBDE, as this evaluation determined magnesium hydroxide to be "potentially unproblematic" while decaBDE was determined to be "potentially problematic."

3.1.3 Comparison of Available Toxicological Data

3.1.3.1 Human Data

Studies on the health effects of BDE-209 in humans are not available (US EPA, 2008a) and therefore a direct comparison of human effect levels cannot be made between BDE-209 and magnesium hydroxide. Information on the toxicity of magnesium hydroxide in humans is available. In addition, since oral exposure to magnesium hydroxide results in absorption of divalent magnesium ions (i.e., Mg^{2+}) into the body (NRC, 2000), toxicity data for other divalent

magnesium compounds can provide additional insight on magnesium hydroxide and are included in this review.

Mg^{2+} is an essential nutrient that is required for normal functioning of the human body, and is ubiquitous in the environment. Magnesium ions are used for processes such as anaerobic and aerobic energy generation. More than 300 enzyme systems in the body require magnesium ions (IOM, 1997). The recommended dietary allowances of nutritional magnesium for children older than one year and adults (including pregnant and lactating women) range from 80 mg/day to 420 mg/day (IOM, 1997). For infants, 30 mg/day to 75 mg/day of dietary magnesium is considered an adequate intake (IOM, 1997). According to the NHANES, estimates of usual daily magnesium intakes are 187 mg/day and 211 mg/day for children aged 1–3 years and 4–8 years, respectively (USDA/ARS, 2009). For males and females 9 years of age or older, usual daily magnesium intakes range from 239 mg/day to 373 mg/day and 211 mg/day to 317 mg/day, respectively (USDA/ARS, 2009).

3.1.3.1.1 Human Health Effects of Divalent Magnesium Compounds

Typical dietary levels of magnesium have not been reported to cause adverse health effects (IOM, 1997). Magnesium toxicity in humans appears to occur more often in people with impaired kidney function or gastrointestinal disease (NRC, 2000). Healthy individuals with normal kidney and gastrointestinal function can typically maintain magnesium homeostasis (i.e., the maintenance of relatively stable internal physiological conditions over a wide range of magnesium intakes [IOM, 1997]).

Information on the toxicity of divalent magnesium compounds in humans is available from studies of people given large amounts of magnesium ions in medicinal treatments such as tocolytics (medication to treat preterm labor in pregnant women). Magnesium sulfate is currently the most frequently used tocolytic agent in the United States (Blumenfeld and Lyell, 2009). A tocolytic treatment typically consists of a bolus dose of 4,000 to 6,000 milligrams of magnesium sulfate followed by maintenance doses of 2,000 to 4,000 milligrams per hour for 24 hours or more (Mittendorf and Pryde, 2005). At these elevated magnesium intakes (e.g., 10,100–19,800 milligrams per day), maternal side effects such as nausea, vomiting, lethargy, dizziness, blurry vision and headache are not unusual, and are reported to occur in about one third to two

thirds of women undergoing tocolytic treatment with magnesium sulfate (Blumenfeld and Lyell, 2009; Mercer and Melino, 2009). Some studies also suggest an increased risk for adverse effects on the fetus or newborn infants of women who were given large oral doses of magnesium sulfate in tocolytic treatments (Elimian et al., 2002; Mittendorf et al., 2002, 2006), but whether the increased risks are caused by magnesium or by some other factor is not clear (Blumenfeld and Lyell, 2009). Tocolytic treatment with magnesium sulfate rarely results in serious complications (Mercer and Melino, 2009).

Gastrointestinal symptoms have been reported in adults ingesting high levels of magnesium in laxative or antacids (NRC, 2000). Diarrhea is reported to be the most sensitive effect in humans, with a reported LOEL of 360 mg/day of magnesium for mild symptoms in cardiac patients receiving magnesium chloride therapy for six weeks, although many people can have higher magnesium intakes with no effects (IOM, 1997). Magnesium intoxication, which is characterized by symptoms such as diarrhea, nausea, muscle weakness, difficulty breathing, low blood pressure, and irregular heartbeat, has been reported in infants who have been given much higher levels of magnesium in laxatives (1,075–4,400 mg/day) over short periods of time (NRC, 2000). Important magnesium intake levels are summarized in Table 3.1.3.1.1-1.

Table 3.1.3.1.1-1: Summary of Important Magnesium Intakes

Description	Magnesium Intake (mg/day)	Comment
Recommended Daily Allowance (IOM, 1997)	80–420	
Usual Daily Intake (USDA/ARS, 2009)	187 (children, 1–3 years old) 211 (children, 4–8 years old) 239–373 (males, > 9 years old) 211–317 (females, > 9 years old)	
Tocolytic Intake (Mittendorf and Pryde, 2005; Blumenfeld and Lyell, 2009; Mercer and Melino, 2009)	10,100–19,800	Total intake over 24 hours associated with health effects (e.g., nausea, vomiting, lethargy, dizziness, blurry vision and headache) in about one third to two thirds of treated women but rarely serious complications.
Intake reported to cause magnesium toxicity in infants (NRC, 2000)	1075–4,400*	Effects seen in infants 2 to 42 days old (e.g., drowsiness, lethargy, difficulty breathing, low blood pressure, irregular heartbeat) given magnesium in laxatives for 2 to 11 days.
Lowest-observed-effect level (IOM, 1997)	360	Magnesium intake, in addition to dietary intake, that caused mild, reversible gastrointestinal symptoms (abdominal pain and diarrhea) in 6 of 21 cardiac patients treated with magnesium chloride for 6 weeks.

*Based on a dose range of 224–917 mg/kg/day (NRC, 2000), assuming an average body weight of 4.8 kg for an infant up to one month old (US EPA, 2008e).

3.1.3.1.2 Magnesium Hydroxide Reference Dose

In a report assessing alternative flame retardants to decaBDE in upholstered furniture, the NRC (2000) Subcommittee on Flame Retardant Chemicals recommended an oral reference dose of 12 mg/kg/day for magnesium hydroxide. The reference dose is defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of noncancer adverse health effects during a lifetime. The reference dose for magnesium hydroxide is based on the LOEL of 360 mg/day for mild diarrhea and gastrointestinal symptoms from ingestion by magnesium salts (IOM, 1997) assuming a body weight of 70 kg and adjusting for the relative amount of magnesium in magnesium hydroxide (ratio of 58.3/24.3). An uncertainty

factor of 1 was applied based on the mild, reversible nature of the effects. The reference dose corresponds to a magnesium hydroxide intake of 840 mg/day for a 70 kg adult, and 120 mg/day for a 10 kg child.

3.1.3.2 Animal Data

A brief comparison of the available toxicological studies in laboratory animals for magnesium hydroxide, other divalent magnesium compounds and BDE-209 is shown in Table 3.1.3.2-1.

Table 3.1.3.2-1: Comparison of Toxicological Studies in Laboratory Animals for Magnesium Hydroxide, Divalent Magnesium Compounds and BDE-209

Type of Study	Magnesium Hydroxide and Divalent Magnesium Compounds*	BDE-209
Subchronic Toxicity	$MgCl_2.6H_20$: 13-Week dietary study in mice (Tanaka et al., 1994). $Mg(OH)_2$: 8-Week dietary study in rats (Wang et al., 1994). $MgSO_4$: 6-Week study in rats (Ścibior et al., 2006).	13-Week dietary study in rats (NTP, 1986). 13-Week dietary study in mice (NTP, 1986).
Chronic Toxicity	$MgCl_2.6H_20$: 96-Week dietary study in mice (Kurata et al., 1989).	2-Year dietary study in rats (NTP, 1986). 2-Year dietary study in mice (NTP, 1986).
Reproductive Toxicity	No data	Gavage study in mice exposed on PNDs 21–70 (Tseng et al., 2006).
Developmental Toxicity	No data	Gavage study in pregnant rats exposed on GDs 0–19 (Hardy et al., 2002).**
Developmental Neurotoxicity	$Mg(C_2H_3O_2)_2.4H_20$: Dietary study of rats exposed on GD 5 – PND 40 (Newman et al., 2002). $MgSO_4$: Mice exposed (via intraperitoneal injections) on PNDs 3, 7 and 14 (Dribben et al., 2009).	Gavage study in mice exposed on PNDs 3, 10, or 19 (Viberg et al., 2003b). Gavage study in rats exposed on PND 3 (Viberg et al., 2007). Oral study (MP) in mice exposed on PNDs 2–15 (Rice et al., 2007). Gavage study in mice exposed on PND 3 (Johansson et al., 2008). Gavage study in rats exposed during gestation and through lactation day 21 (Biesemeier et al., (2011).
Immunotoxicity	No data	No data
Carcinogenicity	$MgCl_2.6H_20$: 96-Week dietary study in mice (Kurata et al., 1989).	2-Year dietary study in rats (NTP, 1986). 2-Year dietary study in mice (NTP, 1986).

GD = gestation day; PND = postnatal day; MP = micropipette

* $Mg(OH)_2$: magnesium hydroxide
$MgSO_4$: magnesium sulfate
$MgCl_2.6H_20$: magnesium chloride hexahydrate
$Mg(C_2H_3O_2)_2.4H_20$: magnesium acetate tetrahydrate

**Study used commercial decaBDE.

The database for magnesium hydroxide and divalent magnesium compounds does not include animal studies that evaluate reproductive and developmental toxicity although some human data are available (see Section 3.1.3.1.1). The NOELs and LOELs from toxicological studies for magnesium hydroxide, other divalent magnesium compounds and BDE-209 are shown in Table 3.1.3.2-2.

Table 3.1.3.2-2: No-Observed Effect Levels (NOELs) and Lowest-Observed Effect Levels (LOELs) from Available Toxicological Studies for Divalent Magnesium Compounds and BDE-209

Type of Study*	Divalent Magnesium Compounds**			BDE-209		
	Description (Reference)	NOEL (mg/kg/day)	LOEL (mg/kg/day)	Description (Reference)	NOEL (mg/kg/day)	LOEL (mg/kg/day)
Subchronic Toxicity	$MgCl_2.6H_20$: 13-Week dietary study in mice (Tanaka et al., 1994)	5,410	11,400	13-Week dietary study in mice (NTP, 1986)	11,566 (females)	Not identified
Chronic Toxicity	$MgCl_2.6H_20$: 96-Week dietary study in mice (Kurata et al., 1989)	730	3,930	2-Year dietary study in rats (NTP, 1986)	1,120 (males)	2,240 (males)
					---	419 (males)†
Reproductive Toxicity	No data	No data	No data	Gavage study in mice exposed on PNDs 21-70 (Tseng et al., 2006)	100	500
Developmental Toxicity	No data	No data	No data	Gavage study in pregnant rats exposed on GDs 0-19 (Hardy et al., 2002)***	1,000	Not identified
Developmental Neurotoxicity	$Mg(C_2H_3O_2)_2.4H_20$: Dietary study of rats exposed on GD 5 – PND 40 (Newman et al., 2002).	Not identified	170	Single dose gavage study in mice exposed on PND 3 (Johansson et al., 2008)	1.34	2.22
				Gavage study in rats exposed from GD 6 and through nursing until 3 weeks old (Biesemeier et al., 2011)	1000	Not identified
Immunotoxicity	No data	No data	No data	No data	No data	No data
Carcinogenicity	$MgCl_2.6H_20$: 96-Week dietary study in mice (Kurata et al., 1989)	3,930	Not identified	2-Year dietary study in rats (NTP, 1986)	Not identified	1,120

GD = gestation day; PND = postnatal day

*Studies reporting the highest NOEL or the lowest LOEL for each toxicological endpoint are shown

**$Mg(OH)_2$: magnesium hydroxide
$MgSO_4$: magnesium sulfate
$MgCl_2.6H_20$: magnesium chloride
$Mg(C_2H_3O_2)_2.4H_20$: magnesium acetate tetrahydrate

***Study used commercial decaBDE.

[†]$BMDL_{10}$: Lower bound estimate on the dose associated with a 10 percent response for liver effects in male rats (Hardy et al., 2009). The $BMDL_{10}$ is a mathematically modeled dose derived from the experimental data and takes into account the shape of the dose response curve.

The most sensitive noncancer effect associated with either magnesium ion or BDE-209 is developmental neurotoxicity reported in mice at a dose of 2.22 mg/kg/day of BDE-209 (Johansson et al., 2008). This level is about 75 times lower or more toxic than the lowest effect level (impaired learning in a water maze test at 170 mg/kg/day in rats) reported for magnesium ion. If the Johansson et al., study is not considered because of study design issues, then the lowest LOEL for BDE-209 is 419 mg/kg/day, about 2.5 times higher or less toxic than the 170 mg/kg/day LOEL for magnesium ion.

The NRC recommended an oral reference dose of 12 mg/kg/day for magnesium hydroxide. Several reference doses have been suggested for BDE-209 (see section 2.9.6). Hardy et al., derived a reference dose of 4 mg/kg/day and the US EPA derived one of 0.007 mg/kg/day. The reference doses for BDE-209 are lower, suggesting greater toxicity for BDE-209 than that for magnesium ion.

Carcinogenicity studies have been carried out with BDE-209 and with magnesium chloride. Exposure to divalent magnesium compounds is not associated with an increased cancer risk. A 96-week study of mice exposed to high levels of magnesium chloride in the diet (Kurata et al., 1989) reported no statistically significant increases in tumor incidence. For BDE-209, NTP reported some evidence of carcinogenicity based on increased incidences of liver neoplastic nodules in rats with equivocal evidence of carcinogenicity in male mice and no evidence in female mice exposed to high levels for their lifetimes (NTP, 1986; US EPA, 2008a,b).

3.1.3.3 Human Exposure Estimate for Magnesium Hydroxide Used as a Flame Retardant

The NRC (2000) estimated an average oral daily magnesium hydroxide dose of 0.021 mg/kg/day from its use as a flame retardant in upholstered furniture. The estimate assumes a one-year old child sucks on 50 cm^2 of magnesium hydroxide-treated fabric for one hour every day for two years. This estimated magnesium hydroxide exposure level can be used to calculate a magnesium intake (in mg/day) which can then be compared to typical magnesium intakes from other sources. Assuming a body weight of 10 kg for a one-year old child and that magnesium makes up 41.6 percent of magnesium hydroxide (based on the ratio of atomic and molecular

weights of magnesium and magnesium hydroxide, respectively [24.3/58.3]), the corresponding magnesium intake is 0.09 mg/day:

$$0.021 \text{ mg/kg/day} \times 10 \text{ kg} \times 0.416 = 0.09 \text{ mg/day}$$

As shown in Table 3.1.3.3-1, the estimated magnesium intake resulting from the use of magnesium hydroxide as a flame retardant (0.09 mg/day) is a small fraction of the recommended magnesium daily allowance and the magnesium usual daily intake (children 1–3 years old). This indicates that the use of magnesium hydroxide as a flame retardant in upholstery is unlikely to contribute significantly to typical magnesium exposures from other sources.

Table 3.1.3.3-1: Comparison of Estimated Magnesium Intake from Use of Magnesium Hydroxide as a Flame Retardant in Upholstery to Other Magnesium Intakes

Description	Intake (mg/day)	Number of Times Higher than Upholstery Intake
Estimated magnesium intake from magnesium hydroxide in upholstery (NRC, 2000)	0.09	----
Recommended daily allowance (IOM, 1997)	80–420	890–4670
Usual daily intake for children 1–3 years old (USDA/ARS, 2009)	187	2,080

The estimated magnesium hydroxide dose from its use as a flame retardant in upholstered furniture (0.021 mg/kg/day) can also be used to calculate a magnesium exposure dose (in mg/kg/day) for a child that can be compared to magnesium exposure levels that are known to cause adverse health effects. The magnesium hydroxide dose is converted to a magnesium dose by adjusting (as above) for the amount of magnesium in magnesium hydroxide (41.6 percent):

$$0.021 \text{ mg of magnesium hydroxide/kg/day} \times 0.416 = 0.009 \text{ mg of magnesium/kg/day}$$

As shown in Table 3.1.3.3-2, the estimated magnesium dose for a child from the use of magnesium hydroxide as a flame retardant (0.009 mg/kg/day) is much lower than any adverse

effect levels identified for divalent magnesium compounds in animals or humans. The large difference between the estimated magnesium dose from upholstery and magnesium doses that cause adverse health effects suggests that use of magnesium hydroxide as a flame retardant in upholstery poses a minimal risk to human health.

Table 3.1.3.3-2: Comparison of Estimated Magnesium Dose from Use of Magnesium Hydroxide as a Flame Retardant in Upholstery to Effect Levels for Divalent Magnesium Compounds

Description	Dose (mg/kg/day)[a]	Number of Times Higher than Upholstery Dose
Estimated magnesium dose from upholstery (NRC, 2000)	0.009	----
LOEL for developmental neurotoxicity in animals (Newman, et al., 2002)	20[b]	2,200
LOEL for chronic toxicity in animals (Kurata et al., 1989)	470[c]	52,200
LOEL for subchronic toxicity in animals (Tanaka et al., 1994)	1,360[d]	150,000
LOEL for gastrointestinal effects in humans (IOM, 1997)	36[e]	4,000

LOEL = lowest-observed effect level.
[a] Doses adjusted based on the relative amount of magnesium in the respective parent compound.
[b] Corresponds to LOEL dose of 170 mg/kg/day for magnesium acetate tetrahydrate (Table 3.1.3.2-2).
[c] Corresponds to LOEL dose of 3,930 mg/kg/day for magnesium chloride hexahydrate (Table 3.1.3.2-2).
[d] Corresponds to LOEL dose of 11,400 mg/kg/day for magnesium chloride hexahydrate (Table 3.1.3.2-2).
[e] Based on intake of 360 mg/day and assumes a 1 year old child weighs 10 kg (Table 3.1.3.1.1-1).

3.1.3.4 Conclusion for Toxicity

Magnesium is an essential nutrient, required for normal functioning of the human body. Magnesium hydroxide is in foods and many commercial products such as toothpaste and over the counter medicines such as antacids and laxatives. It is designated a ―generally recognized as a safe" as a direct human food substance for its use as a nutrient supplement, pH control agent and processing aid.

The estimated magnesium intake for a child from magnesium hydroxide used as a flame retardant is about 0.09 mg/day. This is a small fraction of the recommended daily allowance for magnesium (80–420 mg/day) and the usual daily intake (187 mg/day for children),

The LOELs for magnesium ion are higher or less toxic than the LOELs for BDE-209 if the studies of Viberg, Johansson, Rice and related neurotoxicity studies for BDE-209 (see Section 2.9.1) are considered and about 2.5 times lower or more toxic than the LOELs when these studies are not considered. The NRC reference dose for magnesium ion is higher suggesting less toxicity than the reference doses for BDE-209.

NTP concluded that BDE-209 has some evidence of carcinogenicity based on increased incidences of liver neoplastic nodules in rats with equivocal evidence of carcinogenicity in male mice and no evidence in female mice. Having some, although limited, evidence in mice of carcinogenicity in two species increases the concern about BDE-209‘s carcinogenic activity. Exposure to divalent magnesium compounds is not associated with an increased cancer risk.

Considering that magnesium is an essential nutrient and the toxicity information as a whole, magnesium hydroxide generally appears to be less toxic than BDE-209. The data for this comparison are convincing. The lack of reproductive and developmental toxicity information for magnesium ion, immunotoxicity information for both chemicals and inconsistent developmental neurotoxicity information for BDE-209 contributes some uncertainty to the conclusion.

3.1.4 Comparison of Data on Persistence

Magnesium hydroxide is relatively insoluble in water (9 mg/L at 18 ^{o}C [Hodgman, 1959]). Magnesium hydroxide that dissolves in water will dissociate to magnesium and hydroxide ions. Hydroxide ions will combine with available hydrogen ions to form water. Magnesium ions from magnesium hydroxide or its decomposition products will persist in the environment as magnesium hydroxide, as magnesium ions or as other, insoluble magnesium compounds (e.g., combine with silicate ions to form insoluble magnesium silicate). Magnesium compounds are present in significant quantities in environmental media (e.g., generally ranging from 200 ppm to 10,000 ppm in soil, see Section 3.1.1) and the use and production of magnesium hydroxide may change the distribution of magnesium, but not the global amount of magnesium.

Measured half-life data for BDE-209 in air, soil water and sediment were not found. Estimates of environmental half-lives of BDE-209 in air, water, soil and sediment are available using the PBT Profiler, which was developed by the Syracuse Research Corporation under contract to the US EPA's Office of Pollution Prevention and Toxics (US EPA, 2002). In the absence of measured data, the PBT Profiler is a model that uses chemical structure to estimate a chemical's potential to be persistent and bioaccumulative. Using the PBT Profiler, BDE-209 was estimated to have half-lives of 180 days, 360 days and 1,600 days in water, soil and sediment, respectively (see Section 3.3.1.6 for more information on the PBT Profiler), which are fairly long half-lives. BDE-209 can breakdown in the environment although how fast and how much breakdown occurs is not clear. The use and production of BDE-209 increases the global amount of BDE-209 and its breakdown products. Possible BDE-209 breakdown products (e.g., tetra through octa BDEs) are considered persistent chemicals (see Sections 2.5, 2.11.3, Appendix 8).

Overall, the available information suggests that both magnesium hydroxide and BDE-209 have the potential to persist in the environment. The production and use of BDE-209 increases the global amount of BDE-209; the production and use of magnesium hydroxide does not increase the global amount of magnesium although the distribution may change.

3.1.5 Comparison of Data on Bioaccumulation

Bioaccumulation is a measure of the amount of a contaminant in the organism compared to the amount of the contaminant in the environment around it. Bioaccumulation occurs through the uptake and retention of a chemical by an organism from its surrounding medium (air, soil, sediment or water) and from food. Bioaccumulation is often evaluated by measuring a chemical's uptake in fish and calculating a bioconcentration factor based on the concentration in fish tissue and the concentration in the water. Bioconcentration differs from bioaccumulation in that it refers to the uptake of substances into the organism from water alone.

Two studies were located that calculated bioaccumulation factors for magnesium in fish. One study evaluated the effects of metal contamination in an Egyptian Lake on bioaccumulation in fish and invertebrates (Ali and Fishar, 2005). The authors calculated bioaccumulation factors for magnesium based on the ratio between metal concentrations in sediment and fish (i.e., tilapia, sole and mugil) or three bottom invertebrates (i.e., mollusca, crustacea and annelida). The range

of calculated magnesium bioaccumulation factors was 0.12 to 0.22. Another study evaluated the bioaccumulation of magnesium and other heavy metals in fish (i.e., C. *carassius*, C. *nasus*, L. *cephalus* and A. *alburnaus*) in a Turkish lake based on metal concentrations in lake water and metal concentrations in fish (Uysal et al., 2009). The calculated magnesium bioaccumulation factors in this study ranged from 0.6 to 3.1. Overall, these studies suggest that magnesium hydroxide has a low potential to bioaccumulate.

Two bioconcentration studies report that BDE-209 does not accumulate to an appreciable extent in rainbow trout over 48 hours (Norris et al., 1973, 1974) or in carp over 6 weeks (CITI, 1992). Measured bioconcentration factors for BDE-209 were less than 5 at an initial water concentration of 60 mcg/L, and less than 50 at an initial water concentration of 6 mcg/L in the 6-week study in carp (CITI, 1992). These experiments were done above the water solubility limits of BDE-209 (less than 0.1 mcg/L [ECB, 2002]) and the reported concentrations of BDE-209 in water are questionable; therefore the calculated bioconcentration factors may be underestimated. Feeding studies with rainbow trout (*Oncorhynchus mykiss*) and carp report that the uptake of decaBDE is low, and the concentration of BDE-209 in the test species was generally lower than the concentration of BDE-209 in the food (Kierkegaard et al., 1999; Stapleton et al., 2004, 2006). In addition, at least some fish species are capable of metabolizing BDE-209 to lower brominated congeners (see Section 2.5.) which accumulate in the fish (Stapleton et al., 2006; Kierkegaard et al., 1999). Thus, the total bioaccumulation potential of BDE-209 may not have been recognized in the laboratory studies.

Overall, bioaccumulation and bioconcentration factors for magnesium and BDE-209 are not directly comparable due to differences in study methods, study parameters and fish species. However, the available studies suggest a low potential for both magnesium hydroxide and BDE-209 to build up in aquatic species.

3.1.6 Comparison of Data on Ecotoxicity

One way to evaluate ecotoxicity is to use data for aquatic species. Available experimental ecotoxicity data on aquatic species for divalent magnesium compounds (US EPA, 2008d) and BDE-209 (WWDE/WDOH, 2006) are in Table 3.1.6-1.

Table 3.1.6-1: Ecotoxicity Data for BDE-209, Magnesium Hydroxide and Divalent Magnesium Compounds

Chemical	Algae	Crustacean	Fish	Bacteria
Magnesium Hydroxide	EC_{50} = 2,111 mg/L (96 hours)*	LC_{50} = 648 mg/L (48 hours, *Daphnia*)**	LC_{50} = 1,110 mg/L (96 hours, Fathead Minnow)**	NA
BDE-209	NA***	NA***	LOEL = 7.5–10 mg/kg/day (120 days, rainbow trout)	NOEC $\geq$ 15 mg/L (3 hours, activated sludge micro-organisms)

EC_{50} = concentration causing an effect in 50 percent of the test subjects; LC_{50} = concentration causing lethality in 50 percent of the test subjects; NOEC = no-observed effect concentration; LOEL = lowest-observed effect level; NA = not available.

*Estimated value by US EPA (2008d) from a chronic study assuming an acute to chronic ratio of 4.

**Estimated by US EPA (2008d) from measured LC_{50}s for magnesium chloride and magnesium sulfate, adjusted for differences in molecular weight.

***An EC_{50} for algae and an LC_{50} for fish have been reported, but the experiments were done above the solubility limit for BDE-209 (less than 0.1 mcg/L [ECB, 2002]), which reduces the confidence in these experimental results.

The available experimental information on aquatic species for divalent magnesium compounds and BDE-209 is for disparate species, exposure times and reported parameters. The data are not directly comparable for drawing conclusions about the relative risks divalent magnesium compounds and BDE-209 might pose to aquatic species.

3.1.7 Comparison of Possible Breakdown Products

As discussed in Section 3.1.4, magnesium hydroxide is relatively insoluble in water. Magnesium hydroxide that dissolves in water forms divalent magnesium ions and hydroxide ions. Hydroxide ions will combine with available hydrogen ions to form water. Magnesium from magnesium hydroxide or its breakdown products will persist in the environment as magnesium hydroxide, as magnesium ions or as other, insoluble magnesium compounds. BDE-209 can break down to less brominated PBDEs certain ones are persistent, bioaccumulative and toxic (see Sections 2.5, 2.11.3, Appendix 8) so that BDE-209 in the environment could be a long-term source of these breakdown products,. How much and how fast debromination occurs under environmental conditions is unclear. Considering the major breakdown products of magnesium hydroxide and

BDE-209, the breakdown products of magnesium hydroxide generally appear to be less toxic and bioaccumulative than the breakdown products of BDE-209. The breakdown products of both magnesium hydroxide and BDE-209 are persistent.

3.1.8 Availability of Magnesium Hydroxide

The Task Force charge requires an assessment of the availability of alternative flame retardants to decaBDE. Magnesium hydroxide is used as a flame retardant in a wide range of electronic applications. In wire and cable, magnesium hydroxide is an alternative flame retardant to decaBDE in polyolefin resins (DEPA, 2006; MDEP/MCDCP, 2007; SCI, 2005). Several magnesium hydroxide flame retardants are commercially available, including products specifically marketed for use in wire and cable (e.g., MAGNIFIN® products) and polyolefins (e.g., FR-20 and Kisuma® 5). By one estimate, magnesium hydroxide accounted for approximately 4 percent of flame retardant consumption in Europe in 2005 (KRC, 2007). An earlier estimate from 2001 suggests a similar consumption rate of nearly 5 percent in Europe (DEPA, 2006). In 1993, about 3,000 tons of magnesium hydroxide flame retardants were marketed in the United States (NRC, 2000). This amount was an increase from previous years, suggesting a growing use of magnesium hydroxide flame retardants (NRC, 2000; IPCS, 1997). The German Federal Agency‘s report on flame retardant alternatives to decaBDE (GFEA, 2000) reported predictions of annual growth for magnesium hydroxide flame retardant consumption in Europe. Annual growth rates were projected at approximately 15 percent between 1995 and 2000, and 10 percent between 1998 and 2003. Annual growth rate predictions were considerably higher for magnesium than for other types of flame retardants (e.g., halogenated compounds, phosphorous compounds, melamine and other inorganic flame retardants).

Based on its presence in commercial flame retardants currently in use in Europe and the United States and that the use of these products is increasing, magnesium hydroxide is a technically feasible and available flame retardant alternative to decaBDE.

3.1.9 Reliability of Magnesium Hydroxide

The Task Force charge requires an assessment of the reliability (flame retardant effectiveness) of alternatives to decaBDE. The UL 94 Test for Flammability of Plastic Materials for Parts in

Devices and Appliances is used to evaluate the ability of plastics to withstand combustion following direct contact with an open-flame. Plastics that show the highest degree of flame retardancy (i.e., resistance to combustion) are rated V-0 based on a three tiered rating system (V-2, V-1 and V-0). The V-0 rating means that burning stops within 10 seconds after two applications (10 seconds each) of a flame to the test material, with no flaming material dripping from the surface of the tested plastic. The UL 94 flammability test is often used for plastics and as a screening tool in the development of wire and cable formulations (Lee et al., 2008) and provides useful information on the flame retardant properties of magnesium hydroxide.

Magnesium hydroxide is an alternative flame retardant to decaBDE in wire and cable made of polyolefin (e.g., polypropylene and polyethylene) (DEPA, 2006; MDEP/MCDCP, 2007; SCI, 2005). Polyolefins and other plastics used in cable and wire applications (e.g., polypropylene, low density polyethylene, polyamide, ethyl vinyl acetate and polybutylene terephthalate) can meet a fire safety rating of V-0 when used in combination with magnesium hydroxide flame retardants (Chen et al., 2007b; DEPA, 2006; Du et al., 2006; Fu and Qu, 2004; KRC, 2007; Levchik and Weil, 2005; Wang et al., 2002). This is an equivalent performance rating to decaBDE in polyolefin plastics, which also meets the V-0 fire safety rating (DEPA, 2007). Typical loadings of decaBDE in V-0 grade polyolefins range from 20 to 30 percent (DEPA, 2007). Higher loadings of magnesium hydroxide (45 to 65 percent) and/or chemical enhancers that increase the flame retardant properties of magnesium hydroxide are used to achieve a V-0 flammability rating in polyolefins and other resins used in wire and cable applications (Chen et al., 2007b; DEPA, 2006; Du et al., 2006; Fu and Qu, 2004; KRC, 2007; Levchik and Weil, 2005; Wang et al., 2002). High loading of magnesium hydroxide in wire and cable can result in poor processibility (i.e., the relative ease of making final products of acceptable quality from raw materials, including flame retardants) and physical properties (e.g., flexibility and resistance to damage) (Lee et al., 2008). However, chemical enhancers (e.g., expandable graphite) can reduce the levels of magnesium hydroxide required to achieve a V-0 fire safety rating (Chen et al., 2007b; Levchick and Weil, 2005). In addition, technologies are available to address the high loading of metal hydroxide flame retardants (e.g., magnesium hydroxide). For example, surface treatments (or coatings) of metal hydroxide flame retardants can improve their processing behavior in wire and cable, and the use of nanoparticles can enhance thermo-mechanical properties, barrier properties and flame retardancy (KRC, 2007).

Magnesium hydroxide is found in many commercial flame retardants in use in Europe and the United States and is equally effective as decaBDE in the UL 94 test (i.e., meets the V-0 rating) in polyolefins and other plastics. The available information suggests that magnesium hydroxide can be a reliable and effective alternative flame retardant to decaBDE.

3.1.10 Cost-Effectiveness of Magnesium Hydroxide

The Task Force charge requires an assessment of the cost-effectiveness of flame retardant alternatives to decaBDE. The cost-effectiveness of decaBDE alternatives is influenced by the cost of the alternative flame retardant and use of host materials (i.e., plastics) or chemical flame retardant enhancers when necessary. An evaluation of the cost effectiveness of magnesium hydroxide as a flame retardant is available from the Danish Environmental Protection Agency (DEPA, 2006), which evaluated the costs of decaBDE alternatives in V-0 grade glass-fiber-reinforced polyamide (PA 6 GF). Polyamide is mainly used in small electrical parts such as connectors or switches (DEPA, 2006) and can also be used in wire and cable applications (e.g., BASF Ultramid® Polyamide 6) (BASF Corporation, 2009). The Danish EPA's cost estimates are based on the prices of raw materials and are in Table 3.1.10-1.

Table 3.1.10-1: Cost Comparison of Magnesium Hydroxide and DecaBDE in V-0 Grade Glass-Fiber-Reinforced Polyamide 6 (PA 6 GF)[1]

Flame Retardant (FR)	FR Price (bulk) (€/kg)	FR Loading in Host Material	FR Cost (adjusted for loading in host material) (€/kg PA 6 GF)	Cost of Host Material (adjusted for FR loading)* (€/kg PA 6 GF)	Cost of FR and host material combined (€/kg of flame retarded PA 6 GF)
magnesium hydroxide	1.00–1.50	45%	1.25 (average price) x 45% = 0.563	2.23 x 55% = 1.23	0.563 + 1.23 = 1.79
decaBDE (with ATO)	decaBDE: 2.75 ATO: 2.75	decaBDE: 14% ATO: 5%	2.75 x 19% = 0.52	2.23 x 81% = 1.81	0.52 + 1.81 = 2.33

[1]Table adapted from DEPA (2006). All prices are listed in euros (€).
*The estimated raw material cost of PA 6 GF plastic (without added flame retardants) is 2.23 €/kg.

Based on the Danish EPA's cost estimates shown in Table 3.1.10-1, the cost of the flame retardant and host material combined in PA 6 GF is about 23 percent lower using magnesium hydroxide rather than decaBDE. Total cost estimates for decaBDE include the use of a chemical

enhancer, ATO, which increases the flame retardant properties of decaBDE. Although bulk magnesium hydroxide was estimated to be about 2 to 3 times cheaper per unit weight than decaBDE and ATO, high loading of magnesium hydroxide in the host material result in a similar costs per kilogram PA 6 GF (i.e., 0.563 euro per kilogram [€ /kg] for magnesium hydroxide and 0.52 € /kg for decaBDE and ATO). The Danish EPA's cost estimates for polyamide and flame retardant suggests that magnesium hydroxide is a cost-effective alternative flame retardant to decaBDE despite high loadings in the host material in this application. The availability of commercial magnesium hydroxide flame retardants specifically marketed for use wire and cable, polyolefins or other applications further supports the conclusion that magnesium hydroxide is a cost-effective replacement to decaBDE.

3.1.11 Conclusions

The Task Force is to –evaluate the availability of safer alternatives…including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes." The toxicity, persistence, and bioaccumulation of magnesium hydroxide and BDE-209 and their breakdown products were compared to evaluate if magnesium hydroxide is a safer alternative for BDE-209 (see Section 1.3 for a definition). The Task Force also assessed the reliability, ready availability and cost-effectiveness of magnesium hydroxide.

Toxicity

Magnesium is an essential nutrient, required for normal functioning of the human body. Magnesium hydroxide is in foods and many commercial products such as toothpaste and over the counter medicines such as antacids and laxatives. It is designated a –generally recognized as safe" as a direct human food substance for its use as a nutrient supplement, pH control agent and processing aid.

The estimated magnesium intake for a child from magnesium hydroxide used as a flame retardant is about 0.09 mg/day. This is a small fraction of the recommended daily allowance for magnesium (80–420 mg) and the usual daily intake (187 mg for children),

The most sensitive noncancer effect associated with either magnesium ion or BDE-209 is developmental neurotoxicity reported in mice at a dose of 2.22 mg/kg/day of BDE-209. This

level is about 75 times lower or more toxic than the lowest effect level (impaired learning in a water maze test at 170 mg/kg/day in rats) reported for magnesium ion. If the studies of Viberg, Johansson, Rice and related neurotoxicity studies (see Section 2.9.1) are not considered because of study design issues, then the lowest LOEL for BDE-209 is 419 mg/kg/day, about 2.5 times higher or less toxic than the 170 mg/kg/day LOEL for magnesium ion.

The NRC recommended an oral reference dose of 12 mg/kg/day for magnesium hydroxide. Several reference doses have been suggested for BDE-209 (see section 2.9.6). Hardy et al., derived a reference dose of 4 mg/kg/day and the US EPA derived one of 0.007 mg/kg/day. The reference doses for BDE-209 are lower, suggesting greater toxicity for BDE-209 than that for magnesium ion.

Carcinogenicity studies have been carried out with BDE-209 and with magnesium chloride. Exposure to divalent magnesium compounds is not associated with an increased cancer risk. A 96-week study of mice exposed to high levels of magnesium chloride in the diet (Kurata et al., 1989) reported no statistically significant increases in tumor incidence. For BDE-209, NTP reported some evidence of carcinogenicity based on increased incidences of liver neoplastic nodules in rats and equivocal evidence of carcinogenicity in male mice and no evidence in female mice exposed to high levels for their lifetimes (NTP, 1986; US EPA, 2008a,b). Having some evidence of carcinogenicity in two species increases the concern about BDE-209‘s carcinogenic activity.

In comparing all endpoints for toxicity, noncancer and cancer effects need to be considered together. Considering that magnesium is an essential nutrient and the toxicity information for both noncancer and cancer endpoints, magnesium hydroxide generally appears to be less toxic than BDE-209. The data for this comparison are convincing. The lack of reproductive and developmental toxicity information for magnesium ion, immunotoxicity information for both chemicals and inconsistent developmental neurotoxicity information for BDE-209 contribute some uncertainty to the conclusion.

Persistence

Overall, the available information suggests that both magnesium hydroxide and BDE-209 have the potential to persist in the environment. The production and use of BDE-209 increases the

global amount of BDE-209; the production and use of magnesium hydroxide does not increase the global amount of magnesium although its distribution can change and its bioavailability may increase to some extent (see Section 3.1.3.3). The uncertainty in this conclusion is fairly small.

Bioaccumulation

Overall, bioaccumulation and bioconcentration factors for magnesium and BDE-209 are not directly comparable due to differences in study methods, study parameters and fish species. However, the available studies suggest a low potential for both magnesium hydroxide and BDE-209 to build up in aquatic species.

Breakdown Products

Magnesium hydroxide is relatively insoluble in water. Magnesium hydroxide that dissolves in water forms divalent magnesium ions and hydroxide ions. Hydroxide ions will combine with available hydrogen ions to form water. Magnesium from magnesium hydroxide or its breakdown products will persist in the environment as magnesium hydroxide, as magnesium ions or as other, insoluble magnesium compounds. BDE-209 can break down to less brominated PBDEs and certain ones are persistent, bioaccumulative and toxic (see Sections 2.5, 2.11.3, Appendix 8) so that BDE-209 in the environment could be a long-term source of these breakdown products. How much and how fast debromination occurs under environmental conditions is unclear. Considering the major breakdown products of magnesium hydroxide and BDE-209, the breakdown products of magnesium hydroxide generally appear to be less toxic and bioaccumulative than the breakdown products of BDE-209 and thus the BDE-209 breakdown products are generally more of a concern. The breakdown products of both magnesium hydroxide and BDE-209 are persistent. The uncertainty in this conclusion is relatively small.

Overall Conclusion

For this report, an alternative is deemed to be safer if the characteristics of persistence, bioaccumulation and toxicity considered together of the alternative and its environmental breakdown products generally appear to be less than the characteristics of persistence, bioaccumulation and toxicity of BDE-209 and its breakdown products (see section 1.3 for more details). BDE-209 generally appears to be more toxic than magnesium hydroxide, both

compounds are persistent, and neither compound tends to bioaccumulate. The breakdown products of BDE-209 are persistent, bioaccumulate and toxic (see Sections 2.5, 2.11.3, Appendix 8). The breakdown products of magnesium hydroxide are persistent, but do not bioaccumulate and are not toxic. In considering all these factors together, magnesium hydroxide generally appears to be a safer alternative for BDE-209. This conclusion has some uncertainty because of data limitations, although the uncertainty is relatively small. One example of uncertainty is that the interpretations of the developmental neurotoxicity data for BDE-209 differ.

Magnesium hydroxide is available and has been used as a flame retardant in products (e.g., cables and wires) in Europe and the United States. Products containing magnesium hydroxide were resistant to combustion, demonstrating its reliability. Magnesium hydroxide is also cost effective as its cost to treat polyamide plastic is about 23 percent lower than the cost of using decaBDE. These data show that magnesium hydroxide is available, reliable and cost effective.

These conclusions have strengths and limitations (see previous sections for more detail). For example, persistence has a different context for magnesium hydroxide and its breakdown products than persistence for BDE-209 and its breakdown products, making comparisons difficult. The cost effectiveness evaluation is limited. In contrast, the database for evaluating magnesium hydroxide‘s toxicity is strong.

3.2 Material Substitution in Electronic Products

The primary alternatives to decaBDE in electronic products are other chemical flame retardants (CPA, 2007; LCSP, 2005; WDOH/DEC, 2006). However, additional options that eliminate the need for decaBDE or other chemical flame retardants are available. Metal or inherently flame-resistant plastic can be substituted in certain electronic products. Options that eliminate the need for flame retardants through material substitution while meeting fire safety standards and performance specifications are considered preferable, particularly when product materials are derived from chemicals of lower toxicity and the products and/or materials are able to be recycled or composted (CPA, 2007).

An example of a material substitution that eliminates or reduces the need for chemical flame retardants is provided by Apple Inc., which recently phased out brominated flame retardants

(including decaBDE) in many of its computer products (Apple Inc., 2010a). Apple Inc. replaced electronic encasements previously made of polycarbonate with encasements made of aluminum alloy, thereby eliminating the need to use flame retardants (CPA, 2004, 2007). Aluminum casings have been adopted for several Apple Inc. products including laptops, computer monitors, central processing units and servers (Apple Inc., 2009a,b,c,d). The phase out of brominated flame retardants by Apple Inc. covers all parts of new Apple product designs manufactured after December 31, 2008 (Apple Inc., 2010b).

Specific information on the reliability (i.e., flame retardant effectiveness) and cost effectiveness of substituting plastic encasements containing decaBDE with aluminum alloy casings is not available. However, aluminum alloys are known to be able to withstand high temperatures without igniting for the purposes of molding and casting. Further, according to one estimate, Apple Inc. is currently among the top five computer vendors in the United States, making up nearly 8 percent of the consumer market in 2009 (IDC, 2009), and aluminum enclosures have become a signature feature of Apple Inc. products (Apple Inc., 2005a,b). Thus, reliability and cost effectiveness of these products that have been redesigned through material substitution can be inferred since Apple Inc. products are commercially available and constitute a significant portion of the United States computer market. An additional environmental attribute associated with the use of highly recyclable materials such as aluminum is that it minimizes material waste at the end of a product's life cycle (Apple Inc., 2009a,b,c,d).

In summary, the use of aluminum alloy casings in certain laptop computers, computer monitors, central processing units and servers is an example of an available and cost effective material substitution that makes using decaBDE unnecessary, while still maintaining flame retardant properties.

3.3 Combinations of Chemical and Material Substitution: Resorcinol Bis(diphenyl phosphate) in Electronic Enclosures and Boric Acid in Fire Barriers for Mattresses

3.3.1 Resorcinol Bis(diphenyl phosphate)

3.3.1.1 Background

Resorcinol bis(diphenylphosphate), or RDP, is a non-halogen, phosphorus-containing flame retardant. The chemical name for RDP is phosphoric acid, 1,3-phenylene tetraphenyl ester (CAS number 57583-54-7). Its chemical structure is shown in Figure 3.3.1.1-1.

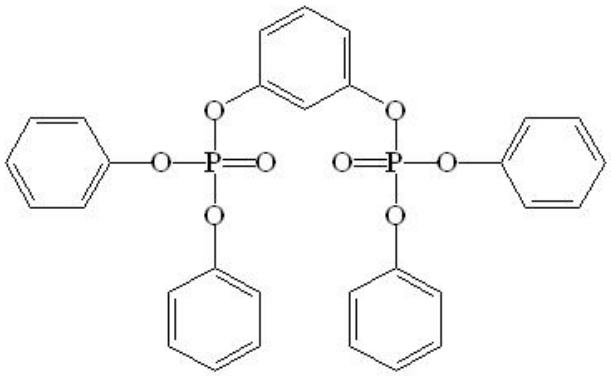
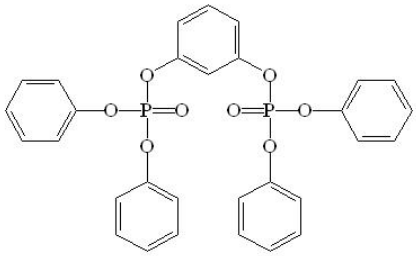
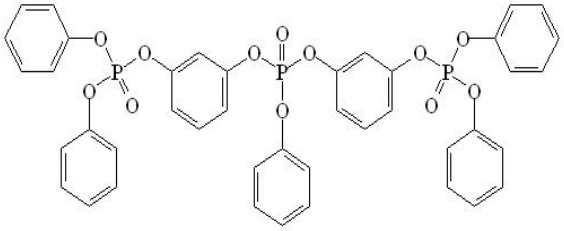
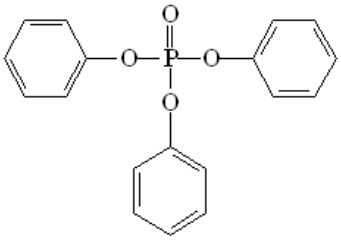


Figure 3.3.1.1-1: Resorcinol Bis(diphenylphosphate)
CAS Number 57583-54-7
Empirical Formula: $C_{30}H_{24}P_2O_8$
Molecular Weight 574.46

Commercial flame retardants containing RDP include Fyrolflex® RDP and Reofos® RDP. The commercial RDP formulations are mixtures of chemicals. Bright et al. (1997) and Henrich et al. (2000a) report that Fyrolflex® RDP is made up of 60–80 percent RDP, less than 5 percent triphenyl phosphate (CAS number 115-86-6), and 15 to 30 percent higher oligomers (small polymers). According to SRC (2006b) and CPA (2007), most of the higher oligomeric portion is expected to be phosphoric acid, bis[3-[(diphenoxyphosphinyl)oxy]phenyl] phenyl ester (CAS number 98165-92-5), which is the dimer of RDP (i.e., has two repeating units). SRC (2006b) reports that other higher molecular weight oligomers are also expected to be present, but probably constitute less than 2 percent of the entire mixture. The main chemicals in Fyrolflex® RDP are shown in Table 3.3.1.1-1.

Table 3.3.1.1-1: Chemicals in Commercial Fyrolflex® RDP

Chemical	CAS Number	Composition	Structure
RDP (phosphoric acid, 1,3-phenylene tetraphenyl ester)	57583-54-7	60–80%	
RDP dimer (phosphoric acid, bis[3-[(diphenoxy-phosphinyl)oxy]phenyl] phenyl ester)	98165-92-5	15–30%	
triphenyl phosphate	115-86-6	< 5%	

Unless otherwise noted, RDP in this document will refer to the commercial product, as most of the available information (particularly toxicological studies) is for the commercial flame retardant.

RDP is an alternative flame retardant to decaBDE for television encasements and consumer electronics. It is not a direct replacement for decaBDE in high impact polystyrene, which is typically used for television encasements when decaBDE is the flame retardant. For RDP to be used in television encasements, the manufacturer must use different plastics such as blends of high impact polystyrene/polyphenylene oxide or polycarbonate/acrylonitrile-butadiene-styrene (LCSP, 2005; WDE/WDOH, 2006).

The following sections summarize the recent evaluations of RDP as an alternative flame retardant for decaBDE, compare the available toxicity, environmental fate (including breakdown products) and persistence information for RDP and BDE-209, and summarize the available information on RDP's availability, efficacy and cost-effectiveness.

3.3.1.2 Previous Evaluations

Several agencies and organizations have evaluated RDP as an alternative to decaBDE (LCSP, 2005; WDE/WDOH, 2006, 2008; SRC, 2006b; CPA, 2007; IEPA, 2007; KRC, 2007; MDEP/MCDCP, 2007). The more recent and/or extensive evaluations are briefly summarized in Table 3.3.1.2-1. Detailed information on the methods used in these reports is available (WDE/WDOH, 2006, 2008; MDEP/MCDC, 2007; IEPA, 2007; CPA, 2007).

Table 3.3.1.2-1: Evaluations of RDP as an Alternative to DecaBDE

Agency/Organization	Description	Conclusion
Washington State Departments of Ecology and Health (2006, 2008)	Evaluation based on qualitative ratings (high, moderate, low) for potential for human toxicity, ecotoxicity, human exposure, persistence and bioaccumulation.	RDP is a safer and technically feasible alternative to decaBDE. RDP's low environmental persistence, moderate bioaccumulation potential and moderate toxicity make it a safer alternative than decaBDE for use in electronic enclosures.
Maine Departments of Environmental Protection and Center for Disease Control and Prevention (2007)	Evaluation based on available data on persistence, bioconcentration, mammalian and aquatic toxicity.	RDP presents a significantly lower threat to the environment and human health than does decaBDE or other brominated chemicals.
Illinois Environmental Protection Agency (2007)	Evaluation based on qualitative scoring (i.e., high, moderate, low, or of no concern) for human toxicity, ecotoxicity, persistence and bioaccumulation. Qualitative scoring formed the basis for designations of overall levels of concern (i.e., potentially unproblematic, potentially problematic, insufficient data, and not recommended	RDP is potentially unproblematic, based on low concern for most health effects using existing data and professional judgment. Key data deficiencies include cancer, chronic systemic effects, and chronic aquatic toxicity studies. The same evaluation concluded decaBDE is potentially problematic, based on moderate concern for developmental neurological effects, high dose pre-cancerous liver lesions, and concern about breakdown products.
Clean Production Action (2007)	Evaluation based on results of SRC (2006b) report and adaptation to Green Screen evaluation. SRC report used qualitative hazard concern ratings (high, moderate, low) for toxicity to human health, ecotoxicity, bioaccumulation, persistence, potential routes of exposure and aquatic toxicity. Additional evaluations for endocrine disruption and RDP breakdown products and metabolites. Computer modeling, structure activity relationships, and professional judgment used where specific data unavailable.	The constituents of RDP are for the most part chemicals of moderate concern and are grouped in Green Screen Benchmark 2: Use but Search for Safer Substitutes. The same evaluation grouped decaBDE in Green Screen Benchmark 1: Avoid - Chemical of High Concern.

- These evaluations and their conclusions are described in Table 3.3.1.2-1 In general the overall conclusion is that RDP poses less risk to human health and the environment than

decaBDE. Some limitations are associated with this conclusion such as some parameters for RDP and its constituent chemicals are based on structure activity relationships rather than being direct measurements and RDP has not been studied for some important toxicological endpoints such as chronic toxicity and carcinogenicity. In contrast the toxicological data for BDE-209 are relatively complete and the persistence information for both chemicals is convincing.

3.3.1.3 Comparison of Available Toxicological Data

Studies on the toxicological effects of RDP and decaBDE in humans are not available. A brief comparison of the available toxicological studies in laboratory animals for RDP and BDE-209 is shown in Table 3.3.1.3-1.

Table 3.3.1.3-1: Comparison of Toxicological Studies of RDP and BDE-209 in Laboratory Animals

Type of Study	RDP*	BDE-209
Subchronic Toxicity	4-Week inhalation study in rats (Henrich et al., 2000a) 4-Week gavage study in rats (Arthur D. Little Inc., 1989)**	13-Week dietary study in rats (NTP, 1986) 13-Week dietary study in mice (NTP, 1986)
Chronic Toxicity	No data	2-Year dietary study in rats (NTP, 1986) 2-Year dietary study in mice (NTP, 1986)
Reproductive Toxicity	Two-generation dietary study in rats exposed prior and during mating, throughout gestation and lacation (Henrich et al., 2000b)	Gavage study in mice exposed on PNDs 21-70 (Tseng et al., 2006)
Developmental Toxicity	Gavage study in rabbits exposed on GD 6 to 28 (Ryan, et al., 2000)	Gavage study in pregnant rats exposed on GDs 0-19 (Hardy et al., 2002)***
Developmental Neurotoxicity	No data	Gavage study in mice exposed on PNDs 3, 10, or 19 (Viberg et al., 2003b) Gavage study in rats exposed on PND 3 (Viberg et al., 2007) Oral study (MP) in mice exposed on PNDs 2–15 (Rice et al., 2007) Gavage study in mice exposed on PND 3 (Johansson et al., 2008) Gavage study in rats exposed during gestation and through lactation day 21 (Biesemeier et al., (2011).
Immunotoxicity	4-Week gavage study in mice (Sherwood et al., 2000)	No data
Carcinogenicity	No data	2-Year dietary study in rats (NTP, 1986) 2-Year dietary study in mice (NTP, 1986)

GD = gestation day; PND = postnatal day; MP = micropipette

* Unless otherwise noted, a commercial mixture of RDP (Fyrolflex® RDP) was used. One study (Henrich et al., 2000a) reports the mixture contained 65─80 percent RDP, less than 5 percent triphenyl phosphate, and 15─30 percent higher oligomers.

**Study apparently used monomeric RDP.

***Study used commercial decaBDE

The database for RDP does not include studies that evaluate chronic toxicity, carcinogenicity (lifetime bioassay), and developmental neurotoxicity. The NOELs and LOELs from the available toxicological studies for RDP and BDE-209 are shown in Table 3.3.1.3-2.

Table 3.3.1.3-2: No-observed Effect Levels (NOELs) and Lowest-Observed Effect Levels (LOELs) from Available Toxicological Studies for RDP and BDE-209

Type of Study*	RDP**			BDE-209		
	Description (Reference)	NOEL (mg/kg/day)	LOEL (mg/kg/day)	Description (Reference)	NOEL (mg/kg/day)	LOEL (mg/kg/day)
Subchronic Toxicity	4-Week gavage study in rats (Arthur D. Little Inc., 1989)***	Not identified	100	13-Week dietary study in mice (NTP, 1986)	11,566 (females)	Not identified
Chronic Toxicity	No data	No data	No data	2-Year dietary study in rats (NTP, 1986)	1,120 (males)	2,240 (males)
					---	419 (males)††
Reproductive Toxicity	Two-generation dietary study in rats exposed prior and during mating, throughout gestation and lacation (Henrich et al., 2000b)	1,305 (females)	Not identified	Gavage study in mice exposed on PNDs 21–70 (Tseng et al., 2006)	100	500
Developmental Toxicity	Gavage study in rabbits exposed on GD 6 to 28 (Ryan, et al., 2000)	1,000	Not identified	Gavage study in pregnant rats exposed on GDs 0–19 (Hardy et al., 2002) †	1,000	Not identified
Developmental Neurotoxicity	No data	No data	No data	Single dose gavage study in mice exposed on PND 3 (Johansson et al., 2008)	1.34	2.22
				Gavage study in rats exposed from GD 6 and through nursing until 3 weeks old (Biesemeier et al., 2011)	1000	Not identified
Immunotoxicity	4-Week gavage study in mice (Sherwood et al., 2000)	5,000	Not identified	No data	No data	No data
Carcinogenicity	No data	No data	No data	2-Year dietary study in rats (NTP, 1986)	Not identified	1,120

GD = gestation day; PND = postnatal day.

*Studies reporting the highest NOEL or the lowest LOEL for each toxicological endpoint are shown

**Unless otherwise noted, a commercial mixture of RDP (Fyrolflex® RDP) was used. One study (Henrich et al., 2000a) reports the mixture contained 65–80 percent RDP, less than 5 percent triphenyl phosphate, and 15–30 percent higher oligomers.

*** Study apparently used monomeric RDP.

†Study used commercial decaBDE.

††$BMDL_{10}$: Lower bound estimate on the dose associated with a 10 percent response for liver effects in male rats (Hardy et al., 2009). The $BMDL_{10}$ is a mathematically modeled dose derived from the experimental data and takes into account the shape of the dose response curve.

The most sensitive noncancer effect associated with either RDP or BDE-209 is developmental neurotoxicity reported in mice at a dose of 2.22 mg/kg/day of BDE-209. This level is about 45 times lower or more toxic than the lowest effect level reported for RDP of 100 mg/kg/day in rats. The effects at the 100 mg/kg/day dose were an increase in mean liver weights and changes in levels of liver markers (alkaline phosphatase) in females, with no gross or histopathological

lesions. These effects are generally considered to be mild. No effects were seen on mortality, clinical signs, body weight or food consumption. If the studies of Viberg, Johansson, Rice and related neurotoxicity studies (see Section 2.9.1) are not considered because of study design issues, then the lowest LOEL for BDE-209 is 419 mg/kg/day, roughly 4 times higher or less toxic than the 100 mg/kg/day LOEL for RDP.

Carcinogenicity studies have been carried out with BDE-209, but not RDP. NTP concluded that BDE-209 has some evidence of carcinogenicity based on increased incidences of liver neoplastic nodules in rats with equivocal evidence of carcinogenicity in male mice and no evidence in female mice.

In comparing all endpoints for toxicity, noncancer and cancer effects need to be considered together. Considering the toxicity information as a whole, RDP generally appears to be less toxic than BDE-209. BDE-209 shows some evidence of carcinogenicity and the lowest effect levels for noncancer endpoints for BDE-209 are either lower or not much higher than those for RDP (and the effects at 100 mg/kg/day are mild). Cancer data or not available for RDP; however, RDP gave negative results in *in vitro* gene mutation tests and in *in vivo* and *in vitro* chromosomal aberration tests (US EPA, 2012a). The lack of chronic toxicity and carcinogenicity studies for RDP is a major contributor to uncertainty in comparing the toxicity of the two chemicals.

3.3.1.4 Comparison of Data on Persistence

The relative potential for RDP to persist in the environment compared to BDE-209 can be evaluated by comparing the measured half-life for each chemical under similar conditions. The only measured half-life data located for RDP are for its chemical hydrolysis half-life in water, and are found in Table 3.3.1.4-1:

Table 3.3.1.4-1: Available Measured Half-Life Data in Water for RDP*

Half-life (days)	pH	Reference
11	4	HPVIS (2009)
17	7	HPVIS (2009)
21	9	HPVIS (2009)

*Oligomeric form (CAS Number 125997-21-9, which refers to a commercial mixture of small RDP polymers containing up to seven repeating units)

In hydrolysis experiments, no major degradation products were found for BDE-209 at pH 5 and 7 at 100°C after six weeks (ECB, 2002). In addition, BDE-209 is not very soluble in water (less than 0.1 mcg/L at 25°C) and does not have functional groups that readily hydrolyze (UK, 2012). The EPA concluded that, ―The persistence concern for decabromodiphenyl ether is very high" (US EPA 2012).

Considering the measured data on persistence, RDP generally appears to be less persistent than BDE-209. Although the data for the two chemicals are slightly different, the uncertainty in this conclusion is fairly small.

3.3.1.5 Comparison of Data on Bioaccumulation

Bioaccumulation is a measure of the amount of a contaminant in the organism compared to the amount of the contaminant in the environment around it. Bioaccumulation occurs through the uptake and retention of a chemical by an organism from its surrounding medium (air, soil, sediment or water) and from food. Bioaccumulation is often evaluated by measuring a chemical's uptake in fish and calculating a bioconcentration factor based on the concentration in fish tissue and the concentration in the water. Bioconcentration differs from bioaccumulation in that it refers to the uptake of substances into the organism from water alone. In the absence of experimental data, bioconcentration factors are sometimes estimated from the chemical's K_{ow}.

Bioconcentration factors calculated from experimental data with fish are not available for RDP. Washington State estimated a value of 316 for RDP based on its measured K_{ow} of 3.9 to 4.8 (WDE/WDOH, 2008). This considers only hydrophobic partitioning behavior and does not take chemical degradation or metabolism into account. Simple hydrolysis at physiological pH (Table 3.3.1.4-1) is relatively rapid and would decrease net bioconcentration.

As discussed in Section 3.1.5, two bioconcentration studies report that BDE-209 does not accumulate to an appreciable extent in rainbow trout over 48 hours (Norris et al., 1973, 1974) or in carp over 6 weeks (CITI, 1992). Measured bioconcentration factors for BDE-209 were less than 5 at an initial water concentration of 60 mcg/L and less than 50 at an initial water concentration of 6 mcg/L in the 6-week study in carp (CITI, 1992). As discussed previously (Section 3.1.5), these experiments were done above the water solubility limits of BDE-209 and

may underestimate the bioconcentration factors for BDE-209. Feeding studies with rainbow trout and carp report that the uptake of decaBDE is low, and the concentration of BDE-209 in the test species was generally lower than the concentration of BDE-209 in the food (Kierkegaard et al., 1999; Stapleton et al., 2004, 2006). In addition, at least some fish species are capable of metabolizing BDE-209 to lower brominated congeners (see Section 2.5) which accumulate in the fish (Stapleton et al., 2006; Kierkegaard et al., 1999). Thus, the total bioaccumulation potential of BDE-209, primarily because of its metabolites, may not have been recognized in the laboratory studies. The bioaccumulation potential of BDE-209 from field studies is examined in detail in Section 2.7.3.1.

Considering measured data on bioaccumulation, RDP is more bioaccumulative than BDE-209. The bioconcentration factor estimated for RDP is 316; the measured factors for BDE-209 in carp are low and feeding studies in rainbow trout and carp found little uptake. Comparing bioconcentration factors that differ in their derivation and the lack of similar data for the two chemicals are major contributors to uncertainty. On the other hand, hydrolysis products of RDP (phenol, resorcinol and phosphate) are unlikely to bioaccumulate (ATSDR, 2008; WHO, 2006).

3.3.1.6 Estimates of Persistence and Bioaccumulation Potential

The Syracuse Research Corporation developed the PBT Profiler under contract to the US EPA‘s Office of Pollution Prevention and Toxics (US EPA, 2002). The PBT Profiler is a modeling approach based on chemical structure that is used as a screening tool to predict a chemical‘s potential to be persistent and bioaccumulative in the absence of measured data. Results from the PBT Profiler for RDP and BDE-209 are in Table 3.3.1.6-1 along with parameters derived from experimental data discussed in the previous section.

Table 3.3.1.6-1: Half-Lives and Bioconcentration Factors for RDP and BDE-209 Estimated by the US EPA PBT Profiler (US EPA, 2002) and Available Parameters from Experimental Data

Parameter	RDP		BDE-209	
	PBT Profiler*	Experimental	PBT Profiler	Experimental
Half-Life in Water	38 days	11–21 days**	180 days	NA
Half-Life in Soil	75 days	NA	360 days	NA
Half-Life in Sediment	340 days	NA	1,600 days	NA
Bioconcentration Factor	3,000	316***	3.2	<5–<50****

NA = not available.
*Profiler results based on monomeric form, CAS number 57583-54-7.
**Range of values at various pH (see Table 3.3.1.4-1).
***Value calculated from measured K_{ow}.
****Experiments done above the water solubility limits of BDE-209, which may underestimate the bioconcentration factors

The results from the PBT Profiler suggest that RDP has a greater potential for bioaccumulation but a lower potential to be persistent compared to BDE-209. These results should be viewed with caution because they are modeled estimates and not measured values (Aronson et al., 2006; Zachary and Greenway, 2009). According to the US EPA website, the PBT Profiler, like all screening methods, has limitations and is used as a means to identify and prioritize chemicals that may need further evaluation for potential persistence, bioaccumulation and toxicity. These estimates of persistence and bioaccumulation support the conclusions that were based on measured data.

3.3.1.7 Comparison of Data on Ecotoxicity

One way to evaluate ecotoxicity is to use data for aquatic species. The available experimental ecotoxicity data on aquatic species for RDP and BDE-209 (WWDE/WDOH, 2006) are in Table 3.3.1.7-1, and in greater detail for BDE-209 in Section 2.7.

Table 3.3.1.7-1: Ecotoxicity Data for RDP and BDE-209

Chemical	Algae	Crustacean	Fish	Bacteria
RDP*	LOEC = 48.6 mg/L (96 hours)	EC_{50} = 0.76 mg/L (48 hours, *Daphnia magna*)	LC_{50} = 12.4 mg/L (96 hours, Brachydanio)	EC_{10} > 121.6 mg/L (Robra test)
BDE-209	NA**	NA**	LOEL = 7.5–10 mg/kg/day (120 days, rainbow trout)	NOEC $\geq$ 15 mg/L (3 hours, activated sludge micro-organisms)

LOEC = lowest-observed effect concentration; EC_{50} = concentration causing an effect in 50 percent of the test subjects; LC_{50} = concentration causing lethality in 50 percent of the test subjects EC_{10} = concentration causing an effect in 10 percent of the test subjects; NOEC = no-observed effect concentration; LOEL = lowest-observed effect level.

*Data are for Reofos® RDP.

**An EC_{50} for algae and an LC_{50} for fish have been reported, but the experiments were done above the solubility limit for BDE-209 (less than 0.1 mcg/L [ECB, 2002]), which reduces the confidence in these experimental results.

The available experimental information on aquatic species for RDP and BDE-209 is for disparate species, exposure times and reported parameters. The data are not directly comparable for drawing conclusions about the relative risks RDP and BDE-209 might pose to aquatic species.

3.3.1.8 Comparison of Possible Breakdown Products

Some governmental agencies and environmental groups have evaluated the potential breakdown products for BDE-209 and RDP. The evaluations of Washington State (WDE/WDOH, 2008) and Clean Production Action (CPA, 2007) are summarized in Table 3.3.1.8-1.

Table 3.3.1.8-1: Evaluations of Potential Environmental Breakdown Products for RDP and BDE-209

Chemical	Breakdown Products	Evaluations	
		Washington	Clean Production Action
RDP	resorcinol phenol diphenylphosphate	RDP breakdown products are not persistent, bioaccumulative and toxic chemicals under Washington's PBT Rule. RDP breakdown products pose low to medium concern for aquatic toxicity, except for diphenyl phosphate for which data are insufficient.	Resorcinol is classified as –—Use, but still opportunity for improvement." Phenol is classified as –—Use, but search for safer substitutes." Diphenyl phosphate was not classified due to insufficient data.
BDE-209	penta- and octa-PBDE congeners	BDE-209 breakdown products are persistent, bioaccumulative and toxic chemicals under Washington's PBT Rule. BDE-209 breakdown could increase exposure to penta- and octa-PBDE congeners.	BDE-209 breakdown products are classified as –Avoid – Chemical of High Concern."

Washington's evaluation depended on whether the potential breakdown products were classified as persistent, bioaccumulative and toxic chemicals (PBTs) based on specific criteria for half-lives, bioaccumulation and toxicity set forth in the state's PBT initiative (WDE, 2006). The potential breakdown products of BDE-209 (specifically penta- and octaBDE congeners) are classified as PBTs, and those of RDP are not (WDE/WDOH, 2008).

The Clean Production Action evaluation placed BDE-209, RDP and their potential breakdown products in one of four categories based on the value of parameters for toxicity, persistence and bioaccumulation. The categories were 1) Avoid: Chemical of high concern; 2) Use but search for safer substitutes; 3) Use but still opportunity for improvement; and 4) Prefer: Safer chemical. The potential breakdown products of BDE-209 (penta- and octaBDE congeners) were classified as —Avoid: Chemical of High Concern" while those of RDP were classified in categories of lesser concern (–Use, but still opportunity for improvement" and –Use, but search for safer substitutes") (CPA, 2007).

Both evaluations are hazard-based screening methods meant to help inform decisions about the relative risks of chemicals. They take into account the properties of the breakdown products, the

kinds of health effects they may cause as well as available data on effect levels, but not the potential levels of exposure resulting from the use of BDE-209 and RDP as flame retardants or the accompanying risks. Additional uncertainties associated with these evaluations are data limitations such as limitations in the toxicity data for diphenyl phosphate.

The European Union has developed risk statements (also known as R-phrases or risk phrases) for many chemicals under Annex III of European Union Directive 67/548/EEC. The risk statements were developed for the purpose of chemical product labeling, and each statement is based on specific criteria (EC, 2001). There are over 100 different risk statements and risk statement combinations. Risk statements for the potential environmental breakdown products of BDE-209 and RDP (ECB, 2001; EC, 2003; ECA, 2011; EC, 2011b) are summarized in Table 3.3.1.8-2. The statements are hazard-based, that is, they are based on the types of health effects the breakdown products cause without regard to potential exposure levels for a particular use. In addition, some of the statements (e.g., "Causes burns" for phenol) are based on direct contact with high concentrations of the pure chemical, which is unlikely to occur through the environmental breakdown of a flame retardant.

Table 3.3.1.8-2: Risk Statements Developed by the European Union For Potential Breakdown Products of BDE-209 and RDP

Flame Retardant	Potential Breakdown Product	Risk Statement
BDE-209	pentaBDE	Harmful: Danger of serious damage to health by prolonged exposure in contact with skin and if swallowed May cause harm to breast-fed babies Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment
	octaBDE	May cause harm to the unborn child Possible risk of impaired fertility
RDP	resorcinol	Harmful if swallowed Irritating to eyes and skin Very toxic to aquatic organisms
	phenol	Toxic by inhalation, in contact with skin and if swallowed Causes burns Harmful: Danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed Possible risk of irreversible effects

WHO (2006) reports that resorcinol, a potential breakdown product of RDP, is classified as being of low toxicity to most aquatic organisms (including fish, invertebrates, and plants) except the water flea (*Daphnia magna*). Both WHO and the ATSDR report that resorcinol and phenol are expected to biodegrade relatively rapidly in environmental media under aerobic and anaerobic conditions (WHO, 2006; ATSDR, 2008), while lower brominated PBDEs such as penta- and octaBDE tend to be relatively persistent in soil and water (WHO, 1994; ATSDR, 2004a).

Several agencies/organizations have evaluated the potential for the breakdown products of RDP and BDE-209 to be persistent, bioaccumulative and toxic or of concern. Washington State classified the breakdown products of BDE-209 as PBTs (penta and octa BDE congeners) and those of RDP were not. Clean Production Action classified BDE-209 breakdown products as ―Avoid: Chemical of High Concern" while those of RPD were ―Use, but still opportunity for improvement" and ―Use, but search for safer substitutes. Both WHO and ATSDR report that resorcinol and phenol biodegrade relatively rapidly and WHO reported that resorcinol is of low toxicity to most aquatic organisms.

The EU has classified certain BDE-209 breakdown products as PBTs (See Appendix 8 for additional information). The risk statements of the EU for resorcinol and phenol in Table 3.3.1.8-2 are not as clear; some statements appear to be based on assuming direct contact with the pure chemical, which is unlikely through the environmental breakdown of the RDP's use as a flame retardant in a product. Phenol and resorcinol are unlikely to persist or bioaccumulate although they can be acutely toxic in some situations.

Considering the major breakdown products for RDP and BDE-209, the breakdown products of RDP generally appear to be less toxic, persistent, and bioaccumulative than the breakdown products of BDE-209. Differences in the databases and in gaps in the databases for the different breakdown chemicals (both their properties and the extent and rate of degradation) contribute to uncertainty in this comparison.

3.3.1.9 Availability of RDP

The Task Force charge requires an assessment of the availability of alternative flame retardants to decaBDE. RDP is currently used in at least two commercially available flame retardant products. Fyrolflex™ RDP is made by ICL Industrial Products (ICL, 2013) and Reofos® RDP is made by Chemtura Corporation (Chemtura Corporation, 2007, 2009b). Halogen-free organic phosphorus compounds (including RDP), made up an estimated 9 percent of flame retardants consumed in Germany in 1997 (GFEA, 2000) and an estimated 8 percent of flame retardants consumed in Europe in 2005 (KRC, 2007). By one estimate, roughly 20,000 metric tons of RDP were used in television enclosures in the European market in 2004 (LCSP, 2005). Finally, one report (Kingsbury, 2002) indicates that RDP is the dominant flame retardant for polyphenylene oxide/high-impact polystyrene and polycarbonate/acrylonitrile-butadiene-styrene (plastics that can be used in electronic enclosures) on the American market. Based on its presence in available commercial products and that it is marketed and used in Europe and the United States, RDP is a technically feasible and available flame retardant alternative to decaBDE for electronic enclosures.

3.3.1.10 Reliability of RDP

The Task Force charge requires an assessment of the reliability (flame retardant effectiveness) of alternatives to decaBDE. For components such as an electronic enclosure, the UL 94 Test for Flammability of Plastic Materials for Parts in Devices and Appliances is used. The test evaluates the ability of plastics to withstand combustion following direct contact with an open-flame. Plastics that show the highest degree of flame retardancy (i.e., resistance to combustion) are rated V-0 based on a three tiered rating system (V-2, V-1 and V-0). The V-0 rating means that burning stops within 10 seconds after two applications (10 seconds each) of a flame to the test material, with no flaming material dripping from the surface of the tested plastic.

Since RDP is not a direct replacement for decaBDE in high impact polystyrene plastics, different polymers must be used to achieve the same level of fire safety (LCSP, 2005). RDP meets the UL 94 V-0 rating when used in blends of polyphenylene oxide/high-impact polystyrene and polycarbonate/acrylonitrile-butadiene-styrene (LCSP, 2005; KRC, 2007; WDE/WDOH, 2008). In addition, the current extensive use of RDP in European markets suggests that RDP also meets

applicable international fire safety standards. The available information indicates that for electronic enclosures, RDP used in polyphenylene oxide/high-impact polystyrene or polycarbonate/acrylonitrile-butadiene-styrene is an effective flame retardant as decaBDE used in high impact polystyrene.

3.3.1.11 Cost-Effectiveness of RDP

The Task Force charge requires an assessment of the cost-effectiveness of flame retardant alternatives to decaBDE. With respect to electronic products, the cost-effectiveness of an alternative is influenced by the cost of the alternative flame retardant and the cost of changing to different plastics when necessary. For instance, decaBDE is used in high impact polystyrene plastics, whereas RDP must be used with blends of polycarbonate and acrylonitrile-butadiene-styrene or blends of high-impact polystyrene and polyphenylene oxide.

A report by the Lowell Center for Sustainable Production considered the costs of alternatives within the context of raw materials (i.e., alternative flame retardants and the plastics needed for their use) (LSPC, 2005). The report identified three cost-effective non-halogenated alternatives to high impact polystyrene plastic containing decaBDE. One of the three alternative options identified was RDP in blends of high-impact polystyrene and polyphenylene oxide. The high impact polystyrene/polyphenylene oxide blends were estimated to be the most expensive of the three alternative plastics costing roughly $1.90/lb compared to roughly $0.90/lb for high impact polystyrene, which is the plastic that is used with decaBDE. The estimated cost of using any of the three alternatives ranged from about $0.40 to $1.00 more per pound than the cost of using decaBDE in high impact polystyrene. Use of polycarbonate/acrylonitrile-butadiene-styrene blends (a plastic resin that can be used with RDP or other decaBDE alternatives) instead of decaBDE/high impact polystyrene would increase the cost of a 27-inch television with a market value of $300 by $4.40 to $7.50 or 1.5 to 2.5 percent of total purchase price (LSPC, 2005).

Additional information on the cost effectiveness of RDP is limited. Although manufacturers may provide generic information such as whether or not products meet fire safety standards (e.g., the V-0 rating in the UL 94 test), information on the precise constituents manufacturers use to make their products (i.e., the type and amount of flame retardant and other components), as well as the specific costs of production, is not typically shared since it is usually considered

confidential business information. Several reports on flame retardants from organizations and government agencies were reviewed, and companies known to use decaBDE alternatives were also contacted. However, additional quantitative information on the cost effectiveness of RDP was not available.

The analysis done by the Lowell Center suggests that RDP can be used to make electronic enclosures without substantially increasing the overall cost of the final product. On a qualitative basis, there is evidence to support that RDP is a cost effective alternative to decaBDE. Several major United States manufacturers such as Dell and Phillips are phasing out (or have phased out) using decaBDE and other brominated flame retardants in electronic enclosures (Dell, 2009; KPE, 2013) and RDP is a commonly used alternative (CPA, 2004; LCSP, 2005) with one report indicating it is the dominant United States flame retardant for polyphenylene oxide/high-impact polystyrene and polycarbonate/acrylo-nitrile-butadiene-styrene resins (Kingsbury, 2002). As RDP is extensively used in Europe, it is reasonable to conclude that it can be used as a cost-effective alternative in the United States as well. This general conclusion about cost effectiveness of RDP is consistent with those made by other state organizations (IEPA, 2007; Washington, 2008).

3.3.1.12 Conclusions

The Task Force is to ~~"~~evaluate the availability of safer alternatives…including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes." The toxicity, persistence, and bioaccumulation of RPD and BDE-209 and their breakdown products were compared to evaluate if RDP is a safer alternative for BDE-209 (see Section 1.3 for a definition). The Task Force also assessed the reliability, ready availability and cost-effectiveness of RDP.

Toxicity

The most sensitive noncancer effect associated with either RDP or BDE-209 is developmental neurotoxicity reported in mice at a dose of 2.22 mg/kg/day of BDE-209. This level is about 45 times lower or more toxic than the lowest effect level for RDP (100 mg/kg/day - an increase in mean liver weights and changes in levels of liver markers (alkaline phosphatase) in females, generally considered mild effects). If the studies of Viberg, Johansson, Rice and related

neurotoxicity studies (see Section 2.9.1) are not considered because of study design issues, then the lowest LOEL for BDE209 is 419 mg/kg/day, roughly 4 times higher or less toxic than the 100 mg/kg/day LOEL for RDP.

Carcinogenicity studies have been carried out with BDE-209, but not RDP. NTP concluded that BDE-209 has some evidence of carcinogenicity based on increased incidences of liver neoplastic nodules in rats. NTP also concluded that the evidence of carcinogenicity for male mice was equivocal with no evidence of carcinogenicity in female mice. Having some evidence of carcinogenicity in two species increases the concern about BDE-209‘s carcinogenic activity. Although RDP has not been studied for carcinogenicity, it gave negative results in *in vitro* gene mutation tests and in *in vivo* and *in vitro* chromosomal aberration tests (US EPA, 2012a).

In comparing all endpoints for toxicity, noncancer and cancer effects need to be considered together. Considering the toxicity information as a whole, RDP generally appears to be less toxic than BDE-209. The combination of BDE-209 showing some evidence of carcinogenicity and the lowest effect levels for noncancer endpoints for BDE-209 being either lower or not much higher than those for RDP (and the effects at 100 mg/kg/day are mild) lead to the conclusion that RDP generally appears to be less toxic than BDE-209. Data gaps primarily for RDP (no chronic toxicity or carcinogenicity studies) are major contributors to uncertainty in comparing the toxicity of the two chemicals. If chronic toxicity or carcinogenicity data for RDP became available and showed effects at much lower levels than BDE-209, the conclusion for toxicity might differ.

Persistence

RDP‘s half-life in water is relatively short, e.g., 11 – 21 days, depending on pH. In hydrolysis experiments, no major degradation products were found for BDE-209 (ECB, 2002), suggesting that BDE-209 is persistent. In addition, BDE-209 is not very soluble in water and does not have functional groups that readily hydrolyze (UK, 2012) or breakdown. The EPA concluded that, ―The persistence concern for decabromodiphenyl ether is very high‖ (US EPA, 2012a).

Considering the measured data on persistence, RDP generally appears to be less persistent than BDE-209. Although the data for the two chemicals are slightly different, the uncertainty in this conclusion is fairly small.

Bioaccumulation

Neither RDP nor BDE-209 appear to bioaccumulate to a great degree. Considering measured data on bioaccumulation, RDP appears to be more bioaccumulative than BDE-209.

The bioconcentration factor estimated for RDP is 316; the measured factors for BDE-209 in carp are low, but uncertain , and feeding studies in rainbow trout and carp found little uptake. Estimated bioconcentration factors also suggest that RDP is expected to bioaccumulate more (3,000 for RDP versus 3.2 for BDE-209). Comparing bioconcentration factors that differ in their derivation and the lack of similar data for the two chemicals are major contributors to uncertainty. This considers only hydrophobic partitioning behavior and does not take chemical degradation or metabolism into account. Simple hydrolysis at physiological pH (Table 3.3.1.4-1) is relatively rapid and would decrease net bioconcentration.

Breakdown Products

Several agencies/organizations have evaluated the potential for the breakdown products of RDP and BDE-209 to be persistent, bioaccumulative and toxic or of concern. Washington State classified the breakdown products of BDE-209 as PBTs (penta and octa BDE congeners) and those of RDP were not. Clean Production Action classified BDE-209 breakdown products as ―Avoid: Chemical of High Concern" while those of RPD were ―Use, but still opportunity for improvement" and ―Use, but search for safer substitutes. Both WHO and ATSDR report that resorcinol and phenol (RDP breakdown products) biodegrade relatively rapidly and WHO reported that resorcinol is of low toxicity to most aquatic organisms.

The EU has classified certain BDE-209 breakdown products as PBTs (See Appendix 8 for additional information). The risk statements of the EU for resorcinol and phenol in Table 3.3.1.8-2 appear to be based on assuming direct contact with the pure chemical, which is unlikely through the environmental breakdown of the RDP's use as a flame retardant in a product. Phenol and resorcinol are unlikely to persist or bioaccumulate although they can be acutely toxic in some situations.

Considering the major breakdown products for RDP and BDE-209, the breakdown products of RDP generally appear to be less toxic, persistent, and bioaccumulative than the breakdown products of BDE-209. Differences in the databases and in gaps in the databases for the different breakdown chemicals (both their properties and the extent and rate of degradation) contribute to uncertainty in this comparison.

Overall Conclusion

For this report, an alternative is deemed to be safer if the characteristics of persistence, bioaccumulation and toxicity considered together of the alternative and its environmental breakdown products generally appear to be less than the characteristics of persistence, bioaccumulation and toxicity of BDE-209 and its breakdown products (see section 1.3 for more details). BDE-209 generally appears to be more toxic (if the studies of Viberg, Johansson, Rice and related neurotoxicity studies (see Section 2.9.1) are considered), more persistent and may be less bioaccumulative than RDP based on hydrophobic partitioning properties. The breakdown products of BDE-209 generally appear to be more bioaccumulative and persistent than the breakdown products of RDP and the breakdown products of both BDE-209 and RDP can be toxic. In considering all these factors together, RDP generally appears to be a safer alternative to BDE-209. Concerns over data gaps and interpretation of studies meant that representatives from the brominated flame retardant manufacturing industry did not agree with the conclusion for RDP.

RDP has been used instead of decaBDE in products produced in the United States and Europe and is available. Products containing RDP were resistant to combustion, demonstrating RDP‘s reliability. Using RDP with high impact polystyrene/polyphenylene oxide blends versus using decaBDE with high impact polystyrene was estimated to increase the cost of a 27-inch television by about 2 percent, a fairly small increase. Thus, RDP is available, reliable and cost effective.

These conclusions have strengths and limitations (see previous sections for more detail). For example, studies that evaluate chronic toxicity, carcinogenicity and developmental neurotoxicity weren‘t available for RDP. In other cases, modeled data rather than measured data were used, or data were compared that weren‘t obtained using the same protocol and the bioaccumulation data are limited and in some cases uncertain. In contrast, the toxicology database for BDE-209 is relatively complete and the data on persistence, both measured and estimated, were strong.

3.3.2 Boric Acid

3.3.2.1 Background

Boric acid (H_3BO_3; CAS Number 10043-35-3) is an inorganic chemical that contains boron as the borate ion. Boron exists in the environment primarily as borates and is found in air, water, soil and plants. Boron is an essential nutrient in plants, including all fruits, vegetables, nuts, legumes and grains. Boron is also found in animal food products. In soils, boron concentrations generally range from less than 20 mg/kg to 300 mg/kg, and average about 30 mg/kg (Eckel and Langley, 1988; USGS, 1984). In 25 soil samples taken during construction of the New York State Thruway, boron levels ranged from less than 20 mg/kg to 150 mg/kg, and averaged 51 mg/kg (USGS, 1984). Boric acid is formed in the environment primarily through the natural weathering of rocks (ATSDR, 2010).

The chemical structure of boric acid is shown in Figure 3.3.2.1-1:

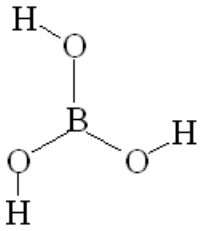


Figure 3.3.2.1-1: Boric Acid
CAS Number: 10043-35-3
Empirical Formula: BO_3H_3
Molecular Weight: 61.83

Boric acid has been used in pharmaceuticals, soaps and cosmetics, as an insecticide, and therapeutically as an astringent and an antiseptic (NRC, 2000; ATSDR, 2010). Boric acid is regulated as an indirect food additive by the Food and Drug Administration and is permitted in adhesives and paper used to package food (FDA, 2006). Boric acid and its sodium salts are regulated under the Federal Insecticide, Fungicide, Rodenticide Act and are used as pesticides on sewage systems, food and non-food crops, outdoor residential areas, and indoor sites such as homes, hospitals, and commercial buildings (US EPA, 1993).

Boric acid is used in the fire barriers of mattresses and is an alternative flame retardant to decaBDE, which was used in the backcoatings or resin binders that line the fabric covering of mattresses. The Lowell Center for Sustainable Production estimated that the manufacture of products that use textiles such as mattresses, upholstered furniture and draperies made up approximately 10 to 20 percent of decaBDE use in the United States (LCSP, 2005). The following sections compare the available toxicity, environmental fate and persistence information for boric acid and BDE-209, and summarize the available information on boric acid‘s availability, reliability and cost-effectiveness.

3.3.2.2 Human Exposure

Since boron compounds exist in many forms, exposure estimates are often given as boron equivalents (i.e., mg boron/kg/day). The general population is exposed to boron compounds primarily through food, beverages and drinking water. Estimates of daily boron intake in the United States are available (see Table 3.3.2.2-1; data on infants not found), and generally range from about 1 mg/day to 2 mg/day for most people. People who take body-building supplements (which contain boron compounds) have higher daily boron intakes. Boron does not exist in its elemental form in foods or beverages, but is present as one or more inorganic, oxygen-containing borates (Rainey et al., 1999).

Table 3.3.2.2-1: Estimates of United States Daily Boron Intakes from the Diet.

Study	Group	Average Dietary Intake (mg/day)
Estimates based on FDA Total Diet Study Methodology and boron content in composite samples of 201 representative foods. Includes drinking water intakes. (Neilsen, 1992; Iyengar et al., 1990; Moore et al., 1997)	Adult Males	1.9
Estimates based on Boron Nutrient Database (1944 individual foods) and 3-day food records of 11,009 survey respondents in Continuing Survey of Food Intakes (Rainey et al., 1999)*	Adult Males (n = 3433)	1.17
	Adult Females (n = 4881)	0.96
	Children (ages 14–18, n = 759)	0.88
	Children (ages 9–13, n = 943)	0.91
	Children (ages 4–8, n = 993)	0.85
Estimates based on Boron Nutrient Database (1944 individual foods) 2-day food records of 22,279 respondents to NHANES III (1988–1994) and Continuing Survey of Food Intakes (IOM, 2001)*	All Individuals (ages 6 and older)	1.15

NHANES III = Third National Health and Nutrition Examination Survey
*Estimates do not include drinking water intakes.

3.3.2.3 Reference Dose for Boron Compounds

The US EPA evaluated the scientific literature and derived a reference dose for boron and boron compounds (US EPA, 2004). As stated previously, the reference dose is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to people (including sensitive subgroups) that is likely to be without an appreciable risk of noncancer adverse health effects during a lifetime. The reference dose, 0.2 mg/kg/day, is expressed as boron equivalents (i.e., mg boron/kg/day), based on the percentage of boron in boric acid (17.5 percent), or 1 mg/kg/day of boric acid. The reference dose for boron and boron compounds is based on decreased fetal weights in rats whose mothers were exposed to boric acid. A total uncertainty factor of 66 for inter- and intraspecies variability (based on chemical specific adjustment factors) was applied to the 95 percent lower confidence limit on the dose associated with a 5 percent decrease in fetal weight relative to control animals (59 mg boric acid/kg/day or 10.3 mg boron/kg/day).

3.3.2.4 Human Exposure Estimate for Boric Acid Used as a Flame Retardant

In developing performance standards for mattress flammability, the CPSC developed quantitative estimates for human exposure to boric acid and other flame retardants resulting from their use in mattress fire barriers (CPSC, 2006b). CPSC conducted migration/exposure assessment studies to estimate how much flame retardant might be released from the barriers. The three main phases of the study were measuring the amount of boric acid in the barrier, evaluating the potential migration of boric acid from the barrier to estimate dermal and ingestion exposure, and estimating the airborne particle-bound release of boric acid from the barrier after 10 years of normal use. According to CPSC, the analysis applied conservative assumptions in areas of scientific uncertainty that may overestimate rather than underestimate exposure to flame retardants (CPSC, 2006b). The CPSC estimates for exposure to boric acid from its use as a flame retardant in mattress barriers are 0.081 mg/day for adults and 0.08765 mg/day for children (CPSC, 2006b). Since boron makes up 17.5 percent of boric acid (based on the ratio of atomic and molecular weights of boron and boric acid, respectively [10.81/61.83]), the corresponding estimated boron intakes can be calculated and then compared to typical boron intakes from other sources such as the diet. The corresponding estimated boron intakes from mattress barriers are 0.0142 mg/day and 0.0153 mg/day for adults and children, respectively:

Adults: 0.081 mg boric acid/day x 0.175 = 0.0142 mg boron equivalents/day

Children: 0.08765 mg boric acid/day x 0.175 = 0.0153 mg boron equivalents/day

The estimated boron intakes resulting from the use of boric acid as a flame retardant in mattress barriers (0.0142 mg/day and 0.0153 mg/day for adults and children respectively) are about 0.7 to 1.8 percent of the average estimated boron intakes from dietary sources (Table 3.3.2.4-1).

Table 3.3.2.4-1: Comparison of Estimated Boron Intakes from Use of Boric Acid as a Flame Retardant in Mattress Barriers to Boron Dietary Intakes

Description	Children	Adults
Estimated boron intake from boric acid in mattress barriers in mg/day (CPSC, 2006b)	0.0153	0.0142
Fraction of average estimated boron intakes from dietary sources[a]	0.017–0.018	0.007–0.015

[a]From Table 3.3.2.2-1, 0.85 to 0.91 mg/day for children; 0.96 to 1.9 mg/day for adults.

The CPSC risk assessment also provided estimates for exposure to boric acid in mattress barriers expressed in units of dose (mg/kg/day) assuming average body weights of 72.25 kg and 19.2 kg for adults and children, respectively. The reported exposure estimates are 0.00113 mg/kg/day for adults and 0.005 mg/kg/day for children (CPSC, 2006b), which can be compared to boric acid exposure levels (in mg/kg/day) that are known to cause health effects (Table 3.3.2.4-2). In the table below, the estimated dose for children (which is higher than the dose for adults) is used for comparison. As is shown in Table 3.3.2.4-2, the estimated boric acid dose for children from its use as a flame retardant in mattress barriers (0.005 mg/kg/day) is lower than any adverse effect levels identified for boric acid in animals or humans. The difference between the estimated dose from mattress barriers and boron doses that cause adverse health effects suggests that use of boric acid as a flame retardant in mattress barriers poses a minimal risk to human health. The CPSC reached a similar conclusion in its assessment of the potential health effects of flame retardants in mattresses (CPSC, 2006b), stating that boric acid is ―not expected to pose any appreciable risk of health effects to consumers who sleep on treated mattresses."

The CPSC assessment did not directly address the potential dermal irritant effects of boric acid. In studies of laboratory animals, boric acid showed some irritant effects when applied directly to the skin. According to the US EPA, boric acid is not a dermal irritant and is classified as Toxicity Category III (slightly irritating) under the agency's pesticide registration program based on erythema (redness) in one out of six animals exposed to boric acid directly on the skin (US EPA, 1993; 2009f). The US EPA also concluded that there is no evidence that boric acid causes dermal sensitization (US EPA, 1993, 2009f).

Table 3.3.2.4-2: Comparison of Estimated Boric Acid Doses from Its Use as a Flame Retardant in Mattress Barriers to Boric Acid Effect Levels

Description	Dose (mg/kg/day)	Number of Times Higher than Mattress Barrier Dose
Estimated boric acid dose from mattress barriers (CPSC, 2006b)	0.005	---
LOEL for subchronic toxicity in animals (NTP, 1987; Dieter, 1994)[a]	195	39,000
LOEL for chronic toxicity in animals (NTP, 1987; Dieter, 1994)[a]	275	55,000
LOEL for reproductive toxicity in animals (Ku et al., 1993; Chapin and Ku, 1994)[a]	149	29,800
LOEL for developmental toxicity in animals (Price et al., 1996)[a]	76	15,200
$BMDL_{05}$ for developmental toxicity in animals, used as the basis for the US EPA reference dose (US EPA, 2004)	59	11,800
Range of reported human effect levels for acute gastrointestinal symptoms in case reports (ATSDR, 2010)	486–1,053	97,200–210,600

LOEL = lowest-observed effect level.
$BMDL_{05}$ = 95 percent lower confidence limit on the dose associated with a 5 percent response rate for an adverse effect
[a]See next section for details on boric acid effect levels.

3.3.2.5 Comparison of Data on Animal Toxicity

A brief comparison of NOELs and LOELs from the available toxicological studies on boric acid and BDE-209 is shown in Table 3.3.2.5-1. To facilitate the comparison, Table 3.3.2.5-1 includes only the boric acid and decaBDE studies that reported the lowest LOEL or the highest NOEL for each toxicological endpoint. Other studies may have reported LOELs at higher exposure levels or NOELs at lower exposure levels. The database on boric acid toxicity does not include animal studies evaluating developmental neurotoxicity.

Table 3.3.2.5-1: No-Observed Effect Levels (NOELs) and Lowest-Observed Effect Levels (LOELs) from Available Toxicological Studies for Boric Acid and BDE-209*

Type of Study	Boric Acid			BDE-209		
	Description (Reference)	NOEL (mg/kg/day)	LOEL (mg/kg/day)	Description (Reference)	NOEL (mg/kg/day)	LOEL (mg/kg/day)
Subchronic Toxicity	13-Week dietary study in mice (NTP, 1987; Dieter, 1994)	Not identified	195	13-Week dietary study in mice (NTP, 1986)	11,566 (females)	Not identified
Chronic Toxicity	2-Year dietary study in mice (NTP, 1987; Dieter, 1994)	Not identified	275	2-Year dietary study in rats (NTP, 1986)	1,120 (males)	2,240 (males)
					---	419 (males)†
Reproductive Toxicity	9-Week dietary study in rats (Ku et al., 1993 and Chapin and Ku, 1994)	Not identified	149	Gavage study in mice exposed on PNDs 21–70 (Tseng et al., 2006)	100	500
Developmental Toxicity	Dietary study in pregnant rats exposed on GDs 0-20 (Price et al., 1996)	55	76 59**	Gavage study in pregnant rats exposed on GDs 0–19 (Hardy et al., 2002)***	1,000	Not identified
Developmental Neurotoxicity	No data	No data	No data	Single dose gavage study in mice exposed on PND 3 (Johansson et al., 2008)	1.34	2.22
				Gavage study in rats exposed from GD 6 and through nursing until 3 weeks old (Biesemeier et al., 2011)	1000	Not identified
Immunotoxicity	No data	No data	No data	No data	No data	No data
Carcinogenicity	2-Year dietary study in mice (NTP, 1987)	550	Not identified	2-Year dietary study in rats (NTP, 1986)	Not identified	1,120

GD = gestation day; PND = postnatal day

*Studies reporting the highest NOEL or the lowest LOEL for each toxicological endpoint are shown.

**$BMDL_{05}$, or 95 percent lower confidence limit on the dose associated with a 5 percent response rate for an adverse effect. The $BMDL_5$ is a mathematically modeled dose derived from the experimental data and takes into account the shape of the dose response curve.

***Study used commercial decaBDE.

†$BMDL_{10}$: Lower bound estimate on the dose associated with a 10 percent response for liver effects in male rats (Hardy et al., 2009). The $BMDL_{10}$ is a mathematically modeled dose derived from the experimental data and takes into account the shape of the dose response curve.

The most sensitive noncancer effect associated with boron or BDE-209 is developmental neurotoxicity in mice at a dose of 2.22 mg/kg/day. This level is about 27 times lower or more toxic than the lowest effect level for boric acid (a $BMDL_{05}$ for decreased fetal weights in rats whose mothers were exposed). If the studies of Viberg, Johansson, Rice and related neurotoxicity studies (see Section 2.9.1) are not considered because of study design issues, then

the lowest LOEL for BDE-209 is 419 mg/kg/day, about 7 times higher or less toxic than the 59 mg/kg/day LOEL for boric acid.

The US EPA recommended an oral reference dose of 0.2 mg boron equivalents/kg/day which corresponds to 1 mg/kg/day of boric acid. Several reference doses have been suggested for BDE-209 (see Section 2.9.6). Hardy et al. derived a reference dose of 4 mg/kg/day (4 times greater or less toxic than boric acid) and the US EPA derived one of 0.007 mg/kg/day (142 time less or more toxic than boric acid).

Carcinogenicity studies have been carried out with BDE-209 and boric acid. For BDE-209, NTP reported some evidence of carcinogenicity based on increased incidences of liver neoplastic nodules in rats with equivocal evidence of carcinogenicity in male mice and no evidence in female mice exposed to high levels for their lifetimes (NTP, 1986; US EPA, 2008a,b). NTP stated that, "there was no evidence of carcinogenicity of boric acid" at the doses studied (NTP, 1987). In a reassessment of boron, the US EPA concluded that the "data are inadequate for an assessment of human carcinogenic potential for boron" (US EPA, 2004). The US EPA also noted an increase in the incidence of some tumors at the low, but not the high, dose with the increases being statistically significant with one test, but not another. It also recognized that the genotoxicity for boron compounds "were overwhelmingly negative, including studies in bacteria, mammalian cells and mice *in vivo*."

In comparing all endpoints for toxicity, noncancer and cancer effects need to be considered together. BDE-209 shows some evidence of carcinogenicity while the data for boric acid generally are not suggestive of carcinogenic activity; thus, boric acid is less of a toxicity concern for carcinogenicity. If the studies of Viberg, Johansson, Rice and related neurotoxicity studies (see Section 2.9.1) and the carcinogenicity studies are considered, boric acid generally appears to be less toxic than BDE-209. If the studies of Viberg, Johansson, Rice and related neurotoxicity studies (see Section 2.9.1) are not considered and the carcinogenicity studies are considered, it is difficult to say one is more or less toxic than the other. These conclusions have limitations because of the lack of developmental neurotoxicity studies and weaknesses in the cancer bioassay for boric acid and different interpretations of the developmental neurotoxicity studies for BDE-209. In contrast, the database for BDE-209 is relatively complete and human data for boric acid are available.

3.3.2.6. European Union Evaluation

Boric acid is classified as a Category 2 chemical (suspected human reproductive toxicant) by the European Union under Regulation (EC) No 1272/2008 of the European Parliament and of the Council, which covers the classification, labeling and packaging of substances and mixtures (OJ 31.12.2008 L353/1; ECA, 2010a). The classification is for chemicals having evidence from humans or experimental animals, possibly supplemented with other information, of an adverse effect on sexual function and fertility, or on development (OJ 31.12.2008 L353/1). The primary basis for the classification of boric acid as a suspected human reproductive toxicant are animal studies that report adverse effects on reproduction (infertility, testicular atrophy, reduced sperm motility, increased uterine weight and decreased estrus cycle) in rats or mice exposed to dietary boric acid prior to and during mating (Weir and Fisher, 1972; NTP, 1990). These studies were considered when comparing effect levels for boric acid and BDE-209 (Table 3.3.2.5-1). The lowest reported boric acid effect level for reproductive toxicity comes from a study which reported reduced release of mature spermatids in rats exposed to 149 mg/kg/day boric acid in their diets (Ku et al., 1993; Chapin and Ku, 1994), and is included in Table 3.3.2.5-1.

Based on the Category 2 classification, boric acid is identified under the criteria of Article 57 (c) of Regulation (EC) 1907/2006 (Registration, Evaluation, Authorisation and Restriction of Chemical substances [REACH]) as toxic for reproduction (ECA, 2010a), and is included on the European Chemicals Agency‘s Candidate List of Substances of Very High Concern (ECA, 2010b).

3.3.2.7 Comparison of Data on Persistence

Boron in the environment is primarily in the form of inorganic borates, which are naturally-occurring and widespread. Boron compounds in the environment are not expected to undergo significant transformation or breakdown (WHO, 2004). Under natural aqueous conditions, boron exists mostly as boric acid, with small amounts of borate ions being formed as the pH increases. In soils, most boron exists as borates and small amounts of boron can be released through weathering processes (ATSDR, 2010; NPIC, 2001). The use and production of boric acid may change the distribution of boron, but not the global amount of boron.

As discussed in Sections 3.1.4 and 3.3.1.4, measured half-life data for BDE-209 in air, soil, water and sediment were not found. Estimates of environmental half-lives of BDE-209 in air, water, soil and sediment using the PBT Profiler are 180 days, 360 days and 1,600 days in water, soil and sediment, respectively (see Section 3.3.1.6 for more information on the PBT Profiler), which are fairly long half-lives.

Overall, the data suggest that borates, boric acid and BDE-209 all have the potential to persist in the environment. The production and use of BDE-209 increases the global amount of BDE-209; the production and use of boric acid does not increase the global amount of boron although the distribution may change.

3.3.2.8 Comparison of Data on Bioaccumulation

Bioaccumulation is a measure of the amount of a contaminant in the organism compared to the amount of the contaminant in the environment around it (see Sections 3.1.5 and 3.3.1.5 for additional discussion). A chemical's K_{ow} is sometimes used as an indicator of bioaccumulation potential in the absence of data.

Two studies were located that evaluate the bioaccumulation of boron compounds in aquatic species. Thompson et al. (1976) studied boron uptake from water in juvenile Pacific oysters and sockeye salmon. The authors reported that after exposure of the oysters for 47 days (at levels of 1 mg/L and 10 mg/L above boron background levels in seawater) and the salmon for 21 days (at 10 mg/L above background), the boron levels in both species approximated the boron water concentrations, and there was no evidence for active bioaccumulation of boron. Suloway et al. (1983; cited in WHO, 2004) reported that boron in a coal fly ash extract did not significantly accumulate in fathead minnows and green sunfish after each was exposed for 30 days in water. The reported bioaccumulation factor was 0.3 for both species. These studies, along with the reported K_{ow} for boric acid of 0.175 (Barres, 1967 cited in WHO, 2004), support the conclusion that the potential for bioaccumulation of boron compounds is low.

As discussed in Sections 3.1.5 and 3.3.1.5, two bioconcentration studies report that BDE-209 does not accumulate to an appreciable extent in rainbow trout over 48 hours (Norris et al., 1973,

1974) or in carp over 6 weeks (CITI, 1992), with measured bioconcentration factors for BDE-209 ranging from less than 5 to less than 50 in the 6-week carp study (CITI, 1992). Since the experiments were done above the water solubility limits of BDE-209 and the reported concentrations of BDE-209 in water are questionable, the calculated bioconcentration factors may be underestimated. Feeding studies with rainbow trout and carp report that the uptake of BDE-209 is low (Kierkegaard et al., 1999; Stapleton et al., 2004, 2006). Some fish are able to metabolize BDE-209 to lower brominated congeners which accumulate (Stapleton et al., 2006; Kierkegaard et al., 1999). Thus, the total bioaccumulation potential of BDE-209, primarily because of its metabolites, may not have been recognized in the laboratory studies. The bioaccumulation potential of BDE-209 from field studies is examined in detail in Section 2.7.3.1.

Overall, bioconcentration and bioaccumulation factors for boron compounds and BDE-209 are not directly comparable due to differences in study methods, study parameters and fish species. However, the available studies suggest a low potential for both to build up in aquatic species.

3.3.2.9 Comparison of Data on Ecotoxicity

Ecotoxicity can be evaluated using toxicity data for aquatic species. Available experimental data for boric acid (WHO, 2004; Grzybowska et al., 2007) and BDE-209 (WWDE/WDOH, 2006) and aquatic species are in Table 3.3.2.8-1 and in greater detail for BDE-209 in Section 2.7.

Table 3.3.2.8-1: Ecotoxicity Data for BDE-209 and Boric Acid

Chemical	Algae	Crustacean	Fish*	Bacteria
Boric Acid	NA**	LC_{50} = 760–1300 mg/L (48 hour, *Daphnia*) LC_{50} = 300 mg/L (21 day, *Daphnia*) NOEC = 34 mg/L (21 day, *Daphnia*)	LC_{50} = 570–860 mg/L (24 hour, rainbow trout) LC_{50} = 450–570 mg/L (28 day, rainbow trout) LC_{50} = 790 mg/L (32 day, rainbow trout) NOEC > 0.05 mg/L LOEC = 0.6 mg/L	MIC = 800–12,800 mg/L (Gram-negative); 1,600–6,400 mg/L (Gram-positive)
BDE-209	NA**	NA**	LOEL = 7.5–10 mg/kg/day (120 days, rainbow trout)	NOEC $\geq$ 15 mg/L (3 hours, activated sludge micro-organisms)

LC_{50} = concentration causing lethality in 50 percent of the test subjects; NOEC = no-observed effect concentration; LOEC = lowest-observed effect concentration; MIC = minimum inhibitory concentration; LOEL = lowest-observed effect level; NA = not available.

*Only data for rainbow trout shown. Data on boric acid are available for other fish species (WHO, 2004)

**An EC_{50} for algae and an LC_{50} for fish have been reported, but the experiments were done above the solubility limit for BDE-209 (less than 0.1 mcg/L [ECB, 2002]), which reduces the confidence in these experimental results.

The available experimental information on the ecotoxicity of boric acid and BDE-209 consists of data for disparate species, exposure times and reported parameters. The data are generally not directly comparable for the purpose of drawing conclusions about the relative risks boric acid and BDE-209 might pose to the environment.

3.3.2.10 Comparison of Possible Breakdown Products

Boron occurs in the environment primarily as borates and environmental boron compounds are relatively stable (US EPA, 1993; WHO, 2004). In water, boron compounds generally exist as the undissociated boric acid and borate ions. In sediments and soil, inorganic borate compounds can slowly release boron in low concentrations through weathering processes (ATSDR, 2010). BDE-209 can breakdown to less brominated PBDEs, certain of these PBDEs are persistent, bioaccumulate and toxic (see Sections 2.5, 2.11.3, Appendix 8) so that BDE-209 in the environment could be a long-term source of these breakdown products. How much and how fast debromination occurs under environmental conditions is not clear. Considering the major breakdown products of boric acid and BDE-209, the breakdown products of boric acid generally

appear to be less toxic and bioaccumulative than the breakdown products of BDE-209. The breakdown products of both boric acid and BDE-209 are persistent. The production and use of BDE-209 increases the global amount of BDE-209; the production and use of boric acid does not increase the global amount of boron. Overall, the breakdown products of BDE-209 are more of a concern than the breakdown products of boric acid

3.3.2.11 Availability of Boric Acid

Boric acid is used in mattress fire barriers, which are found in several products on the consumer market. Serta, one of the largest mattress manufacturers in the United States, uses a fire barrier called FireBlocker® in all its mattresses. FireBlocker® is made up of a blend of natural and synthetic fibers treated with boric acid (MDEP/MCDCP, 2007; Serta International, 2005). Other mattress companies such as NaturaWorld use a cotton barrier treated with boric acid. The cotton barrier is placed inside the mattress beneath a layer of wool and the mattress ticking (NaturaWorld, 2010). Jones Fiber Products makes a fire barrier consisting of cotton batting treated with boric acid (A. Posner, personal communication, July 8, 2010). The presence of these products on the consumer market demonstrates the availability of boric acid for use as a flame retardant in mattresses.

3.3.2.12 Reliability of Boric Acid

The Task Force charge requires an assessment of the reliability (flame retardant effectiveness) of alternatives to decaBDE. In 2006, the CPSC established a new flammability standard for mattresses (16 CFR Part 1633, 2006; IEPA, 2007). The standard sets flammability test criteria and performance requirements that all mattress sets must meet before being introduced into the consumer market. The test protocol calls for exposing mattress materials (i.e., mattress alone and mattress set) to an open flame and allowing them to burn freely for 30 minutes. To comply with the standard, the mattress material must not exceed specified rates of heat release from the fire (test criteria[1] - 16 CFR Part 1633, 2006). Previous flammability standards focused on cigarette-type ignition sources rather than testing mattresses under an open flame (LCSP, 2005). The CPSC estimates that improved mattresses resulting from compliance with the new standard

[1] The peak rate of heat release for the mattress must not exceed 200 kilowatts at any time during the 30 minute test and the total heat release must not exceed 15 megajoules for the first 10 minutes of the test.

could significantly reduce deaths (up to 270 annually) and injuries (up to 1,330 annually) from mattress fires (16 CFR Part 1633, 2006).

Fire barriers are fire-resistant materials that are placed between the surface fabric and the interior mattress padding to protect the mattress padding from combustion. Although no specific test data could be located, Serta, a leading national mattress manufacturer that uses fire barriers with boric acid in all its mattresses (Serta International, 2005), states on its website that its mattresses meet the federal mattress flammability standard for open flame fire resistance (Serta International, 2009).

3.3.2.13 Cost-Effectiveness of Boric Acid

Quantitative information on the cost effectiveness of fire barriers treated with boric acid is not available. An article in an industry publication characterizes the use of fire barriers in mattresses as having widespread availability, relative affordability, and acceptability among mattress manufacturers (Mayberry and Franken, 2005). In addition, mattresses that use fire barriers treated with boric acid are available on the consumer market, suggesting that these products are cost effective.

3.3.2.14 Conclusions

The Task Force is to, ~~"~~evaluate the availability of safer alternatives…including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes." The toxicity, persistence, and bioaccumulation of boric acid and BDE-209 and their breakdown products were compared to evaluate if boric acid is a safer alternative for BDE-209 (see Section 1.3 for a definition). The Task Force also assessed the reliability, ready availability and cost-effectiveness of magnesium hydroxide.

Toxicity

Boron exists in the environment primarily as borates and is in food, both plant and animal. The Food and Drug Administration regulates boric acid as an indirect food additive and it is allowed

in adhesives and paper used to package food. The estimated boron intake from boric acid in mattress barriers ranges from about 1 to 2 percent of the dietary intake.

The most sensitive noncancer effect associated with boron or BDE-209 is developmental neurotoxicity in mice at a dose of 2.22 mg/kg/day. This level is about 27 times lower or more toxic than the lowest effect level for boric acid (a $BMDL_{05}$ for decreased fetal weights in rats whose mothers were exposed). If the studies of Viberg, Johansson, Rice and related neurotoxicity studies (see Section 2.9.1) are not considered because of study design issues, then the lowest LOEL for BDE-209 is 419 mg/kg/day, about 7 times higher or less toxic than the 59 mg/kg/day LOEL for boric acid.

The US EPA recommended an oral reference dose of 0.2 mg boron equivalents/kg/day which corresponds to 1 mg/kg/day of boric acid. Several reference doses have been suggested for BDE-209 (see Section 2.9.6). Hardy et al. derived a reference dose of 4 mg/kg/day (suggesting that BDE-209 is 4 times less toxic than boric acid) and the US EPA derived one of 0.007 mg/kg/day (suggesting that BDE-209 is 142 times more toxic than boric acid).

Carcinogenicity studies have been carried out with BDE-209 and boric acid. For BDE-209, NTP reported some evidence of carcinogenicity based on increased incidences of liver neoplastic nodules in rats and that the evidence of carcinogenicity for male mice was equivocal with no evidence of carcinogenicity in female mice exposed to high levels for their lifetimes (NTP, 1986; US EPA, 2008a,b). Having some evidence of carcinogenicity in two species increases the concern about BDE-209's carcinogenic activity. NTP stated that, "there was no evidence of carcinogenicity of boric acid" at the doses studied (NTP, 1987). In a reassessment of boron, the US EPA concluded that the "data are inadequate for an assessment of human carcinogenic potential for boron" (US EPA, 2004). It also recognized that the genotoxicity tests for boron compounds "were overwhelmingly negative, including studies in bacteria, mammalian cells and mice *in vivo*."

In comparing all endpoints for toxicity, noncancer and cancer effects need to be considered together. BDE-209 shows some evidence of carcinogenicity while the data, including mutagenicity data, for boric acid generally are not suggestive of carcinogenic activity; thus, boric acid is less of a toxicity concern for carcinogenicity. If the studies of Viberg, Johansson, Rice

and related neurotoxicity studies (see Section 2.9.1) and the carcinogenicity studies are considered, boric acid generally appears to be less toxic than BDE-209. If the studies of Viberg, Johansson, Rice and related neurotoxicity studies (see Section 2.9.1) are not considered and the carcinogenicity studies are considered, it is difficult to say one is more or less toxic than the other. These conclusions have limitations because of the lack of developmental neurotoxicity studies and weaknesses in the cancer bioassay for boric acid and different interpretations of the developmental neurotoxicity studies for BDE-209. In contrast, the database for BDE-209 is relatively complete and human data for boric acid are available since people are exposed to boron every day in their diet.

Persistence

Boron is naturally-occurring and widespread in the environment, primarily in the form of inorganic borates. Under natural aqueous conditions, boron exists mostly as boric acid and in soils, most boron exists as borates. Boron compounds are not expected to undergo significant transformation or breakdown in the environment (WHO, 2004).

As discussed in Sections 3.1.4 and 3.3.1.4 measured half-life data for BDE-209 in air, soil, water and sediment were not found. Estimates of environmental half-lives of BDE-209 in air, water, soil and sediment using the PBT Profiler are 180 days, 360 days and 1,600 days in water, soil and sediment, respectively (see Section 3.3.1.6 for more information on the PBT Profiler), which are fairly long half-lives.

Overall, the data suggest that borates, boric acid and BDE-209 all have the potential to persist in the environment. The production and use of BDE-209 increases the global amount of BDE-209; the production and use of boric acid does not increase the global amount of boron, but may increase its bioavailability to some extent (see Section 3.3.2.4).

Bioaccumulation

Overall, bioconcentration and bioaccumulation factors for boron compounds and BDE-209 are not directly comparable due to differences in study methods, study parameters and fish species. However, the available studies suggest a low potential for both to build up in aquatic species.

Breakdown Products

Boron occurs in the environment primarily as borates and environmental boron compounds are relatively stable (US EPA, 1993; WHO, 2004). BDE-209 can breakdown to less brominated PBDEs and certain ones are persistent, bioaccumulate and toxic (see Sections 2.5, 2.11.3, Appendix 8) so that BDE-209 in the environment could be a long-term source of these breakdown products. How much and how fast debromination occurs under environmental conditions is not clear.

Considering the major breakdown products of boric acid and BDE-209, the breakdown products of boric acid generally appear to be less toxic and bioaccumulative than certain breakdown products of BDE-209 and thus BDE-209 breakdown products could be more of a concern. The breakdown products of both boric acid and certain breakdown products of BDE-209 are persistent. Uncertainty in these conclusions arises from uncertainty in the toxicity databases.

Overall Conclusion

For this report, an alternative is deemed to be safer if the characteristics of persistence, bioaccumulation and toxicity considered together of the alternative and its environmental breakdown products generally appear to be less than the characteristics of persistence, bioaccumulation and toxicity considered together of BDE-209 and its breakdown products (see Section 1.3 for more details). Comparing the toxicity of BDE-209 and boric acid is difficult, both compounds are persistent and neither compound tends to bioaccumulate. Certain breakdown products of BDE-209 are persistent, bioaccumulate, and toxic (see Section 2.5, 2.11.3, Appendix 8) while the breakdown products of boric acid are persistent, can be toxic, but do not bioaccumulate. The production and use of BDE-209 increases the global amount of BDE-209; the production and use of boric acid does not increase the global amount of boron, but may change its distribution. In considering all these factors together, boric acid generally appears to be a safer alternative for BDE-209, primarily because of BDE-209 breakdown products. Concerns over data gaps and interpretation of studies meant that representatives from the brominated flame retardant manufacturing industry did not agree with the conclusion for boric acid.

Boric acid is used as a flame retardant in fire barriers for several commercial mattresses, which demonstrates that it is available and suggests that it is cost-effective. Mattresses with flame barriers containing boric acid are also stated to meet the current flammability standards established by the CPSC, suggesting that boric acid is a reliable flame retardant for this application. Boric acid is available, reliable and cost effective as an alternative to BDE-209 in mattresses.

This conclusion has some uncertainty because of data limitations. For example, boric acid hasn't been studied for developmental neurotoxic effects, the carcinogenicity study for boric acid has some weaknesses, interpreting the effect levels of the two compounds is difficult and interpretations of the developmental neurotoxicity data for BDE-209 differ.

3.4 Product Redesign

Product redesign is changing the organization, structure or form of a product so that a chemical flame retardant such as decaBDE is not needed. Some reports on decaBDE alternatives have cited examples of redesign strategies for electronics such as separating ignitable plastic from current-carrying or heat-generating parts (GFEA, 2000, 2001; LCSP, 2005; MDEP/MCDCP, 2007). Another example of a redesign strategy is introducing an inner metal housing in computer monitors as a flame resistant barrier that eliminates the need for flame retardants such as decaBDE in exterior encasements (GFEA, 2000). However, specific examples of electronic or textile products that have been redesigned to eliminate the need for decaBDE were not found. As product manufacturers pursue innovative design and technologies, product redesign strategies (such as those mentioned above or others) could be employed to eliminate the need for flame retardants in electronics and textile products.

3.5 Conclusions

Based on the information reviewed by the Task Force, alternatives for the major uses of decaBDE are available that are cost effective, have reliable flame retardant properties and are safer alternatives to BDE-209. For this report, an alternative is defined as being safer if the characteristics of persistence, bioaccumulation and toxicity considered together of the alternative and its environmental breakdown products generally appear to be less problematic than the

characteristics of persistence, bioaccumulation and toxicity considered together of BDE-209 and its breakdown products. Making such a conclusion requires some judgment since the alternatives and BDE-209 are being compared for a number of characteristics (e.g., persistence, etc.) and the databases and their strengths and weaknesses for the different properties will vary.

Manufacturers can develop alternatives to decaBDE in its applications by using:

- chemical substitution (replacing decaBDE with an alternative flame retardant),
- material substitution (replacing a material that requires decaBDE with a material that does not),
- combinations of chemical and material substitution (using an alternative flame retardant in a different host material), and
- product redesign (changing a product‘s organization, structure or form so that a flame retardant chemical is not necessary).

Magnesium hydroxide in coatings of wires and cables is an example of a safer chemical that can be substituted for decaBDE. Aluminum alloy casings (rather than plastic) in computer products is an example of a material substitution that eliminates the need for a chemical flame retardant. RDP in electronic enclosures and boric acid in fire barriers of mattresses are examples of safer chemical and material substitution combinations that make decaBDE unnecessary. Specific examples of products that have been redesigned to eliminate decaBDE were not found. The ultimate selection of alternatives requires an evaluation of toxicity, environmental impacts, potential human exposure, overall data limitations, flame retardancy and cost.

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Appendix 1. Law Establishing the New York State Task Force on Flame Retardant Safety

LAWS OF NEW YORK, 2004

CHAPTER 387

AN ACT to amend the environmental conservation law, in relation to restricting the use of certain flame retardants and relating to the creation of a state task force on flame retardant safety

Became a law August 17, 2004, with the approval of the Governor.
Passed by a majority vote, three-fifths being present.

The People of the State of New York, represented in Senate and Assembly do enact as follows:

Section 1. Section 37-0101 of the environmental conservation law is amended by adding three new subdivisions 4, 5 and 6 to read as follows:

4. "Brominated flame retardants" refers generally to any product containing a mixture of chemicals known as brominated diphenyl ether to prevent, reduce or retard the risk of fire in electronic devices, furniture, and textiles.

5. "Polybrominated diphenyl ether" (PBDE) is a mixture of brominated diphenyl ethers, usually marketed as pentabromodiphenyl ether or octabromodiphenyl ether, according to how many hydrogen atoms in the diphenyloxide structure are replaced with bromine atoms.

6. "Process", as used in section 37-0111 of this title, shall not include the processing of metallic recyclables containing pentabrominated diphenyl ether or octabrominated diphenyl ether that is conducted in compliance with all applicable federal, state, and local laws.

§ 2. The environmental conservation law is amended by adding a new section 37-0111 to read as follows:

§ 37-0111. Prohibition against brominated flame retardants.

1. Manufacture, process, or distribution of brominated flame retardants. (a) No person shall manufacture, process or distribute in commerce a product, or a flame-retardant part of a product, containing more than one-tenth of one per centum of pentabrominated diphenyl ether or octabrominated diphenyl ether, by mass.

(b) The commissioner may waive the provisions of this section in whole or in part upon a finding by the commissioner, in consultation with the commissioners of health and labor in a particular instance that there is no significant threat to the public health.

2. Administration. (a) The commissioner is hereby authorized and directed to prescribe such rules and regulations, including provisions for maintenance of records relating to products, fabrics or related materials, and for the labeling for a product, fabric or related material, as may be necessary and proper for purposes of administration and enforcement of this article.

(b) The commissioner is hereby empowered to order the recall of or confiscation of consumer products offered for sale which do not meet the standards set forth in or pursuant to this section.

(c) The commissioner may obtain from any person by regulation or subpoena issued pursuant thereto such information in the form of testimony, books, records, or other writings as is pertinent to the findings or determinations which he or she is required or authorized to make pursuant to this section.

All information reported to or otherwise obtained by the commissioner

or his or her representative pursuant to this section which information contains or relates to a trade secret shall be considered confidential, except that such information may be disclosed to other officers or employees concerned with carrying out this section or when relevant in any proceeding under this section.

3. Guaranty. (a) No person shall be subject to prosecution under this section if such person: (1) establishes a guaranty received in good faith signed by and containing the name and address of the person residing in the United States by whom the product, fabric or related material guaranteed was manufactured or from whom it was received, to the effect that reasonable and representative tests showing that the product, fabric or related material covered by the guaranty, or used in the product, fabric or related material covered by the guaranty meets the requirements of this section; and (2) has not, by further processing, affected the flammability of the product, fabric or related material covered by the guaranty which he or she received. Such guaranty shall be either: (A) a separate guaranty specifically designating the product, fabric or related material guaranteed, in which case it may be on the invoice or other paper relating to such product, fabric or related material; or (B) a continuing guaranty filed with the department or with the federal trade commission applicable to any product, fabric or related material handled by a guarantor, in such form as the department or the federal trade commission by rules or regulations may prescribe; or (C) a continuing guaranty given by seller to buyer applicable to any product, fabric or related material sold or to be sold to buyer by seller in a form as the department or the federal trade commission by rules or regulations may prescribe.

(b) The furnishing with respect to any product, fabric or related material, of a false guaranty, except by a person relying upon a guaranty to the same effect received in good faith and signed by and containing the name and address of the person residing in the United States by whom the product, fabric or related material guaranteed was manufactured or from whom it was received, with reason to believe the product, fabric or related material falsely guaranteed may be introduced, sold or transported in commerce, is unlawful.

4. Exclusions. (a) The provisions of this section shall not apply to any common carrier, contract carrier or freight forwarder with respect to a product, fabric or related material shipped or delivered for shipment through the state for commerce in another state or country.

(b) As used in this section, "distribute in commerce" shall not include the resale of products manufactured prior to January first, two thousand six or replacement parts manufactured prior to January first, two thousand six.

5. Violations. A violation of any provision of this section or any rule or regulation of the commissioner promulgated hereunder is a misdemeanor. Each product, fabric or related material made, sold or exposed for sale, shall constitute a separate violation.

6. Severability. The provisions of this section shall be severable and and if any portion thereof or the applicability thereof to any person or circumstances shall be held to be invalid, the remainder of this title and the application thereof shall not be affected thereby.

§ 3. Creation of state task force on flame retardant safety. 1. There is hereby created the "state task force on flame retardant safety", referred to hereafter as the task force. Such task force shall consist of thirteen members: seven of whom shall be appointed by the governor and shall include the commissioner of health, the commissioner of environmental conservation and the secretary of state; three of whom shall be appointed by the temporary president of the senate; and three of whom shall be appointed by the speaker of the assembly.

2. The ten at large members of the task force should include: two representatives of organizations whose prime function is the enhancement

of the environmental quality of the state; two representatives from the brominated flame retardant manufacturing industry; two representatives with expertise in the area of environmental health from academic institutions; two representatives from industries that manufacture products that use flame retardants and two health care professionals with expertise in the area of environmental health.

3. The commissioner of health, or his or her designee, shall serve as the chair of the task force.

4. The members of the task force shall receive no compensation for their services, but shall be allowed their actual and necessary expenses incurred in the performance of their duties.

5. The task force shall, at a minimum:

a. review and report on relevant studies, risk assessments, findings or rulings in connection with the flame retardant decabrominated diphenyl ether, including but not limited to any issued by the United States Environmental Protection Agency and the European Union; and

b. evaluate the availability of safer alternatives to the flame retardant decabrominated diphenyl ether, including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes.

6. The task force shall issue its findings, in the form of a report, no later than December 31, 2005.

7. The task force shall utilize the resources of the department of health, the department of environmental conservation and any other state agencies with relevant information or expertise.

8. The task force may consult with any person, organization, educational institution, or governmental entity including but not limited to the United States Environmental Protection Agency and the European Union.

9. The department of health and the department of environmental conservation shall provide the task force with such facilities, assistance, and data as will enable the task force to carry out its powers and duties. Additionally, all other agencies of the state or subdivisions thereof shall, at the request of the chair provide the task force with such facilities, assistance, and data as will enable the task force to carry out its powers and duties.

§ 4. This act shall take effect immediately; provided, however, that sections one and two of this act shall take effect January 1, 2006; provided, however that effective immediately, the addition, amendment and/or repeal of any rule or regulation necessary for the implementation of this act on its effective date is authorized and directed to be made and completed on or before such effective date.

The Legislature of the STATE OF NEW YORK ss:

Pursuant to the authority vested in us by section 70-b of the Public Officers Law, we hereby jointly certify that this slip copy of this session law was printed under our direction and, in accordance with such section, is entitled to be read into evidence.

JOSEPH L. BRUNO
Temporary President of the Senate

SHELDON SILVER
Speaker of the Assembly

Appendix 2. Members of the New York State Task Force on Flame Retardant Safety

Commissioner, New York State Department of Health
Staff: Nancy K. Kim, Ph.D., Chair

Commissioner, New York State Department of Environmental Conservation
Staff: Pamela Hadad-Hurst

Secretary, New York State Department of State
Staff: John Mueller (2008 - 2011)

Manuel R. Acevedo, M.D. (2010 - present)
Columbia University
Harlem Hospital Center

Kathleen A. Curtis, L.P.N.
Clean and Healthy New York, formerly Clean New York

Raymond B. Dawson, Ph.D.
Albemarle Corporation

Maida P. Galvez, M.D.
Mount Sinai School of Medicine

Ken Geiser, Ph.D.
University of Massachusetts Lowell

John P. Hassett, Ph.D.
State University of New York College of Environmental Science and Forestry

David Sanders, Ph.D.
DCS Consulting
Chemtura Corporation

Suzanne M. Snedeker, Ph.D. (2008 - 2012)
Cornell University

Dennis Sweeney
New York State Professional Fire Fighters Association

Leonardo Trasande, M.D. (2008 - 2010)
Mount Sinai School of Medicine

Appendix 3. Agendas for Meetings and Conference Calls of the New York State Task Force on Flame Retardant Safety

New York State Task Force on Flame Retardant Safety
The Century House
Conference Room B
Route 9, Latham, New York
Thursday, September 11, 2008
8:00 am – 3:30 pm

Objectives:

- **Provide overview and discussion of decabromodiphenyl ether.**
- **Provide overview and discussion of available alternatives to decabromodiphenyl ether.**
- **Obtain committee input on proposed report strategy.**

8:00 am Continental Breakfast

8:30 am Introductions
- Nancy Kim, New York State Department of Health

8:45 am Meeting Agenda
Format of Meetings
Legislation and Charge to the Task Force
- Nancy Kim, New York State Department of Health

9:15 am Overview of Decabromodiphenyl Ether
- Thomas Johnson, Research Scientist, New York State Department of Health
- Daniel Axelrad, Environmental Scientist, United States Environmental Protection Agency

10:15 am Break

10:30 am Discussion

11:30 am Lunch

12:30 pm Overview of Available Alternatives to Decabromodiphenyl Ether
- Ken Geiser, Co-Director, Lowell Center for Sustainable Production

1:30 pm Discussion

2:15 pm Public Comment Period

2:45 pm Draft Strategy for Completing Report Followed by Discussion
- Nancy Kim, New York State Department of Health

3:15 pm Meeting Wrap-up, Action Items
- Nancy Kim, New York State Department of Health

3:30 pm Adjourn

New York State Task Force on Flame Retardant Safety
New York State Department of Health
Center for Environmental Health, Conference Room 4 A/B
547 River Street, Troy, New York
Friday, November 7, 2008
8:00 am – 3:30 pm

Objectives:

- **Provide perspective of brominated flame retardant manufacturers on issues relevant to Task Force charge.**
- **Provide perspective of industries that manufacture products that use flame retardants on issues relevant to Task Force charge.**
- **Discuss strategy for satisfying Task Force charge and completion of Task Force report.**

8:00 am	Continental Breakfast
8:30 am	Introductions and Meeting Agenda • Nancy Kim, New York State Department of Health
9:00 am	Presentation by Brominated Flame Retardant Manufacturers on Decabromodiphenyl Ether • Raymond Dawson, Albemarle Corporation • David Sanders, Chemtura Corporation
10:00 am	Questions/Answers/Discussion
10:15 am	Break
10:30 am	Presentation on Decabromodiphenyl Ether and Alternatives from a Textile Industry Perspective • Phillip Wakelyn, Wakelyn Associates
11:15 am	Questions/Answers/Discussion
11:30 am	Public Comment Period
12:00 pm	Working Lunch

1:00 pm Presentation on Decabromodiphenyl Ether and Alternatives from an Electronics Industry Perspective
- Chris Cleet, Director, Environmental Affairs, Information Technology Industry Council

1:45 pm Questions/Answers/Discussion

2:00 pm Break

2:15 pm Discussion of Task Force Report Strategy
- Nancy Kim, New York State Department of Health
- Thomas Johnson, New York State Department of Health

3:15 pm Meeting Wrap-up, Action Items
- Nancy Kim, New York State Department of Health

3:30 pm Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Wednesday, January 28, 2009
10:00 am – 12:00 pm

Objectives:

- **Obtain information from the states of Maine and Washington on their evaluations of decabromodiphenyl ether and alternatives.**
- **Obtain information from an electronics manufacturer that uses alternatives to decabromodiphenyl ether.**

Note*: Both objectives are relevant to the Task Force charge, "Review and report on relevant studies, risk assessments, findings, or rulings in connection with the flame retardant decabromodiphenyl ether," and "evaluate the availability of safer alternatives to the flame retardant decabromodiphenyl ether, including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes."*

10:00 am Welcome, Introductions and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:10 am Presentation on Evaluations of Decabromodiphenyl Ether and Alternatives in Maine
- John James, Environmental Specialist, Maine Department of Environmental Protection

10:30 am Questions/Answers/Discussion

10:45 am Presentation on Use of Alternative Flame Retardants in the Electronics Industry
- Jose Reyes, Chief Technology Officer, JJI Technologies

11:05 am Questions/Answers/Discussion

11:20 am Presentation on Evaluations of Decabromodiphenyl Ether and Alternatives in Washington State
- Alex Stone, Senior Chemist, Washington State Department of Ecology

11:40 am Questions/Answers/Discussion

11:55 am Meeting Wrap Up/Action Items
- Nancy Kim, New York State Department of Health

12:00 pm Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Thursday, May 21, 2009
9:00 am – 11:00 am

Objectives:

- **To get comments about the overall approach to both sections covering issues such as the language level (too technical, not technical enough, okay, etc.), level of detail (too little, too much, okay), etc.**
- **Identify missing information for the background and health effects information on decaBDE.**
- **Critique the format used for the RDP write up as a model for other possible alternatives.**
- **Identify major omissions/problems with the RDP write up.**

9:00 am Welcome, Introductions and Meeting Agenda
- Nancy Kim, New York State Department of Health

9:10 am Discuss write-up for the background and health effects information on deca.
- Question 1. Is the overall approach (language and technical level) okay?
- Question 2. Any missing information?
- Question 3. Any other major comments?

10:00 am Discuss write-up for RDP
- Question 1. Is the overall approach (language and technical level) okay?
- Question 2. Is the format acceptable as a model for discussing alternatives?
- Question 3. Identify major omissions/problems with the RDP write-up?
- Question 4. Any other major comments?

10:50 am Meeting Wrap Up/Action Items
- Nancy Kim, New York State Department of Health

11:00 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Tuesday, July 28, 2009
10:00 am – 11:15 am

Objectives:

- **Sharing of draft documents.**
- **Discuss criteria for choosing alternatives and the choice of a decaBDE alternative for coated wire and telecom cable applications.**
- **Get general comments about new draft sections of report.**
- **Provide status update on sections of report.**

10:00 am Welcome, Introductions and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:10 am Sharing Draft Documents.

10:20 am Comments on Draft Report
- Thomas Johnson, New York State Department of Health
  - Criteria Choosing DecaBDE Alternatives (Section IIIA)
  - Basis for Magnesium Hydroxide as a DecaBDE Alternative for Coated Wiring and Telecom Cable Applications
- Suzanne Snedeker, Cornell University
  - US EPA Actions on DecaBDE (Section II.J.)
- Pamela Hadad-Hurst, NYS Department of Environmental Conservation
  - State Legislative Initiatives

10:50 am Status of Report Sections Being Drafted
- Thomas Johnson, New York State Department of Health
- John Hassett, SUNY College of Environmental Science and Forestry
- Kathleen Curtis, Clean and Healthy New York
  - Sections on Presence in Environment, Environmental Fate, Debromination, Presence in Humans, Human Exposure Pathways, Ecotoxicity
- Raymond Dawson, Albemarle Corporation
- David Sanders, Chemtura Corporation
  - European Union Actions on DecaBDE
- Ken Geiser, University of Massachusetts Lowell
  - DecaBDE Production and Use

11:00 am Meeting Wrap Up/Action Items/Availability for Next Meeting or Conference Call
- Nancy Kim, New York State Department of Health

11:15 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Thursday, September 24, 2009
10:00 am – 11:00 am

Objectives:

- **Discuss the choice of alternative decaBDE flame retardants to evaluate in Task Force report.**
- **Provide status update on various decaBDE sections of report.**

10:00 am Welcome, Introductions and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:10 am Choice of DecaBDE Alternatives for Report
- Thomas Johnson, New York State Department of Health

10:30 am Status of DecaBDE Sections
- John Hassett, SUNY College of Environmental Science and Forestry
- Kathleen Curtis, Clean and Healthy New York
  - Sections on DecaBDE Environmental Fate, Debromination
- Raymond Dawson, Albemarle Corporation
- David Sanders, Chemtura Corporation
  - European Union Actions on DecaBDE

10:45 am Other (–Big Picture") Comments on Task Force Report

10:55 am Meeting Wrap Up/Action Items/Availability for Next Meeting or Conference Call
- Nancy Kim, New York State Department of Health

11:00 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Wednesday, October 28, 2009
10:00 am – 11:00 am

Objectives:

- **Obtain summary and big picture comments on the Task Force report draft section on decaBDE environmental fate and debromination, and determine an approach for debromination.**
- **Obtain big picture comments on the Task Force report draft section on magnesium hydroxide.**
- **Provide status update on various decaBDE sections of report.**

10:00 am Welcome, Introductions and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:10 am DecaBDE Environmental Fate and Debromination
- John Hassett, SUNY College of Environmental Science and Forestry

10:30 am Magnesium Hydroxide
- Tom Johnson, New York State Department of Health

10:45 am Status of Report Sections under Preparation

10:55 am Meeting Wrap Up/Action Items/Availability for Next Meeting or Conference Call
- Nancy Kim, New York State Department of Health

11:00 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Thursday, December 3, 2009
1:00 pm – 2:00 pm

Objectives:

- **Obtain big picture comments on the Task Force report draft introductory section to decaBDE alternatives and report outline.**
- **Obtain big picture comments on the Task Force report draft section on decaBDE ecotoxicity.**
- **Provide status update on various decaBDE sections of report.**

1:00 pm Welcome, Introductions and Meeting Agenda
- Nancy Kim, New York State Department of Health

1:05 pm Revised Draft Introductory Section for DecaBDE Alternatives and Report Outline
- Tom Johnson, New York State Department of Health

1:20 pm DecaBDE Ecotoxicity
- Pamela Hadad Hurst, New York State Department of Environmental Conservation
- Tim Sinnott, New York State Department of Environmental Conservation

1:35 pm Status of Report Sections under Preparation

1:50 pm Meeting Wrap Up/Action Items/Availability for Next Meeting or Conference Call
- Nancy Kim, New York State Department of Health

2:00 pm Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Wednesday, January 13, 2010
10:00 am – 11:15 am

Objectives:

- **Identify an approach for finalizing the Task Force report given the recent decision to phase out of decabromodiphenyl ether.**
- **Provide status update/comments on various sections of report.**

10:00 am Welcome, Introductions and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:05 am Phase out of DecaBDE
- Ray Dawson (Albemarle Corporation)
- Dave Sanders (Chemtura Corporation)

10:15 am Task Force Report Format
- Nancy Kim, New York State Department of Health

10:35 am Identify Actions Needed for Finalizing Report/Assignment of Tasks

10:50 am Status/Comments on Report Sections
- Magnesium Hydroxide
- Actions by European Union
- Environmental Fate
- Ecotoxicity
- Fire Safety in New York

11:10 am Wrap-up/Schedule Next Conference Call

11:15 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Thursday, February 25, 2010
12:30 pm – 2:00 pm

Objectives:

- **Obtain information from US EPA staff on the decaBDE phase out and the agency's PBDE action plan.**
- **Identify remaining tasks for completion of draft Task Force report.**

12:30 pm	Welcome, Introductions and Meeting Agenda • Nancy Kim, New York State Department of Health
12:35 pm	Presentation on Deca BDE Phase Out and PBDE Action Plan • Ward Penberthy, Deputy Director of Chemical Control Division, United States Environmental Protection Agency
12:50 pm	Questions/Answers
1:10 pm	Description of Draft Task Force Report and Remaining Tasks for Completion • Tom Johnson, New York State Department of Health
1:55 pm	Identify Action Items/Schedule Next Conference Call
2:00 pm	Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Wednesday, March 31, 2010
12:30 pm – 1:30 pm

Objectives:

- **Agree on tentative schedule for completion of Task Force report.**

12:30 pm	Welcome and Meeting Agenda • Nancy Kim, New York State Department of Health
12:35 pm	Status of Various Task Force Report Sections • Tom Johnson, New York State Department of Health
1:00 pm	Tentative Schedule for Completion of Task Force Report • Task Force on Flame Retardant Safety
1:25 pm	Identify Action Items/Schedule Next Conference Call
1:30 pm	Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Tuesday, May 4, 2010
10:30 am – 11:30 am

Objectives:

- **Update status and changes in Task Force Report Sections.**
- **Go over current schedule for submission of comments and completion of Task Force report.**

10:30 am	Welcome and Meeting Agenda • Nancy Kim, New York State Department of Health • Tom Johnson, New York State Department of Health
10:35 am	Status/Changes for Task Force Report Sections Schedule for Submission of Comments and Completion of Task Force Report • Tom Johnson, New York State Department of Health
11:15 am	Identify Action Items/Schedule Next Conference Call
11:20 am	Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Thursday, June 3, 2010
10:00 am – 11:00 am

Objectives:

- **Update status of Task Force Report Sections and Schedule.**
- **Provide overview of Product Redesign Section.**
- **Identify process for release of report and potential reviewers of public comment draft.**

10:00 am Welcome and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:05 am Status of Task Force Report Sections
- Review of DecaBDE Section
- Introductory Sections
- Magnesium Hydroxide
- Appendices 2, 4 and 5
- Glossary

10:25 am Overview of Product Redesign Section
- Tom Johnson, New York State Department of Health

10:30 am Report Release Process and Potential Reviewers
- Nancy Kim, New York State Department of Health

10:45 am Identify Action Items/Schedule Next Conference Call

10:50 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Friday, July 9, 2010
10:00 am – 11:00 am

Objectives:

- **Update status of Task Force Report Sections and Schedule.**
- **Identify remaining issues for completion of full draft report.**

10:00 am Welcome and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:05 am Status of Task Force Report Sections
- Tom Johnson, New York State Department of Health

10:25 am Remaining Issues
- Nancy Kim, New York State Department of Health

10:45 am Identify Action Items/Schedule Next Conference Call

10:50 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Thursday, September 23, 2010
10:00 am – 11:00 am

Objectives:

- **Present options for addressing industry comments received on the decaBDE section of the Task Force report.**

10:00 am Welcome and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:05 am Options for Addressing Industry Comments on DecaBDE Section of Final Report
- Nancy Kim, New York State Department of Health
- Tom Johnson, New York State Department of Health

10:55 am Identify Action Items/Schedule Next Conference Call

11:00 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Thursday, October 28, 2010
2:00 pm – 3:00 pm

Objectives:

- **Present status of revisions to decaBDE section of Task Force report and obtain comments/suggestions.**

2:00 pm	Welcome and Meeting Agenda • Nancy Kim, New York State Department of Health
2:05 pm	Administrative Items • Nancy Kim, New York State Department of Health
2:20 pm	Status of DecaBDE Section of Task Force Report
2:45 pm	Identify Action Items/Schedule Next Conference Call
2:50 pm	Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Thursday, December 13, 2010
9:30 am – 11:30 am

Objectives:

- **Discuss overarching comments on November 30th draft of Task Force report, develop approaches for addressing those comments and discuss approach for report's conclusions and Executive Summary.**

9:30 am	Welcome and Meeting Agenda • Nancy Kim, New York State Department of Health
9:35 am	Administrative Items • Nancy Kim, New York State Department of Health
9:45 am	Comments and discussion on the November 30th Draft of Task Force Report - Section 1: Introduction - Section 2: Decabromodiphenyl Ether - Section 3: Decabromodiphenyl Ether Alternatives
11:00 am	Next Steps
11:10 am	Date for Next Conference Call Week of February 28th
11:15 am	Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Wednesday, April 13, 2011
10:30 am – 12:30 pm

Objectives:

- **Revise/finalize the conclusions of the draft Task Force report.**
- **Finalize the process for releasing the report as a draft for public comment.**

10:30 am Welcome and Meeting Agenda
- Nancy Kim, New York State Department of Health

10:35 am Revise/Finalize Conclusions for the Draft of Task Force Report
- Nancy Kim/All
  - Executive Summary
  - Section 1: Introduction
  - Section 2: Decabromodiphenyl Ether
  - Section 3: Decabromodiphenyl Ether Alternatives

12:00 pm Releasing the Draft for Public Comment
- Nancy Kim, New York State Department of Health

12:15 pm Next Steps

12:30 pm Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Wednesday, September 7, 2011
10:00 am – 11:00 am

Objectives:

- **Update panel members on preparations for releasing public comment review draft of the draft Task Force report.**
- **Obtain suggestions on mailing list for draft report.**

10:00 am	Welcome and Meeting Agenda • Nancy Kim, New York State Department of Health
10:05 am	Update on Releasing Public Comment Draft • Nancy Kim, New York State Department of Health
10:25 am	Mailing List for Draft Report • Tom Johnson, New York State Department of Health
10:45 am	Next Steps
10:50 am	Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Friday, October 7, 2011
2:00 pm – 3:00 pm

Objective:

- **Determine an approach for addressing the San Antonio Statement on Brominated and Chlorinated Flame Retardants in the context of the public comment review draft of the Task Force report.**

2:00 pm	Welcome and Meeting Agenda • Nancy Kim, New York State Department of Health
2:05 pm	Information on Stakeholder Task Force Processes • Diana Yang, Division of Legal Affairs, New York State Department of Health
2:15 pm	Approaches for Addressing San Antonio Statement
2:50 pm	Next Steps
3:00 pm	Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Wednesday, September 19, 2012
9:30 am – 11:30 am

Objective:

- **To discuss and determine an approach for revising draft report.**

9:30 – 9:40 Introductions, agenda, objectives

- Nancy Kim, New York State Department of Health

9:40 – 10:00 Briefing on April 1, 2013 implications

- Holly Dellenbaugh
  - Questions/answers/discussion
- All

10:00 – 11:15 Possible approach – responding to comments

- Nancy Kim, New York State Department of Health

Suggested responses/discussion

- Nancy Kim, New York State Department of Health
- Tom Johnson, New York State Department of Health

Responses to comments for report

- Nancy Kim, New York State Department of Health/All

11:15 – 11:30 Next steps

- Nancy Kim, New York State Department of Health

11:30 am Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Wednesday, December 13, 2012
3:00 pm – 5:00 pm

Objective:

- **To agree on revisions/approaches to revising draft report.**

3:00 – 3:15 Roll Call, Objective and Update from last conference call

- Nancy Kim, New York State Department of Health

3:15 – 4:45 Review Sections

- Nancy Kim, New York State Department of Health/All
  - Section 1.3 Strategies for reporting on deca
  - Section 1.4 Fire Safety Efforts
  - Sections 2.10.3 - 2.10.6 EPA Actions
  - Section 2.11 European Union Actions
  - Section 2.12 Conclusion decaBDE
  - Section 3.3.1 RDP
  - Section 3.5 Conclusions - Alternatives
  - Executive Summary

4:45 – 5:00 Follow up Actions

- Nancy Kim, New York State Department of Health

New York State Task Force on Flame Retardant Safety
Conference Call
Friday, February 1, 2013
1:00 pm – 4:00 pm

Objectives: **Clarify any legal issues for finalizing the report**
Make decisions to finalize the content of the report

1:00 – 1:10 Roll call and schedule
- Nancy Kim, New York State Department of Health

1:10 – 1:30 Clarify legal issues, including voting
- Diana Yang, Division of Legal Affairs/All

1:30 – 1:45 Ray Dawson‘s Comments/Clarification
- Ray Dawson, Albemarle Corporation/All

1:45 – 2:00 Plan for voting on the report
- Nancy Kim, New York State Department of Health/ Diana Yang, Division of Legal Affairs/All

2:00 – 2:50 Review/Comment on report as needed
- Nancy Kim, New York State Department of Health/All
  - Finalize Approach to safer alternatives
  - Alternatives
  - Executive Summary
  - Other Sections

2:50 – 3:00 Follow-up actions
- Nancy Kim, New York State Department of Health

3:00 Adjourn

New York State Task Force on Flame Retardant Safety
Conference Call
Tuesday, March 12, 2013
2:00 pm – 3:00 pm

Objective:

- **To schedule another conference call and to identify any unresolved issues.**

2:00 – 2:15 Roll call and objective
- Nancy Kim, New York State Department of Health

2:15 – 2:30 Schedule another meeting
- Nancy Kim, New York State Department of Health

2:20 – 2:55 Any unresolved issues
- Nancy Kim, New York State Department of Health /All

2:55 – 3:00 Follow-up actions and close
- Nancy Kim, New York State Department of Health

New York State Task Force on Flame Retardant Safety
Conference Call
Monday, March 25, 2013
11:00 pm – 12:00 pm

Objective:

- **To approve the final Report of the New York State Task Force on Flame Retardant Safety.**

11:00 – 11:15 Roll call, summary and objective
- Nancy Kim, New York State Department of Health

11:15 –11:20 Approval of minutes for last conference call
- Nancy Kim, New York State Department of Health//Diana/All

11:20 – 11:25 Call for a motion on report to approve and second
- Nancy Kim, New York State Department of Health

11:25 – 11:45 Discussion of motion to approve
- Nancy Kim, New York State Department of Health//Diana
- All

11:45 – 11:50 Vote on motion
- Nancy Kim, New York State Department of Health

11:50 – 12:00 Follow-up actions and close

Appendix 4. Public Comments on Draft Report

Response to Public Comments on the Draft Report of the New York State Task Force on Flame Retardant Safety

Five comment letters were received on the draft report of the New York State Task Force on Flame Retardant Safety. The draft version of the report was released for public comment on April 30, 2012 and written public comments were accepted on the draft through July 31, 2012.

Letter 1, Emma Lavoie, US EPA Design for the Environment Program
Letter 2, Catherine O'Dell, Minnesota Pollution Control Agency
Letter 3, Alex Stone, Washington Department of Ecology
Letter 4, Robert Sweeney, New York State Assembly
Letter 5, Bobbi Chase-Wilding, Clean and Healthy New York, and 49 other organizations:

New York organizations:

David Levine, President
The American Sustainable Business Council
New York, New York

Margaret Roberts, Director
Capital Region Action Against Breast Cancer (CRAAB!)
Albany, New York

Barbara Warren, Executive Director
Citizens' Environmental Coalition
Albany, New York

Cynthia Wilson, President
Citizens for a Clean Environment
Cobleskill, New York

Erin Heaney, Executive Director
Clean Air Coalition of Western New York
Buffalo, New York

Judy Braiman, President
Empire State Consumer Project
Rochester, New York

Saima Anjam, Government Affairs Program Associate
Environmental Advocates of New York
Albany, New York

Judith M. Anderson, Executive Director
Environmental Justice Action Group of Western New York
Buffalo, New York

Laura Weinberg, President
Great Neck Breast Cancer Coalition
Great Neck, New York

Karen Joy Miller, President
Huntington Breast Cancer Action Coalition
Prevention is the Cure
Huntington, New York

David O. Carpenter, M.D., Director
Institute for Health and the Environment
University at Albany
Rensselaer, New York

Stephen Boese, Executive Director
Learning Disabilities Association of New York State
Latham, New York

Christine Brouwer, Founder
Mira's Movement – Supporting Families Facing Childhood Cancer
Ithaca, New York

Miranda Massie, Interim Executive Director/ Legal Director
New York Lawyers for the Public Interest
New York, New York

Dennis Sweeney, Health & Safety Representative
NY State Professional Fire Fighters Association
Albany, New York

See attached letter
Russ Haven, Legislative Counsel
New York Public Interest Research Group (NYPIRG)
Albany, New York

Kathleen M. Donahue, Vice President
New York State United Teachers
Latham, New York

Jeff Bohner, Chair
Sierra Club Atlantic Chapter
New York, New York

Cecil Corbin-Mark, Deputy Director
WE ACT for Environmental Justice
New York, New York

National organizations:

Katie Huffling, RN, MS, CNM, Director of Programs
The Alliance of Nurses for Healthy Environments
Baltimore, Maryland

Jeanne Rizzo, RN, Executive Director
The Breast Cancer Fund
San Francisco, California

Judy Levin, Pollution Prevention Co-Director
Center for Environmental Health
San Francisco, California
New York, New York

Lois Gibbs, Executive Director
Center for Health, Environment and Justice
Falls Church, VA

Lauren Heine, Consulting Co-Director
Clean Production Action
Anchorage, Alaska

Eve C. Gartner, Staff Attorney
Earthjustice
New York, New York

Richard Denison, Senior Scientist
Environmental Defense Fund
Washington, D.C.

Rick Hind, Legislative Director
Greenpeace
Washington, D.C.

Claire Barnett, Executive Director
Healthy Schools Network
Albany, New York

Arlene Blum
Arlene Blum, PhD, Executive Director
Green Science Policy Institute
Berkeley, California

Gigi Lee Chang
Gigi Lee Chang, CEO
Healthy Child Healthy World
Los Angeles, California

Tom Lent, Policy Director
Healthy Building Network
Berkeley, California

Authorized via communication with Nancy Kim
Claire Barnett, Executive Director
Healthy Schools Network
Albany, New York

Russell Long, PhD, Board member
Friends of the Earth
Washington, DC

Jose Bravo, Executive Director
Just Transition Alliance
San Diego, California

Erin Switalski
Executive Director
Women's Voices for the Earth
Missoula, MT

Organizations from other states:

Pamela Miller, Executive Director
Alaska Community Action on Toxics
Anchorage, Alaska

Mark Mitchell, MD, Senior Policy Advisor,
Connecticut Coalition for Environmental Justice
President
Mitchell Environmental Health Associates
Hartford, CT

Anne Hulick, Coordinator
Coalition for a Safe and Healthy Connecticut
Hartford, CT

Rebecca Meuninck, Environmental Health
Campaign Director
Ecology Center
Ann Arbor, Michigan

Mike Belliveau
Environmental Health Strategy Center
Bangor, Maine

Kathleen Schuler, MPH, Co-Director
Healthy Legacy
Institute for Agriculture and Trade Policy
Minneapolis, Minnesota

Sandra Cort, Board Member, Past President
Learning Disabilities Association of Maine
Windham, Maine

Richard Moore, Coordinator
Los Jardines Institute (The Gardens Institute)
Albuquerque, New Mexico

Cindy Luppi, New England Director
Clean Water Action
Boston, MA
Abby King, Policy Advocate

Natural Resources Council of Maine
Augusta, Maine

Colin Price, Director of Research & Market Innovation
Oregon Environmental Council
Portland, Oregon

Ana Mascareñas, Policy & Communications Director
Physicians for Social Responsibility - Los Angeles
Los Angeles, CA

Andrew McGuire, Executive Director
Trauma Foundation
San Francisco General Hospital
San Francisco, California

Lauren Hierl, Environmental Health Advocate
Vermont Public Interest Research Group
Montpelier, Vermont

Laurie Valeriano, Executive Director
Washington Toxics Coalition
Seattle, Washington

Cc: Governor Andrew Cuomo

Senator Mark Grisanti, Environmental Conservation Committee Chair

Assemblyman Robert Sweeney, Environmental Conservation Committee Chair

Senator Carl Marcellino

NYPIRG
NEW YORK
PUBLIC INTEREST
RESEARCH GROUP

VIA EMAIL

January 23, 2013

New York State Department of Health
Corning Tower
Albany, New York

Dear Sir/Madam:

This is to confirm that the New York Public Interest Research Group (NYPIRG) endorses certain comments submitted with respect to the New York State Taskforce on Flame Retardant Safety to which NYPIRG's name was listed, but no signature from an authorized representative was provided.

In particular, NYPIRG was among fifty organizations from New York and across the nation submitting comments by letter dated July 31, 2012 and addressed to Michelle Lavigne, New York State Department of Health, Bureau of Toxic Substance Assessment.

Please accept this letter and my signature below as our enthusiastic endorsement of and signature upon such comments. If there is any issue with respect to NYPIRG's endorsement of the above-referenced letter and comments, please contact me.

Sincerely,

Russ Haven, Esq.
Legislative Counsel

107 WASHINGTON AVE, 2ND FLOOR • ALBANY, New York 12210-2270 • 518-436-0876 • Fax 518-432-6178
Offices In: Albany, Buffalo, Binghamton, Cortland, Long Island, New Paltz, New York City, Purchase & Syracuse

The comments are briefly summarized below along with a description of how the panel addressed them. Comments with similar themes or content are grouped together. The letters in their entirety are included after the summary.

Summary

1. **Letter 1,5:** The draft report should acknowledge the July 2012 release of the US EPA draft document "An Alternatives Assessment for the Flame-Retardant Decabromodiphenyl Ether (DecaBDE)." Release of the draft report should be delayed until the US EPA finalizes the document (Letter 5).
   Response: A description of the US EPA draft report was added to Section 2.10 (Actions by the US EPA).

2. **Letter 1:** The reason why the alternatives were chosen is not explicitly stated. Doing so would provide clarity to the report.
   Response: The rationale for choosing which alternatives to cover in the report was in Section 1.3. This section was revised to clarify the rationale and make the reasoning more explicit.

3. **Letters 2, 3 and 5:** The draft report needs to put greater emphasis on debromination of decaBDE, and state more explicitly that this is a crucial factor in evaluating the risks from decaBDE. Letter 2 states the need for directly linking these two areas especially in the Executive Summary and also states that the debromination studies are accurately reported. Letter 3 comments on the "lack of emphasis on the persistence, bioaccumulation of degradation products of decaBDE". Letter 5 states "the Task Force failed to fully evaluate the health and environmental impacts of other" PBDEs from the debromination of decaBDE .
   Response: Section 2.5 of the draft report covered debromination and summarized the studies in some detail. The draft report also stated the importance of debromination in the introduction to this section (Section 2.5) and also concluded that the degradation products from decaBDE may pose a greater risk than the degradation products of the alternatives (see Sections 3.1.7, 3.3.1.8 and 3.3.2.10.). The draft report was edited in the mentioned sections, Executive Summary and other appropriate areas to help the reader understand the connection between debromination products and the risks from decaBDE. Adding a section that fully evaluates the health and environmental impacts of the debromination products of decaBDE as requested by Letter 5 was not feasible by April 1, 2013. However, debromination information based on work by the European Union was added to Section 2.11 and in Appendix 8.

4. **Letter 2:** "Acknowledging the changing paradigms in toxicology and risk assessment, as well as the consensus among scientists that current tools do not adequately describe the risks from chemical exposures, provides important context to the information provided in the NYS draft report."
   Response: Paragraphs about the limitations of risk assessment and its new directions were added to the introduction to the report.

5. **Letter 2:** The US EPA's Voluntary Children's Chemical Evaluation Program (VCCEP) has been discontinued. The US EPA Office of the Inspector General reported in 2011 that the program did not meet its goals, is no longer operational and that the agency has "no plans to revive, replace or terminate the program."
   Response: The section on the VCCEP program was removed from the report.

6. **Letter 3:** "Concerns with some research" were noted and recommended stressing that the 2002 European Risk Assessment was too old and the report should not rely on its conclusions. The report should include the US EPA's recently released 2012 report on decaBDE.

Response: The charge required that European Union actions be summarized so the 2002 risk assessment was included. The draft report gave some of the 2002 European Union conclusions and recommendations, but used updates to this report and newer data/reports to reach conclusions. The draft report was edited to clarify the review of the 2002 European Risk Assessment and information was added about the 2012 European Union identification of BDE-209 as a PBT/vPvB substance. The recent US EPA report on decaBDE was also added (see response to Comment 1).

7. **Letters 3, 4 and 5:** "Perceived bias"
Response: These three letters mentioned in different ways that the report appeared to have a bias. One letter specifically mentioned the discussion of the developmental neurotoxicity studies. The draft report was edited to try to reduce the perception of bias, although we recognize that we may not be able to eliminate the possibility that some bias may be perceived. One comment mentioned the connotation of the words "some scientists believe" and those phrases were removed or revised. The discussion of the neurotoxicity studies was not revised. The limitations of the studies reporting positive neurotoxic effects of decaBDE were included in the US EPA risk assessment of decaBDE. The US EPA noted the same limitations discussed in the task force report, and concluded that confidence in the primary study serving as the basis for its decaBDE reference dose (as well as the reference dose itself) was low. The study quality and design issues, according to the US EPA, constituted a scientifically valid reason for having lower confidence in the results and would be an important part of an evaluation. The draft report presented this, as well as stating that the studies provide evidence that decaBDE may be a developmental neurotoxin.

   The wording of the conclusions in the different sections and the overall conclusions in the draft report were revised to make them clearer and more definitive. These revisions may also help to address the perception of bias.

   A statement was added to the draft report about the existence of non-peer reviewed studies although they were not used in report.

8. **Letter 5:** The draft report fails to reach clear conclusions that identify hazards posed by decaBDE and its breakdown products and fails to explicitly conclude that alternatives presented are less toxic. A statement regarding the relative safety of alternatives must be clearly made in both the Executive Summary and in the final conclusion of the report.
Response: The Task Force revised the draft report defined the term safer alternative, and that term included breakdown products for BDE-209 and the alternative chemical flame retardants. The draft report was revised to include explicit conclusions and provided the strengths and limitations of those conclusions. Revisions were made to the alternatives sections, overall conclusion and to the Executive Summary.

9. **Letter 5:** The draft report misperceives the Legislature's charge, fails to reach clear conclusions that identify hazards and is significantly out of step with other agency drafted reports.
Response: The charge to the Task Force is to "a. review and report on relevant studies, risk assessments, findings or rulings in connection with the flame retardant decabrominated diphenyl ether, including but not limited to any issued by the United States Environmental Protection Agency and the European Union; and b. evaluate the availability of safer alternatives to the flame retardant decabrominated diphenyl ether, including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes." The report addresses all these issues. The report uses information from the other reports, including those from the US EPA and the European Union. Although the specific language differs, the conclusions, within the charge, are consistent with the other reports.

10. **Letter 5:** The section on fire safety, fire deaths and use of chemical flame retardants in fire prevention is outside the charge of the Task Force and should be removed. If it remains, the report should present an accurate state of the science regarding the use of chemical additives as flame retardants. The CPSC recently testified to a Senate panel that chemical flame retardants do not provide any meaningful fire safety protection.
Response: The section on fire safety in New York discusses fire issues and the laws/regulations related to fires. It provides background information for the report and some of that information is used in the evaluation of the reliability of alternatives. That section makes no statement about the need for flame retardants. The section was edited and shortened to make it more concise and many of the tables were removed and placed in Appendix 5.

11. **Letter 5:** The draft report fails to consider environmental and health impacts of combustion of decaBDE in its review of potential hazards.
Response: The draft report did not present a detailed discussion of this topic, but did state in Section 1.4 that burning materials that contain brominated flame retardants, including polybrominated diphenyl ethers such as decaBDE, can form brominated compounds such as hydrogen bromide (a corrosive, irritating gas) and brominated dibenzo-*p*-dioxins and dibenzofurans.

12. **Letter 5:** There are numerous instances in which statements are made regarding lack of scientific consensus, when in fact there is broad agreement among independent researchers without a financial tie to flame retardant makers. If in fact the Task Force doesn't agree upon how to report the findings, this statement should be made instead of simply using a blanket statement of "lack of scientific consensus," or a dismissal of areas of broad agreement with phrases like, "some scientists believe…"
Response: The statements concerning the lack of scientific consensus referred to studies in the peer reviewed scientific literature that gave disparate results. The report was edited to avoid the words lack of consensus, leaving the studies to speak for themselves. Also, see response to Comment 7.

13. **Letters 4 and 5:** California is currently redrafting furniture flammability standards such that the use of chemical flame retardants can be reduced or eliminated. The finalization of the report should be delayed until this process is complete.
Response: Information about California's process in the final report is now included in the report.

14. **Letter 5:** The draft report avoided reaching conclusions because the EPA reached a voluntary agreement for the phase out of decaBDE with the leading three flame retardant makers. However, Americans will still be exposed to decaBDE if overseas decaBDE makers sell their chemical to product makers who add it to products made overseas. There is no prohibition in the federal phaseout agreement of final products containing decaBDE from being imported into the United States and sold through retail venues. Therefore, this draft report must remain germane to ongoing discussions about how states may act to protect their residents from toxic chemicals.
Response: See response to Comment 8 for revisions to conclusions. The report acknowledged the limitations of the decaBDE phase out in Section 2.10.2. Additional information was added to the limitations and these limitations were added to the overall conclusion and the Executive Summary.

15. **Letter 5:** Based on recent Senate hearings which probed the chemical industry's misrepresentation of fire safety studies, there is concern about the favoring of industry-sponsored studies in the report, and it is even more important that this report is accurate. As the report stands, the chemical industry will gloss over details and focus on summaries and use the report as a means of defending the ongoing use of decaBDE around the world. Task Force and

Department should not be complicit in this by putting a stamp of approval on industry's sponsored research.
Response: Comment noted.

16. **Letter 5:** The draft report does not live up to mission of the New York State Department of Health, to "protect, improve and promote the health, productivity and well-being of all New Yorkers." The report should put public health first, not the wishes of two flame retardant chemical industry Task Force members. To simply include these comments and publish the report "as is" is unacceptable. The report should be revised to correct the previously mentioned items by this commenter.
Response: Comment noted.

Public Comments on the Draft Report of the New York State Task Force on Flame Retardant Safety

Letter 1

Emma T. Lavoie, PhD, US EPA Design for the Environment Program

Dear Madam or Sir,

Thank you for the opportunity to provide comments on the Draft Report of the New York State Task Force on Flame Retardant Safety.

We offer two recommendations that we believe will help improve transparency and clarity in the report regarding available information on alternatives and why a select few were reviewed. The recommendations are in the document attached to this email.

If there is a need to discuss our comments, please contact me at the number below.

Best regards,

-Emma

Emma T. Lavoie, PhD
Design for the Environment Program - US EPA
Office of Pollution Prevention and Toxics
Tel: 202.564.0951 www.epa.gov/dfe

To:	The New York State Department of Health
From:	Design for the Environment Branch Economics, Exposure, and Technology Division Office of Pollution Prevention and Toxics U.S. Environmental Protection Agency
Subject:	Comments on the Draft Report of the New York State Task Force on Flame Retardant Safety
Date:	July 5, 2012

As the Draft Report of the New York State Task Force on Flame Retardant Safety is still open for public comment, the Design for the Environment Branch would like to offer the following recommendations:

I. A brief description of the 2005 report published by the Swedish Chemicals Inspectorate is included within the draft report to elucidate the number of commercially available alternatives to decaBDE (section 1.3; p.4). Within the same timeframe of New York producing the draft report, the U.S. EPA has identified a list of 30 alternatives to decaBDE.[1] As such, we believe the report would be strengthened by acknowledging this list of alternatives and the ongoing decaBDE alternative assessment by the U.S. EPA's Design for the Environment Program. We also think it would be useful to provide a weblink where the EPA report can be located once it is released this year.[2]

   Addition of this information regarding EPA's alternatives assessment on decaBDE would further meet the requirement of the task force: ~~"r~~eview and report on relevant studies, risk assessments, findings or rulings in connection with the flame retardant decabrominated diphenyl ether, including but not limited to any issued by the United States Environmental Protection Agency …" (section 1.1; p.1).

II. While the draft report makes it clear that there is an array of limitations and uncertainties regarding magnesium hydroxide, resorcinol bis(diphenylphosphate), boric acid and aluminum casings as the alternatives that have been chosen, the specific reason(s) why these four were selected from a multitude of commercially available alternatives is not explicitly stated.

   The Design for the Environment Branch believes that clarifying the reason for only including four alternatives would better support the requirement of the task force to: ~~"e~~valuate the availability of safer alternatives to the flame retardant decabrominated diphenyl ether, including an assessment of the reliability, ready availability and cost-effectiveness of such substitutes" (section 1.1; p.1).

We hope that the two recommendations provided above will help improve transparency and clarity in the draft report regarding available information on alternatives and why a select few were reviewed.

[1] http://www.epa.gov/dfe/pubs/projects/decaBDE/deca_bde_alternatives.pdf
[2] http://www.epa.gov/dfe/pubs/projects/decaBDE/about.htm

Letter 2

Catherine O'Dell, PG, Minnesota Pollution Control Agency

From: O'Dell, Catherine (MPCA)
Sent: Monday, July 30, 2012 5:04 PM
To: 'flametf@health.state.my.us'
Subject: Comments on NYS Task Force draft report on DecaBDE

Dear Ms Lavigne:

I am attaching an unsigned copy of my comments on the draft report referenced above. A signed copy of the letter will go out in the mail to you tomorrow. Thank you very much for the opportunity to comment!

Best regards,

Cathy O'Dell

Cathy O'Dell, PG
Principal Planner/Hydrogeologist
Environmental Reporting and Special Studies Unit
Minnesota Pollution Control Agency
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catherine.odell@state.mn.us
www.pca. state. mn.us

Minnesota Pollution Control Agency
520 Lafayette Road North | St. Paul, Minnesota 55155-4194 | 651-296-6300
800-657-3864 | 651-282-5332 TTY | www.pca.state.mn.us | Equal Opportunity Employer

July 31, 2012

Ms. Michelle Lavigne
Bureau of Toxic Substances Assessment
Flanigan Square Room 300
547 River Street
Troy, NY 12280

Dear Ms. Lavigne:

Thank you for the opportunity to review and comment upon the New York State (NYS) Flame Retardant Task Force draft report, dated December 2011. I want to begin with a note of congratulations to the Task Force and NYS Department of Health staff. Having been involved in a similar effort in Minnesota in 2008, I have a level of appreciation for the time and effort that went into developing this report. Overall, I found the report carefully researched and reported. The focused section on alternatives is especially useful, and demonstrative of progress made in this area over the past few years. I do, however, have a few suggestions that would make the report better reflect current discussions on the topics of chemical risk, toxicity and assessment. These are provided below, with one additional comment.

1. Paragraphs 4-8 of the Executive Summary, discussing Decabromodiphenyl ether (DecaBDE) in the environment and toxicology:

These paragraphs summarize current knowledge about DecaBDE. Paragraphs 4 and 5 note that "numerous studies" have evaluated DecaBDE's potential to debrominate and form more toxic polybrominated diphenyl ether (PBDE) congeners. Paragraphs 6, 7, and 8 then summarize studies that focus exclusively on DecaBDE and its effects. These studies are accurately reported, yet full comprehension of the presented information requires one to connect it with the debromination discussion in Paragraphs 4 and 5. The reader needs to know that debromination of DecaBDE, whether in the environment or in living organisms, is an important element of assessing the potential harm posed by DecaBDE; this is the case even though the conditions and extent to which debromination occurs is not fully understood.

2. Paragraphs 4-8 of the Executive Summary, discussing DecaBDE in the environment and toxicology:

Within toxicology there is growing recognition that the current approach of testing chemical safety using traditional dose-response tests does not reveal the whole story of a chemical's potential for harm. There is increasing evidence that health effects may occur at low chemical doses that have not traditionally been included in dose-response studies; these health effects are real, but we do not yet have the research or tools to understand and make use of this information.

Relating to this, epidemiology studies are showing stronger associations between chemical exposure and human health effects. Linda Birnbaum, Director of the National Institute of Environmental Health Studies (NIEHS) and the National Toxicology Program (NTP) of the National Institutes of Health, has written a number of articles on this topic in the last few months. PBDEs are on a short list of ten chemical groups cited by Ms. Birnbaum that are widely distributed in the environment and are suspected of causing neurodevelopmental toxicity in human beings; these chemicals have been identified as priorities for future, focused research. The article cited was published online on April 25, 2012 in *Environmental Health Perspectives* and is available at the following link: http://dx.doi.org/10.1289/ehp.1104285.

Acknowledging the changing paradigms in toxicology and risk assessment, as well as the consensus among scientists that current tools do not adequately describe the risks from chemical exposures, provides important context to the information provided in the NYS draft report.

3. Paragraph 9 notes U.S. actions with regard to DecaBDE, and cites U.S. Environmental Protection Agency's (EPA's) Voluntary Children's Chemical Evaluation Program (VCCEP) and its work on DecaBDE with industry. My understanding is that this program has been discontinued. According to a July 2011, report from the EPA Office of Inspector General:

"The VCCEP pilot did not achieve its goals to design a process to assess and report on the safety of chemicals to children. The pilot's design did not allow for desired outcomes to be produced. Specifically, the pilot had a flawed chemical selection process and lacked an effective communication strategy. Programmatic effectiveness was hampered by industry partners who chose not to voluntarily collect and submit information, and EPA's decision not to exercise its regulatory authorities under the Toxic Substances Control Act to compel data collection. EPA has not demonstrated that it can achieve children's health goals with a voluntary program. The VCCEP is no longer operational, and the Agency has no plans to revive, replace, or terminate the program. As a result, the Agency is not meeting the intent of EO 13045, ChemRTK, or the VCCEP pilot, and there remains no readily understandable source of chemical exposure information that the general public can access to determine potential risks to children."

This report is available at the following link: http://www.epa.gov/oig/reports/2011/20110721-11-P-0379.pdf.

Again, I appreciate the opportunity to comment upon the NYS draft report on flame retardant safety.

Sincerely,
Catherine O'Dell, P.G.
Planner Principal
Water Assessment and Environmental Information Section
Environmental Analysis and Outcomes Division
CO:jab

Letter 3

Alex Stone, Sc.D. Washington Department of Ecology

Ms. Lavigne,

I am one of the authors of the Washington State Deca-BDE Alternatives Assessment and wished to provide three general comments on the NY State report from my admittedly hasty and cursory review of the NY State report. Unfortunately due to other work commitments and the fact that Washington has banned deca-BDE in several applications, I was unable to give the report as detailed a review I would like. In any case, I've attached my three general comments. Please let me know if you have any questions or concerns.

Sincerely,
Alex

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Comments on the NY State Deca-BDE Alternatives Assessment

Lack of emphasis on the persistence, bioaccumulation of degradation products of decaBDE: One of the deciding factors in the Washington decaBDE alternatives assessment was the identification that the degradation products from RDP are not persistent and have only limited if any bioaccumulation potential. Therefore even if the chemicals were of identical toxicity (WA made the determination that the toxicity of RDP and its degradation products were less than identified in this report), the long-term impacts of RDP degradation products compared with the PBT characteristics of the degradation products of decaBDE are an important factor in replacing decaBDE with a safer alternative like RDP. In addition, the report fails to emphasize the

importance of lower brominated PBDE species as degradation products in this evaluation. Another factor in the Washington assessment of alternatives to Deca-BDE was the persistence, bioaccumulation and toxicity of the penta and octaBDE congeners. As the report indicates, there is clear evidence of degradation to the octaBDE congeners and supporting evidence that degradation proceeds to lower brominated congeners as well. Although the toxicity of decaBDE might be debated by some, there is no question concerning the PBT effects of these lower molecular weight PBDE congeners. In addition, there is some concern about the toxicity of unique congeners and other oxygenated degradation products produced from these degradation processes as their toxicity has not been evaluated. Regardless, the PBT degradation products of decaBDE are an important factor in the decision by Washington to ban the use of decaBDE either in specific or all applications. This report should be updated to emphasize these considerations.

Concerns with some research: Although the New York Legislation requires the Task Force consider the European Union Risk Assessment and Addendum, the Task Force should also identify that considerable research has conducted on BDE-209 since the EU RA work was completed. For example, the EU addendum to the RA states that _*Date of Last Literature Search: January 2004 (environment). A small number of important papers published since then have also been reviewed.*' Although Washington used the EU RA report as a good foundational document, it realized that even in 2008 during its alternatives assessment, recent studies on decaBDE and BDE-209 were not included in the EU RA evaluation and conclusions. Therefore the EU RA conclusions had to be adapted based upon more recent work. This document should reflect the lack of recent data used in the EU RA and not emphasize conclusions that may no longer be valid. EPA recently released an updated decaBDE alternatives assessment based upon the most current research and hazard evaluation. The report should be updated to incorporate this information and greater emphasis should be placed upon this work for the conclusions reached and not depend as much upon a dated EU RA report.

Perceived Bias: The document appears to be biased toward a specific perspective. For example, emphasis is placed upon identifying the problems associated with various studies and evaluation of the potential impacts of BDE-209 upon human health and the environment. Terms such as _Some scientists believe…' are used throughout the document to summarize commonly held opinions on the concerns associated with BDE-209. Unless the document can quantify what is meant by _some', it suggests that the opinions that follow are in the minority while, in fact, they are most often the majority opinion of independent and unbiased scientific researchers. The specific terms and the way the information is presented provide an impression that the opinions that follow are not valid and should be dismissed. In addition, the document goes to great length to point out the defects associated with specific studies but fails to capture the big picture. It is important to address conflicting results using a weight-of-evidence approach. If you have ten studies that show a chemical is a problem and two that suggest it isn't, the weight-of-evidence approach identifies it is likely a problem exists. Even if the studies were not _perfect', the results are still conclusive. The only exception is the concern about bias associated with a study. Bias is a valid reason for eliminating a study from consideration. The long history of the Tobacco Institute has shown that science can be manipulated to get a desired result. If, however, there are no bias issues, even an imperfect study is important to the overall evaluation. This report appears to suggest that any potential faults with a study automatically invalidates its results but appears to be less reflective over studies that support a biased point of view. No study is perfect and the refusal to accept important conclusions from less than perfect studies is a serious limitation of the report. Lastly, the report repeatedly emphasizes that health and environmental effects are not _proven' with what appears to be 100% assurance. If a conclusion is not proven

with 100% assurance, it is often trivialized in the language used. Unfortunately science does not provide such assurance. There will always be variable and conflicting results which is why scientists typically use the weight-of-evidence approach to address this uncertainty. The document should be updated to remove this biased and emphasize the larger picture that the problems associated with decaBDE are the reason for several states to ban its use and for the manufacturers to stop importing or selling it in the U.S.

Letter 4
Hon. Robert K. Sweeney, New York State Assembly (11th District), Chair, Environmental Conservation Committee

THE ASSEMBLY
STATE OF NEW YORK
ALBANY

ROBERT K. SWEENEY
Assemblyman 11TH District

CHAIRMAN
Environmental Conservation

COMMITTEES
Education
Rules
Veterans' Affairs

July 30, 2012

Michelle Lavigne
New York State Department of Health
Bureau of Toxic Substance Assessment
Flanigan Square Room 300
547 River Street
Troy, New York 12180

Re: Draft Report of the New York State Task Force on Flame Retardant Safety

Dear Ms. Lavigne:

Eight years have passed since the creation of the Task Force, and in those eight years the amount of information available about flame retardants has increased significantly. Most importantly, the Environmental Protection Agency, after conducting a thorough review, entered into an agreement with manufacturers that will result in the cessation of the production and use of DecaBDE. Despite the importance of this agreement and the accompanying science, the Task Force's proposed report does not fully reflect recent scientific advances and seems intended to downplay concerns about health impacts associated with DecaBDE. For example, the EPA includes the following statement "Studies have been conducted in laboratory animals to gain a better understanding of the potential health risks of PBDEs. These studies have suggested potential concerns about liver toxicity, thyroid toxicity, developmental toxicity, and developmental neurotoxicity — especially about potential risks in children. The carcinogenic potential of some PBDEs have been studied. In a review of toxicological studies as part of a toxicological review for EPA's Integrated Risk Information System (U.S. EPA, 2006), EPA has found that the data for decabromodiphenyl ether (the commercial formulation composed mostly of BDE 209) support a finding of "suggestive evidence of carcinogenic potential" according to EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005). Instead of relying on this information, the Task Force includes the tepid statement "These studies have led some scientists to conclude that the continued use of decaBDE as a flame retardant may pose risks to human health and to the environment."

The Assembly has been proactive on the issue of flame retardants, and has passed legislation that subsequently became law concerning the use of TRIS (TCEP). In addition, the Assembly has also passed legislation that would codify the EPA's voluntary agreement and prohibit the production and use of DecaBDE. It is unfortunate that the

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E-MAIL: sweeney@assembly.state.ny.us

Task Force does not recognize this precedent and issue a stronger endorsement of such a ban.

In addition to the increased scrutiny in New York, the use of flame retardants is also receiving nationwide attention. The Chicago Tribune published a series of articles, available at www.chicagotribune.com/news/watchdog/flames, examining the activities of the flame retardant industry. In this series they identified a number of questionable tactics and studies by the flame retardant industry. For example, they cited a misleading study concerning the impact of DecaBDE on children's health by Dennis Paustenbach, a toxicologist and industrial hygenist. I urge you to read the series and take a new look at the proposed draft report.

The Chicago Tribune series is also credited with prompting California's Governor to revisit the State's flammability standards. In light of the reliance of the industry on California's standard, such a re-examination could radically change the evaluation of DecaBDE alternatives. I urge you to consider delaying the report until California has completed its process.

I would be interested in knowing more about the process used by the Task Force in creating the report, including what process, if any, was used to obtain the consent of all of the Task Force members prior to the issuance of the report.

I look forward to hearing from you and thank you for your consideration of my comments.

Very truly yours,

Robert K. Sweeney, Chair
Environmental Conservation Committee

RKS/mm

Letter 5

Bobbi Chase-Wilding, M.S., Clean and Healthy New York

Dear Michelle,

Attached please find comments on behalf of fifty organizations calling for the Taskforce on Flame Retardant Safety to reopen its report on decaBDE and alternatives and revise it, in light of clear cases of chemical industry bias in the report, and in light of the newly released Alternatives Assessment issued by the U.S. EPA's Design for the Environment program.

We look forward to a reply explaining how the Department of Health and the Taskforce will address these defects.

Best,
Bobbi

--
Bobbi Chase Wilding, MS
Deputy Director
Clean & Healthy New York

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-- Celebrating five years of promoting safer chemicals,
a sustainable economy and a healthier world --

VIA EMAIL

July 31, 2012

Michelle Lavigne
New York State Department of Health
Bureau of Toxic Substance Assessment
Flanigan Square Room 300
547 River Street
Troy, New York 12180

Re: Public Comment: Draft Report of the New York State Task Force on Flame Retardant Safety

Dear Ms. Lavigne,

The fifty organizations listed below are providing comments on the Draft Report of the New York State Taskforce Report on Flame Retardant Safety.

We find this draft deeply disturbing. If released as drafted, this report will severely damage the Department of Health's credibility. Shockingly, the report, almost seven years late of its 2005 deadline, misperceives the Legislature's charge and conflates industry perspective with an objective, science-based perspective. The Legislature did not charge the Task Force with creating a political document in which the chemical industry's viewpoint—backed by the research it paid for--consistently dilutes independent scientific and agency findings.

Moreover, the report fails to reach clear conclusions that identify hazards posed by decaBDE (and its breakdown PBDE congeners), and fails to explicitly conclude that alternatives presented are less-toxic, available, effective and affordable. The Taskforce must revise their report before publishing it as final, as there are significant problems that must be addressed for this report to be considered credible.

Our organizations take a back seat to no one—particularly the chemical manufacturers and their trade associations—when it comes to fire prevention and fire safety. The Department should not fall prey to the industry's argument that fire safety and chemical safety are mutually exclusive. Indeed, independent agencies have reviewed this issue and concluded that reducing or eliminating toxic exposures will not undermine fire safety. And as recent studies have noted, the real-world fire safety benefits of suffusing consumer products with toxic flame retardants do not exist.

Among the key problems with the report draft:

I. This report is significantly out of step with the tone and clarity in the independent, agency-drafted reports from Washington, Illinois, Maine, Minnesota and elsewhere. Those reports were released after the legislation setting up New York's taskforce was enacted in 2004, and those agencies were charged with very similar directives as the New York State Taskforce on Flame Retardant Safety. Those reports consistently explicitly stated that decaBDE could pose human health and environmental health hazards and clearly identified safer alternatives. Neither conclusion is evident in this report. Here are some of the findings stated in the Executive Summaries of state-drafted reports:

A. Washington: —Safer, technically feasible alternatives to the use of Deca-BDE in TVs, computers and residential upholstered furniture are available and meet applicable fire safety standards."[1]

B. Illinois:

1. —We find that DecaBDE is bioaccumulating in the environment, and levels are increasing in some types of samples."[2]

2. —We now have additional evidence that DecaBDE, certain DecaBDE breakdown products and other PBDES can cause thyroid, reproductive/developmental, and neurological effects; although there is still uncertainty about DecaBDE's role in these effects, our level of uncertainty has decreased from the 2006 report, and further justifies an in-depth evaluation of potential alternative flame retardants for products still using DecaBDE."[3]

C. Maine: —Safer alternatives are available for TV cabinets and textiles, the applications that consume most decaBDE. In the case of textiles, alternatives that do not require the use of chemical flame retardants already are widely employed in the marketplace. In the case of TVs, the use of safer alternatives to decaBDE will require manufacturers to shift from

[1] Department of Ecology, State of Washington, and Washington State Department of Health, Alternatives to Deca-BDE in Televisions and Computers and Residential Upholstered Furniture, December 2008. https://fortress.wa.gov/ecy/publications/publications/0907041.pdf

[2] Illinois Environmental Protection Agency, *DecaBDE Study: A Review of Available Scientific Research*, January 2006. http://www.epa.state.il.us/reports/decabde-study/available-research-review.pdf

[3] Illinois Environmental Protection Agency, *Report on Alternatives to the Flame Retardant DecaBDE: Evaluation of Toxicity, Availability, Affordability, and Fire Safety Issues*, March 2007. http://www.epa.state.il.us/reports/decabde-study/decabde-alternatives.pdf

using cabinets made of high impact polystyrene (HIPS) to other types of plastic that can be treated to meet flammability standards using phosphorus compounds such as resorcinol bis diphenyl phosphate (RDP). RDP presents a significantly lower threat to the environment and human health than decaBDE."[4]

D. Minnesota: "Effective alternatives for achieving flame retardancy appear to be available for most current Deca-BDE applications. In general, flammability requirements can be met using various strategies that include: using an alternative chemical flame retardant; switching to the use of inherently non-flammable materials; and re-designing the product."[5]

II. Discussion about fire safety, fire deaths, and use of chemical flame retardants in fire prevention is outside the Taskforce's scope. Therefore, references in the Executive Summary and the Section 1.4, "Fire Safety in New York State," should be removed. This is particularly important given the other matters the Taskforce did not include in this report, including decaBDE contamination of the environment due to handling of electronic waste and toxic combustion byproducts when decaBDE is burned. Should such a section remain, it is vital to present an accurate state of science regarding the use of chemical additives as flame retardants. Recently, scientists have refuted the chemical industry's use of studies around the effectiveness of chemical additives, particularly with regard to residential products. In fact, the Consumer Product Safety Commission testified before the U.S. Senate that chemical additives to consumer products do not provide any meaningful fire safety protection in real-world settings. Further, the State of California is currently re-drafting their furniture flammability standards such that chemicals will not be needed to achieve the safety standard.

III. In the New York State Taskforce report, most scientific discussions are slanted toward chemical makers, and often favor industry-funded studies while disparaging independently-funded studies. The most egregious example of this is the discussion surrounding neurotoxicity. Several pages are devoted to the explanation of a recent industry-funded study authored by Dr. Biesemeier, which found no neurotoxicity, and this 2011 study is used to discredit numerous independent studies that found neurological impacts. This section must be redrafted to present an unbiased evaluation of all studies, and should objectively note all cases of financial relationships which exist between study authors and flame retardant makers.

IV. The Taskforce failed to fully evaluate the health and environmental impacts of other polybrominated diphenyl ethers as a result of decaBDE breaking down, despite reviewing and providing evidence that decaBDE debrominates in nature. One study found that decaBDE debrominates to pentaBDE, which is considered to be the most potent congener. This has been an appropriate and important part of other evaluations of decaBDE and its long-term ongoing effects. It also fails to consider the environmental and health impacts of combustion of decaBDE in its review of potential hazards.

V. There are numerous instances in which statements are made regarding lack of scientific consensus, when in fact there is broad agreement among independent researchers without a financial tie to flame retardant makers. If in fact the Taskforce doesn't agree upon how to report the findings, this statement should be made instead of simply using a blanket statement of "lack of scientific consensus," or a dismissal of areas of broad agreement with phrases like, "some scientists believe…"

VI. The Taskforce failed to meet its charge regarding the determination of the availability of **safer** alternatives to decaBDE. Extensive work has been done in this regard by the U.S. EPA and elsewhere. While the body of the document appears to imply that the alternative methods of

[4] Maine Department of Environmental Protection and Maine Center for Disease Control & Prevention, *Brominated Flame Retardants: Third annual report to the Maine Legislature*, January 2007. http://www.maine.gov/dep/waste/publications/legislativereports/documents/finalrptjan07.doc

[5] Minnesota Pollution Control Agency, Decabromodiphenyl Ether (Deca-BDE): A Report to the Minnesota Legislature, January 15, 2008. http://www.pca.state.mn.us/index.php/view-document.html?gid=3942

achieving flame retardancy are safer, this is never stated explicitly. In fact, it is undercut by the conclusion in the Executive Summary, which instead codifies uncertainty and doubt: "Regardless of the conclusions that might be drawn about the amount of debromination and the level of health risk, the voluntary phase out of production, importation, and sales for most decaBDE uses… will limit the amount of decaBDE and any debromination products that can be released to the environment in the future." A statement regarding the relative safety of alternatives must be clearly made in both the Executive Summary and in the final conclusion of the report. In the event that the Taskforce fails to reach conclusions, this should be stated explicitly.

Further, the majority of alternatives to decaBDE were not included in this report. An explanation of why other chemicals and materials were not considered (were they not safer, affordable, available options?) should be made.

The U.S. EPA just released for public comment its extensive assessment on alternatives to decaBDE, in which EPA categorizes decaBDE as high for neurotoxicity and developmental toxicity, very high for persistence and high for bioaccumulation potential. The Taskforce's final report should not be considered complete until this new, superior information on decaBDE and its alternatives is included. Such a delay would not be unprecedented: At the urging of the chemical industry, the Taskforce already delayed finalization of the draft report until an industry-funded study on neurotoxicity was published. Since the significant delay in favor of a single, chemical industry-conducted study was permissible, it should be no problem for the taskforce to await information that is highly relevant to the as-yet incomplete fulfillment of its charge.

One primary excuse that the Taskforce report gives to avoid reaching conclusions is that the EPA reached a voluntary agreement with the leading three flame retardant makers of decaBDE, in which their US decaBDE production is phased out and importation of decaBDE itself is phased out. However, Americans will still be exposed to decaBDE if overseas decaBDE makers sell their chemical to product makers who add it to products made overseas. There is no prohibition in the federal phaseout agreement of final products containing decaBDE from being imported into the United States and sold through retail venues. Therefore, this report must remain germane to ongoing discussions about how states may act to protect their residents from toxic chemicals.

We are especially concerned about the favoring of chemical industry arguments or industry-funded studies in light of recent investigations by the Chicago Tribune, which showed ways in which flame retardant chemical makers have misrepresented fire safety studies in foam (according to Vytenis Babrauskas, the study author) and have used dubious data to justify use of flame retardant chemicals in electronics housing. The problems posed by flame retardant chemicals and the manipulations of evidence by chemical makers have also been probed by not one, but two July 2012 U.S. Senate hearings. Given that manufacturers and their trade associations unrepentantly misuse other studies and reports, it is even more important to ensure that this report is accurate. As it stands, we expect that the chemical industry will gloss over the details and focus on the summaries, using this report as a means of defending the ongoing use of decaBDE around the world. The Taskforce and the Department should not be complicit in this by putting the stamp of approval on the industry's sponsored research.

With its overarching bias toward industry-funded science, its failure to meet the legislative directive by explicitly reaching conclusions either about decaBDE's toxicity or the ready availability of safer, effective alternative means of achieving fire safety, it is critical for this report to be revised to reflect a more unbiased, accurate assessment of decaBDE and its alternatives. This is particularly true because of the report's ongoing relevance to discussions in New York and elsewhere about the public health implications and questionable utility of these chemicals, and the newly-released EPA data.

Ultimately, this report is a product of the NYS Department of Health, which states as its mission to "protect, improve and promote the health, productivity and well being of all New Yorkers." As it stands, this report fails to live up to the Department's mission, and the stakes are especially high given children's disproportionate contact with the chemicals and their vulnerability to the effects. The people of New York State deserve a report that puts public health first, not the wishes of two flame retardant chemical industry

taskforce members. The Taskforce must make revisions to correct the items above before issuing its final report. It is not acceptable simply to include these comments and publish the report as-is.

Sincerely,

Bobbi Chase Wilding, Deputy Director
Clean and Healthy New York
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Albany, NY 12207
518-708-3875

Appendix 5. Tables for Section 1.4 (Fire Safety in New York)

Some fire performance characteristics of materials can be controlled and some cannot. Some fire performance characteristics of materials that can be controlled are 1) ignition resistance, 2) flame spread, 3) heat output and 4) smoke production. These are defined in Table 1:

Table 1: Controllable Fire Performance Characteristics of Materials

Material Characteristic	Definition
Ignition resistance	The ability of a material to resist or delay catching on fire from a heat source.
Flame spread	The distribution of flame over the surface of a material away from an open flame ignition source measured over time or against a standard reference material.
Heat output	The amount of heat produced by a burning material per unit of time (heat release rate), or the total amount of heat produced by a material as it is totally consumed by fire.
Smoke production	The amount of smoke released by a burning material as measured by how much an area‘s atmosphere is visually obscured over time.

Table 2: New York State Residential Code Fire Performance Properties of Building Materials, Building Components and Products in One and Two Family Dwellings and in Multiple Single Family Dwellings (Townhouses)

Material/Product/Component	Fire Performance Property
Foam plastic insulation	Flame spread; Smoke developed
Insulation materials other than foam plastic	Flame spread; Smoke developed
Exposed attic insulation	Flame spread; Smoke developed Critical radiant flux
Cellulose loose fill insulation[1]	Critical radiant flux; Ignition resistance[1]
Foam plastic trim	Minimum density; Flame spread
Wall and ceiling finishes	Flame spread; Smoke developed

[1] Specific requirements established by United States Consumer Product Safety Commission (16 CFR 1209) and referenced by the Residential Code of New York State.

Table 3: Selected NYS Building Code and Fire Code Fire Performance Requirements of Building Materials, Building Components and Products in Buildings other than One and Two Family Dwellings and Multiple Single Family Dwellings (Townhouses)

Material/Product/ Component	Occupancy Group	Fire Performance Property
Interior wall and ceiling finishes	All	Flame spread Smoke development
Curtains, drapes, and decorative materials hanging from walls and ceilings	Assembly Educational Institutional Residential (transient) Residential – Dormitories	Flame resistance
Foam Plastic insulation	All	Flame spread Smoke development
Light transmitting plastics	All	Self-ignition temperature Smoke development index
Foam plastic materials	Assembly – decorative purposes, stage scenery or exhibit booths	Maximum heat release rate
Upholstered furniture	Institutional (medical/surgical/psychiatric/ nursing or custodial care)	Cigarette ignition resistance Heat release
Mattresses	Institutional (medical/surgical/psychiatric/ nursing or custodial care)	Heat release
Upholstered furniture	Institutional (board and care)	Heat release
Mattresses	Institutional (Board and Care)	Cigarette ignition resistance Heat release
Upholstered furniture	Institutional (detention and correction)	Heat release
Mattresses[2]	Institutional (detention and correction)	Cigarette ignition resistance[1] Heat release

[1]Specific requirements established by United States Consumer Product Safety Commission (16 CFR 1633) and referenced by the Fire Code of New York State.

Table 4: Selected Fire Performance Requirements Established by the Federal Government for Clothing, Furnishings, Building Materials, and Transportation

Material/Product/ Component	Federal Agency	Federal Code Section	Fire Performance Property
Clothing textiles	Consumer Product Safety Commission	16 CFR 1610	Burning rate
Children‘s sleepwear sizes 0–6X (other than tight fitting sleepwear)	Consumer Product Safety Commission	16 CFR 1615	Flame resistance
Children‘s sleepwear, sizes 7–14 (other than tight fitting sleepwear)	Consumer Product Safety Commission	16 CFR 1616	Flame resistance
Carpets, rugs	Consumer Product Safety Commission	16 CFR 1630 16 CFR 1631	Ignition resistance
Mattresses, mattress pads	Consumer Product Safety Commission	16 CFR 1632 16 CFR 1633	Cigarette ignition resistance Open flame resistance, heat release
Cellulose Insulation	Consumer Product Safety Commission	16 CFR 1209	Flame resistance
Motor vehicle interiors	National Highway Traffic Safety Administration	49 CFR 571	Burn resistance
Passenger rail car and locomotive cab interiors	Federal Railroad Administration	49 CFR 238	Flammability Smoke emission
Aircraft passenger , baggage and cargo compartments; thermal/acoustic insulation materials	Federal Aviation Administration	14 CFR 25	Flame propagation Flame penetration

Table 5: Selected Voluntary Industry Standards with Fire Performance Requirements for Products and Materials

Product/Material	Standard Issuer	Fire Performance Property
Upholstered furniture	Upholstered Furniture Action Council	Standard for upholstered furniture resistance to ignition from smoldering cigarettes.
Plastic Materials for Parts in Devices and Appliances	Underwriters Laboratories (UL 94)	Tests for flammability of polymeric materials used in parts. Serves as a preliminary indication of acceptability for flammability.
Polymeric Materials – Products, Electrical Products, Printed Wiring Boards, Flexible Interconnects	Underwriters Laboratories (UL 746 A through F)	Standard tests for physical, electrical, flammability, thermal, and other properties of materials for manufacturers, molders, the end-product manufacturers, safety engineers, and others.
Appliance Wiring Material	Underwriters Laboratories (UL 758)	Requirements for appliance wiring (single insulated conductors, multi-conductor cables, optical fibers, individual insulated conductors, and fiber optics used as components in multi-conductor cables).
Electrical Wires, Cables, and Flexible Cords	Underwriters Laboratories (UL 1581)	Specifications, including flammability, for wire and cable standards.
Electrical and Optical-Fiber Cables	Underwriters Laboratories (UL 1685)	Flame exposure fire test for determining cable damage and smoke release from electrical and optical-fiber cables.
Small Polymeric Component Materials	Underwriters Laboratories (UL 1694)	Needle-flame test which simulates the effect of small flames from fault conditions within equipment to assess fire hazard.
Audio/Video and Musical Instrument Apparatus for Household, Commercial, and Similar General Use	Underwriters Laboratories (UL 6500)	Requirements for electronics for receiving, generating, recording or reproducing audio, video and associated signals including resistance to fire of electrical components, mechanical parts and enclosures.

Appendix 6. List of Reports on Decabromodiphenyl Ether and Alternatives

Organization/Agency	Title	Year of Publication*
United States Environmental Protection Agency (US EPA)	An Alternatives Assessment for the Flame Retardant Decabromodiphenyl Ether (DecaBDE)	2012
United Kingdom and Environment Agency	Proposal for Identification of a PBT/vPvB Substance. Bis(pentabromophenyl)ether (decabromodiphenyl ether; decaBDE)	2012
United States Environmental Protection Agency (US EPA)	An Exposure Assessment of Polybrominated Diphenyl Ethers (Final Report)	2010
Environment Agency	Environmental Risk Evaluation report: Decabromodiphenyl Ether (CAS no. 1163-19-5)	2009
Albemarle Corporation/Poland Institute of Public Health and Environmental Protection	Toxicology and Human Health Assessment of Decabromodiphenyl Ether	2009
Michigan Department of Environmental Quality	Polybrominated Diphenyl Ethers: A Scientific Review with Risks and Recommendations	2008
Minnesota Pollution Control Agency	Decabromodiphenyl Ether (Deca-BDE): A Report to the Minnesota Legislature	2008
United States Environmental Protection Agency (US EPA)	Flame Retardants in Printed Circuit Boards. Partnership to Evaluate Flame Retardants in Printed Circuit Boards	2008
United States Environmental Protection Agency (US EPA)	Integrated Risk Information System Summary Information on 2,2',3,3',4,4',5,5',6,6'-Decabromodiphenyl Ether (BDE-209) (CASRN 1163-19-5)	2008
United States Environmental Protection Agency (US EPA)	Toxicological Review of Decabromodiphenyl Ether (BDE-209). In Support of Summary Information on the Integrated Risk Information System	2008
Washington State Departments of Ecology and (Health WDE/WDOH)	Alternatives to Deca-BDE in Televisions and Computers and Residential Upholstered Furniture	2008
Clean Production Action (CPA)	The Green Screen for Safer Chemicals: Evaluating Flame Retardants for TV Enclosures	2007
Danish Environmental Protection Agency (DEPA)	Health and Environmental Assessment of Alternatives to Deca-BDE in Electrical and Electronic Equipment	2007
European Chemicals Bureau (ECB)	Review on Production Processes of Decabromodiphenyl ether (DecaBDE) used in Polymeric Applications in Electrical and Electronic Beguilement, and Assessment of the Availability of Potential Alternatives to DecaBDE	2007
Illinois Environmental Protection Agency (IEPA)	Report on Alternatives to the Flame Retardant DecaBDE: Evaluation of Toxicity, Availability,	2007

Organization/Agency	Title	Year of Publication*
	Affordability, and Fire Safety Issues. A Report to the Governor and the General Assembly.	
Karlsruhe Research Centre (KRC)	Halogen-free Flame Retardants in E & E Applications. A Growing Toolbox of Materials is Becoming Available	2007
Maine Department of Environmental Protection and Maine Center for Disease Control & Prevention (MDEP/MCDCP)	Brominated Flame Retardants. Third Annual Report to the Maine Legislature	2007
Danish Environmental Protection Agency (DEPA)	Deca-BDE and Alternatives in Electrical and Electronic Equipment	2006
Illinois Environmental Protection Agency (IEPA)	A Report to the General Assembly and the Governor in Response to Public Act 94-100. DecaBDE Study: A Review of Available Scientific Research	2006
Swedish Chemicals Inspectorate (SCI)	Cost Benefit Analysis Model for Fire Safety Methodology and TV (DecaBDE) Case Study	2006
United States Environmental Protection Agency (US EPA)	Polybrominated Diphenyl Ethers (PBDEs) Project Plan	2006
Washington State Departments of Ecology and (Health WDE/WDOH)	Polybrominated Diphenyl Ether (PBDE). Chemical Action Plan: Final Plan	2006
Swedish Chemicals Inspectorate (SCI)	Survey and Technical Assessment of Alternatives to Decabromodiphenyl Ether (decaBDE) in Plastics	2005
Lowell Center for Sustainable Production (LCSP)	Decabromodiphenylether: An Investigation of Non-Halogen Substitutes in Electronic Enclosure and Textile Applications	2005
Toxic Use Reduction Institute (TURI)	Alternatives Assessment for Toxics Use Reduction: A Survey of Methods and Tools. Methods and Policy Report No. 23	2005
Clean Production Action (CPA)	Brominated Flame Retardants in Dust on Computers: The Case for Safer Chemicals and Better Computer Design	2004
Washington State Departments of Ecology and (Health WDE/WDOH)	Washington State Polybrominated Diphenyl Ether (PBDE) Chemical Action Plan: Interim Plan	2004
European Chemicals Bureau (ECB), Institute for Health and Consumer Protection, European Commission Joint Research Centre	European Union Risk Assessment Report: Bis(pentabromophenyl) ether	2002
University of Massachusetts Lowell/ Massachusetts Toxic Use Reduction Institute	Environmental, Health and Safety Issues in the Coated Wire and Cable Industry Technical Report No. 51	2002

Organization/Agency	Title	Year of Publication*
(UML/MTURI)		
German Federal Environmental Agency (GFEA)	Substituting Environmentally Relevant Flame Retardants: Assessment Fundamentals	2000
Danish Environmental Protection Agency (DEPA)	Brominated Flame Retardants Substance Flow Analysis and Assessment of Alternatives	1999

*Full citations are available in Section 4 (References) of this document.

Appendix 7. State Legislative Initiatives on Decabromodiphenyl Ether

Source: University of Massachusetts Lowell. 2011. US State Level Chemicals Policy Database. Accessed (June 23, 2011) online at http://www.chemicalspolicy.org/uslegislationsearch.php.

1. Enacted Legislation Involving Decabromodiphenyl Ether

Name	**Supporting the Phase Out of Production and Importation of Decabromodiphenyl Ether in the United States**
State	Hawaii
Region	Pacific
Citation	S.R. 107, 25th Leg., Reg. Sess. (Haw. 2010); H.R. 165, 25th Leg. Reg. Sess. (Haw. 2010).
Status	Enacted—2010
Description	Supports the industry phase out of production and importation of decaBDE in the U.S. Encourages the U.S. EPA to continue its efforts to encourage the remaining minor importers of decaBDE to end their importation of decaBDE into the U.S.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Brominated Flame Retardant Prevention Act**
State	Illinois
Region	Midwest
Citation	H.B. 2572, 94th Gen. Assemb., Reg. Sess. (Ill. 2005).
Status	Enacted—2005
Description	Prohibits the manufacture, processing, or distribution of products or flame retardant parts of a product containing more than one tenth of 1% penta-BDE or octa-BDE. Directs the Illinois Environmental Protection Agency to review current literature on the health impacts of and alternatives available to deca-BDE by 2006 and submit this report to the Governor.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Illinois Governor's Letter Requesting Follow-up Study of Deca**
State	Illinois
Region	Midwest
Citation	Letter from Rod R. Blagojevich, Illinois Governor, to Doug Scott, Director of Illinois Environmental Protection Agency (Mar. 3, 2006).
Status	Enacted—2006
Description	Instructs the Illinois EPA to conduct a follow-up study to answer critical questions that remain about the environmental and health effects of DecaBDE.
Category	Data Collection
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**An Act to Clarify Maine's Phaseout of Polybrominated Diphenyl Ethers**
State	Maine
Region	Northeast

Citation	L.D. 1568, 124th Leg., 2nd Reg. Sess. (Me. 2009).
Status	Enacted—2010
Description	Prohibits the manufacture and sale of shipping pallets or any product manufactured from recycled shipping pallets containing decaBDE. Permits the Department of Environmental Protection to restrict the use of other flame retardants in plastic shipping pallets if the flame retardant is harmful to the public health and the environment and a safer alternative to the flame retardant is available. Requires that decaBDE be replaced with safer alternatives. Prohibits the replacement of decaBDE with a chemical alternative that is a persistent, bioaccumulative, and toxic chemical or another brominated or chlorinated flame retardant. Permits the Department to supervise an alternatives assessment study to determine the availability of safer alternatives to the use of decaBDE in shipping pallets.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Restrictions on Sale and Distribution of Brominated Flame Retardants**
State	Maine
Region	Northeast
Citation	Me. Rev. Stat. Ann. tit. 38, § 1609 (2008).
Status	Enacted—Adopted: 2003; Amended: 2007, 2009
Description	Prohibits the sale or distribution of a product containing more than 0.1% of the "penta" or "octa" mixtures of polybrominated diphenyl ethers. Restricts the manufacturing and sale of any mattress, mattress pad, upholstered furniture for indoor use, television or computer that has a plastic housing or contains plastic fibers with the "deca" mixture of polybrominated diphenyl ethers. Permits the Commissioner of Environmental Protection to adopt rules to prohibit the manufacture, sale or distribution of any other flame retardant in similar products.
Category	Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Concerning Environment - Decabrominated Diphenyl Ether - Prohibitions**
State	Maryland
Region	Northeast
Citation	S.B. 556, 427th Gen. Assemb., Reg. Sess. (Md. 2010).
Status	Enacted—2010
Description	Phases out the use of decaBDE in products. Prohibits the manufacture, lease, sale, or distribution for sale or lease of mattresses, upholstered furniture designed for residential use, and electrical or electronic equipment containing decaBDE by 2010. Prohibits of manufacture, lease, sale, or distribution for sale or lease any product that contains decaBDE by 2012. Prohibits the manufacture, lease, sale, or distribution for sale or lease of transportation equipment, military equipment, or components of transportation or military equipment by 2013.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Pentabrominated and Octabrominated Diphenyl Ether-Prohibition**

State	Maryland
Region	Northeast
Citation	Md. Code Ann., Envir. §§ 1201-1205 (2008).
Status	Enacted—2005
Description	Prohibits the manufacture, processing, sale, or distribution of a new product or flame-retardant part of a new product that contains more than a specified amount of penta- or octa-brominated diphenyl ether. Requires the Department of the Environment, in conjunction with interested parties, to report to the Legislature on the use of decabrominated diphenyl ether (decaBDE) in products sold in the state, any data available on the human body burden or environmental occurrence of decaBDE, recommendations regarding the use, sale, and disposal of products containing decaBDE, and any other recommendations to further protection of public health and the environment from decaBDE.
Category	Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Products Containing Polybrominated Diphenyl Ether**
State	Minnesota
Region	Midwest
Citation	Minn. Stat. §§ 325E.385-325E.388 (2008).
Status	Enacted—2007
Description	Prohibits the manufacturing, processing, or distribution of a product or flame-retardant part of a product containing certain concentrations of pentabromodiphenyl ether or octabromodiphenyl ether. Requires the Commissioner of the Pollution Control Agency to review uses of decabromodiphenyl ether, availability of technically feasible and safer alternatives, fire safety, and any evidence regarding the potential harm to public health and the environment posed by commercial decabromodiphenyl ether and the alternatives. Requires that equipment, supplies, and other products that do not contain polybrominated diphenyl ethers be made available to all state agencies.
Categories	Alternatives Assessment, Data Collection, Environmentally Preferable Purchasing, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Prohibition Against Brominated Flame Retardants**
State	New York
Region	Northeast
Citation	N.Y. Envtl. Conserv. Law § 37-0111 (2008).
Status	Enacted—2004
Description	Prohibits the use of pentabrominated diphenyl ether or octabrominated diphenyl ether in any product or as use as a flame retardant. Creates the state Task Force on flame retardant safety to, at a minimum, review and report on relevant studies, risk assessments, findings, or rulings in connection with the flame retardant decabrominated diphenyl ether and evaluate the availability of safer alternatives to decabrominated diphenyl ether.
Category	Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**A Bill for an Act Relating to Decabrominated Diphenyl Ether**
State	Oregon
Region	West
Citation	S.B. 596, 75th Leg. Assemb., Reg. Sess. (Or. 2009).
Status	Enacted—2009
Description	Prohibits introduction or delivery for introduction into commerce any product containing more than one-tenth of one percent by mass of decaBDE.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Relating to Brominated Flame Retardants; Creating New Provisions; and Amending ORS 453.005, 453.085 and 453.995**
State	Oregon
Region	West
Citation	S.B. 962, 73rd Leg. Assemb., Reg. Sess. (Or. 2005).
Status	Enacted—2005
Description	Restricts the introduction into commerce of any product containing pentabrominated diphenyl ether or octabrominated diphenyl ether.
Category	Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Hazardous Chemicals—Contamination of Breast Milk and the Environment**
State	Rhode Island
Region	Northeast
Citation	R.I. Gen. Laws §§ 23-13.4-1-23-13.4-6 (2008).
Status	Enacted—2006
Description	Restricts the manufacturing or distribution of flame retardants containing pentaBDE or octaBDE. Requires study on decaBDE.
Category	Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Brominated Flame Retardants**
State	Vermont
Region	Northeast
Citation	H. 444, 2009-2010 Leg., Reg. Sess. (Vt. 2009).
Status	Enacted—2009
Description	Prohibits the sale or distribution of a product containing octaBDE or pentaBDE. Prohibits the manufacture, sale, or distribution of a mattress or mattress pad, upholstered furniture intended for indoor use in a home or other residential occupancy, or a television or computer with plastic housing containing decaBDE. Prohibits a manufacturer from replacing decaBDE with a chemical that is classified as "known to be a human carcinogen" or "reasonably anticipated to be a human carcinogen" or is identified by the U.S. EPA as causing birth defects, hormone disruption, or harm to reproduction or development.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)

Types	Electronics Equipment, Furniture

Name	**Polybrominated Diphenyl Ethers—Flame Retardants**
State	Washington
Region	West
Citation	Wash. Rev. Code Ann. §§ 70.76.005-70.76.110 (2008).
Status	Enacted—2007
Description	Restricts the sale of noncomestible products containing PBDEs and mattresses containing commercial decaBDE. Requires the Department of Ecology and the Department of Health to study alternatives to PBDEs and decaBDEs. Restricts the sale of televisions, computers, and residential upholstered furniture containing decaBDE as a result of the Departments' finding that safer and technically feasible alternatives that meet fire safety standards are available.
Categories	Alternatives Assessment, Environmentally Preferable Purchasing, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

2. Proposed Legislation Involving Decabromodiphenyl Ether

Name	**An Act Relating to Flame Retardants and the Manufacture, Sale and Distribution of Products Containing Flame Retardants; Relating to Bioaccumulative Toxic Chemicals; and Providing for an Effective Date**
State	Alaska
Region	Arctic
Citation	H.B. 271, 25th Leg., 2nd Sess. (Alaska 2008).
Status	Proposed—January 15, 2008
	Referred to Labor and Commerce Committee January 15, 2008
Description	Prohibits the sale of a product that contains more than .1% by mass of pentaBDE or octaBDE. Restricts the sale of a mattress, mattress pad or upholstered furniture containing more than .1% decaBDE. Restricts the sale of electronic products with plastic housing that contains more than .1% of decaBDE. Will develop a program to help retailers determine what products violate the restrictions. A list of PBTs will be developed and updated biannually.
Categories	Multiple Chemical Policy, Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Persistent Bioaccumulative Toxins (PBTs), Polybrominated diphenyl ethers (PBDEs)
Type	Furniture

Name	**An Act Relating to Flame Retardants and to the Manufacture, Sale, and Distribution of Products Containing Flame Retardants; Relating to Bioaccumulative Toxic Chemicals; and Providing for an Effective Date**
State	Alaska
Region	Arctic
Citation	S.B. 295, 26th Leg., 2nd Sess. (Alaska 2010); H.B. 385, 26th Leg., 2nd Sess. (Alaska 2010).
Status	Proposed—February 24, 2010 (S.B. 295); February 23, 2010 (H.B. 385)

	Referred to Finance March 23, 2010 (S.B. 295); Referred to Labor and Commerce February 23, 2010 (H.B. 385)
Description	Prohibits the manufacture, sale, or distribution of a product that contains pentaBDE, octaBDE, or a combination of pentaBDE and octaBDE. Prohibits the manufacture, sale, or distribution of a mattress, mattress pad, or upholstered furniture with textile components containing decaBDE. Prohibits the manufacture, sale, or distribution of an electronic product with plastic housing containing decaBDE. Permits the Department of Environmental Conservation to prohibit the manufacture, sale, or distribution of a product that contains a flame retardant that is not a brominated flame retardant if it determines that the flame retardant is harmful to public health or the environment and an alternative to the flame retardant exists, is safer for the public health or the environment, and is available on a nationwide basis. Requires the Department to review the hazards and risks of brominated flame retardants and possible alternatives to brominated flame retardants. Requires the Department, in consultation with the Department
Categories	Alternatives Assessment, Multiple Chemical Policy, Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Persistent Bioaccumulative Toxins (PBTs), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Concerning Toxic Substances**
State	Connecticut
Region	Northeast
Citation	H.B. 5805, 2008 Gen. Assemb., Feb. Sess. (Conn. 2008).
Status	Proposed—February 29, 2008
	Referred by House to Committee on Judiciary April 9, 2008
Description	Phases out the use of alkylphenol ethoxylates in cleaning products, laundry products, and personal care products. Prohibits the sale or distribution of a product containing pentaBDE or octaBDE. Prohibits the manufacture, sale, or distribution of a mattress, mattress pad, upholstered furniture intended for indoor residential or office use, televisions, or computers containing decaBDE. If the Commissioner of Environmental Protection determines that a flame retardant is harmful to public health and the environment, an alternative is nationally available, and the alternative meets applicable fire safety standards, permits the Commissioner to adopt regulations to prohibits the manufacture, sale, or distribution of products containing such flame retardants. Authorizes the Commissioner to take part in the creation and implementation of a regional, multistate organization to help carry out the bill's requirements.
Category	Single Chemical Restriction
Chemicals	Alkylphenol, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Cleaning Products, Electronics Equipment, Furniture

Name	**An Act Phasing Out the Use of Polybrominated Diphenyl Ethers**
State	Connecticut
Region	Northeast
Citation	S.B. 919, 2009 Gen. Assemb., Jan. Sess. (Conn. 2009).
Status	Proposed—February 11, 2009
	Motion failed in Senate Committee on Public Safety and Security April 17, 2009

Description	Prohibits the sale or distribution of a product containing more than fifty parts per million of pentaBDE or octaBDE. Prohibits the manufacture, sale or distribution of a mattress or mattress pad, upholstered furniture intended for indoor residential or office use, or electronic devices that have a plastic housing containing decaBDE. Permits the Commissioner of Environmental Protection to adopt regulations to prohibit the manufacture, sale or distribution of products containing harmful flame retardants if a safer alternative exists.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Phasing Out the Use of Polybrominated Diphenyl Ethers**
State	Connecticut
Region	Northeast
Citation	H.B. 5477, 2009 Gen. Assemb., Jan. Sess. (Conn. 2009).
Status	Proposed—January 22, 2009
	Referred to Joint Committee on Environment January 22, 2009
Description	Phases out the sale or distribution of products containing pentaBDE or octaBDE. Phases out the manufacture and sale of computers, televisions, furniture, textiles or mattresses containing decaBDE.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Human and Environmental Health Protection Amendment Act of 2009**
State	District of Columbia
Region	Northeast
Citation	Council of the District of Columbia, Bill No. B18-0521 (Nov. 6, 2009).
Status	Proposed—November 6, 2009
	Referred to Government Operations and the Environment November 6, 2009
Description	Prohibits the manufacture and sale of any product intended for use by children under the age of 6 that contains bisphenol-A or phthalates. Prohibits the manufacture and sale of any product containing pentBDE or octaBDE. Prohibits the manufacture or sale of a mattress or mattress pad containing decaBDE, upholstered furniture intended for indoor use in a home containing decaBDE, or a television, monitor, or computer that has a plastic housing containing decaBDE. Phases out the use of perchloroethylene in dry cleaning. Prohibits the manufacture and sale of products containing propoxur, permethrin, atrazine, and synthetic exfoliants.
Categories	Product Categories, Single Chemical Restriction
Chemicals	benzyl butyl phthalate (BBP), Bisphenol-A, decabromodiphenyl ether (decaBDE), di-(2-ethylhexyl) phthalate (DEHP), di-n-octyl phthalate (DNOP), dibutyl phthalate (DBP), diisodecyl phthalate (DIDP), diisononyl phthalate (DINP), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Perchloroethylene, Phthalates, Polybrominated diphenyl ethers (PBDEs)
Types	Children's Products, Electronics Equipment, Furniture

Name	**A Bill for an Act Related to Brominated Flame Retardants**
State	Hawaii

Region	Pacific
Citation	H.B. 1881, 2010 Leg., Reg. Sess. (Haw. 2010).
Status	Proposed—January 20, 2010
	Referred to Committee on Health, Committee on Judiciary, Committee on Consumer Protection and Commerce, and Committee on Finance January 20, 2010
Description	Prohibits the sale or distribution of a product containing octaBDE or pentaBDE. Prohibits the manufacture, sale, or distribution of a mattress or mattress pad, upholstered furniture, televisions, or computers containing decaBDE. Prohibits manufacturers from replacing decaBDE with a chemical that classified as a "known" or "reasonably anticipated" human carcinogen on the most recent report on carcinogens by the National Toxicology Program, classified as "carcinogenic to humans" or "likely to be carcinogenic to human" in the US EPA's most recent list of chemicals evaluated for carcinogenic potential, or identified by US EPA as causing birth defects, hormone disruption, or ham to reproduction or development.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Type	Furniture

Name	**A Bill for an Act Relating to Toxic Chemicals**
State	Hawaii
Region	Pacific
Citation	S.B. 814, 25th Leg., Reg. Sess. (Haw. 2009); H.B. 799, 25th Leg., Reg. Sess. (Haw. 2009)
Status	Proposed—January 23, 2009 (S.B. 814); January 26, 2009 (H.B. 799)
	Referred to Committees on Commerce and Consumer Protection and Judiciary and Government Operations January 30, 2009 (S.B. 814); Referred to Committees on Health, Consumer Protection and Commerce, Judiciary, and Finance January 28, 2009 (H.B. 799)
Description	Prohibits the manufactuer, sale, or distribution of products containing PBDEs.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**An Act Related to Polybrominated Diphenyl Ethers**
State	Hawaii
Region	Pacific
Citation	S.B. 1045, 24th Leg., Reg. Sess. (Haw. 2007).
Status	Proposed—January 19, 2007
	Carried over to 2008 Regular Session August 27, 2007
Description	Prohibits the use of decabromodiphenylether in upholstered furniture, televisions, and computers. Requires the Department of Health to study whether there are suitable alternatives to decabromodiphenyl ether.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Relating to Decabromodiphenylether**
State	Hawaii
Region	Pacific

Citation	H.B. 461, 24th Leg., Reg. Sess. (Haw. 2007); S.B. 1109, 24th Leg., Reg. Sess. (Haw. 2007)
Status	Proposed—January 22, 2007 (H.B. 461); January 19, 2007 (S.B. 1109)
	Carried over to 2008 Regular Session August 27, 2007 (H.B. 461); Carried over to 2008 Regular Session August 27, 2007 (S.B. 1109)
Description	Prohibits the manufacture, sale, and distribution of televisions, computers, furniture, mattresses, and mattress pads containing commercial decabromodiphenylether.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Supporting the Phase Out of Production and Importation of Decabromodiphenyl Ether in the United States**
State	Hawaii
Region	Pacific
Citation	S.C.R. 209, 25th Leg., Reg. Sess. (Haw. 2010).
Status	Proposed—March 10, 2010 (S.C.R. 209); March 10, 2010 (H.C.R. 235)
	Referred to Committee on Health and Committee on Economic Revitalization, Business & Military Affairs April 8, 2010
Description	Supports the industry phase out of production and importation of decaBDE in the U.S. Encourages the U.S. EPA to continue its efforts to encourage the remaining minor importers of decaBDE to end their importation of decaBDE into the U.S.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Toxic Fire Retardant Prevention Act**
State	Illinois
Region	Midwest
Citation	H.B. 1421, 95th Gen. Assemb., Reg. Sess. (Ill. 2007).
Status	Proposed—February 21, 2007
	Session Sine Die January 13, 2009
Description	Prohibits a person from manufacturing, processing, or knowingly selling, offering for sale, distributing for sale, or distributing for use a mattress, mattress pad, an article of furniture, or any other product intended for indoor residential use if the product has a textile component containing decaBDE. Requires a manufacturer of certain products restricted under the Act to notify persons that sell or distribute that manufacturer's product of the requirements of the Act. Requires a person who manufactures a product or product component that contains decaBDE and is not regulated under the Act to provide written notice to the Illinois Environmental Protection Agency in accordance with the provision.
Category	Single Chemical Restriction
Chemical	decabromodiphenyl ether (decaBDE)
Type	Furniture

Name	**Toxic Fire Retardant Prevention Act**
State	Illinois
Region	Midwest
Citation	H.B. 5298, 95th Gen. Assemb., Reg. Sess. (Ill. 2008).

Status	Proposed—February 14, 2008
	Session Sine Die January 13, 2009
Description	Prohibits a person from manufacturing, processing, or knowingly selling, offering for sale, distributing for sale, or distributing for use a mattress, mattress pad, an article of furniture, or any other product intended for indoor residential use if the product has a textile component containing decaBDE or a television, computer, or other electronic device if the exterior cases of the devices contains decaBDE. Establishes manufacturer and retailer responsibilities with respect to such products. Permits the Illinois Environmental Protection Agency to participate in the establishment and implementation of a regional, multistate clearinghouse to assist in carrying out the requirements of the Act and help coordinate education and outreach activities, review hazard and alternative assessments, and any other activities related to the administration of the Act.
Category	Single Chemical Restriction
Chemical	decabromodiphenyl ether (decaBDE)
Types	Electronics Equipment, Furniture

Name	**Toxic Fire Retardant Prevention Act**
State	Illinois
Region	Midwest
Citation	H.B. 373, 96th Gen. Assemb., Reg. Sess. (Ill. 2009).
Status	Proposed—January 29, 2009
	Rule 19(a) / Re-referred to Rules Committee March 13, 2009
Description	Prohibits the manufacture, process, sale, or distribution of a mattress, mattress pad, article of furniture or any other product intended for indoor residential use if the product has a textile component containing decaBDE. Prohibits the manufacture, process, sale or distribution of a television, computer or other electronic device if the exterior casing of the device contains decaBDE. Provides that the Illinois Environmental Protection Agency may participate in the establishment and implementation of a regional, multistate clearinghouse to help coordinate education and outreach activities, review hazard and alternatives assessments, and any other activities related to implementation.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Concerning Environment — Brominated Flame Retardants — Decabrominated Diphenyl Ether — Prohibition**
State	Maryland
Region	Northeast
Citation	H.B. 35, 427th Gen. Assemb., Reg. Sess. (Md. 2010); S.B. 353, 427th Gen. Assemb., Reg. Sess. (Md. 2010).
Status	Proposed—January 13, 2010 (H.B. 35); January 28, 2010 (S.B. 353)
	Recommit to Committee Environmental Matters April 5, 2010 (H.B. 35); Hearing March 16, 2010 (S.B. 353)
Description	Prohibits the manufacture, sale, or distribution of a new product or flame-retardant part of a new product that contains decaBDE.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Decabrominated Diphenyl Ether Prohibition**
State	Maryland
Region	Northeast

Citation	S.B. 184, 426th Gen. Assemb., Reg. Sess. (Md. 2009); H.B. 14, 426th Gen. Assemb., Reg. Sess. (Md. 2009).
Status	Proposed—January 22, 2009 (S.B. 184); January 14, 2009 (H.B. 14)
	Hearing March 3, 2009 (S.B. 184); First Reading Senate Education Health and Environmental Affairs February 27, 2009 (H.B. 14)
Description	Prohibits the manufacture, sale, or distribution of a new product or flame-retardant part of a new product that contains more than one-tenth of 1% of decaBDE by mass.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**Environment - Brominated Flame Retardants - Decabrominated Diphenyl Ether - Prohibition**
State	Maryland
Region	Northeast
Citation	H.B. 1, 425th Gen. Assemb., Reg. Sess. (Md. 2008).
Status	Proposed—January 9, 2008
	Hearing April 1, 2008
Description	Prohibits the manufacture, production, sale, or distribution of a new product or a flame-retardant part of a new product that contains a specified amount of decabrominated diphenyl ether.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)

Name	**An Act Relative to Toxic Fire Retardant Prevention**
State	Massachusetts
Region	Northeast
Citation	H. No. 3881, 186th Gen. Ct., Gen. Sess. (Ma. 2009).
Status	Proposed—March 20, 2009
	Public Hearing November 2, 2009
Description	Prohibits the manufacture, processing, or distribution of a product or flame-retarded part of a product containing more than one-tenth of 1% of pentaBDE or octaBDE. Prohibits the manufacture, processing, or sale of a mattress, mattress pad, article of furniture, or products intended for indoor residential use if the product has a textile component containing decaBDE. Prohibits the manufacture, processing, or sale of a television, computer, or other electronic device if the exterior casing of the devices contain decaBDE. Requires manufacturers of products containing decaBDE not regulated by this section to notify the Department of Environmental Protection of the product, how much decaBDE is contained within the product, how many units are sold within the Commonwealth, and the distributors of the product within the Commonwealth.
Categories	Data Collection, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Decabromodiphenyl Ether**
State	Michigan
Region	Midwest
Citation	H.B. 4465, 94th Leg., Reg. Sess. (Mich. 2007).
Status	Proposed—March 14, 2007
	Referred to Committee on Great Lakes and Environment March 14, 2007

Description	Prohibits the manufacture and distribution of a mattress or upholstered furniture designed for residential use that contains deca-BDE. Prohibits the manufacture or distribution of a computer or television that contains deca-BDE. Permits the Department of Environmental Quality to establish a PBDE advisory committee to assist the Department in determining the risk posed by substances used as alternatives to PBDEs.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Mary Beth Doyle PBDE Act**
State	Michigan
Region	Midwest
Citation	H.B. 4699, 95th Leg., Reg. Sess. (Mich. 2009).
Status	Proposed—March 25, 2009
	Passed House January 27, 2010; Referred to Senate Committee on Government Operations February 2, 2010
Description	Prohibits the manufacture, sale, or distribution of a mattress or upholstered furniture designed for residential use that contains decaBDE. Prohibits the manufacture, sale, or distribution of any electrical or electronic equipment that contains decaBDE. Prohibits the manufacture, sale, or distribution of motor vehicles or other transportation equipment, military equipment, or components within motor vehicles or other transportation or military equipment that contain decaBDE. Requires a person that produced decaBDE for use in a product sold in Michigan or that sold decaBDE for use in Michigan to submit a report to the Department detailing the quantity of decaBDE produced or sold during the prior year.
Categories	Data Collection, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**A Bill for An Act Relating to Health; Modifying Provisions Related to Maternity Care; Banning the Use of Certain Phthalates, Flame Retardants, or Other Polymers or Chemicals; Requiring Reports; Appropriating Money; Amending Minnesota Statutes 2007 Supplement, Sections 144.651, Subdivision 9; 325E.386; 325E.387, by Adding a Subdivision; Proposing Coding for New Law in Minnesota Statutes, Chapters 145; 325F.**
State	Minnesota
Region	Midwest
Citation	S.F. 651, 85th Leg., Reg. Sess. (Minn. 2007).
Status	Proposed—Feburary 8, 2007
	Veto by Governor May 12, 2008
Description	Restricts the manufacturing, processing, or distribution of the exterior casing of a television, computer, or computer monitor, upholstered furniture or textiles, mattresses or mattress pads containing decabromodiphenyl ether. Establishes a multistate clearinghouse to assist in carrying out the PBDE restriction provisions. Prohibits the sale or offer of a new children's product that contain certain phthalates. Prohibits the replacement of phthalates with a chemical that is classified as a "known human carcinogen", "reasonably anticipated to be a human carcinogen" or identified as causing birth defects or reproductive or environmental harm. Permits the establishment of a multistate clearinghouse to identify children's products containing bisphenol-A or phthalates and to evaluate safer alternatives that may be substituted for those chemicals.
Categories	Alternatives Assessment, Product Categories, Single Chemical Restriction

Chemicals	benzyl butyl phthalate (BBP), Bisphenol-A, decabromodiphenyl ether (decaBDE), di-(2-ethylhexyl) phthalate (DEHP), di-n-octyl phthalate (DNOP), dibutyl phthalate (DBP), diisodecyl phthalate (DIDP), diisononyl phthalate (DINP), Phthalates
Types	Children's Products, Electronics Equipment, Furniture

Name	**A Bill for An Act Relating to the Environment; Banning Certain Products Containing Commercial Decabromodiphenyl Ether; Providing for Exemptions and Fees; Authorizing Participation in Multistate Clearinghouse; Requiring a Report; Amending Minnesota Statutes 2007 Supplement, Sections 325E.386; 325E.387, by Adding a Subdivision.**
State	Minnesota
Region	Midwest
Citation	H.F. 934, 85th Leg., Reg. Sess. (Minn. 2007).
Status	Proposed—Feburary 13, 2007
	Committee Recommendation and Adoption of Report: To Pass as Amended and Re-referred to the Ways and Means Committee April 21, 2008
Description	Restricts the manufacture, production or distribution of the exterior casing of a television, computer, computer monitor, upholstered furniture, textiles, mattress, or mattress pad containing certain levels of decabromodiphenyl ether. Permits the Commissioner of the Pollution Control Agency to participate in a multistate clearinghouse to assist in carrying out these requirements. Requires the Pollution Control Agency to report to the senate and house of representatives committees with jurisdiction over environment and natural resources and commerce policy regarding flame-retardant alternatives available for decabromodiphenyl ether.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemical	decabromodiphenyl ether (decaBDE)
Types	Electronics Equipment, Furniture

Name	**A Bill for an Act Relating to the Environment; Restricting the Manufacture and Sale of Certain Polybrominated Diphenyl Ethers; Authorizing Participation in Multistate Clearinghouse; Amending Minnesota Statutes 2008, Sections 325E.386, by Adding**
	Subdivisions; 325E.387, by Adding a Subdivision.
State	Minnesota
Region	Midwest
Citation	S.F. 596, 86th Leg., Reg. Sess. (Minn. 2009); H.F. 607, 86th Leg., Reg. Sess. (Minn. 2009).
Status	Proposed—February 12, 2009 (S.F. 596); February 9, 2009 (H.F. 607)
	Referred to Environment and Natural Resources February 12, 2009 (S.F. 596); Referred to Environment Policy and Oversight February 9, 2009 (H.F. 607)
Description	Prohibits the manfacture or distribution of the exterior cases of a television, computer, or computer monitor, upholstered furniture or textiles intended for indoor use in a home or other residential occupancy, or mattresses and mattress pads containing more than one-tenth or one percent of commercial decaBDE by mass. Permits the Commissioner of the Pollution Control Agency to participate in a regional or national multistate clearinghouse to assist in carrying out the requirements of this section.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Toxic Fire Retardant Prevention Act**
State	Missouri
Region	Midwest
Citation	H.B. 1155, 95th Gen. Assemb., 1st Reg. Sess. (Mo. 2009).
Status	Proposed—April 1, 2009
	Referred to Public Safety Committee May 15, 2009
Description	Prohibits the manufacture or distribution of a product containing more than one-tenth of one percent of pentaBDE or octaBDE. Prohibits the manufacture, sale, or distribution of a mattress, mattress pad, article of furniture, or any other product intended for indoor residential use if the product has a textile component containing decaBDE. Prohibits the manufacture, sale, or distribution of a television, computer or other electronic device if the exterior casing contains decaBDE. Permits the Department of Natural Resources to participate in the establishment and implementation of a regional, multistate clearinghouse. Requires the Department to report on the bioaccumulation, routes of exposure, health effects, breakdown products, and available alternatives for decaBDE.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Toxic Fire Retardant Prevention Act**
State	Missouri
Region	Midwest
Citation	H.B. 2084, 95th Gen. Assemb., 2nd Reg. Sess. (Mo. 2010).
Status	Proposed—February 9, 2010
	Referred to House Public Sasfety Committee May 14, 2010
Description	Prohibits the manufacture or distribution of a product containing more than one-tenth of one percent of pentaBDE or octaBDE. Prohibits the manufacture, sale, or distribution of a mattress, mattress pad, article of furniture, or any other product intended for indoor residential use if the product has a textile component containing decaBDE. Prohibits the manufacture, sale, or distribution of a television, computer or other electronic device if the exterior casing contains decaBDE. Permits the Department of Natural Resources to participate in the establishment and implementation of a regional, multistate clearinghouse. Requires the Department to report on the bioaccumulation, routes of exposure, health effects, breakdown products, and available alternatives for decaBDE.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act to Amend the Environmental Conservation Law, in Relation to Restricting the Use of Certain Ethers in Certain Products**
State	New York
Region	Northeast
Citation	S. 177, 232nd Leg., Reg. Sess. (N.Y. 2009).
Status	Proposed—January 7, 2009
	Amend and recommit to Environmental Conservation January 11, 2010

Description	Prohibits the manufacture, processing, or distribution of covered electronic devices, upholstered furniture or textiles intended for indoor use in a home or other residential occupancy, or mattresses and mattress pads that contain decaBDE. Requires the Commissioner of the Department of Environmental Conservation and the Commissioner of Health to review uses of decaBDE, availability of technically feasible alternatives, and any new evidence regarding the potential harm to public health and the environment posed by decaBDE. Authorizes the Commissioner of the Department of Environmental Conservation to participate in a regional or national multistate clearinghouse. Permits the Commissioner to establish a "PBDE-free" product labeling program for products that do not contain PBDEs. Requires the Commissioner of the Office of General Services to give priority and preference to the purchase of equipment, supplies, and other products that do not contain decaBDE.
Categories	Alternatives Assessment, Environmentally Preferable Purchasing, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act to Amend the Environmental Conservation Law, in Relation to Restricting the Use of Certain Ethers in Certain Products**
State	New York
Region	Northeast
Citation	S. 1119, 232nd Leg., Reg. Sess. (N.Y. 2009).
Status	Proposed—January 26, 2009
	Referred to Environmental Conservation January 26, 2009
Description	Prohibits the manufacture, processing, or distribution of covered electronic devices, upholstered furniture or textiles intended for indoor use in a home or other residential occupancy, or mattresses and mattress pads that contain decaBDE. Requires the Commissioner of the Department of Environmental Conservation and the Commissioner of Health to review uses of decaBDE, availability of technically feasible alternatives, and any new evidence regarding the potential harm to public health and the environment posed by decaBDE. Authorizes the Commissioner of the Department of Environmental Conservation to participate in a regional or national multistate clearinghouse. Permits the Commissioner to establish a "PBDE-free" product labeling program for products that do not contain PBDEs. Requires the Commissioner of the Office of General Services to give priority and preference to the purchase of equipment, supplies, and other products that do not contain decaBDE.
Categories	Alternatives Assessment, Environmentally Preferable Purchasing, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act to Amend the Environmental Conservation Law, in Relation to Restricting the Use of Certain Ethers in Certain Products**
State	New York
Region	Northeast
Citation	S. 2515, 232nd Leg., Reg. Sess. (N.Y. 2009).
Status	Proposed—February 23, 2009
	Referred to Economic Development January 6, 2009

Description	Prohibits the manufacture, processing, or distribution of covered electronic devices, upholstered furniture or textiles intended for indoor use in a home or other residential occupancy, or mattresses and mattress pads that contain decaBDE. Requires the Commissioner of the Department of Environmental Conservation and the Commissioner of Health to review uses of decaBDE, availability of technically feasible alternatives, and any new evidence regarding the potential harm to public health and the environment posed by decaBDE. Authorizes the Commissioner of the Department of Environmental Conservation to participate in a regional or national multistate clearinghouse. Permits the Commissioner to establish a "PBDE-free" product labeling program for products that do not contain PBDEs. Requires the Commissioner of the Office of General Services to give priority and preference to the purchase of equipment, supplies, and other products that do not contain decaBDE.
Categories	Alternatives Assessment, Environmentally Preferable Purchasing, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act to Amend the Environmental Conservation Law, in Relation to Restricting the Use of Certain Ethers in Certain Products**
State	New York
Region	Northeast
Citation	A. 7573, 232nd Leg., Reg. Sess. (N.Y. 2009).
Status	Proposed—April 16, 2009
	Passed Assembly. Delivered to Senate. Referred to Environmental Conservation April 20, 2010
Description	Prohibits the manufacture, processing, or distribution of covered electronic devices, upholstered furniture or textiles intended for indoor use in a home or other residential occupancy, or mattresses and mattress pads that contain decaBDE. Permits the Commissioner to establish a "PBDE-free" product labeling program for products that do not contain PBDEs. Requires the Commissioner of the Office of General Services to give priority and preference to the purchase of equipment, supplies, and other products that do not contain decaBDE.
Categories	Environmentally Preferable Purchasing, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Restricts the Use of Decabromodiphenyl Ether in Certain Products**
State	New York
Region	Northeast
Citation	S. 5244, 230th Leg., Reg. Sess. (N.Y. 2007).
Status	Proposed—April 25, 2007
	Referred to Environmental Conservation January 9, 2008

Description	Prohibits the manufacture, processing, or distribution of covered electronic devices, upholstered furniture or textiles intended for indoor use in a home or other residential occupancy, or mattresses and mattress pads that contain decaBDE. Requires the Commissioner of the Department of Environmental Conservation and the Commissioner of Health to review uses of decaBDE, availability of technically feasible alternatives, and any new evidence regarding the potential harm to public health and the environment posed by decaBDE. Authorizes the Commissioner of the Department of Environmental Conservation to participate in a regional or national multistate clearinghouse. Permits the Commissioner to establish a "PBDE-free" product labeling program for products that do not contain PBDEs. Requires the Commissioner of the Office of General Services to give priority and preference to the purchase of equipment, supplies, and other products that do not contain decaBDE.
Categories	Alternatives Assessment, Environmentally Preferable Purchasing, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Restricts the Use of Decabromodiphenyl Ether in Certain Products**
State	New York
Region	Northeast
Citation	A. 7977-B, 230th Leg., Reg. Sess. (N.Y. 2007).
Status	Proposed—May 2, 2007
	Referred to Environmental Conservation April 15, 2008
Description	Prohibits the manufacture, processing, or distribution of covered electronic devices, upholstered furniture or textiles intended for indoor use in a home or other residential occupancy, or mattresses and mattress pads that contain decaBDE. Requires the Commissioner of the Department of Environmental Conservation and the Commissioner of Health to review uses of decaBDE, availability of technically feasible alternatives, and any new evidence regarding the potential harm to public health and the environment posed by decaBDE. Authorizes the Commissioner of the Department of Environmental Conservation to participate in a regional or national multistate clearinghouse. Permits the Commissioner to establish a "PBDE-free" product labeling program for products that do not contain PBDEs. Requires the Commissioner of the Office of General Services to give priority and preference to the purchase of equipment, supplies, and other products that do not contain decaBDE.
Categories	Alternatives Assessment, Environmentally Preferable Purchasing, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**Limit Toxic Flame Retardants Containing PBDEs**
State	North Carolina
Region	Southeast
Citation	H.B. 823, 2009-2010 Gen. Assemb., Reg. Sess. (N.C. 2009); S.B. 993, 2009-2010 Gen. Assemb., Reg. Sess. (N.C. 2009).
Status	Proposed—March 26, 2009 (H.B. 823); March 25, 2009 (S.B. 993)
	Re-Referred to the Committee on Health April 13, 2009 (H.B. 823); Referred to Commerce March 26, 2009 (S.B. 993)

Description	Prohibits the manufacture, sale, or distribution of a product containing more than 50 parts per million of octaBDE or pentaBDE. Prohibits the manufacture, sale, or distribution of a mattress, mattress pad, any type of mattress cover or mattress top, upholstered furniture intended for indoor use in a home or other residential occupancy, and electronic devices containing decaBDE. Prohibits manufacturers from replacing decaBDE with any chemicals of similar concern due to their toxicity, bioaccumulation, persistence, or long-range transport. Permits the Secretary of Environment and Natural Resources to establish a product-labeling program for products that meet fire safety standards and do not contain chemical flame retardants. Provides a tax credit for North Carolina manufacturers who develop alternative product designs in order to meet fire safety standards while removing chemical flame retardants from their products. Permits the Secretary to participate in the establishment and implementation of a regional, mu
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Related to Brominated Flame Retardants**
State	Vermont
Region	Northeast
Citation	S. 109, 2009-2010 Leg., Reg. Sess. (Vt. 2009).
Status	Proposed—February 27, 2009
	Referred to House Committee on Natural Resources and Energy April 3, 2009
Description	Prohibits the sale or distribution of a product containing octaBDE or pentaBDE. Prohibits the manufacture, sale, or distribution of a mattress or mattress pad, upholstered furniture intended for indoor use in a home or other residential occupancy, or a television or computer with plastic housing containing decaBDE. Prohibits a manufacturer from replacing decaBDE with a chemical that is classified as "known to be a human carcinogen" or "reasonably anticipated to be a human carcinogen" or is identified by the U.S. EPA as causing birth defects, hormone disruption, or harm to reproduction or development.
Categories	Alternatives Assessment, Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Relating to Brominated Flame Retardants and Bisphenol A**
State	Vermont
Region	Northeast
Citation	S. 76, 2009-2010 Leg., Reg. Sess. (Vt. 2009).
Status	Proposed—February 10, 2009
	Referred to Senate Committee on Natural Resources and Energy February 10, 2009

Description	Prohibits the sale or distribution of a product containing octaBDE or pentaBDE. Prohibits the manufacture, sale, or distribution of a mattress or mattress pad, upholstered furniture intended for indoor use in a home or other residential occupancy, or a television or computer with plastic housing containing decaBDE. Prohibits a manufacturer from replacing decaBDE with a chemical that is classified as "known to be a human carcinogen" or "reasonably anticipated to be a human carcinogen" or is identified by the U.S. EPA as causing birth defects, hormone disruption, or harm to reproduction or development. Permits the Secretary of Natural Resources to participate in the establishment and implementation of a regional multistate clearinghouse. Prohibits the manufacture, sale or distribution of any food or beverage container, child care article, or toy containing bisphenol A in any concentration. Requires manufacturers to use the least toxic alternative when replacing bisphenol A.
Categories	Alternatives Assessment, Product Categories, Single Chemical Restriction
Chemicals	Bisphenol-A, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Children's Products, Electronics Equipment, Furniture, Packaging, Toys

Name	**An Act Relating to the Sale or Distribution of Brominated Flame Retardants**
State	Vermont
Region	Northeast
Citation	H. 589, 2007-2008 Leg., Reg. Sess. (Vt. 2008).
Status	Proposed—January 11, 2008
	In House Commerce Committee January 11, 2008
Description	Restricts the sale of products containing OctaBDE or PentaBDE. Prohibits the sale a mattress, mattress pad, upholstered furniture, a television or computer with a plastic housing containing DecaBDE. Permits the Secretary of Natural Resources to participate in the establishment and implementation of a regional, multistate clearinghouse to assist in the implementation of the requirements of this section.
Category	Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Relating to the Sale or Distribution of Brominated Flame Retardants**
State	Vermont
Region	Northeast
Citation	S. 260, 2007-2008 Leg., Reg. Sess. (Vt. 2008).
Status	Proposed—January 8, 2008
	In Senate Natural Resources and Energy January 8, 2008
Description	Restricts the sale of products containing OctaBDE or PentaBDE. Prohibits the sale a mattress, mattress pad, upholstered furniture, a television or computer with a plastic housing containing DecaBDE. Permits the Secretary of Natural Resources to participate in the establishment and implementation of a regional, multistate clearinghouse to assist in the implementation of the requirements of this section.
Category	Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

Name	**An Act Relating to Testing the Chemical Content of Products Sold at Retail; Amending RCW 70.76.030; and Creating New Sections.**
State	Washington
Region	West
Citation	S.B. 5977, 61st Leg., Reg. Sess. (Wa. 2009).
Status	Proposed—February 11, 2009
	Reintroduced and retained in present status. January 11, 2010
Description	Directs the Joint Legislative Audit and Review Committee to review commercially available testing methods for the detection of decaBDE in proposed banned products and a reasonable time frame for the full integration of such testing by retailers.
Category	Data Collection
Chemical	decabromodiphenyl ether (decaBDE)

Name	**An Act to Amend Sections 108921 and 108922 of the Health and Safety Code, Relating to Hazardous Chemicals.**
State	California[6]
Region	West
Citation	A.B. 513, 2007-08 Leg., Reg. Sess. (Cal. 2007).
Status	Proposed—February 20, 2007
	Failed*—Died on third reading file February 4, 2008
Description	Amends the existing PBDE prohibition to include decaBDE in electronic products.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Type	Electronics Equipment

Name	**An Act to Reduce Contamination in the Home from the Release of Brominated Flame Retardants**
State	Maine
Region	Northeast
Citation	L.D. 1488, 123rd Leg., 1st Reg. Sess. (Me. 2007).
Status	Proposed—March 20, 2007
	Failed*—Placed in Legislative Files (Dead) April 24, 2007
Description	Prohibits the sale, offer for sale, or distribution of a mattress, an article of furniture, or any other product intended for household use if the product has a textile component containing more than .1% by mass of the "deca" mixture of polybrominated diphenyl ethers. Prohibits the sale, offer for sale, or distribution of a television, computer, or other electronic device if the exterior casing of the device contains more than .1% by mass of the "deca" mixture of polybrominated diphenyl ethers. Authorizes the Department of Environmental Protection to require a manufacturer to provide a certificate of compliance if there are grounds to suspect that a product is being offered for sale in violation of the law.
Category	Single Chemical Restriction
Chemicals	Brominated Fire Retardants, decabromodiphenyl ether (decaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

[6] The State of California is currently reviewing its flammability standards for upholstered furniture with the goal of reducing or eliminating flame retardants while continuing to ensure fire safety.

Name	**An Act Prohibiting the Manufacture, Sale, or Distribution of Certain Products Containing Polybrominated Diphenyl Ethers; Defining Terms; Providing Certain Exemptions and Exceptions; Requiring that Certain Reports be Made to the Legislature; Requiring A Manufacturer to Provide Notification of Restrictions to Persons Selling Products that Contain Polybrominated Diphenyl Ethers; Providing Compliance Mechanisms; Authorizing Civil Penalties; Requiring the Department of Environmental Quality to Assist Other State Agencies in Identifying Certain Products for Purchase; Authorizing the Department of Environmental Quality to Adopt Rules; And Providing and Immediate Effective Date**
State	Montana
Region	West
Citation	H.B. 560, 60th Leg., Reg. Sess. (Mont. 2007).
Status	Proposed—February 3, 2007
	Failed*—Died in Standing Committee April 27, 2007
Description	Restricts the sale of noncomestible products containing polybrominated diphenyl ethers. Prohibits the sale of a mattress containing decabromodiphenyl ether. Prohibits the sale of residential upholstered furniture that contains commercial decabromodiphenyl ether or any television or computer that has an electronic enclosure that contains commercial decabromodiphenyl ether if there is a safer alternative. Requires the Department of Environmental Quality and the Department of Health and Human Services to annually review risk assessments, peer-reviewed scientific studies, and other relevant findings regarding alternatives to the use of decabromodiphenyl ether in residential upholstered furniture, televisions, and computers.
Category	Single Chemical Restriction
Chemicals	decabromodiphenyl ether (decaBDE), octabrominated diphenyl ether (octaBDE), pentabrominated diphenyl ether (pentaBDE), Polybrominated diphenyl ethers (PBDEs)
Types	Electronics Equipment, Furniture

*In some states, "failed" legislation refers to bills not voted on in a particular legislative session. These bills may or may not be re-introduced in later sessions.

Appendix 8. European Union Conclusions for Lower Brominated Congeners

The following is the summary of the persistent, bioaccumulative and toxic (PBT) and very persistent and very bioaccumulative (vPvB) conclusions as given in the Proposal for Identification of a PBT/VPvB substance decaBDE (UK, 2012).

–Summary of PBT profiles for specific congener groups

- TetraBDE congeners meet the PBT and vPvB criteria.
- PentaBDE congeners meet the PBT and in some cases the vPvB criteria.
- HexaBDE congeners meet the PBT and in some cases the vPvB criteria.
- HeptaBDE congeners meet the vP and T criteria. They do not appear to meet the B or vB criteria based on an estimated fish BCF, but the balance of available evidence suggests that they can be considered to be B. HeptaBDEs are therefore considered to be a PBT substance.
- OctaBDE congeners meet the vP criteria, but probably do not meet the B criteria. They possibly meet the T criteria.
- NonaBDE congeners meet the vP criteria, but probably do not meet the B criteria. There are insufficient data to conclude on T.

The PBT/vPvB nature of the tetra-, penta-, hexa- and heptaBDE congeners has already been recognize by listing them as persistent organic pollutants (POPs) on Annex A of the Stockholm Convention, implemented in the EU as Commission Regulation (EU) No. 757/2010.

Experiments have shown that nonaBDEs can be degraded to octaBDEs by anaerobic bacteria (Gerecke et al., 2005 and 2006). He et al. (2006) and Lee and He (2010) have shown that octaBDE can be biodegraded by anaerobic bacteria collected from a range of locations to hexa-, penta- and tetraBDEs after around six months‘ incubation at 30O. Robrock et al. (2008) also elucidated the likely reaction pathway. Stapleton et al. (2004b) showed that Common Carp (*Cyprinus carpio*) exposed to BDE-99 and BDE-183 (a penta- and heptaBDE, respectively) via the diet could metabolize these substances to BDE-47 (a tetraBDE) and BDE-154 (a hexaBDE) respectively. It therefore seems likely that nona- and octaBDEs an also act as precursors to the PBT/vPvB congeners. Indeed, UNEP (2007) concluded that the octa- and nonaBDE congeners

are also likely to lead to significant adverse human health and/or environmental effects, such that global action is warranted, due to their transformation to other PBDEs."