

217/782-3397

January 25, 2006

The Honorable Rod R. Blagojevich  
Governor of Illinois  
207 State House  
Springfield, Illinois 62706

Dear Governor Blagojevich:

On July 1, 2005, you signed House Bill 2572 into law, creating the Brominated Fire Retardant Prevention Act (P.A. 94-100). Through this Act, the General Assembly stated its desire for the state to develop a precautionary approach regarding the production, use, storage, and disposal of products containing brominated fire retardants (BFRs).

BFRs are chemicals that are widely used in the United States by manufacturers in order to meet stringent fire standards. BFRs are added to a multitude of products including plastic housings of electronics and computers, circuit boards, and the foam and textiles used in furniture.

Polybrominated diphenyl ether (PBDE), which is a subcategory of BFRs, has the potential to disrupt thyroid hormone balance and contribute to a variety of developmental deficits, including low intelligence and learning disabilities. PBDE may also have the potential to cause cancer.

P.A. 94-100 requires the Illinois EPA to submit to the General Assembly and the Governor a report that reviews the latest available scientific research to address the following issues regarding decabromodiphenyl ether (decaBDE):

- levels of bio-accumulation in humans and the environment;
- health effects resulting from human exposure;
- health effects resulting from degradation products; and
- available effective alternatives.

To that end, enclosed is a copy of "DecaBDE Study: A Review of Available Scientific Research." If you have any questions or comments, please contact me or Tom Hornshaw, Manager, Toxicity Assessment Unit, at 217/785-0832.

Very truly yours,

Douglas P. Scott

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**State of Illinois**  
Rod R. Blagojevich, Governor

**Illinois Environmental Protection Agency**  
Douglas P. Scott, Director



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**A Report to the General Assembly and the Governor  
In Response to Public Act 94-100**

*“DecaBDE Study: A Review of Available Scientific Research”*

**Illinois Environmental Protection Agency**  
**January 2006**

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## **ACKNOWLEDGEMENTS**

The Illinois Environmental Protection Agency gratefully acknowledges the efforts of the Illinois Public Interest Research Group and the Bromine Science and Environmental Forum to provide the Agency with important information regarding the polybrominated diphenyl ethers, especially decabromodiphenyl ether, in meetings on July 25, 2005 and September 8, 2005, respectively, and through personal contacts. The information provided is greatly appreciated.

## EXECUTIVE SUMMARY

This report has been prepared to address the five issues posed by the Illinois Legislature to the Illinois Environmental Protection Agency in HB2572 regarding the use of Decabromodiphenyl ether (DecaBDE). The Agency reviewed numerous data sources, including some very recent information, pertaining to the five issues in order to respond in as thorough a manner as possible. However, data gaps exist in certain key areas that have hampered our ability to fully address some issues. The five issues and our findings follow.

The first issue asks whether DecaBDE is bio-accumulating in the environment, and if so, whether the levels of DecaBDE are increasing, decreasing, or staying the same. We find that DecaBDE is bioaccumulating in the environment, and levels are increasing in some types of samples (sediments, some top predators, and possibly human blood and breast milk).

The second issue asks how humans are exposed to DecaBDE. We find that humans are exposed to decaBDE from many sources including the diet, workplace, and home, with diet the primary source for adults and breast milk and house dust important sources for infants and small children.

The third issue asks what health effects could result from exposure to DecaBDE, and are current levels of exposure at levels that could produce these effects. We find that the most important health effects from exposure to decaBDE and/or lower-brominated congeners appear to be liver, thyroid, reproductive/developmental, and neurological effects, although the relevance of some of the effects reported in animal studies for human health risks has been questioned, and significant data gaps in the decaBDE toxicity database have been identified; estimates of current human exposures to the PBDEs indicate that effects on the liver should not be occurring, but two recent studies suggest that exposures could be occurring that are in the range of doses causing adverse effects in laboratory animals.

The fourth issue asks whether DecaBDE breaks down into more harmful chemicals that could damage public health. We find that DecaBDE can be broken down by ultraviolet light and direct sunlight, and also by metabolic processes in animals and microorganisms, but uncertainty and controversy exists about the extent of breakdown by light under environmentally relevant conditions and the human health implications of the breakdown products; therefore, we believe that the information available at this time regarding DecaBDE's breakdown products is not sufficient to allow us to confidently address this issue.

The fifth issue asks whether effective flame retardants are available for DecaBDE uses, and whether the use of available alternatives reduces health risks while still maintaining an adequate level of flame retardant performance. We find that effective, though more costly, alternatives exist for most of the plastics and textiles/fabrics uses of DecaBDE, and these alternatives will likely reduce risks while maintaining an adequate level of flame retardant performance; however, significant toxicity data gaps exist for many of the main potential alternatives, and further research is needed to better evaluate the health and environmental consequences of these alternatives.

We also reviewed the actions of other jurisdictions regarding the polybrominated diphenyl ethers (PBDEs). USEPA's Voluntary Children's Chemical Evaluation Program has determined that a significant data gap exists regarding the environmental transport and fate of decaBDE, and DecaBDE manufacturers will soon begin studies to fill these gaps. The European Union (EU) has included the PBDEs on a list of chemicals to be phased out of use in electrical and electronic equipment, but DecaBDE manufacturers have successfully petitioned for an exemption for DecaBDE from this ban. This exemption may be challenged in the European Court of Justice. The EU will also conduct studies of the reproductive/developmental and neurological effects of decaBDE to fill important gaps in the toxicity database. Several states have recently legislated bans on the use of the Penta- and OctaBDE flame retardant formulations in products, and Maine will ban DecaBDE in 2008 if effective alternatives to DecaBDE are identified. Some states have also required studies of DecaBDE to help decide what actions, if any, are appropriate for DecaBDE.

The research noted above on the potential for reproductive/developmental and neurological effects of decaBDE and the studies on the environmental transport and fate of decaBDE, plus other on-going or planned studies, should provide valuable information to assist in evaluating the issues raised in HB2572.

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## 1.0 INTRODUCTION

This report has been prepared by the Illinois Environmental Protection Agency (Agency) at the request of the Illinois Legislature, via HB2572, to address concerns about polybrominated diphenyl ether (PBDE) flame retardants. These concerns have arisen as more research has been conducted on their transport and fate in the environment and on their health effects. Recent studies have shown that the PBDEs are becoming widespread in the environment and in human blood, milk, and tissues, and that some of them have produced adverse effects in laboratory animals. These concerns have driven the Legislature to request this report.

It is our intent with this report to provide a general overview of the information relevant to the five issues to be addressed by the Agency in HB2572, and highlight what we see as the key issues specific to Decabromodiphenyl ether. It is not our intent to provide a wide-ranging summary of all that is known about the environmental chemistry and toxicology of the PBDEs. If the reader is interested in further pursuing information about specific topics, several recent in-depth reviews/reports can be consulted (ATSDR, 2004; Gill et al., 2004; Darnerud et al., 2001; Washington, 2005; Maine, 2005; Michigan, 2004).

This introductory section will present a brief overview of the PBDEs and discuss HB2572. Subsequent sections will address each of the five issues contained in HB2572 and present a brief overview of actions taken by other jurisdictions regarding the PBDEs.

### 1.1 PBDE Flame Retardants

Flame retardants are chemicals that are added to many materials and products to prevent or suppress ignition of the materials and products or to limit the spread of fire once ignition occurs. They have been credited with saving many lives and preventing injuries and loss of property as a result of mandated or voluntary use. They can be either reactive flame retardants, in which the retardant reacts chemically with the components of the product, or additive flame retardants, in which the retardant is merely added to the product without reacting. Products containing the element bromine comprise a significant portion of the flame retardant market due to this element's effectiveness at suppressing ignition and stopping the spread of flame, and relatively low cost.

The PBDEs are one of the main classes of brominated flame retardants. There are theoretically 209 individual brominated diphenyl ethers (called congeners), differing in the number (1 to 10) of bromine atoms attached to the diphenyl ether "backbone" and in the positions of attachment of the bromines to the backbone. The only uses of the PBDEs are as additive flame retardants, and they have been produced primarily in three technical formulations, PentaBDE, OctaBDE, and DecaBDE, under a variety of trade names. The Penta-, Octa-, and DecaBDE formulations have an average of five, eight, and ten bromine atoms attached to the diphenyl ether molecule, although each product is a mixture of several closely related congeners. Information about these formulations is listed in Table 1. To avoid confusion, the remainder of this report will refer to the commercial PBDE formulations as PentaBDE, OctaBDE, and DecaBDE; to the general classes of congeners having the same number of bromine atoms but different points of

attachment to the diphenyl ether backbone as monoBDE through decaBDE; and to individual congeners by their accepted numbering system (for example, BDE-47 is 2,2',4,4'-tetrabromodiphenyl ether, and BDE-209 is decabromodiphenyl ether).

The PBDE formulations have been used in a wide variety of products, including many types of plastics (often as housings for and components of electronic equipment), textiles, packaging materials, furniture, and upholstery. Typically, the PBDE formulation is added to the product or material in the range of 5-30% by weight. DecaBDE has been the most widely used of the three formulations, both in the United States and worldwide. Recent production of DecaBDE has exceeded 60,000 metric tons worldwide, and over 40% of the use of DecaBDE occurs in North America. This production may increase due to voluntary or regulatory curtailment of the uses of the Penta- and OctaBDE formulations.

## **1.2 HB2572**

HB2572, the “Brominated Fire Retardant Protection Act,” was passed by the Illinois Legislature in 2005 due to concerns about increasing levels of brominated flame retardants, especially the PBDEs, and potential health consequences of these chemicals. The Act notes that PBDEs have increased forty-fold in human milk since the 1970s, and they have the potential to disrupt thyroid hormone balance, contribute to developmental deficits, and possibly cause cancer. Because of these concerns, the Act prohibits the manufacture, processing, or distribution in commerce of products containing more than 0.1% of the PBDE formulations PentaBDE and OctaBDE as of January 1, 2006. It also requires the Agency to submit to the Legislature and the Governor a DecaBDE study by January 2, 2006. This study is to review the latest available scientific research on DecaBDE and respond to five issues:

- Whether DecaBDE is bio-accumulating in the environment, and if so, whether the levels of DecaBDE are increasing, decreasing, or staying the same;
- How humans are exposed to DecaBDE;
- What health effects could result from exposure to DecaBDE, and are current levels of exposure at levels that could produce these effects;
- Whether DecaBDE breaks down into more harmful chemicals that could damage public health; and
- Whether effective flame retardants are available for DecaBDE uses, and whether the use of available alternatives reduces health risks while still maintaining an adequate level of flame retardant performance.

The Act also requires the Illinois Department of Public Health to review the Agency’s report by February 28, 2006 and report their findings to the Legislature and the Governor.

## **2.0 PRESENCE OF DECABROMODIPHENYL ETHER IN HUMANS AND THE ENVIRONMENT**

DecaBDE is increasingly being detected in the North American environment including Illinois. Detections in living organisms including humans are also becoming more frequent. Concentrations in the environment tend to be much higher than those detected in organisms. The pattern of deposition of decaBDE in environmental media and organisms also varies from that of the lower-brominated congeners.

### **2.1 Presence of DecaBDE in Environmental Media**

The physical properties of decaBDE determine its partitioning into environmental media. Based upon decaBDE's organic carbon partition coefficient (K<sub>oc</sub>) of 1.67E+12 it is expected to attach strongly to soil and sediment, which are both high in organic carbon. Modeled environmental partitioning of decaBDE predicts binding of 57% to sediment, 42% to soil, 1.0% to water and 0.1% to air (BSEF, 2005). Evidence of the accumulation of decaBDE is abundant. Many studies include examination of media within or that are important to the State of Illinois.

Because of its low volatility, transportation of decaBDE in the air as a vapor is expected to be minimal. However, due to its strong attraction to organic carbon, decaBDE has been detected in ambient atmospheric particulates although transport by this means should not be over any significant distance. The presence of decaBDE in the particulate phase was documented in 1997-1999 air samples from the Chicago area (Strandberg et al., 2001). The absence of higher brominated congeners (hexa- through decaBDEs) at other more remote Great Lakes sample sites permitted the authors to confirm assumptions regarding the aerial transport of decaBDE. Air concentrations of decaBDE in the Chicago area during 2002-2003 were found to be "relatively abundant" (Hoh and Hites, 2005). DecaBDE concentrations within the Chicago area were the highest of those tested at about 10 times higher than other Midwestern sites, including an Arkansas site near two PBDE manufacturing plants. No sources of the Chicago concentrations were identified.

The throughput of PBDEs in the City of Palo Alto, California wastewater treatment plant was recently studied (North, 2004). Effluent and sewage sludge were collected and analyzed for all major PBDE congeners including decaBDE. The researcher found that decaBDE was the predominant PBDE congener in the sludge; however, decaBDE was only a minor constituent (6%) of the total PBDEs in the effluent. Due to their strong attraction to organic carbon and their extremely low solubility, PBDEs in the effluent were minute. North estimated that the Palo Alto treatment plant discharged approximately 2 lb/yr total PBDEs into the San Francisco Estuary.

As a consequence of its physical properties and its continuing release into the environment during its synthesis, manufacturing, end use, and disposal, the presence of decaBDE in sewage sludge is inevitable. Hale et al. (2003) cited unpublished results regarding sewage sludge from eight Lake Michigan communities. The concentrations of decaBDE in sewage sludges from these communities were very similar to those of the other less brominated BDEs, which were about 30-50 times greater than the most abundant polychlorinated biphenyl (PCB) congener. Application of sewage sludges to agricultural fields creates the potential for contamination of

plants destined to become animal or human foodstuffs. This is a possible explanation for the presence of PBDEs (including decaBDE) in infant soy formula (Schechter et al., 2004). Concentrations of higher-brominated PBDEs in soils including sewage sludge amended soils were correlated to concentrations of these compounds in earthworms living in the subject soils (Sellstrom et al., 2005). DecaBDE was determined to have the lowest biota-soil accumulation factor of the tetra- through decaBDE congeners. Nevertheless, decaBDE was determined to be bioavailable from the soil and to accumulate in earthworms. The authors concluded that the presence of PBDEs in soil invertebrates introduces an exposure pathway into terrestrial food webs.

Some of the highest environmental concentrations of decaBDE are found in the sediments of lakes, rivers, streams, and estuaries. The accumulation of PBDEs in the sediment of Lakes Michigan and Huron was recently investigated (Song et al., 2005). Seventy-five samples were obtained from sediment cores at three locations each within the two lakes. Results of the subsampling within the cores showed a dramatic increase in total PBDEs at all lake locations as subsamples progressed toward the surface of the sediment core, which corresponded to present time. The authors were able to calculate a doubling time for total PBDEs of 10 to 13 years. The total accumulated loading to Lake Michigan was estimated to be 29 to 50 metric tons with a 2002 loading rate of 0.37 to 1.14 metric tons. Other researchers studied sediment cores in Lakes Michigan and Erie (Zhu and Hites, 2005). They also observed the apparent rapid increase of total PBDEs and estimated a total accumulated load to the entire Great Lakes system of 200 metric tons. The latter researchers calculated a doubling time for decaBDE in Lake Michigan of 7.5 years and a 2003-2004 loading rate of 2.8 metric tons. Both sets of researchers found that decaBDE was the overwhelmingly predominant congener in sediment comprising  $\approx 95\%$  of the total PBDE load.

## **2.2 Presence of DecaBDE in Organisms**

Sediments in industrial areas and near waste treatment facilities are the environmental media most contaminated by PBDEs. Animals living in these sediments would be expected to reflect their environment. A study of temporal trends of PBDEs in marine mussels was conducted at the industrialized Seine estuary in France from 1981 to 2003 (Johansson et al., 2004). DecaBDE was quantified in the tissue samples in each year of the investigation, however, the levels of decaBDE were very low in comparison to the other BDE congeners and do not reflect the trend toward greater decaBDE contamination of the sediments, a consequence of its increasing use.

Although decaBDE is the predominant congener found in PBDE contaminated sediments, it seems to be a minor contaminant in fish. Because pike is a very stationary species they were selected to correlate sediment concentrations of PBDEs with fish tissue concentrations (Sellstrom et al., 1998). These researchers concluded that decaBDE does not seem to be bioavailable to fish. These same researchers point out that decaBDE is a superlipophilic compound (a substance that is highly attracted to organic carbon) and suggest that bioaccumulation is inhibited in the wild due to the chemical's very strong attraction to the sediments. A variety of fish were evaluated in an Indiana lake heavily contaminated by activities of a PBDE manufacturer (Dodder et al., 2002). In this investigation, no decaBDE was detected in any of the fish samples except for a trace amount in one smelt sample. These researchers

concluded that decaBDE does not seem to be bioavailable. More recent studies seem to contradict these findings.

DecaBDE's uptake into fish has also been evaluated in controlled laboratory feeding studies. The biological uptake of decaBDE was investigated in the laboratory using rainbow trout (Kierkegaard et al., 1999). DecaBDE spiked food was given to test trout for 120 days followed by a 71-day depuration period. During dosing, steadily increasing muscle and liver decaBDE concentrations were observed at 16, 49, and 120 days. Additionally, increasing concentrations of several hexa- to nonaBDE congeners were observed. During depuration, decaBDE concentrations decreased significantly, however concentrations of the lower-brominated congeners were unaffected or increased. The authors attribute these observations to biological metabolism (debromination) of the decaBDE, selective uptake of the lower-brominated congeners present as contaminants of the DecaBDE formulation, or as breakdown products produced during the analytical process. Assuming metabolism and based on the sum of all PBDE congeners in the fish muscle, uptake in trout was estimated to be approximately 0.02-0.13% after 120 days of exposure. Higher absorption was calculated in a more recent feeding study (Stapleton et al., 2004). This study found no accumulation of decaBDE in juvenile carp but the results confirmed limited bioavailability of decaBDE from food in the form of lower-brominated congeners. The latter researchers calculated bioavailability of decaBDE from food to be 0.44% in juvenile carp. They caution that although bioavailability of <1.0% may seem insignificant, appreciable accumulation could occur in aquatic organisms exposed to highly contaminated sediments or sewage sludge.

The bioaccumulation and biomagnification of lower-brominated BDEs has been well demonstrated in aquatic (Stapleton and Baker, 2003) and aquatic-terrestrial foodwebs (Wolkers et al., 2004). In spite of the implication of limited bioavailability of decaBDE in fish and the near absence of absorption from ingested carbon rich sediments, a recent study provides evidence of increasing concentrations of decaBDE in a Florida aquatic foodweb (Johnson-Restrepo et al., 2005a). Several feeding levels were examined within this foodweb. The results suggest taxon-specific selective metabolism of the various PBDE congeners. The absence of specific lower-brominated congeners in the tissues of certain classes of fish suggests rapid metabolism. DecaBDE illustrates this circumstance very well. Except for the hardhead catfish, bony fish that feed low on the food web were nondetect for decaBDE; however, decaBDE was the prevalent PBDE detected among the top predator sharks. In contrast, the higher feeding level carnivorous dolphins were nondetect for decaBDE. This information indicates that generalizations regarding bioaccumulation and biomagnification of decaBDE in the environment should be made with extreme caution.

A study of decaBDE in birds compared birds that feed in aquatic environments to those that feed in terrestrial environments (Jaspers et al., 2006). It also showed varying results with respect to tissue concentrations of decaBDE. In this study decaBDE was only detected in the tissues of the terrestrial-feeding birds. This either points to an absence of decaBDE in the prey of the aquatic-feeding birds or a metabolic difference between the two groups of birds based on their feeding habits. Two recent studies show that decaBDE is bioavailable to birds and that it can be taken up by peregrine falcons (*Falco peregrinus*) and transferred to their eggs (Lindberg et al., 2004; Vorkamp et al., 2005). The latter study showed a statistically significant temporal trend toward

increased decaBDE concentrations. Unhatched eggs were collected between 1986 and 2003 from a South Greenland study area. Peregrine falcons are top predators that, during breeding, prey on medium-sized birds that feed in terrestrial environments. Peregrine falcons do migrate to Central and South America during the winter and thus they are exposed to unknown food sources.

The ubiquity of the environmental contamination by PBDEs (including decaBDE) can be illustrated by their presence in glaucous gulls (*Larus hyperboreus*) and polar bears (*Ursus maritimus*) on the remote island of Svalbard in the Norwegian Arctic (Verreault et al., 2005). Glaucous gulls are top predators consuming a wide range of food items including eggs, chicks, fish, carrion, crustaceans, and adult birds. Polar bears also feed at the top of their marine foodweb and prey exclusively on ringed seals. All congener groups from mono- to decaBDEs were detected in ringed seal blubber (Ikonomou et al., 2002). Temporal trends from 1981 to 2000 showed exponential increases in tissue levels. At current bioaccumulation rates, the authors estimated that PBDE levels would surpass PCB concentrations by the year 2050 and become the most prevalent organohalogen compound in Canadian arctic ringed seals.

### **2.3 Presence of DecaBDE in Humans**

Rising environmental concentrations and concomitant bioaccumulation and biomagnification in global foodwebs signals concern for human populations and, indeed, PBDEs have been detected in human tissues. A study of Swedish clerks working full-time at computer screens plus electronic equipment dismantling workers found numerous PBDE congeners present in all workers and decaBDE present in 30 of 39 samples (Sjodin et al., 1999). Surprisingly, the targeted PBDEs (including decaBDE) were also detected in 14 of 20 samples from a control group of hospital cleaners. This study included a resample of the electronics dismantling workers following an exposure-free vacation standardized to a 30-day period. Following removal from exposure, the drop in the decaBDE concentrations was the greatest of the PBDE congeners investigated indicating that decaBDE is eliminated more rapidly than the lesser-brominated PBDEs.

While decaBDE was not detected in the majority of samples of human fat from New Yorkers that had undergone cosmetic surgery, levels of BDE-153 and several unidentified PBDE compounds were elevated (Johnson-Restrepo et al., 2005b). None of these compounds originate from known commercial formulations. The authors speculate possible occupational exposure of certain subjects in their study. Another possible explanation for the presence of these compounds is that they are the products of metabolism of decaBDE.

In a study of blood samples from 1988 United States donors, five of 12 samples were found to contain decaBDE (Sjodin et al., 2001). The main author cites his earlier work establishing a half-life (the length of time required for an initial concentration to decrease by one-half) for decaBDE of 6.8 days in serum. This study concludes that to sustain detectable levels of decaBDE in 1988 samples of human serum there must have been low-level chronic exposures. A study correlating maternal and fetal blood concentrations of PBDEs was conducted on subjects in Indiana (Mazdia et al., 2003). Although this study did not look for decaBDE, it did conclude that fetal blood concentrations of PBDEs did not differ from corresponding maternal

concentrations. Another source of PBDE (including decaBDE) to infants was investigated by analyzing breast milk samples from nursing mothers from the U.S. Pacific Northwest (She et al., 2005). DecaBDE was evaluated in this study and it was detected although at very low concentrations. This investigation found that PBDEs, primarily lower-brominated congeners, were the major persistent organic pollutant – surpassing PCBs – in the 40 samples examined.

Finally, a study of the blood of a California family that avoided chemical cleaners and pesticides showed the unavoidable nature of PBDEs and decaBDE (Inside Bay Area, 2005). The Hammond Hollands family avoided chemicals, ate organic foods, had no new appliances, and eschewed wall-to-wall carpeting yet all four family members had high levels of PBDEs and decaBDE in their blood. The highest level of decaBDE was found in the male toddler. Levels of PBDEs and decaBDE were so elevated that Swedish researcher Ake Bergman was asked to confirm the PBDE results. A subsequent blood collection three months later showed that concentrations of the lower-brominated PBDE were about the same, however, the decaBDE levels had diminished to about a tenth of the original. Dr. Bergman explains laboratory error is not likely based on the similarity in the concentrations of the lower-brominated congeners. He suggests that the drop in decaBDE was due to metabolism and that the initial high concentrations were due to an unknown major exposure.

### **3.0 HUMAN EXPOSURE TO DECABROMODIPHENYL ETHER**

As is the case with PCBs and dioxins, the primary route of exposure to PBDEs in humans is by ingestion of fatty animal foods such as meat, fish, poultry, eggs, and dairy products. However, there are additional exposure routes for PBDEs that are not found for PCBs and dioxins. PBDEs are found in many consumer products in our homes and places of work. Many of these applications are for products that we use on a daily basis. Additionally, we may unknowingly expose our children to PBDEs at a very early age.

#### **3.1 DecaBDE in Our Diets**

For most people, the primary route of exposure to PBDEs (including decaBDE) is through their diet. In a marketbasket survey of 30 food types from major supermarket chains in the Dallas, Texas area elevated levels of PBDEs (including decaBDE) were detected (Schechter et al., 2004). Contaminant concentrations were higher than those established for Spain and Japan, the only other national surveys available. Samples were exclusively from foods of animal origin and included fish, meat, and dairy products. In the fish samples, varying PBDE congener profiles were observed. DecaBDE accounted for over 50% of the total PBDE concentrations in the two freshwater catfish samples. DecaBDE was the prominent congener in the calf's liver, soy instant formula, cheese, and margarine samples and third most prevalent in the chicken liver sample. These results suggest that decaBDE may be a significant contributor to total PBDE levels in some food supplies. The authors explain that as decaBDE becomes the only commercial PBDE flame retardant manufactured in the US beginning in 2005, the PBDE profiles in food will change, probably shifting toward increases in decaBDE and the higher-brominated PBDEs.

As discussed earlier, PBDE levels in wild sport fish are well established with concentrations of decaBDE consistently low. Controlled feeding studies show that fish do absorb decaBDE but rapid metabolism (debromination) apparently keeps body burdens low. A study of farm-raised fish may shed light on the elevated levels of decaBDE found in the marketbasket survey (Hites et al., 2004). This study looked at 51 different salmon farms in three geographical areas (European Union, North America, and Chile) and compared fish PBDE concentrations to those determined in salmon fillets purchased from supermarkets and 135 wild caught fish representing five species of Pacific salmon. Although decaBDE was not investigated, the researchers found that farm-raised and supermarket salmon have much higher levels of total PBDEs than do wild salmon. The differences between the farmed and wild salmon were attributed to their diets. Farmed salmon are fed a concentrated feed high in fish oil and fishmeal obtained from small open ocean fishes.

#### **3.2 DecaBDE in the Workplace**

Some occupations have a potentially high likelihood of exposure to PBDEs and to decaBDE. We previously reported on decaBDE exposures to a group of Swedish workers in the electronics-dismantling industry (Sjodin et al., 1999). DecaBDE is incorporated into the plastics used in electronic circuit boards and in the casings of televisions, computers, and other electronic equipment (Darnerud et al., 2001). It is reasonable to assume that anyone working in the

electronics production and dismantling industries are potentially exposed to elevated amounts of decaBDE. Routes of exposure would include inhalation and incidental ingestion.

PBDEs, including decaBDE, are also used extensively in the household furnishing industry (USEPA, 2005b). DecaBDE formulations are used as fire retardants in backings of draperies, upholstery, and carpeting. Industries that manufacture these materials would be potential sources of worker exposure to decaBDE. In addition, individuals employed in the removal and disposal of the materials are also potentially exposed.

As shown in the study of sewage sludge (North, 2004), high concentrations of decaBDE are present in municipal sewage sludge and workers in sludge-related activities are potentially exposed to very high concentrations, primarily through inhalation.

A study of contaminant concentrations in dust was conducted in Parliament buildings in eight EU countries and an Internet office in the Netherlands (Santillo et al., 2001). DecaBDE was detected in all dust samples and was the most predominant of the PBDEs detected. This is a reflection of its abundant use in office products such as carpets, furnishings, and electronic equipment and cables.

### **3.3 DecaBDE in the Home**

Many of the products containing DecaBDE used in the office are also commonly used in our homes. The same DecaBDE material used as a fire retardant in office carpeting, furniture upholstery, and draperies is used in the domestic market for home use. For the most part, there is no difference between office PCs, printers, and electronic cables and those used in homes.

Three recent studies point to house dust as a potentially large source of exposure to PBDEs, especially for young children. Greenpeace conducted a study of house dust from 100 households in the United Kingdom and Scotland (Santillo et al., 2003). DecaBDE was the most abundant of the PBDEs detected with levels ranging higher than those observed in an earlier evaluation of work place dust (Santillo et al., 2001). A similar study was undertaken on households in Ottawa, Canada (Wilford et al., 2005). Their results showed that decaBDE comprised, on average, 42% of the total PBDE content of all house dust samples. A study of 16 homes in the Washington, DC area revealed similar results (Stapleton et al., 2005). All dust samples collected by vacuuming carpets and hardwood floors proved to contain decaBDE, which represented 8 to 88% of the total PBDE content. Evidence of children's exposure to contaminants through house dust is well documented. Studies of lead contaminated homes have demonstrated that house dust levels and children's blood lead levels are positively correlated. It is estimated that a child's hand-to-mouth activity can account for up to two-thirds of its daily exposure to lead.

Newborn exposures to decaBDE in the home are also possible. Human milk has been studied for organohalogen contamination in Sweden since 1972 (Meironyte and Noren, 1999). While most organochlorine compounds, e.g., PCBs, DDT, dieldrin, etc., have decreased to about one-tenth of their 1972 levels, PBDE concentrations have increased about 60 times. DecaBDE levels have not been reported in the Swedish studies, however, a study of nursing mothers in the Dallas, Texas area does include decaBDE (Schechter et al., 2003). DecaBDE was detected in 7 of the 23

breast milk samples ranging between 2 and 13% of the total PBDE levels. These results also show greater total PBDE elevations compared to European levels, and raise a special concern for nursing infants.

## 4.0 HEALTH EFFECTS OF DECABROMODIPHENYL ETHER

In order to answer the questions posed in HB2572 about what health effects could result from exposure to DecaBDE, and if current levels of exposure could produce these effects, this section will concentrate on the most relevant health effects attributed to DecaBDE. Where necessary, health effects reported for the Penta- and OctaBDE formulations and individual congeners will be discussed. Surprisingly little human data are available considering the large volume of PBDEs produced and used since their introduction in the 1970s, while there have been numerous laboratory animal studies published in recent years due to the increased interest in these chemicals. Readers seeking an in-depth evaluation of the health effects of the PBDEs are referred to recent reviews of the toxicology of the PBDEs (ATSDR, 2004; Gill et al., 2004; Darnerud et al., 2002).

### 4.1 Health Effects from Exposure to DecaBDE

The toxicology of decaBDE is determined in part from its physical and chemical nature. Since all ten possible attachment positions around the diphenyl ether “backbone” of the molecule are occupied by the relatively large bromine atom, the decaBDE molecule is very large and bulky. This physical characteristic causes the molecule to be hindered in passing through the membranes of cells, resulting in oral absorption rates in rodents of less than 10% and dermal absorption much less than 1% (ATSDR, 2004). Retention of decaBDE in the body after absorption is short in most animals since it is easily excreted, with greater than 99% of the administered decaBDE eliminated after 24-72 hours (NTP, 1986; El Dareer et al., 1987). Furthermore, enzymatic removal of one or more bromine atoms from the molecule is relatively easy, aiding in the excretion from the body and also leading to the formation of metabolic breakdown products. However, the full range of these breakdown products is not clear at this time, nor is their fate in the body. Since it is possible that some of these breakdown products may be the more toxic congeners, it will be necessary to include some discussion of the toxicology of these congeners in this report.

In contrast to decaBDE, the PBDEs with lower bromine content are well absorbed after oral exposure. For example, one group of researchers (Klassen-Wehler et al., 1996; Orn and Klassen-Wehler, 1998; Hakk et al., 2002) report that greater than 90% of the congener 2,2',4,4'-tetraBDE (BDE-47) is absorbed in mice and rats, with 86% retained by the rats and 47% by the mice, and over 50% of the dose of the congener 2,2',4,4',5-pentaBDE (BDE-99) retained in rats. Thus, while nearly all decaBDE is eliminated in 1-3 days, the half-lives (the length of time required to eliminate one-half of the body burden) of tetra- to hexaBDE congeners in rats are in the range of 19-119 days (Hakk et al., 2003). The lower brominated congeners are also more likely to accumulate in fatty tissues than decaBDE.

The PBDEs exhibit very low toxicity with respect to lethality, with oral doses of thousands of milligrams of PBDE per kilogram of body weight (mg/kg) producing no or limited mortality in rodents. For example, single doses of OctaBDE (IRDC, 1974) and DecaBDE (IRDC, 1975) of 5000 mg/kg produced no deaths in rats, while the LD50 (the dose that kills 50% of test animals) for PentaBDE in rats is 5000 mg/kg (BIBRA, 1977). Doses causing adverse effects for most

organs and tissues are also relatively high for the PBDEs, therefore this report will focus on those organs and tissues for which effects are seen at lower doses. This report will also focus mainly on oral exposures due to the much larger database and the assumed primary route of most non-occupational exposures.

Liver Effects – Numerous studies of subchronic and longer duration have shown effects of PBDEs on the liver in laboratory animals, ranging in severity from enlargement without other signs of toxicity to limited evidence of carcinogenicity. It also appears that the lower-brominated PBDEs are more toxic than DecaBDE. In studies conducted in one laboratory (IRDC, 1976), the Lowest Observable Adverse Effect Level (LOAEL) for liver enlargement in rats from 28-day exposures to Penta- and OctaBDE was 9 mg/kg/d, whereas the LOAEL for DecaBDE was 90 mg/kg/d. The reduced toxicity of DecaBDE compared to lower-brominated congeners is further illustrated by studies using an earlier formulation of DecaBDE with a significant proportion of lower-brominated components versus a later formulation of higher decaBDE purity. In studies with the earlier formulation, which contained 21.8% nonaBDE and 0.8% octaBDE congeners in addition to 77.4% decaBDE, enlarged livers in rats were seen at 80 mg/kg/d for 30 days and more serious effects at 800 mg/kg/d (Norris et al., 1973, 1975a,b), whereas a study conducted with the later formulation having a decaBDE content of 94-97% showed no serious effects after 90 days exposure to 2000-8000 mg/kg/d (NTP, 1986).

USEPA has used liver effects as the basis for oral Reference Doses (RfD; an estimate of daily exposure that is thought to be without any adverse effects for a lifetime of exposure) for Penta-, Octa-, and DecaBDE (IRIS, 2005). These values reflect the relative toxicity of the lower-brominated BDEs versus DecaBDE. The RfD for PentaBDE is 0.002 mg/kg/d, based on increased levels of liver enzymes at 3.5 mg/kg/d; the RfD for OctaBDE is 0.003 mg/kg/d, based on increased liver enzymes and other cellular effects at 5 mg/kg/d; and the RfD for DecaBDE is 0.02 mg/kg/d, based on liver enlargement at 80 mg/kg/d. It should be noted that these RfDs were published in 1990 (Penta- and OctaBDE) and 1995 (DecaBDE), prior to several studies published after 1995 that may show adverse effects in other organs or systems at doses lower than those causing liver effects.

USEPA has also evaluated the cancer-causing potential of DecaBDE based on the results of 2-year studies of rats and mice exposed to very high levels of DecaBDE. In these studies (NTP, 1986), rats and mice were exposed to a DecaBDE formulation of 94-97% purity at dosages ranging from 1120-2550 mg/kg/d in rats and 3200-7780 mg/kg/d in mice. The incidence of liver neoplastic nodules (groups of cells thought to be pre-cancerous) was significantly increased in male and female rats, although the rates of liver tumors were not increased, and there was a nonsignificant trend of increased liver tumors in male mice. Based on the lack of human data and the limited evidence from rats and mice, USEPA determined that DecaBDE is a possible human carcinogen (IRIS, 2005). This conclusion has been criticized because of the very high dosages used in the studies and because the studies only produced pre-cancerous effects. It should also be noted that other cancer authorities (the US National Toxicology Program and Occupational Safety and Health Administration and the International Agency for Research on Cancer) have not at this time ranked DecaBDE for carcinogenicity.

Thyroid Effects – In an occupational study of 35 men working with DecaBDE and another flame retardant product, polybrominated biphenyls (PBBs), four were diagnosed as being hypothyroid (Bahn et al., 1980). These results can only be considered suggestive because of the low number of subjects and the lack of exposure levels. It is also not possible to determine whether exposure to DecaBDE, PBBs, or both products could be associated with hypothyroidism. However, it is also not appropriate to rule out an association with PBDE exposure and hypothyroidism because of results from animal studies.

Several studies, by both the inhalation and oral routes, have reported decreases in thyroid hormone levels after exposure to PBDEs. In an inhalation study in which rats were exposed to OctaBDE for 90 days at concentrations of 0, 1.1, 16, or 202 mg/m<sup>3</sup>, decreased levels of the thyroid hormone thyroxin (T<sub>4</sub>) were seen in both sexes at 16 and 202 mg/m<sup>3</sup>, and increased levels of thyroid stimulating hormone (TSH) were seen in males at the two highest concentrations and in females at 202 mg/m<sup>3</sup> (Great Lakes Chemical Corporation, 2001a,b). In one oral exposure study, weanling female rats were exposed for four days to DecaBDE or OctaBDE at doses ranging from 0.3-100 mg/kg/d and to PentaBDE from 0.3-300 mg/kg/d. No changes in thyroid hormone levels were found for DecaBDE, while decreases in T<sub>4</sub> were seen at 10, 30, 60, and 100 mg/kg/d for OctaBDE and 30,100, and 300 mg/kg/d for PentaBDE (Zhou et al., 2001). These authors also found decreased levels of the thyroid hormone triiodothyronine (T<sub>3</sub>) in the PentaBDE study at 100 and 300 mg/kg/d and in the OctaBDE study at 60 and 100 mg/kg/d. In another study by these authors, female rats were exposed to 1, 10, or 30 mg/kg/d of PentaBDE during days 6-21 of gestation. Both the females and their offspring were found to have decreased T<sub>4</sub> levels, with the females decreased at the 30 mg/kg/d dose and the offspring decreased at 10 and 30 mg/kg/d (Zhou et al., 2002). In another study of the effects of PentaBDE on the thyroid, mice were given either single oral doses ranging from 0.8-500 mg/kg or repeated doses of 18, 36, or 72 mg/kg/d for 14 days. In the single dose study, decreased levels of T<sub>4</sub> were seen at 0.8, 4, 20, and 500 mg/kg, but not at 100 mg/kg, and decreases in T<sub>4</sub> were seen at all three doses in the 14-day study (Fowles et al., 1994).

Decreased thyroid hormone level is not the only thyroid effect seen following exposure to PBDEs. In the DecaBDE cancer study discussed above (NTP, 1986), enlarged thyroids, a potential indication of thyroid gland toxicity, were among the effects reported for male mice after two years of exposure, although enlargement was not seen in female mice or either sex of rats. The 30-day exposure to the older formulation of DecaBDE containing lower-brominated congeners also discussed above (Norris et al., 1973, 1975a,b) found enlarged thyroids, whereas a 90-day exposure to the higher-purity formulation in the NTP study did not. In other studies, enlarged thyroids were found in rats exposed to OctaBDE at 50 (males) and 70 (females) mg/kg/d (IRDC, 1977) and PentaBDE at 100 mg/kg/d (WIL Research Laboratories, 1984). In an *in vitro* study, Meerts et al. (2000) demonstrated that PBDE breakdown products containing the hydroxyl (OH) group are able to displace T<sub>4</sub> from the blood transport protein, transthyretin, suggesting that these breakdown products may be able to disrupt normal thyroid hormone balance and function.

Thyroid hormones have been shown to play a critical role in the development of the nervous system. Clinical hypothyroidism during pregnancy has resulted in cretinism, a disorder characterized by severe mental retardation and many other physiological problems, and even

mild hypothyroidism during critical periods of gestation has been associated with developmental deficits (Porterfield, 1994; Haddow et al., 1999). Thus, the decreases in thyroid hormones in rodents may be important for the neurotoxic effects attributed to the PBDEs (discussed below). However, it must be pointed out that there are key differences between the rodent and human thyroid system, including much smaller reserve levels of hormones in the gland and more rapid turnover of circulating hormones in rodents, and primary reliance on transthyretin for transport in rodent blood versus thyroid binding globulin protein in humans (ATSDR, 2004). These differences may make prediction of human thyroid effects from rodent data problematic.

Reproductive/Developmental Effects – One of the outcomes of USEPA’s Voluntary Children’s Chemical Evaluation Program (VCCEP), discussed in Section 7.1, is the identification of data gaps in the database for reproductive and developmental effects for the PBDEs. The only reproductive effects study completed to date is a one-generation study in rats, using the DecaBDE formulation of lower decaBDE purity described in the Liver Effects section above, at doses of 3, 30, or 100 mg/kg/d. There were no effects on reproductive parameters or other adverse effects on adults or offspring reported (Dow Chemical Co., 1975; Norris et al., 1975b). As noted in USEPA’s report on the VCCEP, the database still requires a one-generation reproductive/developmental toxicity study in a second species, a two-generation reproductive/developmental toxicity study, a neurotoxicity screening battery, and the developmental neurotoxicity study that will be conducted by the European Union (discussed in Section 7.2) to fill in the reproductive/developmental effects data gaps.

There are a few recent studies in rodents that provide suggestive evidence that the lower-brominated PBDEs, at relatively low exposures, can impair the development and function of the reproductive system. Lichtensteiger et al. (2003) reported that exposure of young male rats to doses of BDE-99 as low as 1 mg/kg/d caused reduced weights of the epididymis. Also using BDE-99, Kuriyama et al. (2005) found decreases in spermatids and sperm counts in male rats whose mothers were exposed to a single dose of 0.06 mg/kg on a critical day of gestation, although these decreases did not affect the reproductive performance of the offspring in subsequent tests. In another study from this laboratory, Talsness et al. (2003) found changes in the structure of ovary cells of female rats exposed to doses as low as 0.06 mg/kg/d of BDE-99. In two studies with a PentaBDE formulation, Stoker et al. (2004, 2005) report effects on the male and female rat reproductive system. In the first study (2004), 30-day exposures to doses as low as 30 mg/kg/d prior to puberty caused decreases in the weights of seminal vesicles and prostates in males, and delays in reaching puberty in males and females. In the second study (2005) that follows up on the results found in the male rats in the first study, the delay in reaching puberty was confirmed and potentially attributed to changes in male reproductive hormone levels and blocking of the binding of these hormones to their receptor. The results of these recent studies, if proven to be reliable predictors of reproductive/developmental effects in humans, may be a cause for concern since some of the effects occurred at exposures less than those upon which the current USEPA Reference Doses for the PBDEs are based.

Neurological Effects – The PBDEs do not appear to be overtly toxic to the nervous system of adult animals, since symptoms of neurological damage have not been reported in many studies with exposures to relatively high levels for extended durations. However, there are several

studies in which neurological effects are found in animals exposed in the womb and/or postnatally.

A Swedish laboratory has reported on the effects of several PBDE congeners in a series of studies of newborn mice exposed during a critical period of neurological development called the brain growth spurt. This period is marked by extensive growth and connection of nerves in the brain, and is accompanied by the development of motor and sensory abilities (Davison and Dobbing, 1968). These studies used a similar experimental procedure in which 10-day old mice were exposed to the test chemical, and then evaluated for activity levels in an enclosure for three consecutive 20-minute periods at 2, 4, and 6 months of age. This test evaluates spontaneous behavior and habituation (a decrease in activity level over time as the animal becomes accustomed to a new environment), with the normal pattern in untreated animals being high levels of spontaneous behavior during the first 20-minute period as the animal explores its surroundings and lower levels in the last 20-minute period as it becomes used to the surroundings.

The Swedish laboratory tested BDE-47, BDE-99 (using two different strains of mice), the congener 2,2',4,4',5,5'-hexabromodiphenyl ether (BDE-153), and decaBDE (BDE-209) and found similar results for all four congeners. The treated mice displayed reduced activity levels in the first 20 minutes and increased levels in the last 20 minutes, opposite of the normal behavior, indicating impaired neurological development. The differences from the control animals also increased from the tests at 2 months to those at 4 and 6 months, suggesting that the damage is permanent. The lowest doses causing these effects for the congeners were 10.5 mg/kg for BDE-47 (Eriksson et al., 2001), 0.8 mg/kg for BDE-99 (both strains of mice; Eriksson et al., 2001; Viberg et al., 2004), 0.9 mg/kg for BDE-153 (Viberg et al., 2003a), and 2.22 mg/kg for BDE-209 (Viberg et al., 2003b). This laboratory also exposed mice to BDE-99 plus the polychlorinated biphenyl congener 2,2',5,5'-tetrachlorobiphenyl (PCB-52) at doses of 0.4 mg/kg for each chemical, that had no effects in this test when given singly but which had the same effects on activity levels as described for the BDE congeners when given together (Eriksson et al., 2003). This result suggests that PBDEs and PCBs affect the same target(s) in the nervous system and that the impacts are additive.

It is necessary to provide additional discussion of the Viberg et al. (2003a) study with decaBDE. In contrast to the studies with the other three BDE congeners, in which dosing occurred only at age 10 days, in this study the mice were dosed at age 3, 10, and 19 days. Effects were only seen in mice dosed at age 3 days, strongly suggesting that a breakdown product(s) is the active compound since the parent decaBDE is inactive at age 10 and 19 days and uptake of decaBDE into the brain in this study did not occur until age 7 days. This result illustrates the importance of identifying key decaBDE breakdown products in the body and in the environment.

It must also be pointed out that this study, in particular, and this testing procedure, in general, have been criticized for certain procedural and statistical problems (Piccirillo, 2003; DeSesso and Mavis, 2003). Among other things, the decaBDE study is criticized for:

- selecting mice from only 3-4 litters per treatment group instead of at random from all available litters, as is common for other studies;

- calculating study statistics on the basis of individuals instead of litters, as is common in other studies;
- dosing the mice at age 3 days when mice from the other congener tests were dosed at 10 days, which may cause problems due to dosing at such an early age and for which little historical data are available for reference;
- the method used to calculate the uptake of decaBDE into the brain; and
- the lack of positive controls.

The general testing procedure is criticized for using doses much greater than the anticipated range of human exposures, and questioned as to the biological relevance of the endpoints measured in the test for predicting effects in humans. These criticisms raise valid points, although it should be noted that the effects reported in the studies are consistent and the statistical evaluations still show significant differences, thus the effects are likely not spurious. They also highlight the critical importance of the planned full developmental neurotoxicity study to be conducted by the European Union to address some of these issues.

There are several other studies that suggest that the lower-brominated BDEs can affect the developing nervous system. In a study similar to those conducted by the Swedish laboratory but measuring activity levels and neurological development with a different testing protocol, Branchi et al. (2002) also found neurological effects in the offspring of treated mice. Mice were exposed to 0.6, 6, or 30 mg/kg/d of BDE-99 from day 6 of pregnancy through day 21 post-pregnancy, and their offspring were subjected to various neurodevelopmental tests at ages 2-20 days. Activity levels were subsequently measured at ages 22, 34, 60, and 120 days. Delays in the neurodevelopmental tests were seen at the 30 mg/kg/d dose, and spontaneous activity and habituation changes were seen at 6 and 30 mg/kg/d. In the Kuriyama et al. (2005) study discussed above regarding reproductive/developmental effects, the authors also evaluated the neurological effects of BDE-99 in the offspring of rats whose mothers were given doses of 0.06 or 0.3 mg/kg on day 6 of pregnancy and tested for activity levels at ages 36 and 71 days. In this portion of the study, the offspring were found to be hyperactive on day 36 at the 0.3 mg/kg/d dose and at both doses at 71 days. In another study, rats were exposed for one week to 30 mg/kg/d of a PentaBDE formulation beginning at age 6 days and subjected to visual recognition (a test of learning ability) and sustained attention tests at ages 30-83 days. The rats were found to have deficits in the visual recognition tests (Dufault et al., 2005). As was the case in the discussion of reproductive/developmental effects, if these neurological effects are proven to be reliable predictors of neurological effects in humans, this may be a cause for concern since some of these effects occur at doses less than those upon which the current Reference Doses are based.

A summary of the information presented above appears in Table 2.

#### **4.2 Are Current Levels of Exposure at Levels That Could Produce Effects?**

This is a difficult question to answer, especially regarding decaBDE. Significant problems in measuring or estimating levels of exposure to the PBDEs result in a fairly large level of uncertainty. Specific to decaBDE, there is a notable lack of quantification of decaBDE in studies monitoring the presence of PBDEs in the environment and in humans (due to analytical problems in earlier studies and also due to the often low decaBDE levels in some environmental

compartments), and as discussed above the identity and fate of decaBDE breakdown products is a major data gap. Regarding PBDEs in general, studies often do not include key parameters needed to do an in-depth exposure assessment, such as BDE concentrations in fat, blood, and milk in mothers and fat and blood concentrations in offspring, and BDE exposures due to non-dietary sources can be difficult to estimate. Nevertheless, this report will evaluate current levels of PBDE exposures and compare them against levels shown to cause health effects in animals.

One recent review contains a summary of published estimates of human exposures to PBDEs. The Washington State PBDE Chemical Action Plan (2005) has summarized the efforts of several recent studies to estimate daily human intake of PBDEs from different countries. These estimates range from an adult daily intake of 0.00000019 mg/kg/d in the Netherlands from food (sum of 6 BDE congeners, not including decaBDE) to 0.0026 mg/kg/d for a child's intake from air, water, food, breast milk, and house dust in Canada (sum of tetra- to decaBDE congeners). In the four studies from the United States, intake estimates ranged from 0.000003 mg/kg/d for adults from food to 0.000355 mg/kg/d for nursing infants from breast milk (multiple congeners, including decaBDE). In another recent study, McDonald (2005) derived estimates of daily PBDE intake (five congeners, not including decaBDE) from various sources in four countries, with the U.S. estimate being the highest at 0.000016 mg/kg/d. This estimate is within the range of the four U.S. studies cited in the Washington Chemical Action Plan.

Two other recent studies reporting the results of monitoring PBDE levels in humans in the United States can be used, with reasonable assumptions, to estimate the daily intake of PBDEs that would result in these levels. Gill et al. (2004) evaluated the levels of five BDE congeners (not including decaBDE) reported in studies from several countries, including a study by She et al. (2002) that found an average and maximum PBDE concentration in the breast fat of US women of 0.086 and 0.46 mg/kg, respectively. In the McDonald (2005) study cited above, the author estimated from several studies of PBDE levels in blood, milk, and fat that a daily PBDE intake of 0.177 nanogram per kilogram of body weight (ng/kg/d) will result in a PBDE content in fat of 0.001 mg/kg. Applying this ratio to the She et al. fat concentrations results in an estimate of daily PBDE exposure of approximately 15 and 82 ng/kg/d, or 0.000015-0.000082 mg/kg/d, for the average and maximum fat contents, respectively. These values fall well within the range of the U.S. studies in the Washington report.

In the other recent study, the Maine (2005) report on brominated flame retardants summarized measured levels of total PBDEs in breast milk from several countries, including four studies from the U.S. The U.S. studies had milk PBDE levels ranging from 6.2-1078 micrograms of PBDEs per kilogram (ug/kg) of milk fat. An infant's daily intake of PBDEs can be estimated from these values, using the average content of milk fat of 3.7% and an infant's average daily milk consumption of 0.7 kilograms to approximate the infant's daily intake of milk fat of 0.026 kilogram per day. Multiplying the milk fat intake by the milk fat PBDE levels, and assuming an average infant's weight to be 10 kilograms, results in an estimated range of daily intakes of 0.016-2.8 ug/kg/d, or 0.000016-0.0028 mg/kg/d. The larger value is nearly identical to the estimated child's intake from all sources in Canada included in the Washington report, and is greater than the value estimated for the nursing infant in the U.S. study cited in that report.

One other recent report can be used to estimate a child's daily PBDE intake from house dust ingestion. Stapleton et al. (2005) estimate that a child's dust ingestion of 0.02-0.2 grams per day from ages 1-4 years could result in a PBDE intake of 120-1180 nanograms per day, based on their average of 5900 nanograms of PBDEs per gram of dust. Assuming an average weight for ages 1-4 of 15 kilograms, the daily PBDE intake would be in the range of 8-80 ng/kg/d, or 0.000008-0.00008 mg/kg/d. Stapleton et al. also report that an average of 40% of the total PBDE content is decaBDE, which results in an intake of decaBDE of 0.0000032-0.000032 mg/kg/d.

The above results for PBDE daily intakes are summarized in Table 3. As can be seen, there is a wide range of estimated intakes, ranging from 0.00000019-0.0028 mg/kg/d or an almost 15,000-fold difference. Some of this variability can be explained by the differences in sources of the PBDEs and in the congeners evaluated, but there is also a true difference in the daily exposures experienced by people. In order to compare these daily intakes to intakes that have resulted in health effects, this report will concentrate on the values from the upper end of the range of daily intakes.

From the studies evaluating multiple routes of exposure in U.S. children and adults, the estimates of daily exposure are fairly consistent, in the range of 0.000014-0.0009 mg/kg/d. These values suggest that there is very little concern for liver toxicity, when the values are compared to USEPA's Reference Doses for the PBDEs, since the upper end of the intake range is less than the lower end of the range of RfDs (0.002-0.02 mg/kg/d). As noted above, the RfDs are based on liver effects, and daily exposures less than the RfDs are assumed to have no effects. However, as also noted above, some of the studies described for thyroid, reproductive/developmental, and neurological effects have found health effects at exposures less than the effect levels used to develop the RfDs. Among these studies, the lowest exposure shown to have an effect is 0.06 mg/kg. The Kuriyama et al. (2005) study found decreases in spermatids and sperm count at puberty and hyperactivity at age 71 days in male rats whose mothers had been given 0.06 mg/kg of BDE-99 on day 6 of pregnancy. The Talsness et al. (2003) study found changes in the structure of ovary cells of female rats, also given 0.06 mg/kg/d of BDE-99. In both studies, 0.06 mg/kg was the lowest dose given, thus a No Observable Adverse Effect Level (NOAEL) has not been determined for these effects. This raises the concern that exposures in the range of those estimated for infants, as high as 0.0028 mg/kg/d, could be at a level that could produce these effects as the child reaches puberty. It must be reiterated that the results from these studies must be shown to be reliable predictors of human effects before this concern can be fully evaluated.

## 5.0 BREAKDOWN OF DECABROMODIPHENYL ETHER

The fourth issue raised in HB2572 concerns whether decaBDE breaks down into more harmful chemicals that could damage public health. In the past, it had been thought that decaBDE in the environment was resistant to breakdown due to its chemical structure and unreactive nature. However, this issue has been the subject of considerable research over the past several years, with studies examining the fate of decaBDE in the environment and in organisms. This section briefly summarizes the ways that decaBDE is broken down and what it breaks down to, and whether the breakdown products can damage public health.

### 5.1 Breakdown Mechanisms and Products

Sunlight – Numerous studies have demonstrated that decaBDE can be broken down by ultraviolet light and direct sunlight. In this reaction, the energy from the light dislodges bromine from the diphenyl ether backbone, creating a BDE congener with fewer bromines and, potentially, also other brominated compounds. This reaction is faster when decaBDE is present in organic solvents and slower when in water. For example, in laboratory studies in which decaBDE was dissolved in xylene one-half of the initial concentration was broken down within 15 hours, whereas three months were required to break down decaBDE in water (Norris et al., 1973). Palm et al. (2002) report similarly fast breakdown for decaBDE dissolved in toluene, with complete disappearance of decaBDE in sunlight after two days. These authors identified penta- through nonaBDE congeners as breakdown products. When decaBDE was dissolved in a mixture of hexane, benzene, and acetone it was found to break down quickly into a mixture of congeners having 3-8 bromines (Watanabe and Tatsukawa, 1987). The latter study also identified brominated dibenzofurans among the breakdown products, raising an additional issue for potential concern since these chemicals may have dioxin-like activity.

In a report to the Brominated Flame Retardant Industry Panel, Jafvert and Hua (2001) conducted a series of studies on the light-induced breakdown of decaBDE in various scenarios. In studies with natural sunlight, the authors found minimal if any breakdown when decaBDE was adsorbed to sterile sand, and very little change when humic acids (natural organic acids present in surface waters) were added to the sand. However, when decaBDE was applied by water to clear quartz tubes, approximately 50% of the decaBDE was broken down after 72 hours. When decaBDE was applied by water plus humic acids, the breakdown rate was slowed to only about 10% after 72 hours. When the quartz tube studies were conducted using ultraviolet light, the breakdown rates were faster, with approximately 70% of the decaBDE applied by water broken down after 60 hours. The authors found small concentrations of nona- and octaBDE congeners and no tetra- or pentaBDEs, as well as a number of unidentified products, were produced as a result of the breakdown of decaBDE.

In another study, Stapleton (2005) evaluated the breakdown in sunlight of decaBDE added to house dust and found a 30% decrease in decaBDE after 90 hours. The author reported that 83% of the breakdown products were lower-brominated congeners and 17% of the loss could not be accounted for. Hepta-, octa-, and nonaBDE congeners were identified as breakdown products. This study is being repeated with a longer exposure to sunlight to determine if additional

breakdown products can be identified. House dust in which the decaBDE is present “naturally” instead of being added to the dust in the laboratory will also be used to determine if there are differences in the breakdown rate and/or products.

Metabolism – Several studies have demonstrated that decaBDE can be broken down as a result of metabolic processes in animals and microorganisms. As discussed in Section 2.2, rainbow trout (Kierkegaard et al., 1999) and carp (Stapleton et al., 2004) were exposed by diet to decaBDE and the build-up and breakdown of decaBDE was followed for several months. Hexa- through nonaBDE congeners were found in trout and penta- through nonaBDEs were found in carp. Morck et al. (2003) have identified several breakdown products in the bile, feces, liver, and intestines of rats dosed with decaBDE, containing hydroxyl structures indicative of enzymatic breakdown. Similar results have been reported by Sandholm et al. (2003), who found hydroxyl breakdown products in the blood of rats dosed with decaBDE. DecaBDE has been shown to be broken down to octa- and nonaBDE congeners by microbes in sewage sludge (Gerecke et al., 2005). In contrast to the studies with animals described above, in this study the breakdown occurred in the absence of oxygen, suggesting a different metabolic pathway for removal of bromine from decaBDE. This pathway may be indicative that decaBDE can also be broken down in other anaerobic compartments of the environment, such as some soils and sediments.

## **5.2 Potential for DecaBDE Breakdown Products to Damage Public Health**

At this time, there is considerable uncertainty and controversy regarding the extent of breakdown of decaBDE and what are its stable breakdown products. It appears that there is consensus that decaBDE breaks down in ultraviolet and natural light, and that nona- octa- and heptaBDE congeners are formed. The identification of two heptaBDE congeners (BDE-181 and BDE-190; Rice et al., 2002) in fish that have not been identified in any commercial BDE formulation strongly suggests that these two congeners are breakdown products of decaBDE. However, there is disagreement whether any of the more toxic lower-brominated congeners are also produced by light under environmentally relevant conditions (i.e., other than in the presence of organic solvents), and whether brominated dioxins and furans are produced in meaningful quantities in light. Thus, it is unclear to what extent the presence of the more toxic lower-brominated BDEs is due to the use of the Penta- and OctaBDE formulations and what derives from the breakdown of decaBDE.

It is also unclear at this time what potential harm might derive from the metabolic breakdown products of decaBDE. It has been theorized that the addition of hydroxyl groups to the PBDEs might impart a structure that is similar to estrogen and/or the thyroid hormones (Birnbaum and Staskal, 2004), possibly leading to disruption of the normal function of these hormones. Further research would be necessary to evaluate this possibility. Additional studies of the environmental fate of decaBDE are on-going or planned in the near future, which will provide important information on which breakdown products can accumulate in the environment. Therefore, we believe that the information available at this time regarding decaBDE’s breakdown products is not sufficient to allow us to confidently answer this question.

## 6.0 FLAME RETARDANT ALTERNATIVES

This section addresses the last issues posed to the Agency in HB2572, which asks the following questions:

- Are there effective flame retardants available for current uses of Decabromodiphenyl ether?
- Will the use of available alternatives reduce health risks while still maintaining an adequate level of flame retardant performance?

DecaBDE is known to leach out of plastic and textile materials, and enter the environment and food chain (Lowell, 2005). As discussed in the previous section, decaBDE can then be broken down in sunlight and metabolized by living organisms into additional brominated compounds, which may or may not be a cause for health concern. This section examines whether alternatives are available to potentially reduce this load of brominated compounds in the environment and organisms.

### 6.1 Background

The term “brominated flame retardant” covers a large number of organic substances, all with bromine in their molecular structure. Bromine has an inhibitory effect on the formation and propagation of fire in organic materials. The most widely used substances among the brominated retardants are Tetrabromobisphenol A (TBBPA), polybrominated biphenyls (PBBs), and the three PBDEs already described in this report (Danish EPA 1999).

There are three general flame retardant alternatives to using PBDEs (Washington Department of Health, 2005):

- Substituting non-brominated chemical additives
- Substituting product materials that don't require PBDEs
- Changing design and construction of products so they are inherently less flammable

Research shows that the flame retardant of concern, DecaBDE, is used in two primary arenas, electronics enclosures and textiles. There are some small fractional uses of DecaBDE, but for purposes of this section, the focus will be on the majority usage of DecaBDE in electronics and the minority usage in textiles.

The Lowell Center for Sustainable Production, at the University of Massachusetts at Lowell, reports in their March 2005 investigation that roughly 80% of the DecaBDE used in the United States is believed to be in electronics. The primary use for this flame retardant is in the black plastic high impact polystyrene (HIPS) electronic enclosures used in the rear of television sets. DecaBDE is an inexpensive, highly efficient flame retardant, and highly compatible with the inexpensive HIPS. It should be noted that only a small percentage of DecaBDE treated HIPS is used in computer monitors.

Approximately 10 to 20 percent of the flame retardant is used in the textile industry with the primary uses being mattresses, drapery, commercial upholstered furniture, and transportation, particularly the automotive and airline industries. The remaining textile applications include tents, awnings, and related fabric applications (Lowell, 2005).

The remaining minor DecaBDE applications include rubber products, wire and cable, and uses in paper and mineral wool. The Danish Environmental Protection Agency identified DecaBDE applications in sockets for incandescent and fluorescent lighting, wall sockets and mounting boxes for house wiring, and relays, circuit breakers, contactors and starters used in power supply systems and for industrial automation (Danish EPA, 1999). Further applications of DecaBDE are found in expanded polystyrene and extruded polystyrene foam building materials, rigid polyurethanes, and foils for roofing materials (Lowell, 2005). These applications are considered to be a small fraction of DecaBDE use and will not be considered for this section.

## **6.2 Electronic Enclosure Plastics**

At the present time, it appears questionable whether cost-effective non-halogen flame retardants are available for use in electronic enclosures, although it is beyond the Agency's expertise to conduct cost-effectiveness evaluations. There are a number of phosphorus-based flame retardants that can serve as effective non-halogen substitutes in HIPS blends and other resin systems, and certain inorganic compounds can be used also. The Lowell Center lists four of the chief non-halogen flame retardants for use with HIPS and other resins in electronic enclosures, the organic phosphorus compounds:

- Triphenyl phosphate (TTP)
- Resorcinol bis(diphenylphosphate) (RDP)
- Bisphenol A diphosphate (BAPP)
- Bisphenol A bis diphenyl phosphate (BDP)

However, the non-halogen systems are more costly than DecaBDE HIPS systems. For example, the organic phosphorus-based compounds increase the cost of the front and rear enclosure for an average 27-inch television by 57%. Thus, the higher cost and lack of regulatory drivers has limited the widespread adoption of phosphorus-based flame retardants in the United States (Lowell, 2005).

There are substitute resin systems available for HIPS which expand the availability of alternatives to utilizing DecaBDE HIPS. The Lowell Center reports that the most cost-effective substitute for DecaBDE HIPS involves changing the resin system and the use of phosphorus-based flame retardants, and lists the three following resin substitutes:

- Blends of polycarbonate and acrylonitrile-butadiene-styrene (PC/ABS)
- Polycarbonate (PC)
- Blends of HIPS and polycarbonate oxide (HIPS/PPO)

Regarding the toxicity of alternatives to DecaBDE in electronics enclosures, there were few human health concerns noted from the studies available for these compounds. However, more data on human and environmental toxicity of the compounds is needed. For example, the four organic phosphorus compounds listed above as potential alternatives for electronics enclosures appear to suffer some of the same database deficiencies as decaBDE regarding reproductive/developmental and neurological effects. In addition, issues with aquatic toxicity were noted since the compounds can be released into the environment and affect algae, invertebrate, fish and other aquatic species (Lowell, 2005).

### 6.3 Textiles

Compared to electronics enclosures, there are many more options for DecaBDE substitution in textiles. Strategies for substituting DecaBDE in textiles include the redesign of products to reduce their fuel load by eliminating the use of foam (i.e., office chairs such as the Aeron from Herman Miller contain no polyurethane foam). This approach provides a comfortable product without contributing to the fuel load, and is a potential research and innovation area in the furniture industry (Lowell, 2005). Other strategies include the application of other inorganic and organic chemical flame retardants, the incorporation of barrier layers in products, and utilizing inherently fire-resistant fabrics.

There are several chemically applied non-halogen DecaBDE substitutes available for natural cellulose fibers such as cotton, wool, rayon, and linen. They include:

- Ammonium polyphosphates
- Dimethylphosphono (N-methylol) propionamide
- Phosphonic acids such as (3-{[hydroxymethyl]amino}-3oxopropyl)-dimethyl ester
- Tetrakis (hydroxymethyl) phosphonium urea ammonium salt

In the case of chemically applied flame retardants, natural fibers tend to be easier to flame retard than synthetic. Some DecaBDE substitutes exist for synthetics, but their water solubility results in limited durability as they “wash out” during laundering. In these cases, dry cleaning may be required. One approach is to use a blend of natural and synthetic fibers since the natural fibers are more effectively flame retarded. Some fire resistant fibers require no added flame retardant, and these inherently fire resistant fibers can be used as DecaBDE substitutes for high durability synthetic fibers. In addition, some synthetic fibers can be made fire resistant through the addition of non-halogen phosphorus-based additives. These previous two approaches can be applied to draperies used in public places required to meet flame retardant standards. Another substitution approach involves the use of fire barriers. The barrier technology is “sandwiched” between the surface fabric and the interior foam core in furniture and mattress construction or bonded to the back of fabrics to achieve fire standard compliance (Lowell, 2005).

Unfortunately, there is little definitive information about the costs related to DecaBDE substitutes/alternatives for fabrics, since they are very application-specific and difficult to generalize. In addition, few manufacturers provide data on the costs of alternatives. One manufacturer, which did provide data to the Lowell Center, revealed that phosphate-based

replacements cost 2 to 2.5 times as much as DecaBDE. Other concerns with textile substitute flame retardants include considerations of product comfort and wear and tear.

As was the case with DecaBDE substitutes for electronics enclosures, the human and ecological toxicity risks associated with the DecaBDE substitutes for fabrics are not well known, despite their widespread, and in some cases, long term use (Lowell, 2005). However, based on the data available, the phosphorus-based alternatives appear to have fewer health and environmental concerns in general than the PBDEs, since they do not appear to be bioaccumulative or break down into toxic and bioaccumulative chemicals, and the inorganic chemicals also appear to have few toxicity concerns.

As a final note, we raise one other potential issue that may become an indirect consequence of switching to phosphorus-based alternatives to DecaBDE, in cases where fires occur in spite of flame-retarded materials. In all fires, smoke and carbon monoxide are generated and are responsible for many fire-related deaths and injuries. In addition, many other irritant gases are generated during a fire, and these gases can contribute to the adverse effects experienced by fire victims. For example, hydrogen chloride and hydrogen bromide are potent irritants formed in fires involving materials containing chlorine and bromine (including from flame retardant use). Similarly, fires involving materials that have phosphorus-containing compounds may produce phosphine gas, which is not only an irritant but also a highly toxic gas with known human fatalities due to accidental exposures from its use as a pesticide. For purposes of comparing the acute lethality of hydrogen bromide versus phosphine gases, we note that the one-hour LC-50 (the concentration in air that results in 50% mortality during a one-hour exposure) in rats is 2858 part per million (ppm) for hydrogen bromide (MacEwen and Vernot, 1972) and 134 ppm for phosphine (Muthu, 1980). Based on these LC-50s, phosphine is at minimum 21 times as acutely toxic as hydrogen bromide gas. We are concerned that a widespread switch to phosphorus-based flame retardants could result in an increase in fire-related deaths and injuries due to the increased level of the toxicity of gases generated during fires. We have searched for information on the concentrations of phosphine that may result from the various stages of a fire, but to date searches of the literature and inquiries to the U.S. Consumer Product Safety Commission have produced no results.

In summary, the Agency's review of DecaBDE alternatives permits us to respond, tentatively, to the two issues raised in HB2572 in the affirmative. There are effective flame retardant alternatives to most DecaBDE uses, although the cost-effectiveness of these alternatives is beyond the Agency's expertise to determine. Based on the toxicity data available, use of the alternatives will likely reduce risks while maintaining an adequate level of flame retardant performance. Further research is needed, however, to better evaluate the potential health and environmental consequences of many of the major DecaBDE alternatives.

## **7.0 OTHER JURISDICTIONS' ACTIONS REGARDING PBDES**

In order to provide some perspective on concerns regarding the PBDEs, we have reviewed actions taken by several jurisdictions to evaluate and/or regulate exposures to PBDEs. Summaries of these actions are presented for information purposes. The actions of the states of Washington and Maine are presented in some depth, as representative of most of the other states' findings/actions, while the remaining states' legislative actions will be briefly summarized.

### **7.1 USEPA's Voluntary Children's Chemical Evaluation Program (VCCEP)**

The VCCEP is a voluntary pilot program that is part of USEPA's Chemical Right-to-Know Initiative. This Initiative's goal is to assist the public to better understand the risks to children from chemical exposures, and the VCCEP pilot addresses 23 high-priority chemicals that have been identified in humans and the environment. The primary question to be addressed by the VCCEP is whether children's exposures and risks have been adequately characterized, and, if not, what additional data are necessary. Companies that manufacture or import these chemicals are asked to volunteer to develop the appropriate exposure and health effects information, and integrate the information into a risk assessment and data needs evaluation. These assessments are conducted in a tiered manner, with the first tier consisting of all available data for the exposure assessment and a list of basic toxicity information for the health effects assessment. Tiers 2 and 3 for the exposure assessment require increasingly in-depth data, while the requirements for health effects data in these tiers include specific types of toxicity tests that follow up on the basic data.

The PBDEs are one of the VCCEP high-priority chemicals, and the Brominated Flame Retardant Industry Panel (BFRIP) volunteered to conduct the Tier 1 evaluations for Penta-, Octa-, and DecaBDE. The April 2003 BFRIP report on DecaBDE was reviewed by a panel of independent experts, and this panel issued a report containing a range of opinions regarding the exposure and health effects assessments. USEPA then reviewed the BFRIP and panel reports, and issued its own report in August 2005 (VCCEP, 2005). The conclusions of the USEPA review of the exposure assessment recommended a Tier 2 assessment to further evaluate the environmental transport and fate of DecaBDE, especially in the indoor environment, and BFRIP has indicated that they will conduct the studies. The review of the health effects assessment identified several data gaps, and noted that the data need for a Tier 3 developmental neurotoxicity study will be filled by a study to be conducted by the European Union as part of its evaluation of DecaBDE (see below). The report also concluded that the other identified data gaps would not need to be addressed unless the results of the Tier 2 exposure assessment change the exposure estimates. For more information about the VCCEP, the reader may turn to the USEPA website (<http://www.epa.gov/chemrtk/vccep/childhlt.htm>).

### **7.2 European Union**

The European Union (EU) has taken two actions with regard to the PBDEs. In February 2003 the EU passed Directive 2003/11/EC, which banned the marketing and use of all products

containing more than 1% by weight of PentaBDE and OctaBDE by August 15, 2004. In January 2003 the EU passed Directive 2002/95/EC, the “Restriction of Certain Hazardous Substances to Electrical and Electronic Equipment” (RoHS), which contains a list of chemicals to be phased out of use in these types of equipment by July 1, 2006. The PBDEs are included in this list. Since the Penta- and OctaBDE formulations are already banned by Directive 2003/11/EC, there has been no concern with the inclusion of these PBDEs in the RoHS Directive. However, the issues regarding DecaBDE are more complicated.

The RoHS Directive contains a provision allowing for an exemption from the Directive when substitutes for a product are shown to be technically or scientifically impractical or where other negative impacts (health, environmental, or consumer safety) deriving from use of a substitute would outweigh the benefits. An exemption was proposed by DecaBDE producers in March 2005, based in part on the results of updates to the human health (February 2004; European Commission, 2004a) and environmental (May 2004; European Commission, 2004b) risk assessments conducted pursuant to the EU’s 1993 Existing Substances Regulation (793/93/EEC). Both risk assessment updates followed up on the original 2002 assessments that had identified significant data gaps regarding DecaBDE, and both updates concluded that there were at that time no risks that required further regulations regarding DecaBDE.

In March 2005, the Directorate General for the Environment for the European Commission accepted the DecaBDE producers’ arguments and proposed an exemption from the RoHS Directive. The proposed exemption was rejected by the RoHS Technical Advisory Committee, but the European Commission re-introduced the proposal for exemption in June 2005. Following this re-introduction several Member States opposed the exemption, and the European Parliament also voted to continue the phase-out of DecaBDE. However, the vote by the EU Council of Environment Ministers to reject the exemption failed to reach a qualified majority (72.3%), and the European Commission finalized the exemption on October 15, 2005. By provision of the RoHS Directive, all exemptions are to be reviewed after four years, and the DecaBDE exemption will be reviewed in 2009.

There are some further complications to the EU DecaBDE story. Even though the update to the human health risk assessment did not call for further restrictions on DecaBDE, the EU as part of the Existing Substances Regulation has required a developmental neurotoxicity study and a survey of DecaBDE in human blood and breast milk to follow up on identified data gaps. Similarly, while the update to the environmental risk assessment did not call for further regulation of DecaBDE, it did call for further data to determine whether this chemical is persistent, bioaccumulative, and toxic, or whether DecaBDE breaks down into other chemicals that are more bioaccumulative and toxic. Also regarding the 2004 update to the environmental risk assessment, the Scientific Committee on Health and Environmental Risks (SCHER), an advisory committee to the European Commission, released their review of this assessment in March 2005, finding among other things that recent studies have provided evidence that DecaBDE does break down to more harmful compounds and should be subject to risk reduction measures. The SCHER findings and the calls for additional data in the 2004 update are among the reasons for the European Parliament’s vote to continue the DecaBDE phase-out. Furthermore, the proposal for exemption does not contain a comparative assessment of potential substitutes, as required by the RoHS Directive, so the exemption may be taken before the

European Court of Justice. As of the date of this report to the Legislature, there is no further information about potential legal action regarding the DecaBDE exemption from the RoHS Directive in the EU.

### **7.3 Washington**

In January 2004, Governor Locke issued an executive order directing the Departments of Ecology and Health to develop a plan to reduce the threat of PBDEs in the environment. Following a Public Comment Draft (October 2004) and Interim Draft (December 2004), the “Washington State Polybrominated Diphenyl Ether (PBDE) Chemical Action Plan: Draft Final Plan” was issued on December 1, 2005. Among the Plan’s findings:

- PBDEs have been found in the environment, foods, and people, with the highest levels in people occurring in Canada and the U.S.;
- The Penta- and OctaBDE formulations are at higher levels in people and food, while DecaBDE is higher in sediments and household dust;
- PBDE exposure in the womb can disrupt neurological development in rodents, but no data are available for humans, and thyroid, liver, and reproductive effects are also seen in laboratory animals;
- Penta- and OctaBDE are the primary concerns for the above health effects while DecaBDE is the least toxic, however, there is concern that DecaBDE may break down into the Penta- and OctaBDE congeners;
- PBDE levels in people are not yet at levels shown to be toxic in laboratory animals and are not an immediate health threat.

Because of these findings, the Plan recommends among other things that:

- The Legislature should prohibit the manufacture, sale, or distribution of new products containing Penta- and OctaBDE by July 2006;
- The Legislature should also ban DecaBDE if safer alternatives are identified or if additional evidence of harmful effects emerges, and if safer alternatives cannot be identified then incentives/disincentives should be developed to encourage development of safer alternatives or product design changes that eliminate the need for PBDEs;
- The Department of Ecology should establish appropriate recycling or disposal practices for PBDE-containing products at their end-of-life by July 2006;
- The Department of Health should develop public education and outreach programs to help the public minimize exposure to PBDEs;
- The Departments of Health and Labor and Industries should determine ways to minimize workplace exposures to PBDEs.

### **7.4 Maine**

In 2003, the Maine Legislature passed “An Act to Reduce Contamination of Breast Milk and the Environment from Release of Brominated Chemicals in Consumer Products.” This Act prohibits the sale of products containing more than 1% of the Penta- and OctaBDE formulations by January 1, 2006, and also prohibits the sale of products containing more than 1% of DecaBDE by

January 1, 2008 if safer, nationally available alternatives are identified. The Act also requires the Departments of Environmental Protection and Human Services (Bureau of Health) to review relevant information concerning brominated flame retardants and report their findings annually to the Legislature, beginning in January 2005. The first annual report was issued in February 2005. Among the findings are several relevant to DecaBDE:

- DecaBDE is ubiquitous in the environment, being found in wildlife, sewage sludge, indoor air and dust, food, and human tissues;
- DecaBDE bioaccumulates and concentrates up the food chain;
- DecaBDE levels are much higher in the U.S. than in Europe;
- DecaBDE is neurotoxic in developing rodents;
- Total PBDE levels in humans are not yet at levels shown to be harmful to rodents, and DecaBDE's contribution to total PBDE body burdens is unclear now due to incomplete data on the breakdown of DecaBDE;
- DecaBDE is broken down by sunlight and living organisms but the full identity and fate of the breakdown products in the environment are not well studied at present, suggesting that DecaBDE's contribution to total PBDE levels may be underestimated;
- Alternatives are available for all DecaBDE uses, although they are more costly, and they do not appear to have human and environmental concerns as great as DecaBDE.

As a result of these findings, the report recommends that the Legislature keep in place the 2008 ban on DecaBDE while the Departments continue to review the on-going and planned studies of this chemical's toxicity, environmental transport and fate, and alternatives.

## **7.5 California**

In August 2003, the Legislature passed AB302, which prohibits the manufacture, processing, or distribution of products containing more than 0.1% of the Penta- and OctaBDE formulations by January 2008, and requires the Senate Office of Research to submit recommendations for further regulation of the PBDEs. This report was submitted to the Senate in June 2004, and its findings include an opinion that DecaBDE is not presently occurring at levels thought to be unsafe for humans or the environment, although danger could not be ruled out because of uncertainties in the toxicity and exposure data. This report also recommends that the Office of Environmental Health and Hazard Assessment develop a reference dose for DecaBDE and that the state develop a program to monitor PBDE levels in breast milk. The 2008 effective date of the Penta- and OctaBDE ban has since been moved up to June 2006 by AB 2587, passed by the Legislature in 2004.

## **7.6 Michigan**

The Toxics Steering Group of the Department of Environmental Quality (DEQ) issued in January 2004 "Polybrominated Diphenyl Ethers (PBDEs): Background Paper" at the request of DEQ management, to assist management's decision on whether to place PBDEs on Michigan's Critical Materials Register and help form their response to proposed legislation to ban PBDEs in Michigan. This report found that the Penta- and OctaBDE formulations presented unacceptable risks, based on the results seen in laboratory animals and on the increasing levels found in the

environment, wildlife, and human blood and breast milk. The report found limited data for DecaBDE but expressed concerns about the potential for neurotoxicity, and recommended that its presence in humans warranted continued review of the literature. The report also recommended legislation to ban Penta- and OctaBDE and continued monitoring of PBDEs in the environment. As a result, HB 4406 and SB 1458 were passed by the Legislature in January 2005 banning the manufacture, processing, or distribution of products containing more than 0.1% of Penta- or OctaBDE by June 2006. The ban excludes replacement parts and the processing of recyclable products.

### **7.7 Hawaii**

In June 2004, the Legislature passed HB 2013/SD2/CD1, which bans the manufacture, processing, or distribution of products containing more than 0.1% of Penta- or OctaBDE by January 2006. This legislation contains an exemption for the recycling of metal products containing PBDEs.

### **7.8 Maryland**

In May 2005, the Legislature passed HB83, which prohibits the sale, manufacture, processing, or distribution of products containing Penta- or OctaBDE by October 1, 2008. The legislation also requires the Department of Environment to report on uses of DecaBDE and evaluate potential restrictions on its sale and use by January 8, 2007.

### **7.9 New York**

In August 2004, the Legislature passed A 10050/S 7621, which prohibits the manufacture, processing, or distribution of Penta- and OctaBDE, but does not prohibit the sale or use of products containing these chemicals. The legislation also establishes a Task Force on Flame Retardant Safety to study potential risks of and alternatives to DecaBDE.

## **8.0 SUMMARY**

This report has been prepared to address the five issues posed by the Illinois Legislature to the Illinois Environmental Protection Agency in HB2572 regarding the use of Decabromodiphenyl ether. The Agency has tried to respond to these issues in as thorough a manner as possible, relying on very recent information where available. However, data gaps exist in certain key areas that have hampered our ability to fully address some issues. This section summarizes our findings and presents the Agency's responses to the five issues.

### **8.1 Bioaccumulation in the Environment**

The first issue to be addressed is “whether DecaBDE is bio-accumulating in the environment, and if so, whether the levels of DecaBDE are increasing, decreasing, or staying the same.” Numerous studies, some that include data from Illinois, have documented the presence of the PBDEs in various compartments of the environment. DecaBDE has been found in particulates collected in air samples, with the Chicago sample being the highest of the samples analyzed. DecaBDE has also been found at relatively high levels in sewage sludge and sediments, while much lower levels have been found in most samples of soil and water. Other studies have reported relatively high levels of decaBDE in house dust, with decaBDE usually being the predominant BDE congener detected.

DecaBDE has also been detected in samples from various organisms, including humans. Low levels have been reported for most fish species sampled, although DecaBDE levels in sharks tend to be higher. Birds that feed on terrestrial prey can accumulate decaBDE, and decaBDE can also be passed on to birds' eggs. Illustrating that decaBDE has become a world-wide contaminant, one recent study has found decaBDE in glaucous gulls and polar bears from the Arctic. DecaBDE has also been detected in samples of human blood, fat, and breast milk. Thus, the answer to the first part of this issue is that decaBDE is bioaccumulating in the environment.

The answer to the second part of this issue is that levels of decaBDE appear to be increasing in some types of samples. Studies have shown that decaBDE levels are increasing over time in sediments, in certain top predators in aquatic and terrestrial environments, and possibly in human blood. Total PBDE concentrations in breast milk were found to have increased 60-fold in Sweden from 1972 levels, although decaBDE was not measured. DecaBDE was detected, however, in a study of PBDEs in Texas mothers' milk.

### **8.2 Routes of Human Exposure to DecaBDE**

The second issue raised in HB2572 asks how humans are exposed to decaBDE. Humans can be exposed to decaBDE from many sources, including the diet, workplace, and home. Dietary sources are the major contributor to decaBDE body burdens for most adults, with decaBDE detected in many food types, especially in foods and products containing animal fat. Nursing and accidental ingestion of house dust can be significant sources of decaBDE exposure for infants and small children. Workers can be exposed to decaBDE, with certain occupations having a high potential for exposure (for example, workers that manufacture, repair, and

dismantle/recycle electronics equipment, textile and carpet workers, and workers that handle sewage sludge). Lower levels of exposure can be expected for people whose work occasionally brings them in contact with such products, such as sales and stockroom personnel. Since many of these products are also used in the home, low-level exposure occurs in the home as well.

### **8.3 Health Effects and Current Levels of Exposure**

The third issue to be addressed from HB2572 asks “what health effects could result from exposure to DecaBDE, and are current levels of exposure at levels that could produce these effects.” Of the reported health effects from exposure to DecaBDE (mainly from studies with laboratory animals), the Agency has identified liver, thyroid, reproductive/developmental, and neurological effects as the most sensitive endpoints of toxicity. Numerous PBDE studies have reported liver effects, ranging in severity from enlargement without other signs of toxicity to pre-cancerous changes in liver cells suggestive of cancer risk. DecaBDE is much less toxic than the other BDE formulations as shown by several studies. The pre-cancer effects have resulted in USEPA listing DecaBDE as a “possible” carcinogen. Because of the very high dosages used and the production of only pre-cancerous cells at these doses, the Agency believes at this time that the cancer risk from DecaBDE is minimal to nonexistent.

DecaBDE has been associated with hypothyroidism in a small study of workers, who were also exposed to another brominated flame retardant. While this study is only suggestive of thyroid toxicity in humans, this effect cannot be ruled out because results from laboratory animal studies (mainly using BDEs other than decaBDE) also show effects on the thyroid. Decreases in thyroid hormone levels have been reported in several studies, and thyroid gland enlargement (an early sign of hypothyroidism) has been shown in studies of longer duration exposure. Adequate levels of thyroid hormones are critical for the normal development of the fetus, especially development of the nervous system, and the hypothyroidism reported in these studies may help explain some of the neurological effects discussed below. Significant differences between the human and rodent thyroid system exist, making prediction of human effects from the rodent data problematic.

Regarding reproductive/developmental effects, USEPA has determined from a review of data submitted on the PBDEs for the Voluntary Children’s Chemical Evaluation Program (VCCEP) that significant data gaps exist for these effects. The only study available to date is a one-generation reproductive effects study that found no effects on adults or offspring. Still needed are a second one-generation study in another species, a two-generation study, a neurotoxicity screening battery, and a developmental effects study. The need for developmental effects data will be addressed by a study soon to be conducted by the European Union. Other recent studies provide suggestive evidence that the PBDEs can impair the long-term development and function of the reproductive system, with effects on male and female offspring reported for exposures both to the mother during pregnancy and to the offspring shortly after birth.

Regarding neurological effects, it appears that the PBDEs are not toxic to the adult nervous system, but results from several recent studies suggest that the developing nervous system may be very sensitive to PBDE exposure. Changes in activity levels (measures of nervous system development) have been reported in mice whose mothers were exposed to four BDE congeners,

including decaBDE, at relatively low doses, and similar results were reported by another laboratory for BDE-99 using different measures of activity. The decaBDE study has been criticized for several procedural and statistical problems, and the general testing protocol has also been questioned as to the relevance of the results to humans. However, the consistency of the results among the studies from two laboratories strongly suggest that the results in rodents are real, not spurious, and should be evaluated for their relevance to human risks. Results from other studies in rodents report that exposure in the womb or as a newborn results in neurodevelopment delays, hyperactivity, and learning ability deficits as the animal matures.

In order to evaluate the issue of whether current levels of exposure are at levels that could damage public health, the Agency reviewed estimates of daily PBDE exposure from several sources and back-calculated estimates of daily exposure from reported PBDE levels in human fat and milk. These estimates range from 0.00000019 mg/kg/d of five BDE congeners (not including decaBDE) in the Netherlands from food to 0.0028 mg/kg/d in the U.S. for nursing infants from milk. There is an almost 15,000-fold difference in these estimates, although the studies evaluating multiple routes of exposure in U.S. children and adults are more consistent, in the range of 0.000014-0.0009 mg/kg/d. From these estimates, the Agency can fairly confidently state that current exposures should not result in liver toxicity, since the upper end of the intake range is less than the range of the USEPA Reference Doses (which are based on liver effects). However, if the results from the recent studies on the thyroid, reproductive/developmental, and neurological effects of the PBDEs are found to be relevant to human risks, then there may be cause for concern. Two studies report results at the lowest dose tested, 0.06 mg/kg/d, suggesting that effects may be possible at doses lower than the upper end of the daily intake estimates.

#### **8.4 Breakdown of DecaBDE**

The fourth issue to be addressed in HB2572 concerns whether DecaBDE breaks down into more harmful chemicals. Numerous studies have shown that decaBDE can be broken down by ultraviolet light and direct sunlight into lower-brominated congeners and other brominated compounds. Breakdown occurs much more rapidly in the presence of organic solvents versus water and when decaBDE is adsorbed to clear quartz tubes versus sand, raising an issue about whether the results are environmentally relevant. There is also controversy regarding the extent of breakdown under environmentally relevant conditions, since some studies report breakdown to the tetra- to hexaBDEs of greatest toxicological concern while others report breakdown only to the hepta- to nonaBDEs. The lack of information about the extent of breakdown and the breakdown products of decaBDE has been identified as a major data gap in USEPA's VCCEP review, and DecaBDE producers have indicated that they will conduct further studies on its environmental transport and fate in the environment.

DecaBDE can also be broken down by metabolic processes in animals and microorganisms. Trout and carp have been shown to break down decaBDE to hexa- to nonaBDEs and penta- to nonaBDEs, respectively. In addition to lower-brominated BDEs, rats have been shown to produce other brominated breakdown products that contain hydroxyl structures. It has been theorized that these hydroxyl-containing metabolites may be similar in structure to estrogen and/or the thyroid hormones, possibly leading to disruption of the normal function of these hormones, but further research would be needed to evaluate this possibility. Microbes present in

sewage sludge can break down decaBDE to octa- and nonaBDEs in the absence of oxygen, in contrast to the metabolic pathway used by the animals.

As can be appreciated from the above discussion, there is considerable uncertainty and controversy regarding the breakdown of decaBDE in the environment and the body, and what might be the human health implications of the breakdown products. At this time, we believe that the information available on decaBDE's breakdown products is insufficient to allow us to confidently respond to this issue. We anxiously await the results of the planned environmental transport and fate studies to provide further information on this topic.

## **8.5 DecaBDE Alternatives**

The final issue raised in HB2572 is whether effective flame retardants are available as alternatives to DecaBDE uses, and whether the use of alternatives will reduce public health risks while still maintaining an adequate level of flame retardant performance. We reviewed information on alternative flame retardants for DecaBDE's major uses, in plastics and in textiles and fabrics, to determine their effectiveness and potential to cause adverse health effects.

Alternatives to DecaBDE's major uses can be achieved in three general ways: substituting a different chemical; substituting a different product material that doesn't require DecaBDE to achieve a particular level of flame retardation; or changing the design and construction of a product to make it inherently less flammable. Regarding DecaBDE's uses in plastics (especially in electronics equipment), all three general approaches can be used to select alternative flame retardants. Several phosphorus-based flame retardants are available as effective substitutes for DecaBDE, although they are more costly at this time. There are also substitute plastics/resin blends that are compatible with the phosphorus-based retardants and are able to achieve equivalent levels of protection to DecaBDE, and certain inherently less flammable approaches can also be used. In contrast to the relatively straightforward approaches to alternatives for DecaBDE's plastics uses, the approaches for selecting alternatives for its textiles and fabrics uses are quite complicated. The general strategies are the same as for plastics uses, but can be complicated by considerations of product comfort, product wear and tear, wash-out of the flame retardant by cleaning/laundry, and cost. Numerous alternatives are available, including phosphorus-based and inorganic chemicals, fibers or fiber/resin blends that are more easily flame-retarded, and naturally fire-resistant fibers, from which a manufacturer can choose an alternative approach to using DecaBDE.

Regarding the toxicity of chemicals that may be used as alternatives to DecaBDE, we note the relatively limited databases for some of the main candidates as replacements for DecaBDE. For example, it appears that the organic phosphorus compounds suffer from some of the same database deficiencies regarding reproductive/developmental and neurological effects as noted for decaBDE. Nevertheless, in general the phosphorus-based compounds seem to have fewer health and environmental concerns than the PBDEs since they do not appear to be bioaccumulative or break down into toxic and accumulative chemicals, and the inorganic chemicals have few health or environmental concerns.

Based on our review of potential DecaBDE alternatives, the Agency believes that there are effective alternatives to most of DecaBDE's major uses in plastics and textiles/fabrics. Whether these alternatives are cost-effective is beyond the capabilities of the Agency to determine. We also believe that the use of alternative flame retardant approaches will likely reduce risks while maintaining an adequate level of flame retardant performance. Further research is needed, however, to better evaluate the potential health and environmental consequences of many of the DecaBDE alternatives.

## **8.6 Other Jurisdictions' Activities**

Although not requested in HB2572, the Agency has reviewed the actions of certain jurisdictions regarding the PBDE flame retardants for some perspectives on concerns regarding use of these products. As mentioned above, USEPA's VCCEP has identified several data gaps in the decaBDE database for health effects and environmental transport and fate. The European Union has taken two actions with regard to DecaBDE, a Directive that bans the marketing and use of products containing more than 1% of Penta- or OctaBDE as of August 2004 and a Directive (the RoHS Directive) that contains a list of chemicals, including the PBDEs, that will be phased out of use in electrical and electronic equipment by July 2006. The RoHS Directive allows for an exemption from the phase-out if substitutes are shown to be impractical or if the use of a substitute results in negative impacts that outweigh benefits, and DecaBDE manufacturers have petitioned for an exemption for DecaBDE. This petition has been opposed by several bodies in Europe, but it was ultimately approved on October 15, 2005 by the European Commission. This ruling may be taken before the European Court of Justice since the petition did not include an analysis of potential impacts of substitutes, as required by the RoHS Directive.

Several states have recently taken actions to ban or phase out the PBDE flame retardants. Washington issued a Chemical Action Plan for the PBDEs in December 2005 that provides an in-depth review of many topics regarding the PBDEs in support of its many recommendations. Because of concerns for health effects of the PBDEs, the Plan recommends that the Legislature should ban manufacture, sale, or distribution of products containing the Penta- and OctaBDE formulations by July 2006, and ban DecaBDE uses if safer alternatives can be identified or if additional evidence of harmful effects emerges. This Plan also recommends establishment of appropriate recycling and disposal practices for PBDE-containing products, development of public education and outreach programs for PBDE exposure reduction, and determination of ways to minimize work place exposures. The Maine Legislature passed an act in 2003 that prohibits the sale of products containing more than 1% of the Penta- and OctaBDE formulations by January 2006, and DecaBDE by January 2008 if safer, nationally available alternatives are identified. The act also requires the Maine Departments of Environmental Protection and Human Services to report annually on information relevant to brominated flame retardants beginning in January 2005. The first report, like the Washington report, is also an in-depth review of the PBDEs, and contains findings very similar to those of the Washington report. The states of California, Michigan, and Hawaii have passed bans on products containing more than 0.1% of Penta- or OctaBDE, while New York and Maryland have banned these two formulations outright. The California legislation also required a report from the Senate Office of Research on recommendations for further research and possible actions on PBDEs, and this report

recommended development of a Reference Dose for decaBDE and a breast milk monitoring program.

## **8.7 Overall Summary**

The Agency has reviewed numerous data sources regarding the five issues raised in HB2572, and has found much relevant information as well as some significant data gaps. In response to the five issues, we find that:

- DecaBDE is bioaccumulating in the environment, and levels are increasing in some types of samples (sediments, some top predators, and possibly human blood and breast milk).
- Humans are exposed to decaBDE from many sources including the diet, workplace and home, with diet the primary source for adults and breast milk and house dust important sources for infants and small children.
- The most important health effects from exposure to decaBDE and/or lower-brominated congeners appear to be liver, thyroid, reproductive/developmental, and neurological effects, although the relevance of some of the effects reported in animal studies for human health risks has been questioned and significant data gaps in the decaBDE toxicity database have been identified; estimates of current human exposures to the PBDEs indicate that effects on the liver should not be occurring, but two recent studies suggest that exposures could be occurring that are in the range of doses potentially causing adverse effects.
- DecaBDE can be broken down by ultraviolet light and direct sunlight, and also by metabolic processes in animals and microorganisms, but uncertainty and controversy exists about the extent of breakdown by light under environmentally relevant conditions and the human health implications of the breakdown products; thus, at this time we believe the information available does not allow us to confidently respond to this issue.
- Effective alternatives exist for most of the plastics and textiles/fabrics uses of DecaBDE, although they are more costly, and these alternatives will likely reduce risks while maintaining an adequate level of flame retardant performance; however, significant toxicity data gaps exist for many of the main potential alternatives and further research is needed to better evaluate the health and environmental consequences of these alternatives.

Research is planned soon on the potential for reproductive/developmental and neurological effects of decaBDE, and other studies will be conducted to better understand the environmental transport and fate of decaBDE. These studies should provide valuable information to assist in evaluating the issues raised in HB2572.

Table 1. Representative Levels of Brominated Diphenyl Ether Congeners Found in Polybrominated Diphenyl Ether Formulations (%).<sup>(a)</sup>

| Formulation | Congener Percent |           |           |          |           |          |          |          |
|-------------|------------------|-----------|-----------|----------|-----------|----------|----------|----------|
|             | Tri-BDE          | Tetra-BDE | Penta-BDE | Hexa-BDE | Hepta-BDE | Octa-BDE | Nona-BDE | Deca-BDE |
| PentaBDE    | <1               | 24-38     | 50-60     | 4-8      |           |          |          |          |
| OctaBDE     |                  |           |           | 10-12    | 43-44     | 31-35    | 10-11    | <1       |
| DecaBDE     |                  |           |           |          |           | Trace    | <3       | 97-98    |

(a) International Programme on Chemical Safety, World Health Organization, "Environmental Health Criteria 162:Brominated Diphenyl Ethers", [www.inchem.org](http://www.inchem.org) viewed December 8, 2005.

Table 2. Health Effects Reported for PBDEs.

| Health Effect                             | NOAEL<br>(mg/kg)                           | LOAEL<br>(mg/kg)                          | BDE<br>Congeners<br>Tested | Data From                                 |
|---|--|---|----------------------------|---|
| <b>Liver</b>                              |  |   |                            |   |
| Enlargement                               | ND   | 9   | Penta-,<br>OctaBDE         | IRDC, 1976                                |
|   | ND<br>8 <sup>(a)</sup>                     | 90<br>80 <sup>(a)</sup>                   | DecaBDE                    | Norris et al, 1973,<br>1975 a,b           |
|   | 8000<br>(highest<br>dose)                  | —   | DecaBDE<br>(higher purity) | NTP, 1986                                 |
| Increased Enzymes                         | 1.77 <sup>(a)</sup><br>2.51 <sup>(a)</sup> | 3.52 <sup>(a)</sup><br>5.0 <sup>(a)</sup> | PentaBDE<br>OctaBDE        | Carlson, 1980<br>Carlson, 1980            |
| Cellular Changes<br>Cancer <sup>(b)</sup> | 2.51 <sup>(a)</sup><br>ND                  | 5.0 <sup>(a)</sup><br>1120                | OctaBDE<br>DecaBDE         | Carlson, 1980<br>NTP, 1986                |
| <b>Thyroid</b>                            |  |   |                            |   |
| Decreased Hormones                        | 1.1 (mg/m <sup>3</sup> )                   | 16 (mg/m <sup>3</sup> )                   | OctaBDE                    | Great Lakes Chemical<br>Corp, 2001 a,b    |
|   | 10 (Adults)<br>3 (Offspring)               | 30 (Adults)<br>10<br>(Offspring)          | PentaBDE                   | Zhou et al, 2002                          |
| Enlarged Gland                            | 3 (Adults)<br>ND                           | 10 (Adults)<br>0.8                        | OctaBDE<br>PentaBDE        | Fowles et al, 1994                        |
|   | ND   | 3200                                      | DecaBDE                    | NTP, 1986                                 |
|   | ND   | 80  | DecaBDE<br>(lower purity)  | Norris et al, 1973,<br>1975a,b            |
|   | 8000<br>(highest<br>dose)                  | —   | DecaBDE<br>(higher purity) |   |
|   | 5 (Males)<br>7 (Females)                   | 50 (Males)<br>70 (Females)                | OctaBDE                    | IRDC, 1977                                |
|   | 10   | 100                                       | PentaBDE                   | WIL Research<br>Laboratories, 1984        |
|   |  |   |                            |   |
| <b>Reproductive/Developmental</b>         |  |   |                            |   |
| One-Generation<br>Reproductive Study      | 100<br>(highest<br>dose)                   | —   | DecaBDE<br>(lower purity)  | Dow Chem Co, 1975;<br>Norris et al, 1975b |
| Reduced Weight of<br>Epididymus           | ND   | 1   | 99                         | Lichtensteiger et al,<br>2003             |

| Health Effect                                   | NOAEL<br>(mg/kg) | LOAEL<br>(mg/kg) | BDE<br>Congeners<br>Tested | Data From            |
|---|------------------|------------------|----------------------------|----------------------|
| Decreased Spermatids and Sperm Count            | ND               | 0.06             | 99                         | Kuriyama et al, 2005 |
| Changes in Ovary Cells                          | ND               | 0.06             | 99                         | Talsness et al, 2003 |
| Reduced Weight of Seminal Vesicles and Prostate | 3.0              | 30               | PentaBDE                   | Stoker et al, 2004   |
| Puberty Delay (Male & Female)                   | 3.0              | 30               |                            |                      |
| <b>Neurological</b>                             |                  |                  |                            |                      |
| Changes in Spontaneous Behavior, Habituation    | 0.7              | 10.5             | 47                         | Eriksson et al, 2001 |
|   | ND               | 0.8              | 99                         |                      |
|   | 0.45             | 0.9              | 153                        | Viberg et al, 2003a  |
|   | ND               | 2.22             | DecaBDE                    | Viberg et al, 2003b  |
|   | 0.6              | 6                | 99                         | Branchi et al, 2002  |
| Delays in Development                           | 6                | 30               | 99                         | Branchi et al, 2002  |
| Hyperactivity                                   | ND               | 0.06             | 99                         | Kuriyama et al, 2005 |
| Learning Ability Deficits                       | ND               | 30               | PentaBDE                   | Dufault et al, 2005  |

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#### NOTES

NOAEL= No Observable Adverse Effect Level

LOAEL= Lowest Observable Adverse Effect Level

ND= Not Determined

(a)= Used as basis for developing reference dose by USEPA (IRIS, 2005)

(b)= Study only reported pre-cancerous effects

Table 3. Estimated Daily PBDE Intake (mg/kd/d) from Various Sources.

| Daily Intake                         | Source                              | BDE Congeners Included       | Country     | Group Exposed | Data From             |
|--------------------------------------|-------------------------------------|------------------------------|-------------|---------------|-----------------------|
| 0.00000019-0.000003                  | Food                                | 47, 99, 100, 153, 154        | Netherlands | Adults        | Washington, 2005      |
| 0.0002-0.0026                        | Air, Water, Food, Dust, Breast Milk | Multiple, Tetra-Through Deca | Canada      | Children      | Washington, 2005      |
| 0.000355                             | Breast milk                         | Multiple                     | US          | Infants       | Washington, 2005      |
| 0.000004(max)                        | Food                                | Multiple                     | US          | Children      | Washington, 2005      |
| 0.000003(max)                        | Food                                | Multiple                     | US          | Adults        | Washington, 2005      |
| 0.00004-0.0009                       | Multiple                            | Penta-BDE                    | US          | Children      | Washington, 2005      |
| 0.000014-0.000054                    | Multiple <sup>(a)</sup>             | 47, 99, 100, 153, 154        | US          | Adult Women   | Washington, 2005      |
| 0.000016 (median)                    | Multiple                            | 47, 99, 100, 153, 154        | US          | Adult Women   | McDonald, 2005        |
| 0.000015(average)-0.000082(max)      | Multiple <sup>(a)</sup>             | 47, 99, 100, 153, 154        | US          | Adult Women   | Gill et al, 2004      |
| 0.000016-0.0028                      | Breast milk                         | Not Specified                | US          | Infants       | Maine, 2005           |
| 0.000008-0.00008, 0.0000032-0.000032 | Dust                                | Multiple, DecaBDE            | US          | Children      | Stapleton et al, 2005 |

(a) Values back-calculated from BDE concentrations in tissues

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