Appendix C
Comments on
Draft PBDE CAP
(October 2004)

Commenters: Draft PBDE CAP

Click on the commenter name (in blue) to view the selected letter.

AeA (High-Tech electronics)
Aequus Corporation
Association of Washington Business (AWB)
Boeing
Bromine Science and Environmental Forum (BSEF)
City of Seattle – Office of Sustainability and Environment
City of Tacoma Public Works Department
Independent Business Association (IBA)
Institute for Children's Environmental Health
King County - Local Hazardous Waste Management Program
Matsushita Kotobuki Electronics
MBA Polymers, Inc.
Northwest Biosolids Management Association (NBMA)
Northwest Environment Watch (NEW)
People for Puget Sound
Pierce County Public Works and Utilities – Environmental Services
Pierce County Recycling, Composting, and Disposal, LLC dba LRI (LRI)
Puget Sound Action Team (PSAT)
Seattle Chapter Fellowship of Reconciliation
Tacoma-Pierce County Public Health Department (TPCHD)
The Breast Cancer Fund
Thurston County Public Health and Social Services Department
Total Reclaim
Toxic Free Legacy Coalition
Washington Academy of Family Physicians
Washington Association of Churches
Washington Citizens for Resource Conservation (WCRC)
Washington Physicians for Social Responsibility
Washington State Patrol - Office of the State Fire Marshal
Washington State Public Health Association
Washington Toxics Coalition (WTC)
Washington Retail Association
Washington Refuse and Recycling Association
California EPA - Tom McDonald

Commenters at Public Meetings

Seattle – October 19, 2004

Elise Miller
Doreen Smith, salesperson at a natural bedding store

Nancy Evans, health consultant for breast cancer fund in San Francisco, 14-year breast cancer survivor
John Abbots, NW Environmental Watch
David Hayworth, Washington Physicians for Social Responsibility
Elizabeth Davis, League of Women Voters
Jim Mulligan, Earth Ministry
Amy Hirsch, law student
Matthew Cacho, Healthy Building Network –
Tracy Hendershot, health care worker
Bobbi Morgan, Bainbridge retired speech language pathologist
Jennifer Cropack, Burien, Washington Toxics Coalition, Audubon Society
Cindy Chowdry, mother of two
Kelly Faye, mother, toxicology student
Megan Blankwise,
Beth Seltzer, with son
Sarah Augustine
Eldon Wall
Nancy Dickeman, Physicians for Social Responsibility
Ivy Sager-Rosenthal, People for Puget Sound
Lindsey Datelund, Seattle resident
Mary Ann O'Hara, family physician and PBDE Advisory Committee member
Laurie Valeriano, Toxics Coalition
Sybil Diver, Toxic-Free Legacy
John Staltfuss
Linda Boyd

Commenters at Public Meetings

Spokane – October 26, 2004

Jenny Greenwood, parent –
Debbie Boswell, Lands Council, mother of two
Michael Abbier
Mike Peterseon, Lands Council, 1400 members
Linda Greene

Washington Council

AeA

Advancing the Business of Technology

November 10, 2004

Department of Ecology
Attn: Cheri Peele
PO Box 47600
Olympia, WA 98504

Dear Ms. Peele,

The Washington Council of AeA appreciates the opportunity to share our views on the Washington State PDBE Chemical Action Plan draft. The members of AeA believe in environmental policy decisions that are based on sound scientific data and objective criteria, that promote innovation in and the competitiveness of the U.S. high-tech industry, and that benefit the environment. Given the current production realities of Penta-BDE and Octa-BDE we believe a ban on their use may be considered reasonable but is unnecessary and we disagree with the Department of Ecology's assessment for a ban on Deca-BDE in electronic products.

AeA is the nation's largest high-technology trade association and represents more than 3,000 companies with 1.8 million employees. These 3000+ companies, including approximately 170 in Washington State, span the high- technology spectrum, from software, semiconductors, medical devices and computers to Internet technology, advanced electronics and telecommunications systems and services.

Two major issues cause our concern and the basis for AeA's disagreement of a ban on Deca-BDE; 1) inconclusive scientific findings on direct human health impacts, and 2) the plan's failure to study effects and feasibility of alternate substances for Deca-BDE.

DOE's own draft report clearly states (though a disputable point) that while Deca-BDEs are precursors to more bioaccumlative and persistent lower brominated PBDEs and PBDFs, that it is not clear whether this adds to the overall risk to organisms. Notes from the August 25th meeting of the advisory committee indicate a number of unknowns specific to Deca-BDE, including such critical items as; the breakdown of products with Deca-BDE in the environment, lack of data in our state related to Deca-BDE, lack of understanding of how Deca-BDE moves from products to the environment and to humans, and the toxicity of products formed by Deca-BDE breakdown. While some of these questions are addressed in the final draft plan the conclusions do not appear to be fully substantiated. Additionally, other recent reports cited in the draft plan indicate an absence of conclusive data on the necessity to ban Deca-BDE due to human health concerns.

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Not only is this is an important issue, but it is a highly charged one as well; all the ramifications of our actions need to be contemplated lest we do more harm than good. Should we ban Deca what are the unintended consequences? PBDEs were introduced to reduce the very real risk of loss of life due to potential flammability of items often found in our homes. Currently there are limited alternatives available on an industrial or commercial level which are as successful in reducing fire risk for certain products. Also troubling is the lack of scientific research on possible alternatives. We must be sure we are not simply phasing one chemical out and introducing another that has a higher potential for human health risks and negative environmental impacts.

Secondarily we have concerns related to the economic impact a Washington State only ban could have on consumers and businesses in our state. If Deca-BDE containing products are banned in Washington, will non-Deca products be more expensive due to the necessity of using alternate flame retardants and or will product choice be limited? If so, consumers could be forced to buy products outside our state resulting in a loss of sales to retailers and a decrease in tax collection for the State.

AeA opposes the proposed ban on Deca-BDEs; however we look forward to the results of more intensive study on direct human impacts of Deca-BDE, and if in the future a phase out based on scientifically sound criteria is recommended, then further discussion on what human and environmentally friendly alternatives are, will be necessary. The prudent action is to monitor ongoing research with respect to Deca-BDE and other flame retardants and to base any future decision on a sound scientific basis.

Sincerely,

Nancy L Atwood

Nancy Atwood
Director of Policy and Legislative Affairs

November 22, 2004

TO: Mike Gallagher
WA Dpt. of Ecology, PBT Program

From: Randy Ray
AEQUUS Corporation

Re: Comments for PBDE Draft CAP on behalf of Seafood Industry

PROCESS ISSUES

When the PBDE (fire retardant) debate started in the Legislature in 2004, the fishing industry thought it had no involvement. What did flame retardant have to do with fish? Nothing. Then this summer, the front page of the Seattle P-I had a headline with PBDEs in "wild Chinook" in Lake Washington. As a representative for the many of the seafood processors, I and the seafood industry became alarmed. Accusations were being made about our healthy product.

I then began to investigate the PBDE Chemical Action Plan process. I found that health experts project 90% to 93% of a persons exposure to PBDEs comes from food intake. I discovered the PBDE CAP process lacked any representation from the fishing industry or any food producers.

The first thing the PBDE CAP should say is that PBDEs do not pose a health risk to human or other organisms, One can say concerns do exist if PBDE level continue to rise, because animal tests do show an adverse impact to lab animals at high rates. But, presently no health risk has been shown of ingestion of PBDEs.

The scientific reports footnoted in the PBDE CAP say this repeatedly. None of the studies show a present danger from current PBDE levels. Few people have high PBDE levels. The CAP appears to be using scare tactics to sell a political agenda.

I agree other PBTs can cause severe harm. I agree that Europe has banned Penta and Octa PBDEs, but not Deca PBDEs. But, no health impact of an individual has been shown to be harmed from either penta, octa, or deca.

Yet during this process, the Dpt. of Ecology and the Dpt. of Health ran an ad in newspapers across the state. The ad showed a picture of a baby, the headline read: "HE HAS HIS DAD'S EYES AND HIS MOTHER'S PBDES". The ad went on to ask people to come to a meeting to talk about PBDEs. The inflammatory nature of the ad is unprecedented in my 29 years in Olympia. This was not an ad to ask people to come to a meeting, this was an ad that crossed the line. After

Ecology promised to pull the ad, we heard weeks later from others the ad was still appearing in web versions.

The process for the PBDE CAP has not been an inclusive one. Ecology and Health both knew of the link between PBDEs and food. Yet, neither agency bothered to notify the seafood or agriculture industry.

The PBDE CAP process needs to be started over.

TECHNICAL ISSUES

HUMAN EXPOSURE PATHWAYS & DIET

The Draft PBDE CAP centered on fish as the main dietary exposure pathway. I asked Ecology and Health for the scientific reports on which the health impact were based. These were provided. After careful review of each one, what was written in the Draft CAP did not match the backup papers. Staff appears to have cherry picked certain points and ignored others.

Fish in the CAP was cited as a primary source of diet exposure. Yet the paper failed to cite a Japanese study where people were tracked with different diets: high fish intake, medium fish intake, and low fish intake. All people still exhibited evidence of PBDEs. Therefore, fish are not the only dietary source of PBDEs.

The CAP did not cite that PBDEs are not only in found in fish, but in shellfish, Dungeness crab, pork, chicken, cheese, ice cream, eggs, and spinach. Surprisingly to scientists, beef has one of the lowest counts of PBDEs.

Yet, while PBDEs have been detected in all these foods, there still has been no impact on humans or other animals containing PBDEs. This message is not found anywhere in the CAP.

ENVIRONMENTAL EXPOSURE PATHWAYS

How do PBDEs get into the environment and food sources? Ecology seems to be baffled on such. To prevent exposure, Ecology is proposing to ban further production and use of PBDES. Yet, PBDEs are present in millions of pounds of existing products. In many products such as car seats, plastic dashboards, the plastic portion of consumer electronic products, PBDEs can make up 8% to 30% of the product by volume.

Ecology cites in the Draft CAP that “electronic recycling” facilities may represent a source of contamination to the surrounding environment of PBDEs.

In the next paragraph, Ecology cites that Municipal and private landfills, where shredded “auto fluff” is spread on landfills by the ton. Auto fluff is the portion of a

car that is left over after a vehicle is shredded and the metal is removed. The auto fluff would typically contain 8% to 30% PBDEs. This fluff is spread on top of every landfill on a daily basis by the tons. It is also used to create a berm between different cells in a landfill.

In the studies footnoted by Ecology for the Draft CAP, US studies, Swiss studies, Canadian studies, Japanese studies all cited air deposition of as the main source of PBDEs in the environment. While electronic recycling faculties are mentioned as a possible source of PBDEs, Ecology states landfills are said to be unknown in the possible environmental impact. In fact, Ecology says:

> …, it is possible that using auto fluff as a daily cover is the best waste management practice with regard to PBDEs.

One is mystified by an agency that is so worried by a substance that the product must be banned from future use, but dumping tons of the substance shredded into the open air, where scientific reports repeatedly say air-deposition is the main exposure pathway of fish, spinach, eggs, even contamination in pristine areas where no point source of PBDEs exist, would say that such disposal practices are “OK”.

If Ecology moves to ban PBDEs use and production, the substance should be classified as a hazardous waste and treated such. All landfills should be required to eliminate future and existing PBDEs from their facilities.

Another area being ignored by Ecology is “biosolids and sewage sludge”. PBDE testing have found PBDE is every sample of biosolids and sewage sludge. Typically, the PBDE will bind to a sediment particle. When a particle dries out, such as when biosolids and sludge is deposited on agricultural land, the PBDE loses the adhesion, falls off and becomes bioavailable for air dispersion or uptake into a plant. Here again, scientific papers Ecology cite detail such a concern, but no mention of these comments exist in the Draft CAP.

If Ecology moves to ban PBDEs use and production, the biosolids and sewage sludge should be classified as a hazardous waste and treated such. Land application and incineration of biosolids and sewage sludge should be eliminated and consideration given to cleaning up sites where land application has occurred.

As stated above, PBDEs, particularly Deca PBDEs, have not been shown to be harmful to humans or animals. Concern has been raised that if levels increase harm may result. Therefore, if Ecology and Health believe that PBDEs are so harmful the production should be banned, then one must also declare existing products a hazardous waste and not be allowed to be dumped in the environment and made bioavailable. And existing dumps sources should be cleaned up.

BREAKDOWN OF PENTA

Manufacturers of PBDEs are switching from penta and octa formulations of PBDEs to Deca PBDEs. Ecology is stating that all three should be banned. Ecology states Deca PBDEs breakdown into penta and is subsequently just as harmful. Ecology did provide several excellent papers detailing results of how Deca breakdown into Penta.

But, Ecology seemed to have cherry picked a preferred answer again. Papers citing that little breakdown of Deca to Penta in the environment takes place are not mentioned any place.

On the question of breakdown, Ecology has failed to prove its case. What more, Ecology seems to have deliberately only presented evidence in their possession that proves their case and purposely ignored evidence to the contrary.

This is not good science, nor good policy.

CONCLUSION

The Departments of Ecology and Health have appeared to have used a process that was deliberately not inclusive of all relevant parties. Each agency had information of the relevancy of the food industry to the PBDE issue and contacted no one.

Ecology has tried to slant this as a fish issue, where the evidence points such conclusions are totally erroneous.

Ecology and Health have put misleading ads in the media that were highly detrimental to the food industry, but have done nothing to correct such actions.

Ecology and Health have not given accurate health messages in the Draft PBDE CAP. This is highly unfortunate and scares away consumers from dietary foods deemed very beneficial my volumes scientific and medical studies.

Human exposure pathways have been deliberately slanted.

Environmental exposure pathways have just been ignored all together, even though footnoted studies clearly demonstrate concerns.

And, Ecology has failed to make its case on the breakdown of Deca to Penta.

The PBDE Process needs to be started over. The PBDE CAP needs to be seriously revised.

I would hope Governor Locke, the Department of Ecology, and the Department of Health would step back and do this process over. One of the serious flaws was trying to do so much in such a short time. This large issue cannot be covered in the time given.

We look forward to working with all parties on a real Chemical Action Plan that will address the concerns in a realistic manner of PBDEs.

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Washington state's chamber of commerce

November 11, 2004

Department of Ecology
Attn: Cheri Peele
PO Box 47600
Olympia, WA 98504

AWB Comments Regarding Draft PBDE Chemical Action Plan For Washington State

Dear Ms. Peele,

The Association of Washington Business offers the following comments on the October 11, 2004 draft chemical action plan for polybrominated diphenyl ether (PBDE) flame retardants, developed by the departments of Ecology and Health.

AWB has significant concerns regarding the recommendations in the draft chemical action plan and fundamentally, we believe the departments are moving in the wrong direction. Concerns outlined in the following comments have been expressed to the departments of Ecology and Health during advisory committee and other meetings, yet appear to have been ignored in the current draft.

Ecology and Health should withdraw its recommendations to ban PBDEs for the following reasons:

1) Ecology and Health have not completed a science-based risk assessment, cost-benefit analysis and small business economic impact statement, nor have they considered less burdensome recommendations and methods to measure whether proposed recommendations will accomplish the chemical action plan's intended goals. These elements are logical and critical first-steps in developing any chemical action plan.

2) PBDEs are effective flame retardants that save lives and prevent serious injuries from fires by providing products with resistance to flame and providing people more time to escape a burning structure. Ecology and Health's recommendation to

ban PBDEs in certain products may increase injuries and deaths caused by fire. Sweden has documented a 100% increase in fires after they discontinued the use of brominated flame retardants in televisions – Ecology and Health should take prudent steps to ensure a similar situation does not result in Washington State.

3) Penta-BDE and Octa-BDE are no longer being made in the U.S.. Banning Penta and Octa is therefore not necessary and doing so may limit opportunities to recycle certain consumer products. In addition, EPA is promulgating a Significant New Use Rule on Penta and Octa which will make it virtually impossible to manufacture or import products containing Penta and Octa after January 2005.

4) Scientific information regarding Deca-BDE does not support banning products containing Deca. Ecology and Health over-rely on the precautionary principle and the alleged debromination of Deca into lower congeners (Penta and Octa) to justify the agencies recommendations. However, scientific studies show that congeners found in the environment originate from commercial Penta and Octa, not the debromination of Deca. Ecology's selected science advisor from California EPA stated that if the department recommended banning products containing Deca-BDE, it would be relying on the precautionary principle to justify its position. No other state, including California and no other country in the world has determined that Deca presents sufficient risk to warrant regulatory action at this time.

5) Safe and effective alternatives for some products are not available. For certain plastics, there are currently no cost-effective alternative flame retardants which can provide good flame retardancy and good mechanical properties. Manufacturers who are already using different chemicals in the place of PBDEs, or plan to use alternatives in the future, have stated that those alternatives are at least 15% more expensive, may be less effective and their toxicity is unknown. In order to achieve the same level of fire protection compared to brominated flame retardants, higher quantities of alternative chemicals are often needed, which could lead to increased risk to human health and the environment and an increase in manufacturer and consumer costs.

Sincerely,

Grant Nelson
Governmental Affairs Director

November 14, 2004
The Boeing Company

Ms Cheri Peele
Washington Department of Ecology
Lacey, WA.

Dear Ms Peele:

The Boeing Company provides the following comments on the Draft PBDE Chemical Action Plan as posted on the flameretardant.org website as of November 7, 2004. We appreciate that the Department agreed to a stakeholder committee to review the issues surrounding these three chemical formulations. Our involvement in the PBDE Chemical Action Plan stakeholder process has been very beneficial. We are in concurrence with comments provided by the Bromine Chemistry Council and the Association of Washington Businesses on issues not specifically addressed in this letter.

Our detailed review of the proposed PBDE Chemical Action plan identifies a overriding single fatal flaw- a lack of any meaningful risk or impact assessment to support the majority of recommendations. The DOE/DOH (the Agencies) urgency in completing the CAP in the time allotted under the Governor's executive order appears to underlie this failure to provide sufficient analysis of the complex range of technical, social, economic and health issues. A reasoned approach to a risk analysis may have addressed a number of contentious issues that are created with this CAP's recommendations. Hence, instead of identifying actions promote cooperation among the parties involved, this plan creates a divisive formula for future debate and dissention. A plans that is therefore unlikely to be effective in addressing the issues of concern.

Lacking a meaningful risk analysis, the agencies have substituted an extraordinary interpretation of the precautionary principle. The Agencies dependence on a derivative threat from deca-BDE (debromination) is hard to both understand and substantiate. The need for substantially greater research on debromination is documented in the reports from multiple governments. These same governments do not recommend a need to take any specific action, such as a ban, which the Agencies are recommending

A companion flaw is the lack of a systems-thinking approach to the question of economic or social impact. These analyses are as critically important in identifying the likely outcome of any Agency recommendation. This impact analysis is particularly important when considering the impact of a ban on a life saving chemical such as fire retardants The agency's lack of analysis leaves unanswered questions about any increased risks of deaths and injuries in fires, the likelihood of more fires, and the environmental and health effects of combustion products in such fires. For example, the Agencies must determine that reducing the alleged risk by PBDE is greater than the impact of increasing the risk of death by fire. The Agencies must also establish the environmental effects of the additional burn products created by an increase in previously preventable fires further, the environmental and health effects of the alternative

materials are, if anything, less understood than those of the PBDE family. Weighing the risks associated with a ban, the Precautionary Principle would lead one to take no action at this time. Rather, the prudent course of action is to monitor research on deca-BDE and the proposed substitutes so that fire protection is not compromised and environmental and health impacts are minimized.

The economics of the PBDE issue are being underplayed and unanalyzed in this CAP. The Agency's proposals will create one of a kind restriction in Washington State ranging from manufacture buy-back programs to a ban on electronics containing Deca-BDE. Each of these independent actions has a potential for multiple incidental impacts; any or all of which could lead to adverse economic conditions. In the above comment we considered the relative impact on fire safety. We now suggest that the Agencies must look seriously at what will happen to people subject to economic dislocation due to a non-competitive economic sectors resulting from these restrictions.

The multiple, complex and unresolved issue in the PBDE debate reinforce our concern that the Agencies have not had adequate time or resources to work through the myriad issues at play. A remedy to this situation is necessary by taking the following recommend actions:

- The Chemical Acton Plan be rescinded and reissued on a chemical specific basis (effectively two plans).
- Each CAP includes substantive risk and impact analysis.
- Coordinate with the US EPA as to action necessary, including a gap analysis specific to Washington State.
- The WDOE monitor deca-BDE science for emerging trends related to debromination.

Additional specific comments and recommendations are in the attachments.

Please contact the undersigned if you have question about our comments.

Sincerely,

Kirk Thomson
Director- Boeing Environmental Affairs
PO Box 3707 MC 7A-UU
Seattle, WA 98124
206-930-6122

1) The Chemical Acton Plan Lacks Clarity.

Comment: The plan is an incomprehensible mixture of discussions and information on three versions of the PBDE, Octa, Penta, and Deca. Two of these, Octa and Penta, materials have some showing of toxicity and are already being phased out of production by their manufacture. The EPA has taken action through TSCA to prevent their reintroduction into the country. The European Union (EU) has an effective ban on their use under its various rules. Deca-BDE has significantly different properties and regulatory environment than Oct/Penta-BDE. For any Washington Citizen to understand the varying effects between Octa /Penta and Deca requires clear independent presentation with science and technical facts specific to these substances. This report fails in this regard.

Recommendation: Rescind the current draft PDBE chemical action plan. Reissue the plan addressing each of the PBDE's under evaluation. Limit factual information to that which can be specifically correlated to the PBDE formulation under discussion in the plan. Where pertinent cross references between formulations are appropriate be sure that they are clearly identified. In interest of economy it may be feasible to combine the CAP for Octa and Penta –BDE since both are out of production and the challenges will be similarly focused on recycling issues. Deca-BDE however; is considerably different in effects and underlying knowledge. It must be discussed independently of the Oct and Penta versions.

2) Imprecise language adding to confusion:
Comment: The plan as written contains a number of statements that could be confusing to the public and policy makers as to whether the statement was speculation, unsubstantiated extrapolation or assumptions not in fact. The number of these statements occurring creates an inaccurate understanding of the impact of the chemicals under discussion. When combined with the interweaving of discussion of the various chemicals, as noted in attachment 1, it becomes impossible to discern if the discussion is fact, concept, extrapolation or guesswork. Some random example statements taken from the plan are:

- Page 55: In anticipation of the phase-out of Penta-BDE and Octa-BDE, **it is expected** that manufacturers are moving away from these products and identifying alternatives. In addition, a number of electronics manufacturers have been identified that are phasing out of all PBDEs, including Deca-BDE.
  Comment: Expected by who? What evidence do we have that the thousands of manufactures across the world are changing formulations. Was it market forces or better alternatives? What happens if EU does not ban deca-BDE, will manufactures return to this material? What are the net impact of the substitutes on the environment- is the life cycle cost of smelted metal cases greater or less than deca-BDE?

- Page 33: As reductive debromination has been observed in experiments using water with dissolved humic substances**, it must be assumed that this may also occur** in the environment. Other factors, not yet explored, may also influence both photolytic degradation rate and products.
  Comment: Policy decisions can not be based on Agency staff's assumptions. This statement creates an unfounded concern about degradation products.

- Page 27: Butt et al. found indoor levels of PBDEs in Southern Ontario were 1.5 to 20 times greater than outdoor levels on a site-by site basis. **They suggest** that indoor air may serve as a significant source of PBDEs to outdoor air. "
  Comment: Suggest" has no place in a document alleged to be filled with facts- it either is, or is to be confirmed (and hence is not).

- Page 27: **If** brominated dioxins and furans were present in substantial quantities, this could be a pathway for release to the environment.
  Comment: If? The word itself is the essence of speculation. It has no place in a policy document based on scientific analysis.

- Page 56: PBDEs are found in a **vast number** of consumer products, with **vast potential** for continued human exposure.
  Comment: "Vast" is a very hard term to quantify in a meaningful manner.

Such a term is more appropriate to description in tourist advertisements than use in a government policy document.

- Page iv: Each additional year that PBDE products are produced and sold will extend that timetable – and any related costs – **by a decade or more**.
  Comment: Where in the literature is the data to support this statement. A statement designed more to create apprehension and a rush to judgment than understanding of chemical degradation processes, such as half-life.

One further example of inherent failure in the impartiality in this entire PBDE process is the public hearing announcement created and issued by the Agencies with full management approval, and at $20,000 in cost. The advertisement, well known to the department, used language and baby photo that created a fear filled environment at the public hearing. It is this disturbing trend, the advertisement and the CAP language, that fuels a concern that a full and honest evaluation of the PBDE issue may not be possible within the Agencies. Concern that even makes the use of an ".org" website a matter that deserves questioning- not to mention the use of a photo of mother and child- creating an adverse implication for PBDE.

Recommendation: The extent to which imprecise language can be found suggests a need for an independent technical editor. An editor well versed in neutral, factual technical writing may be able to restructure and rewrite the recommended new documents to standards of impartiality & scientific clarity expected from a government agency. This rewrite will provide the policy makers and the public with a factually accurate view of the topic on which to determine actions.

Further, Agency executive management needs to reiterate to its staff that personal perspective on PBDE will not be allowed to influence developing Agency policy. This should include careful over-site of staff actions in all areas affecting CAP content and public notices.

Attachment 3:

National and/or International actions and findings

Comment: Action by Washington State on any of the PBDEs needs to considered as a whole, not selectively, when compared to actions and findings by other government agencies.

- **National Academy of Science:** As noted in the CAP the National Academy of Sciences (NAS). NAS reviewed the toxicological and exposure data of 16 flame retardants, including Deca-BDE, to assess potential health risk to consumers and the general population resulting from potential exposure from the chemicals in residential furniture. Despite the lack of a complete database, the report concluded that Deca-BDE, along with a number of other flame retardants listed in Table 8, could be used on furniture with minimal risk, even under worst-case assumptions. (underlined for clarity)
- **State of California**: It is no surprise that when California considered the issue of banning PBDEs it sought expert evaluation resulting in a California legislatively mandated report:
    - "As required by AB 302, in June 2004 the Senate Office of Research submitted a report entitled "Polybrominated Diphenyl Ethers (PBDEs): Potential Hazards from DecaBDE and Unresolved Issues from AB 302" to the President Pro Tempore of the Senate and the Senate Environmental Quality Committee. The report stated that, based on the "likely potential harm to humans posed by decaBDE and the known human exposures to this chemical, it does not appear that human exposure to decaBDE is occurring at a level that is likely to be unsafe for human health or development." **The report concluded that, at this time, it would be premature to add Deca-BDE** to the list of banned PBDEs contained in AB 302.[181]
- Even the EU, in a somewhat confusing analysis, has determined that there is insufficient information or harm to ban the use of Deca-BDE. EU staff responded to a WDOE request that: "The Conclusion is that further information and testing are required in an attempt to demonstrate whether the substance is or is not a safe product. Hence, Deca-BDE is currently being evaluated by the European Commission for exemption from the ban under the RoHS Directive. Through July 5, 2004, the Commission solicited written stakeholder comments in response to the following questions with regard to Deca-BDE:
    - Do feasible substitutes currently exist in an industrial and/or commercial scale?
    - Do any restrictions apply to such substitutes?
    - What are the costs and benefits and advantages and disadvantages of such substitutes?

The Agencies would have the public believe that it's analysis of the impact of deca-BDE is superior to that of international, Federal and other state organization's with substantially better fiscal and scientific resources.

Yes, we note the derivate analysis that deca degrades into other products that may be more toxic as the sole basis for this recommendation. If this was truly a major concern, with reliable scientific backing, then is it reasonable that other governments would not have acted on this concept to limit the use of deca-BDE. They have not.
The evidence on degradation is tenuous at best. The Agencies analysis focuses on the worst possible case extrapolated from limited scientific evidence. Evidence that is contradicted by other well designed work such as these examples:

- Scientific evidence, by Jacob de Boer, a Dutch environmental scientist, that as Deca-BDE in the environment increases, the components of penta are decreasing. This is evidence based on actual environmental finding in the Scheldt river, and should be most important when considered against laboratory data and speculation.

- Ikonomu also published a paper indicating that deca-BDE in a Canadian river was not responsible for the penta found in the same river.

- Laboratory study, by Cornelius Zetsch, a noted German UV degradation scientist, shows that deca-BDE could degrade, upon exposure to ultraviolet light, by very slowly losing bromine, but that the degradation would proceed sequentially, Br 10 to Br 9, etc., all the way to Br 0, therefore passing through penta, but not stopping. His study therefore concludes that all bromination levels, 9,8,7,6, etc. should be found, in the environment, not just penta/tetra. This is not the case.

- Zetsch also showed that the actual isomers of penta found in the environment (the location of the bromines on the diphenyl ether substructure) would be different that those that are produced by photolytic degradation of Deca. He concludes that it is extremely unlikely that Deca is responsible for the components of penta in the environment.

- Deca does not degrade under the conditions for anaerobic degradation as verified by a Swedish study.

(note: full text of studies are being provided via other commenters)

<u>Recommendation:</u> Washington state should conform to the standards being developed by the United State Environmental Protection Agency for the management of any of the version of PBDE.

- The EPA is already taking action on octa and penta BDE. As quoted in the draft CAP. "EPA is in the process of developing a Significant New Use Rule (SNUR) for Penta- and Octa-BDE. The rule would require notification to EPA prior to manufacture or import of Penta- or Octa-BDE for any use

after January 1, 2005.177 EPA's authority to issue SNUR's comes from The Toxic Substances Control Act section 5(a)."

- The USEPA is studying deca-BDE to determine if any action is warranted. Per the presentation provide by USEPA nothing has thus far indicated that any action is necessary. Hence, Washington state should take no action other than to monitor on-going research and agency actions.

Attachment 4

Precautionary Principle. This concept has been around since the cave man in one form or another. When a real danger can be identified it is better to take some early actions to increase "public" safety. The challenge these days is the threats are subtle and can create fear without having a basis in science. Look at the current definition of Precautionary Principle:

> **Principle 15 (the *Precautionary Principle*) from The Rio Declaration on Environment and Development (1992), reads:** *Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation***.**
>
> **Interpretation found in literature:** *Where there is reasonable certainty of a cause-and-effect relationship resulting in significant harm from a specific, well-defined activity, absolute proof should not be required to initiate cost-effective remedial action***.**

Thus we have a chemical such as deca-BDE which has not been shown in itself to be a hazard to human health in anything close to the dosage that have been observed; or are even likely to occur. Yes, it accumulates, but with a half life of 12 days in human body can not be considered persistent. Nor, has any study found it to be toxic except vague concerns about neurological damage in high dosages in experimental animals. Data poorly translated to humans without much more work. So, lacking a real threat ("threats of serious or irreversible damage") on which to base a claim for deca-BDE restrictions, ban proponents attempt to create a derivative problem by asserting that the breakdown products may create another threat. Please note the phrase- "lack of full scientific certainty" in the definition. Full certainty may not be required, but; some certainty is needed that there is really a threat of serious or irreversible damage. The degradation derivative concept is so scientifically uncertain, as discussed in attachment 3, as to fail even approaching the Rio international standard. The EU may have put a good point on this topic in the response to Ecology: EU staff responded:

<u>"The Conclusion is that further information and testing are required in an attempt to demonstrate whether the substance is or is not a safe product."</u>

Hence, the resources of the EU, the godfather of the Precautionary Principle, is unable to find adequate reason to determine a product is un-safe, including consideration of its derivative products. This raises the question of the standards of certainty under which the Agencies are making their recommendations.

The WDOE continues to lose credibility with reasonable people when it takes actions that are so clearly outside the rational boundary of the precautionary principle. These actions risk the Agencies losing further credibility in the application of this principle in any situation. As described in Governor Locke's blue ribbon report the WDOE already has a unsatisfactory reputation with the

State's regulated community- private and public. This is another action that reinforces that image and will encourage an increased logjam of legal, legislative and public opposition to WDOE actions- good and bad. Such logjams in Agencies activities will adversely affect the protection of the natural and the business environment.

Recommendation: The Agencies should drop the proposed ban on deca-BDE. Instead, consider a proposal to monitoring the science as it develops from the many agencies currently investigating deca-BDE. Should sufficient scientific information surface identifying a realistic problem, then the department can propose actions with-in the frame work of the (under development) PBT rule. The Agencies may wish to look at deca-BDE as though it were subjected to the reviews of a significant new rule by including risk and impact analysis.

Attachement 5.

Social, Safety and Economic Impacts.

Comment: The proposed recommendations on the PBDE's have multiple social, safety and economic impacts that have require extensive additional study. A few of the areas needing attention are:

- **Recycling**: Two sets of recommendations are needed to differentiate between the impact of Octa/Penta-BDE ban and Deca-BDE. The Octa/Penta-BDE issue will quickly become one of recycling and reuse since the supply of these products will end about July 2005. Hence, the question becomes do we accept products that are manufactured from recycled content that may have some level of Octa/Penta-BDE. A strict ban on new products containing these materials would necessarily require that any new product be certified to not have Penta/Octa-BDE recycled content. The recyclers have testified that they have no knowledge of what is in the products they get. Hence, they would of necessity reject everything that could- maybe- have Octa/Penta-BDE in them. So what happens to these products? They end up in land fills, along side roads, dumped on charities and the problem just becomes exacerbated in other area. Eventually, these products end up in landfills- probably safe locations considering current landfill standards; but at what cost to the environment in use of virgin materials instead of recycled products. For example; as recommended "new" products made from recycled materials, such as the plastic "2x4" could not be sold in Washington. Instead we would need to use virgin materials such as forest products or imported oil to make the alternative products. California passed an inclusive Octa/Penta ban, then came back and amended the law to address a range of recycling issues they had not adequately considered. Washington State should learn from California's experience and carefully craft rules on recycling and new products containing recycled content to maximize environmental benefits.

- **Manufacturing buy-back**: In a related concern is the WDOE's proposal to create a "manufacture buy-back" requirement. The topic has been hotly debated at NEPSI meeting held under USEPA auspices. It is also the topic of consideration in a WDOE solid waste advisory group created by legislation in 2004 session. All reference to this approach for management of any material- PBDE or otherwise should be stricken from this CAP pending legislative resolution of the surrounding policy issues.

- Consumer Electronic deca-ban: The proposed ban on deca-BDE in electronic equipment is both inappropriate as discussed above and likely to cause far more harm than good. The category of materials covered in this proposed ban is equivalent to those in the EU ROHS listing. That is just about every electrical and electronic item from basic components such

as wire and transistors (sold to hobbyists) to after-market car components. The deca-ban would affect not only average consumers, but; also small and large businesses that use a range products covered under this definition. Boeing uses thousands of office products ordered from suppliers such as Office-Max, Boise.com and others.

Even if a ban were imposed it is problematic if it is going to be effective in reducing public exposure to deca-BDE. Look at Ecology's own figures on PBDE availability in table 7 of CAP- as expressed as reflection of waste stream: (note: this appears to be all PBDE, not just deca-BDE). Notice that electronics is .3% (.003) of the waste steam! The department needs to demonstrate how eliminating .3 percent of the products from the waste stream, and by implication available products in consumers home, can have any realistic impact on public health. Especially when:

- The Agencies own data suggests that the majority of exposure to PBDE's is through food consumption.
- Food production in the US is so sophisticate that much of it comes from a few central locations (CA, FL, Midwest); hence,
- A majority of food consumed by Washington residents is likely from out of state.
- Thus the proposed ban on PBDE's in electronics is going to have minimal effect on body burden caused by eating food; the alleged major source.

Only an effective Federal ban on a product can create a business environment in which a deca-BDE ban on electronics would be effective. The Montreal Protocol and related EPA action is an example where this can work. However, such a ban is not likely in the foreseeable future as the US EPA does not see a need at this time to ban or restrict the use of deca-BDE (see earlier discussion).

- **Fire Codes**: Boeing also has concerns about fire safety should deca-BDE be unavailable in our offices and factory electronics. The National Fire code has many requirements for flammability standards in the work place that have been adopted by local ordinance. It is entirely feasible that some parts of these codes could not be satisfied with available substitutes.

  Concern for our employees safety is a central ternate of our operations; both at work and home. We have appended several articles from the European press addressing fire safety issues. It is particularly interesting to note that while there are about the same number of fires per-year the number of deaths in Europe is nearly 4 times higher

"The estimated impact of fires in each continent is listed in Table 1. The financial cost of fires is exceptionally high. Direct property losses **amount to 0.2% of total GDP and the total cost of fire to society has been calculated at 1% of GDP.** Protection of life and property from the effects of fire has been a topic of a great deal of research, much of which has focused around prevention of the spread of

flame using barriers and flame retardants. To recognize how these materials work, you have to understand the burn process.

Continent	Population (millions)	Fires per year (millions)	Fire deaths per year (thousands)
Europe	**720**	**2.2**	**25.0**
Asia	3660	1.0	30.0
North America	**470**	**2.3**	**6.5**
South America	340	0.5	2.5
Africa	780	0.8	5.0
Australia	30	0.1	0.3
TOTAL	6000	6.9	69.3

[citation: **Melting in the heat.(International Pages)(Statistical Data Included)** HARDING, PAUL; CROMPTON, GEOFFREY Asia Pacific Coatings Journal, v13, n4, p16 August, 2000]

In the CAP is a discussion of the purpose of flame retardants that can be summarized as: Giving the occupants more time to escape prior to flashover. In a impact analysis the Agencies could advise the public if they are four times as likely to die in a fire due to removal of effective flame retardants- such as deca-BDE; An impact analysis seriously missing in this plan.

Recommendation: The Agencies need to conduct a suitable risk and impact analysis prior to making its recommendations. All recommendations that affect usage of deca-BDE as a flame retardant should be withdrawn until the analysis are completed, reviewed and publicly commented on.

Attachment 6

Exemptions and waivers:
Comment: Washington DOE needs to ensure that an exemption or waiver process is incorporated in any scheme to manage PBDEs. These options are needed to ensure these products can be used when unique applications are required. As a parallel example, the Montreal Protocol on ozone depleters contains specific exemptions for uses such as Space Shuttle and waivers for uses for which there are no substitutes, such as foam blowing in certain missiles. The EU even recognizes this need as they have recently granted a interim waiver for use of Penta-BDE in aircraft escape slides- as there is not alternative available at this time.

- As a follow-up to the July QMI where there was lengthy discussion about the use of pentaBDE in certain escape slides, a meeting was held in Brussels to review the use and make a determination on possible derogation. The DRAFT COMMISSION DIRECTIVE, amending Council Directive 76/769/EEC with respect to restrictions on the marketing and use of pentabromodiphenyl ether in aircraft emergency evacuation systems for the purpose of adapting its Annex: .
- "3. By way of derogation, until 31 March 2006 paragraphs 1 and 2 shall not apply to aircraft emergency evacuation systems."

Recommendation: The Chemical Action Plan should include a discussion of exemptions and waivers processes. These processes should not require the applicant to provide excessive documentation or research data. Rather, they should use the data currently at hand in making determinations. Waivers & exemptions granted by other governments should be de-facto sufficient reason to grant a waiver or exemption in Washington State.

Gale Group Trade & Industry DB

14187015 SUPPLIER NUMBER: 81299682 (THIS IS THE FULL TEXT)

Let's not lose sight of first principles. (Compounding).(Brief Article)

British Plastics & Rubber, 28(2)
Nov, 2001

TEXT:

There is an old proverb about swamps and alligators which demonstrates that no matter what adversity one faces, it is important to keep one's mind on the job in hand. The chemistry surrounding the polymer industry is constantly being attacked for its negative aspects while the overwhelming benefits it may bring are overlooked. **Flame retardant chemicals get a lot of bad press, particularly halogenated materials. But as Anne Noonan of Great Lakes Polymer Additives reminds us, they also do a pretty good job of stopping people being burned to death.**

WHEN politics drives the debate over additives, the discussion often boils down to the call for a ban on all fire-retardant chemicals, starting with halogenated compounds. However, that position is increasingly at odds, not just with trends in the marketplace, but also with recent scientific findings and the resulting adjustments in official attitudes.

When science drives the issue the conclusions are very different. One global trend that is gathering pace is using science to confirm the key reason for using FR polymer additive technology in the first place -- namely, that these products save lives and property.

A look at developments in a number of countries reveals the strength of this trend.

Sweden: Sweden has been generally credited with being most strongly against FR additives, with its Chemical Inspectorate providing the leading critical voice. Yet, in April 2001, the Chemical Inspectorate criticised as 'inconclusive' research by environmental activists in their case against brominated FRs. 'Just because a fire retardant contains bromine doesn't make it dangerous,' said Eva Ljung of the Chemical Inspectorate in remarks published in the April 26 issue of Miljorapporten, an environmental advocacy magazine.

Of course, the Nordic countries are still looking closely and critically at FR products. In the past year, they've raised important questions about antimony, phosphate, and bromine-based FRs. But, again, the prevailing view highlights the role of these products in saving lives and property as the prime considerations.

The European Union: In Brussels, the European Commission and Parliament spent much time and many resources this year looking at brominated FRs in the context of the Waste Electrical and Electronic Equipment (WEEE) and hazardous substance ban (RoHS) directives. At one point, the Commission considered banning all brominated FRS. Instead, the Parliament passed a law that will ban rarely-used PBBs and PBDEs for electrical and electronic equipment.

Risk assessments are underway on two important FR additives -- decabrom and octabrom; scientific data generated to date proves that these chemicals are environmentally acceptable.

Japan: In Tokyo, the Japan Environment Association has recently changed its ecolabel criteria for copiers, printers, and PCs. The change withdraws the exclusion of all BrFRs to just PBBs and PBDEs, products that have little impact on the marketplace. Pressure for this change has filtered up from Japanese OEMs, who recognize the superior recyclability of BrFR plastics and also the consumer demand for greater fire safety.

Note: bolding created by search services

United States: In recent years, the National Academy of Sciences examined the effects on human health of 16 FR chemicals used to meet new furniture fire safety standards. The studies produced enough data for the Academy to definitively evaluate eight of these products -- and it concluded that there were no significant environmental or health concerns related to their use.

Public safety factors

The second major component of FR growth is the increased interest in improving public safety.

The US Consumer Product Safety Commission, the US Fire Administration, and the National Fire Protection Association issue annual estimates on fire losses in the nation. Based on these figures, it's estimated that there are approximately 400,000 residential fires each year requiring a response from firefighters. These fires kill about 4,000 people, with another 20,000 people suffering serious injuries from bums. The fires also result in property losses totalling about $4.5 billion. Of these fires, approximately 70,000 involve electrical distribution and appliances; another 40,000 stem from fires in upholstered furniture and mattresses.

Perhaps the most interesting aspect about these statistics is that they grossly underreport the problem. For example, a congressional investigation revealed that the federal government, which employs two million civilians housed in 8,300 buildings, has no data on even the largest of fires on its own property. Also, and incredibly, there have been years in which large states such as California and Pennsylvania have not reported a single fire. Clearly, this shows that nationwide fire reporting systems are severely inadequate.

In Europe, the system for collecting and reporting fire data is not much better, but even with partial data, the member states of the European Union report about 80,000 people are seriously injured in European fires each year. Of these, some 60,000 are hurt in their homes.

One subset of fires that has attracted attention is the number of European fires involving television sets. According to the Swedish National Testing and Research Institute, about 160 people die each year in Europe as a direct result of TV fires. In Sweden, for example, which has relatively lax fire-safety standards for the plastic housings of TV sets, there are 165 TV set fires per million population. By comparison, the United States, with stringent FR standards in this area, shows less than two TV fires per million in population.

For all the criticism of FRs -- not by the scientists or even the environmentalists, but mainly by politicians -- the European Commission acknowledges there would be 20 per cent more European fire deaths if FRs were not being used. In the same vein, the UK reports that 1,860 lives have been saved in the past decade because of its safety standards for upholstered furniture, due to the use of FRs.

Higher demand, greater safety

The marketplace is reflecting this need to deal aggressively with the massive destruction and cost of fires. Companies in turn are responding with products that use FR additives for extra safety in a vast array of Recent additions to the Great Lakes portfolio of flame retardants for thermoplastics are an intumescent additive for polypropylene and a high bromine content general purpose flame retardant for colour-sensitive compounds.

Reogard 1000 is a phosphorus nitrogen-based intumescent flame retardant that is melt blendable at PP's processing temperature and is said to increase the heat distortion temperature of the compound. It is also

Note: bolding created by search services

non-blooming, has good electrical properties, and has a reduced tendency to absorb water.

PP containing Reogard 1000 is recommended for use in V0 PP homopolymers and low polyethylene content co-polymers. It can also be used in a number of impact modified grades.

The new Firemaster 2100 is a non-diphenyl oxide based brominated flame retardant supplied as a uniform white powder suitable for formulations requiring white or light coloured products.

Applications foreseen include television cabinets, foam insulation and wire and cable.

www.pa.greatlakes.com

Factfinders:

Reogard 1000 122

Firemaster 2100 123

products, including highly combustible plastic outer casings and housings, candles, power cords, and the like.

In the United States, for example, televisions are made with fire-resistant outer housings. In Japan, some of the most prominent TV makers -- Panasonic, Toshiba, and Mitsubishi -- are now following suit. No regulatory agency or governmental power is making them do it. They just want to do the right thing.

This is another prevailing tendency that has recently emerged, what can be called the 'social conscience' trend. Companies are increasingly taking it upon themselves to improve the fire performance of their goods for marketing concerns, surely, but also to a greater extent for ethical reasons. Again, it's being done because it is the right thing to do.

There have been many 'Green Label' attacks on FRs over the years, but the untold story is that many ecolabels are increasingly reflecting the concerns that imposing restrictions on FRs may be creating increased risk of fire. In short, ecolabels often follow a common sense approach in their considerations of FR additives. As a result, more lives will be saved.

For instance, TCO, Sweden's ecolabel for computers, is expected to include fire safety standards in its next set of criteria. Why? **Fire safety officials from Sweden, Finland, France, the UK, Belgium, Germany, Canada, and the United States are worried about TCO's restrictions on FRs as a public safety question. Fire safety advocacy groups worldwide are urging TCO to include effective fire safety standards to help ensure that lives are not lost. Not just in Sweden, but governments worldwide are in greater agreement than ever before: fire safety and environmental concerns must be treated with equal importance.**

Product trends

The trends in upholstered furniture, appliances, and automobiles reflect significant increases in the use of FRs. Manufacturers are likewise responding with an array of new FR products and technologies.

Furniture: The US Consumer Product Safety Commission recently agreed to move forward with tough, new fire safety standards for mattresses. In July, California issued its own laws for improved safety of mattresses and bedding. In addition, a US consortium of companies and associations is now drafting federal legislation for higher safety levels for upholstered furniture, sleep products, candles, and cigarettes.

Appliances: The U.S. Underwriters Laboratory has just adopted new fire safety standards that will result in a significant increase in the use of FR chemical additives. More than 40 other UL standards, governing thousands of products, are directly affected, including hair dryers, toasters, power drills, and electric can openers. Manufacturers will have two choices: re-engineer their products or use fire-resistant outer

Note: bolding created by search services

housings. The early indication is that most manufacturers view FR additives as by far the more cost-effective approach.

Automotive: The US National Highway Transportation Safety Administration plans to change its rear-impact test for cars from 30 to 50 MPH. The implications for fire safety are enormous, as it will affect how fuel systems, interiors, and even some exterior parts are made.

Manufacturers respond

Increasingly, additives manufacturers are heeding the call of the marketplace for improved FR additives that meet customers' exact specifications. This call has been prompted by recent scientific data on the efficacy of such materials as well as a heightened desire for greater public safety.

Manufacturers of FR products are following more sensitive environmental practices. By doing so there will ultimately be far less reluctance by manufacturers to employ FR technology to meet the overwhelming desire for greater fire safety in consumer products.

Note: bolding created by search services

Flame Retardants.(flame retardents industry)
Wigotsky, Victor
Plastics Engineering, v57, n2, p22
Feb, 2001
ISSN: 0091-9578
Language: English Record Type: Fulltext
Document Type: Magazine/Journal; Trade
Word Count: 3925
TEXT:
Comparative photos show the dramatic fire-resistant effect of adding a small amount of nonhalogen Flamestab NOR116 retardant and synergist to the fibers of polypropylene cloth. Thirty-five seconds after flame removal, the test sample without the new additive burns intensely, while the protected material remains intact. Ciba Specialty Chemicals Corp.

The threatened bans on halogen flame retardants have not materialized in North America. These old standbys are holding their own, and new developments and market entries reflect a growing accepted technology. Still, the nonhalogens are making inroads in selected areas, although their cost and performance have not kept pace with those of the more efficient halogen products. In **Europe*****, however, the story is different. The nonhalogen flame retardants are thriving, and the halogen-based additives are under increasing restrictions because of environmental perceptions.

Geographical differences

"The market for flame retardants is a growing at an annual rate of 4% to 6%," says Russ Kidder, executive vice president of the Fire Retardant Chemicals Association (FRCA). **"The Europeans***** are willing to settle for less fire safety rather than to use brominated flame retardants. This is seen most dramatically in television sets, which, for the most part, contain no flame retardant at all. As one might expect, TV fires***** have increased***** greatly since flame retardants have been eliminated from TV sets in Europe*****.** In the U.S., television sets and some unattended appliances, contain halogenated flame retardants; in these applications, fire safety is at high levels. In most appliances where nonhalogenated flame retardants are used, either the flammability rating or physical properties are below U.S. standards. Nonhalogenated flame retardants are receiving more research effort in the United States and have made some inroads in the market place. But cost/performance favors the halogenated products in many applications.

"There have not been many new regulatory standards for flame retardancy or fire safety promulgation in the last several years" Kidder continues. "Meanwhile, the California Bureau of Home Furnishings is trying to modernize and upgrade California Technical Bulletin 117 for upholstered furniture. This state activity should be complete in 2001. The mattress industry, on the other hand, is cooperating with the regulatory groups to upgrade the present mattress standard. Research work is being completed at the National Institute of Standards and Technology (NIST) and a proposed mattress fire safety standard should be issued soon. One major problem with the mattress standard is, how do you factor in the contribution of sheets and blankets to the fire safety of mattresses? Activities in other markets are at the early stages of development. Plenum wire and cable is one of these that could affect the use of flame-retardant chemicals."

Phosphorus-based

Akzo Nobel's Phosphorus Chemicals introduced Fyrolflex BDP (bisphenol A diphenyl phosphate) as the next generation nonhalogenated flame retardant for engineered resins such as PC/ABS and PPO/HIPS blends. Another major product is resorcinol-based Fyrolflex RDP.

Note: bolding created by search services

Demand for bisphosphate nonhalogenated flame retardants continues to grow, especially in areas where environmental concerns are the strongest, says Ted Halchak, marketing and sales manager, The Americas. Capacity expansion and continued development of phosphorus-based approaches demonstrate Akzo's commitment to the future growth of nonhalogen-based products.

Fyrol PNX is an all-phosphorus, nonhalogenated flame retardant added to flexible polyurethane foam; one targeted area is in low-fogging automotive interiors. High efficiency permits reduced use levels. Fyrol CLP, containing chlorine and phosphorus, is being evaluated where flame-retarded flexible polyurethane foams are used.

The company expanded capacity of Fyrol PCF and Fyrol CEF, major flame retardants used in rigid polyurethane foams. The trend toward hydrocarbon blowing agents in place of fluorocarbons to comply with the Montreal Protocol has resulted in the incorporation of additional flame retardant to meet flammability standards. Volume expansion in the first quarter of 2001 will increase the availability of Fyrol FR2 and Fyrol 38, products used in the flexible polyurethane foam market. In the recent past, the growth rate of flame retardants in the flexible foam market has been just above GNP rates. Akzo Nobel's Phosflex flame-retardant plasticizers for flexible vinyl applications experienced moderate growth during the 1990s. A family of materials introduced a few years ago, the Phosflex 300 series, continues to make inroads through improved cost/performance.

Expanded line

Albemarle Corp. has been developing, acquiring, or forming alliances with other companies to expand its line with additional brominated products and new halogen-free technologies, such as phosphorus-based and zinc borate-based flame retardants. NcendX P-30 is phosphorus-based for PC/ABS, PPO/HIPS, and other polymers; Albemarle's first entry with halogen-free products, it is a clear liquid that improves the melt flow of the host resin. NcendX B-1000, UV, chemically, and thermally stable to 290(degrees)C, is another new halogen-free offering; it is zinc-borate-based and used as a flame retardant, smoke and afterglow suppressant, and anti-arcing agent. Available in **Europe***** , it is the product of a commercial agreement between Albemarle and The Borax Polymer Additives Group. Zinc borates have been used primarily in PVC, polyamide, and epoxy potting compound applications. Albemarle sees opportunities, however, in styrenics, engineering plastics, and other resins that take advantage of the low toxicity and flame-reta rdant performance of zinc borates.

Saytex CP-2000 (tetrabromobisphenol-A) is a highly pure reactive or additive flame retardant containing stable aromatic bromine and is produced in the world's first continuous process. As a reactive flame retardant, it finds application in epoxy and polycarbonate polymers and helps circuit boards achieve higher thermal stability and longer-term reliability than with nonhalogen additives. Tetrabromobisphenol A epoxy oligomer made with CP-2000 is widely used as an additive in styrenic polymers and many engineering thermoplastics, because the oligomer offers improved polymer viscosity and UV resistance.

Saytex HP-900 flame retardant is another of Albemarle's new bromine-based products. A high-purity grade of hexabromocyclododecane containing a high amount of aliphatic bromine, HP-900 allows reduced loadings. Its low melting point enhances melt processability, resulting in minimal effects on the mechanical properties of formulated systems. The flame retardant also has high solubility in common solvents, and offers potential to achieve transparent formulations. It can be used without the

addition of antimony oxide, primarily in extruded polystyrene or suspension-polymerized polystyrene foam. With a stabilization package and proper processing conditions, however, it is also usable in polystyrene and polypropylene resins.

Albemarle's Pyro-Chek 68PB, a brominated polystyrene additive for flame retarding a range of polymeric systems, features excellent thermal stability, moldability, and dispersibility. Because it is polymeric, it prevents migration and blooming in compounded resins. The company offers its own line of brominated polystyrene flame retardants as Saytex HP-7010. Saytex 8010 flame retardant responds to the increasing demand for recyclable polymer additives. Thermal stability and recyclability facilitate use in high-performance styrenic polymers, engineering resins, wire & cable, and elastomers. Outside analysis of samples demonstrated that resin compounds containing Saytex 8010 conformed to the German Dioxin Ordinance regulating dioxin and furan contents. Its chemical structure was developed to minimize the formation of brominated dioxins or furans during plastic processing, recycling, or incineration.

Most efficient

"Although there have been many reports, studies, and detailed looks at the overall health, safety, and environmental impact around the world on bromine flame retardants, to date there are no legislated bans or restrictions on the use of any of the commercially available bromine flame retardants," says Glade Squires, vice president, marketing, AmeriBrom, Inc. "The use of bromine flame retardants in all applications has been shown to be the most efficient and cost effective means of providing fire safety." The National Association of State Fire Marshals (NASFM) has publicly supported the use of bromine flame retardants in order to have the proper degree of fire safety built into goods. As a result, the use of bromine flame retardants continues to grow on a global basis.

Currently, the U.S. is poised to legislate the use of flame-retardant furnishings in all homes. The U.S. is behind other developed nations in requiring the use of flame-retardant home furnishings, Squires adds. So far, only California has had legislation covering flame-retardant standards for home furnishings sold in that state. California's fire statistics before and after the legislation testify to the need for this legislation nationwide.

As preparation was under way to draft legislation for furniture flammability standards on a nationwide basis, the Consumer Product Safety Commission (CPSC) enlisted the National Academy of Sciences (NAS) to make recommendations on the overall safety of flame retardants that would find their way into home furnishings. The final report from NAS, after it reviewed the requested data on these flame retardants, supported the complete and unrestricted use of both decabromodiphenyl ether and hexabromocyclododecane (two of the largest volume bromine flame retardants sold globally) in home furnishings.

Squires acknowledges substantial interest among OEMs in eventually incorporating nonhalogen FRs in their goods. "To date, however, there have been many nonhalogen FR products tested, and none has the overall efficiency and cost effectiveness of bromine flame retardants. Maintaining physical properties, particularly of thermoplastics, while achieving flame retardancy, is always the major challenge." Bromine flame retardants have been able to meet this challenge, Squires continues, based on the efficiency of bromine and the resulting low load levels needed to achieve the desired degree of flame retardancy. In the case of nonhalogen FR systems, the high load levels needed for these to perform severely affect physical properties and substantially increase the cost of final

formulations. "Also, the overall environmental impact of many of these nonhalogen FRs remains to be determined, since they have not been as thoroughly investigated and tested as bromine FRs." Nevertheless, to broaden its product portfolio, the Dead Sea Bromine Group (DSBG), a leading producer of magnesium hydroxide, also develops and sell nonhalogen flame retardants.

AmeriBrom's new products include FR-370, a bromine-containing phosphate ester that provides outstanding performance, light stability, and physical properties in polypropylene; and FR-372, an analog of FR-370 designed to meet the needs and demands of polypropylene fiber. FR-720, the 2,3 dibromopropyl ether of tetrabromobisphenol-A, used in polypropylene, provides expanded FR solutions for polypropylene by complementing the FR-370 in the product line.

FR-20 magnesium hydroxide is the Dead Sea Bromine Group's first nonhalogen flame retardant. Although not new, several unique surface treatments have grown sales significantly, particularly in TPOs. FR-6120 melamine cyanurate is DSBG's newest flame retardant. Although not new as a flame retardant to the industry, it provides more FR options for customers.

Effective retardant/synergist

Ciba Flamestab NOR116 is a new nonhalogen flame retardant and synergist for polyolefins. As a NORHALS, it also acts as a light stabilizer. It is effective at low concentrations and exhibits polymer compatibility and excellent extraction resistance. Since it is melt processable, it is less likely to diminish the functional performance of the host material. In addition, significant synergies can be realized when it is used with some traditional flame retardants, and the improved performance of these systems allows passing of some of the more stringent industrial standard flame-retardancy tests. Also, reduction in the level of conventional flame retardants can avoid detrimentally affecting light stability and mechanical properties.

Flame retardance results from the NFPA 701 and MVSS 302 burn tests on polypropylene knitted socks, without any flame retardant, show test failures, based on char length and after-flame criteria. The material, however, passed the tests with addition to the fibers of as little as a 0.5% concentration of Flamestab NOR116. Moreover, with the addition of NOR116 at levels as low as 0.25% in combination with brominated flame retardant, the polypropylene material passed the tests, allowing reduction of the halogen material by more than 50%.

Flamestab NOR116 can also be used as a synergist with traditional FR products in molded polypropylene.

Thermal stability

Great Lakes Chemical Corp. has developed three new products, Firemaster PBS-64, PBS-64HW, and CP-44B, out of its brominated styrene-based technology. Because of lower inherent hydrolyzable bromide content, they possess excellent thermal stability. Molecular weight is controlled to provide melt blendable, low melt viscosity polymers that improve the melt flow of filled and unfilled polyamides and polyesters. Firemaster CP-44B, a copolymer of brominated styrene and glycidyl methacrylate, has improved compatibility in polyamides and polyesters, helping reduce the amount of FR synergist needed.

The planned phase-out of lead-based solders in connectors and printed circuit boards will significantly increase the operating temperatures of the flame-retardant polymers. Flame retardants must not significantly reduce the heat resistance of their polymer hosts. The products show little effect on the HDT of the host polymer. Similar challenges are facing the manufacturers of printed circuit boards in

formulating for higher-temperature solder baths, whether based on tetrabromobisphenol A or the newer halogen-free systems.

Pressure has continued on the brominated **diphenyl***** **ether***** (**PBDE*****) flame retardants in Europe***** .EU***** risk assessment on these products is nearing completion. Keith Hughes, Great Lakes' senior global marketing manager, flame retardants, says it is expected that DecaBDE and OctaBDE will be shown to not pose unreasonable risks to health and the environment. However, it is expected that risk reduction measures will be imposed in **Europe***** on the use of PentaBDE. Great Lakes has developed new flame-retardant technology, Firemaster BZ-54, based on tetrabromobenzoate esters, as an alternative. A low-viscosity liquid, it contains 54% bromine and can replace pentabromodiphenyl-oxide-based flame-retardant systems in flexible polyurethanes and other applications.

Great Lakes has also introduced two new non-halogen systems mainly directed at PC/ABS and PPO/HIPS polymers. Reofos BAPP and Reofos 507 complement the existing Reofos TPP and Reofos RDP. Reofos BAPP, bisphenol A diphosphate, has excellent cost/performance and reduced tendency to migrate. Reofos 507, a proprietary monophosphate, is an alternative to BAPP in formulating low migration, hydrolysis-resistant PC/ABS compositions.

Debate in Sweden*** and Germany***** examines whether perceived environmental issues associated with PBDEs, and flame retardants in general, outweigh their benefits in improving fire safety. A recent Swedish research program demonstrated that the use of flame retardants can actually reduce rather than increase environmental pollutants such as dioxins, dibenzofurans, and polyaromatic hydrocarbons. The two-year study, conducted by the SP (Swedish National Testing and Research Institute) in collaboration with IVL (Swedish Environmental Research Institute), consisted of an extended life cycle analysis comparing a television set manufactured to U.S. standards of fire performance (UL 94V0) with a comparable European***** model with a typically HB-rated cabinet. It included U.S. and European***** TV set fire statistics and full-scale room burns, and investigated recycling, incineration, and landfill options at end of life. The analysis indicated that emissions of key environmental pollutants (such as dibenzodioxins and polyaromatic hydrocarbons) were lower for the TV cabinet containing DecaBDE and antimony trioxide than for a non-flame-retarded HB cabinet. It is estimated that 160 people will die and 2000 will be injured each year in Europe***** as a result of fires in TVs.**

Hughes says that as fabricators and compounders try to improve the efficiency and safety of their operations, many are looking to more formulated products that may offer multifunctional performance, improved handling, and reduced material usage. Great Lakes has introduced two new 100% active FR masterbatches. Fyrebloc 100 and 101 are 70% antimony oxide in a polybromostyrene binder. The nondusting masterbatches can be used in a variety of polyamide and thermoplastic polyester applications. The company also has introduced a briquette form of tribromophenol, PH73-FF, which has much improved flow and anti-caking tendencies over standard tribromophenol.

The National Association of State Fire Marshals, pressing for improved fire performance in upholstered furniture, petitioned the Consumer Products Safety Commission (CPSC) in 1993 to consider a possible flammability standard. In 2000, the National Research Council of the National Academy of Sciences (NAS) completed its assessment of the use of flame retardants for CPSC in its Toxicological Risks of Selected Flame Retardant Chemicals. The report concluded that eight of sixteen retardants that were evaluated pose little or no health risk. The way is now open for

Note: bolding created by search services

the CPSC to consider a small flame test ignition standard for upholstered furniture in the U.S. Meanwhile, an increasing number of the major U.S. furniture retailers have been voluntarily adopting the California CAL 117 furniture flammability standard for polyurethane foam used in furniture sold in the U.S. The Sleep Products Safety Council (SPSC) is working with the CPSC and NIST on the problem of mattress fires with the aim of developing an improved standard. These initiatives, if adopted, should lead to safer furniture and mattresses in the U.S. and an increased market for flame retardants in polyurethane foams and back-coating of fabric.

Broader supply

Harwick Standard is now supplying a full line of flame retardants to the thermoplastic industry--as it has in the past to the elastomer industry--says David R. Schultz, senior technical service representative. The company is a full-service distributor that supplies traditional flame-retardant systems that contain antimony oxide and halogens as well as halogen-free materials. The company has most recently aligned itself with Aluchem, a supplier of alumina trihydrate. Harwick Standard also distributes a range of products that permit a variety of compounding options.

Antimony trioxide

Oxychem is now the leading producer of antimony trioxide in the U.S., after acquiring the Fireshield line of antimony oxide from Laurel Industries and then more recently acquiring the Thermoguard line of antimony oxide from Atochem. Besides antimony trioxide, Oxychem produces the flame retardants Dechlorane Plus, Pyronil 45, and sodium antimonate. An aqueous dispersion of Dechlorane Plus and Fireshield H (antimony oxide) is now available from Oxychem, called Dechlorane Plus AD; this material is 67% active, containing a 3:1 ratio of Dechlorane Plus and Fireshield H. Dechlorane Plus can be used as a flame retardant in nylons and epoxies using several different syngerists to obtain a UL 94 rating. These synergists include different zinc compounds and also iron compounds. By using these different synergists, one can obtain higher CTI values, improved thermal stability on processing, and lower cost formulation.

Flame-retardant masterbatches

PMC Group Polymer Products provides new offerings within its standard and custom Endura flame-retardant masterbatches and has acquired the North American Avantra ignition-resistant HIPS product line from BASF Corp. Early in 2000, the company finished matching its masterbatches to many of the common resin grades, defining loadings that give UL 94V-2 and V-0 ratings, and measured the associated physical properties. Compounders can use the database to shorten time to commercialization of new flame-retardant compounds.

Polymer Products can provide in developmental quantities a highly loaded melamine cyanurate concentrate for unreinforced nylon and polyester, says business manager Don G. Barber, "that eliminates direct-addition processing difficulties and allows the compounder to produce a lower-cost nonhalogen system." Also, available in developmental quantities is a new masterbatch for PC/ABS. In addition, two cost-effective masterbatches designed for the construction film market, Endura PE-101 and PE-102, have recently been commercialized.

Non-lead PVC

PolyOne Wire & Cable says the company sees continued growth in non-lead PVC products. In response to this projected market need, PolyOne has new wet-rated, non-lead compounds for THHN-2 and THWN-2 applications slated for commercialization this year. Demand is also steadily growing for the company's LSFOH (low smoke zero halogen) products introduced into the

U.S. in 2000. While demand for flame-retardant PVC compounds will remain high, PolyOne expects to see continued growth for these materials in specialty contract work sectors such as mass transit, tunnels, airports, and nuclear reactors. The company's ECCOH (Enviro-Care zero-halogen) compounds remain an option for jacketing applications that must meet these requirements. PolyOne supplies standard and custom grades of polyvinyl chloride (PVC) insulation and jacketing materials for a wide range of cable applications, including riser, plenum, and low-acid gas PVC cable.

Plenum cable

The growth of computer and communication networks has greatly increased use of plenum spaces for cabling, says Donald G. Ouellette, industry manager, Vinyl Division, Teknor Apex Co. As one generation of computer and communications technology succeeds another, new cable runs are necessary to accommodate the changes. A typical generation of plenum cable lasts three to five years before replacement. More often than not, installers leave the old cable in place and run the new cable alongside or on top of it, **increasing***** the **fire***** load within the enclosed space. And because plenums facilitate the movement of air within buildings, the fire performance requirements for cables installed there are especially stringent. Teknor Apex developed its PVC-based Fireguard line of low-flame, low-smoke compounds because of the high levels of flame resistance and smoke suppression required in plenums. Ouellette says the company diversified the Fireguard program by developing a series of nonhalogenated Fireguard LSZH (low-smoke, ze ro-halogen) compounds. "While U.S. standards bodies place their greatest emphasis on compounds that pass the most stringent flame test," he comments, "their counterparts in **Europe***** currently emphasize compounds that mitigate or remove the perceived potential for halogenated cables to generate excessive amounts of smoke and irritating or corrosive gases during a fire. Our Fireguard LSZH compounds are selling very well," Ouellette continues, "but not for cable to be installed in the U.S. Most companies that purchase LSZH compounds use them for cables to be installed in **Europe***** . In the U.S., where fire safety standards for communications cable are more stringent, there is no strong opposition to halogenated materials like PVC."

The European*** market for data and communications cable is dominated by nonhalogenated compounds consisting of polyolefin resin (typically polyethylene or its major copolymers) and such flame retardants as aluminum trihydrate and magnesium hydroxide. These materials pass the JEC 60332 test for riser***** cable, which is the most stringent fire***** test mandated in Europe***** , but not the NFPA plenum-cable test. In effect, there is no plenum cable standard in Europe***** . In addition, the standard riser-cable test specified in the U.S., based on UL 1666, is more stringent than that in LEC 60332. "Several of our Fireguard***** LSZH compounds can pass both riser***** tests," says Ouellette.**

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Flame retardants-a cause for debate.

AUTHOR: Tyler, A.
JOURNAL: Performance Chemicals Int., (Performance Chemicals International (PCI)), Volume 14, Issue 2, Page(s) 5
ISSN: 0950-3870
PUBLICATION DATE: Mar/Apr 1999 (19990300) LANGUAGE: English
ABSTRACT**: A report from the University of Surrey on the risks and benefits of flame retardants has boosted the case for brominated flame retardants. Risks and benefits in the use of flame retardants in consumer products was commissioned by the UK Department of Trade and Industry. It indicates that the risk of death or injury from a fire involving consumer products can be reduced by 30-90% by using flame retardants.** A European Union risk assessment of polybrominated diphenyl ether flame retardants is scheduled for publication in summer 1999. Environmental groups in a number of European countries have claimed that there are significant potential risks to human health and the environment linked to the use of flame retardants.
DESCRIPTORS: flame retardants ; risk assessment ;
CHEMICAL SUBSTANCE(S): polybrominated biphenyl ethers
SECTION: Precautions (12)
SECTION CROSS-REFERENCE: Hazardous Waste Management (02)

Note: bolding created by search services

Gale Group PROMT(R)

06920310 Supplier Number: 58418336 (THIS IS THE FULLTEXT)

TESTING/STANDARDS: Forum Attacks Sweden Food Toxin Article.

Flame Retardancy News, v9, n12, pNA
Dec 19, 1999

TEXT:

An Oct. 11 article in Sweden's daily newspaper, Dagens Nyheter, entitled "Environmental Toxin Discovered In Food," inaccurately described tetrabromodiphenylether as a flame retardant, according to Bromine Science and Environmental Forum chair Michael Spiegelstein (BSEF, 118 Ave. de Cortenbergh, 1000 Brussels, Belgium; Tel: 32-2 733 93 70, Fax: 32-2 735 60 63). TeBDE is not a flame retardant used in consumer electronics, says Spiegelstein. TeBDE is called a flame retardant in the article, because it has been used as a minor part of one flame retardant application for other uses, he says.

A possible explanation for the occurrence of TeBDE in the Baltic is its previous use in bell-bore fluids by the oil industry in the North Sea, and for several years in hydraulic fluid used by the mining industry in Northern Europe. All of the use was stopped several years ago, because those using the substance realized how emissive it was.

One of the studies of polybrominated diphenyl ethers (PBDEs) presented at the Dioxin '99 conference recently held in Venice, Italy indicates that the levels of PBDEs in the environment actually are leveling off and in certain cases even diminishing. The logical conclusion of this is that the levels that can be found in the food chain in all probability also will soon drop, asserts Spiegelstein.

If levels in the environment continue falling, as is expected, says Spiegelstein, it is a confirmation that the probable source is the earlier use in the oil and mining industries, and that it has no connection with flame retardants. Blaming every finding of TeBDE on flame retardants is not only factually incorrect-it also causes unnecessary concern about flame retardants, he says. **Flame retardants are not used frivolously in many consumer products. On the contrary, they can mean the difference between survival and death in a fire. After the use of brominated flame retardants in television sets was stopped in Sweden, television fires increased by 100%, he points out.**

BSEF agrees with the authors of the article that TeBDE in the food chain is a serious issue, Spiegelstein says. But, he adds, to automatically blame flame retardant use without investigating other possible explanations risks the existence of such flame retardants on a lack of a scientific base. A balanced scientific approach must be taken-it must not be forgotten that brominated flame retardants save lives in potential fire situations every day, he urges.

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08291668 Supplier Number: 65773465 (THIS IS THE FULLTEXT)
Melting in the heat.(International Pages)(Statistical Data Included)
HARDING, PAUL; CROMPTON, GEOFFREY
Asia Pacific Coatings Journal, v13, n4, p16
August, 2000
TEXT:
Many conventional fire retardants are reduced in effectiveness at elevated temperatures when a fire is at its most dangerous. Special ceramics cause the paint to melt and then fuse to form a permanent, impervious glaze. Fire costs millions of dollars and hundreds of lives every year. Although Asia has a small number of fires in proportion to the population size, the fires can cause much more damage and a higher loss of life. The estimated impact of fires in each continent is listed in Table 1. The financial cost of fires is exceptionally high. Direct property losses **amount to 0.2% of total GDP and the total cost of fire to society has been calculated at 1% of GDP.** Protection of life and property from the effects of fire has been a topic of a great deal of research, much of which has focussed around prevention of the spread of flame using barriers and flame retardants. To recognise how these materials work, you have to understand the burn process.

Continent	Population (millions)	Fires per year (millions)	Fire deaths per year (thousands)
Europe	**720**	**2.2**	**25.0**
Asia	3660	1.0	30.0
North America	**470**	**2.3**	**6.5**
South America	340	0.5	2.5
Africa	780	0.8	5.0
Australia	30	0.1	0.3
TOTAL	6000	6.9	69.3

	Average per year	
	Fires per 1000 people	Fire deaths per 1000 fires
Europe	**3.1**	**11.4**
Asia	0.3	30.0
North America	**4.9**	**2.8**
South America	1.5	5.0
Africa	1.0	6.3
Australia	3.2	3.0
TOTAL	1.2	10.0

Table 1: distribution of fires by continent (Courtesy of the Centre of Fire Statistics of CTIF)

The development of a fire is a cyclic phenomenon. From a small ignition source, enough heat is released to create an initial fire. This initial fire can then increase the ambient temperature to the point at which repeat ignition occurs, leading to fire growth, releasing more heat and combustion side products, including partially oxidised flammable gases. The next stage is where most of the remaining material erupts into flames. This flashover generates a developed, mature fire that quickly becomes difficult to contain. The

mature fire may burn through containing walls, propagating the flame to adjacent areas and leading to re-ignition, starting the cycle. Flame retardants block the growth of flame. The common methods of retarding the flame include:

* halogenated systems: halogenated materials, such as antimony trioxide or pentoxide, chlorinated paraffin and other brominated materials, are considered to be effective flame retardants. There is, however, mounting concern about the toxicity of the gases generated in large quantities during the combustion of halogen-containing polymers. Despite their efficiency, there is growing demand for halogenated systems to be banned and substituted with non-halogenated systems

* non-halogenated systems: the majority of non-halogenated flame retardants produce water vapour or carbon dioxide at elevated temperature to stifle a flame. Typical non-halogenated flame retardants include alumina trihydrate Al((OH).sub.3) (or ATH), huntite MgCa(((CO.sub.3)).sub.4, and magnesium hydroxide Mg((OH)).sub.2. These materials are effective in stifling a burn at low temperatures and over an extended period of time. However, both methods stop acting at elevated temperatures. When a fire is at its most dangerous, and once the extinguishing agent has been exhausted, they often provide a source of fuel for a flame.

At higher temperatures, a greater defence is needed. Materials, such as zinc borate, can help create a glassy char and slow heat flow through the host material. The chars formed by these materials block heat flow and suppress afterglow once a burn has stopped but are brittle and often fall off a substrate, revealing more fresh surface to burn, reducing the host material's integrity. Finally, there is a material type that is active at higher temperatures. It slows or stops entirely the emission of smoke and toxic fumes, forms a stable glassy char to limit heat flow and retains host integrity. This material is called `Ceepree' and is composed of a mixture of glass-like and ceramic-like materials known as flits. Rather than relying on chemical emission degradation, Ceepree works by melting as the temperature increases. The secret to the success of this material lies in its ingredients.

Conventional glass flits, similar to window glass, begin to melt at about 650 (degrees) C and pass through melt, flow and fusion as a steady transition with increasing temperature. Figure 1 shows the behaviour of three frits when heated. The graph represents the typical area of a sample viewed from the side using a thermodilatometer. A conventional frit would undergo the following stages during a melt:

(Figure 1 ILLUSTRATION OMITTED)

* softening: the material begins to melt; the volume has not yet changed significantly (up to 600 (degrees) C in Figure 1)

* sphere: the volume of the frit reduces considerably as the melt progresses and the voids between particles disappear. (600-800 (degrees) C in Figure 1)

* half-sphere: the melt is underway, the volume continues to reduce as the cohesion of the sample pellet is lost entirely. (800-1100 (degrees) C in Figure 1)

* fluid: the frit is now a liquid. As a fire barrier material, a conventional flit would run and drip off the substrate at this point (above 1000 (degrees) C in Figure 1)

To counteract this final stage, Ceepree contains a devitrifying frit that crystallises and sets hard prior to final melt and fusion. Figure 2 shows the action of a devitrifying flit under temperature, softening at 800 (degrees) C, melting and flowing to 950 (degrees) C

at which point it crystallises (devitrifies) and remains in that hard state to 1200 (degrees) C before melting and flowing again to fuse at 1300 (degrees) C.

(Figure 2 ILLUSTRATION OMITTED)

A material that did not begin to protect until a fire was already established and mature would not be useful so, to enable earlier activation of the material, Ceepree often contains a soft frit or one activated at a lower temperature. Figure 3 shows the melt of a typical soft frit occurring at approximately 450 (degrees)C.

(Figure 3 ILLUSTRATION OMITTED)

Combining these materials (Figure 4) gives a product that activates at the same temperature as Iow melt frits but devitrifies when the soft frit begins to flow and stays in this crystalline state to 900 (degrees) C. It flows and fuses between 900 and 1100 (degrees) C. This final flow and fuse, when used in a flammable carrier material, binds the char together and protects substrates by promoting formation of an insulating layer of glass and carbonaceous char.

By adding components, it is possible to modify the behaviour of a Ceepree frit combination to include some intumescence before and during the devitrification stage. The intumescence (foaming) provides more protection to the host material by providing a hard shelled foam of cells filled with carbonaceous char. Formation of this carbonaceous foam results in a significant increase in volume of the material as shown in Figure 4.

(Figure 4 ILLUSTRATION OMITTED)

HOW CEEPREE WORKS

The action and changes in the Ceepree material can be demonstrated by using the simplest grade of Ceepree, `C200', as an example. * below 350 (degrees) C: Ceepree is an inert component in the host material. (Figure 5)

* 350-400 (degrees) C: when heated below its activation temperature of around 350-400 (degrees) C (the soft frit melt point), the low melt components within the Ceepree formulation begin to melt, causing vitreous material to flow over and around the burning host material and beginning to form a char. The picture (Figure 6) shows Ceepree after the melt. The continuous surface forms an effective barrier to fire and smoke. The melt and flow process is endothermic and absorbs the heat from the flame. The char helps to disperse and absorb heat from the flame. The encapsulation of the host also inhibits the access of oxygen to the combustible materials, preventing carbonaceous and volatile decomposition products from being emitted as smoke.

* 750-800 (degrees) C: the devitrifying component of the Ceepree begins to act, passing from a glassy to a crystalline state and remaining in place, maintaining host material integrity and adhering the char to the surface of the host material.

* 1200 (degrees) C: the Ceepree is still continuing to act, retaining its integrity and protecting the host material.

Typical activation profiles for Ceepree flit combinations are shown in Figure 7.

This type of fire barrier solution has a number of uses, from paint to plastics, caulks, mastics and structural panels, and it can save lives.

Ceepree-based systems are effective in small, enclosed areas or where there is potential for a mature flame to spread. It prevents the emission of toxic fumes, and can provide valuable escape time. The formation of a continuous surface to starve the flame and the action of

the char in absorbing heat can also provide time for the fire to be fought. Ceepree-based paints have been used in US Navy submarines, where the suppression of smoke and flame is vital and in multi-story apartments where the potential for the spread of a mature flame from one apartment to another is extremely high. Ceepree-based systems can also be used over existing solvent based paints or onto bare or primed wood.

RECENT DEVELOPMENTS

Ceepree has evolved significantly since it was patented 12 years ago. Despite its advantages, it was limited in application because of some limitations:

* Water solubility: some elements of the frit mixture were initially chosen for their partial water solubility, making Ceepree unsuitable for exterior application. An insoluble mixture of frits has now been formulated for paints, plastics, cable and exposed areas.

* pH of the flit combination: Ceepree in its simplest form is alkaline and can cause stability problems and setting in many acrylic resin systems. The solution to this problem was simple but had evaded many paint formulators. Small amounts of boric acid (itself a fire barrier material) counteract the alkalinity of the Ceepree, and stable grades are now available for use in water-based media.

* particle size: Ceepree was originally only manufactured with a median particle size of 30um. This prohibited its use in paints requiring thin film application, many powder coatings and extruded thermoplastics. Two finer grades of Ceepree - `Microfine', with a (d.sub.50) of 5(micro)m, and `Ultrafine', also with a (d.sub.50) of 5(micro)m but with the coarse tail removed, show promise in thermoplastic cabling and powder coatings. The finer particle sizes give Ceepree Microfine and Ultra fine grades a higher surface area and form the continuous surface more readily.

* high activation temperatures: the original Ceepree frit combination did not become active until 450(degrees)C, at which point the fire was already burning freely. Ceepree Products is currently working on the commercialisation of two patented grades with lower activation temperatures.

Ceepree materials have one other great advantage. Their action is a physical change of state rather than a chemical process so they are compatible with all known fire retardant systems and can be used in conjunction with them.

In many cases, the flame retardant will act synergistically with the Ceepree, lowering the activation of the soft frit and slowing the melt process, providing better encapsulation of the host material. Although the action of Ceepree-based systems is simple, the relationships with other fire barriers and flame retardants can be complex. Ceepree Products offers a high level of technical support to help users to reach an economically viable protection from flames.

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November 11, 2004

Ms. Cheri Peele
Policy Analyst
Washington State Department of Ecology
PO Box 47600
Olympia, WA 98504-7600

Dear Cheri:

Thank you for the opportunity to comment on the Washington State Chemical Action Plan draft on PBDEs.

Based on the overwhelming weight of scientific evidence, the Bromine Science and Environmental Forum respectfully – but strongly – disagrees with the preliminary conclusions of the Department of Ecology on Decabromodiphenyl ether (Deca-BDE).

This evidence demonstrates that:

DecaBDE in the environment does not contribute to environmental PentaBDE levels

- In the instances where Deca-BDE has been detected in sediments, anaerobic degradation studies indicate no significant degradation of Deca-BDE to lower BDEs. These results are confirmed by additional data on anaerobic degradation reported in a study by the Swedish EPA [Prof. Cynthia de Wit of the Institute of Applied Environmental Research (ITM), Stockholm, 2000]

- Experts agree that the vast majority of PBDE's detected in the environment originate from the Penta-BDE flame retardant, whose production is being discontinued voluntarily by its sole manufacturer. [Soderstrom et al, 2004; Prevadourus, 2004]

There is no risk identified for continued use of Deca-BDE

- On May 26, 2004, EU Member State policy regulators issued a favorable European scientific risk assessment of Deca-BDE, reaffirming its use without restrictions. This concluded a 10-year, multi-million dollar Risk Assessment process.
- A report from the California Senate Office of Research, issued in May of 2004, concluded that there was insufficient information to support a ban of deca in California.
- In 2003, the Voluntary Children's Chemical Evaluation Panel (VCCEP) Risk Assessment submitted by industry (and later reviewed by a panel of independent experts) found that Deca-BDE poses no significant risk to children's health.

BSEF ▪ Bromine Science and Environmental Forum
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- The National Academy of Sciences reported in 2000 that the use of Deca-BDE in textile applications was acceptable and posed little risk.
- The World Health Organization concluded of Deca-BDE: *"Risk to the general population from [Deca] is considered to be insignificant."*
- The U.S. Consumer Products Safety Commission concluded: *"[Deca] is not likely to present a hazard to consumers."*

There may be a potential negative impact on fire safety

- Hasty action could create a gap in fire safety should Washington ban the use of Deca-BDE. According to the *2003 Fire in Washington* report from the office of the Washington State Fire Marshal, 1,195 citizens of the state have lost their lives due to fire. Historically, the majority of those who perish in fire are the very young and the elderly. Deca-BDE is critical to the continued fire safety protections for the citizens of Washington, and any ban on this product could set a dangerous precedent that could put more citizens in harm's way.

There may be a potential negative impact on Washington businesses

- Restrictions in Washington may damage the state's competitiveness. If overly stringent regulation is enacted, some manufacturers may be forced to consider Washington a 'separate market' that demands a 'separate product' – one that would likely cost more for consumers since it is tailored to that market. Consumers may therefore be forced to shop out-of-state in order to seek out affordable products or – of greater concern – to guarantee they are purchasing adequately fire-safe products. For current users of Deca-BDE, to force substitution will add unnecessary financial burdens that will render Washington State businesses less competitive.

Deca-BDE alternatives are not proven, studied, available

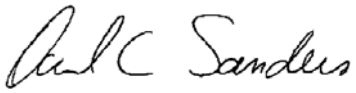
- Forcing substitution to less well understood materials may simply create new problems in the future. Given Deca-BDE's low risk profile, the common sense approach is to maintain use while also monitoring additional information on Deca-BDE and other flame retardants. There are at present no completely acceptable substitutes or alternatives for Deca-BDE that:
  - Are available on an industrial or commercial scale
  - Provide Deca-BDE's flame retardant capabilities in terms of physical and flammability properties
  - Are a cost-effective substitute or alternative for Deca-BDE
  - Have been tested as rigorously as Deca-BDE and found to be safe from both an environmental and human health perspective

Restriction on Deca-BDE will hurt Washington's growing plastics recycling industry and will force used plastics into landfill or other disposal options

- Where mechanical recycling is available, these materials are well-suited for recovery. There is also increasing demand for flame-retarded recycled material in the plastics industries.

Based on this evidence, detailed in the attachment, the Department of Ecology should not recommend restriction of any kind on Decabromodiphenyl ether (Deca-BDE). BSEF does support a conclusion of "continued monitoring" of the Deca-BDE literature on a biannual basis.

Sincerely,



David C. Sanders, Ph.D
Past-Chairman
Bromine Science and Environmental Forum

REFERENCES

www.bsef.com

Closure of Deca-BDE Risk Assessment: UK House of Commons Official report, July 6, 2004

Swedish Environmental Protection Agency, "Brominated Flame Retardants", Cynthia de Wit, Report 5065, 2000

Hale R, La Guardia M, Harvey E, Mainor T. 2002. Potential Role Of Fire Retardant-Treated Polyurethane Foam As A Source Of Brominated Diphenyl Ethers To The US Environment. Chemosphere, Feb, 46(5):729-35.

Hardy M. 2002a. The Toxicology Of The Three Commercial Polybrominated Diphenyl Oxide (Ether) Flame Retardants. Chemosphere 46, 757-777.

Hardy M. 2002b. Properties of the Major Commercial PBDPO Flame Retardant, DBDPO, in Comparison to PBB and PCB. Chemosphere 46, 717-748.

Hardy M. 2001. BFRs In Breast Milk – Which Ones, How Much And What Does It Mean? Proceedings, American Chemistry Council's Brominated Flame Retardant Industry Panel, Brominated Flame Retardants Workshop. Nov 13, 2001. Arlington, VA.

NTP 1986. Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No.1163-19-5) in F344/N Rats and B6C3F1 Mice (Feed Studies). National Toxicology Program Technical Report Series No.398. U.S. Department of Health and Human Sciences. Public Health Service. National Institutes of Health. Research Triangle Park, NC.

Submissions to Voluntary Children's Chemical Evaluation Panel (VCCEP) available at www.tera.org:

- Decabromodiphenyl Ether sponsor-submitted assessment document
- Octabromodiphenyl Ether sponsor-submitted assessment document
- Pentabromodiphenyl Ether sponsor-submitted assessment document

FLAME RETARDANTS MANUFACTURERS COMMENTS

OF NOVEMBER 11, 2004

ON THE

DRAFT

WASHINGTON STATE PBDE CHEMICAL ACTION PLAN

DATED OCTOBER 11, 2004

FLAME RETARDANT MANUFACTURERS' COMMENTS: KEY POINTS

- The Draft PBDE Action Plan of October 11, 2004 addresses PBDEs generically. It does not clearly distinguish between the three PBDE products in composition, volumes, applications, toxicology and detection in the environment. Because of this, the Draft unjustly penalizes the major PBDE product, Decabromodiphenyl ether/oxide (Deca).

- The Pentabromodiphenyl ether (Penta) and Octabromodiphenyl ether (Octa) products will be voluntarily discontinued by their sole U.S. manufacturer by the end of 2004. The U.S. EPA will issue a Significant New Use Rule governing their future use (if any) in the United States.

- Given that the Draft Document was produced over the summer of 2004, after the announcement that production of Penta and Octa would end, Deca should have been the focus of the Draft Document. Instead, the Draft Document addresses 'PBDEs' generically, and presents information that is largely related to the Penta product.

- The Draft's recommendations for action are the result of environmental detection of PBDE congeners[1] that are associated with the Penta product. Appropriate recommendations for Deca cannot be based on the Penta product. Recommendations for action should be based on risks presented by Deca, the PBDE in commerce after 2004.

- Human health risks have not been identified for Deca, and the Draft Document concedes that any concern with Deca rests with its potential degradation in the environment.

- Evidence for Deca's environmental degradation to the congeners of concern is not presented. Evidence indicating that the congeners of concern detected in the environment originated from the Penta and/or Octa products is not considered. This has led to erroneous conclusions regarding Deca's potential for environmental degradation.

- The unintended consequences, if Deca's use is prohibited in Washington State, on human health and the environment are not considered. Deca's use in consumer electronics and upholstery textiles saves lives, reduces property loss, and prevents environmental pollution from fires. Prohibition of Deca could produce opposite effects if no, or a less effective, flame retardant were substituted.

- The risk-benefit ratio is not considered in the recommendations pertaining to Deca. The benefits derived from Deca's use far outweigh any potential risks.

[1] BDE-47, 99, 100, 153, and 154

Page iii-iv, Executive Summary

Please see our detailed comments that follow.

Page v, Recommendations Summary

Two recommendations have to do with either measuring or minimizing workplace exposure to polybrominated diphenyl ethers (PBDEs). The Pentabromodiphenyl ether (Penta) and Octabromodiphenyl ether (Octa) products will not be in commercial production after the end of 2004. The US EPA will issue a SNUR covering any future use of these products. Therefore, there will be no use of these 2 products in the workplace. Efforts to either measure or minimize exposures would appear an unnecessary expenditure of tax dollars. The American Industrial Hygiene Association recommends an occupational exposure limit of 5 mg/m3 for the Decabromodiphenyl ether (Deca) product. This is equivalent to a nuisance dust. Efforts to reduce total dust exposures in the workplace would appear appropriate, but reductions based on concern for potential toxic effects due to Deca would not.

Another recommendation suggests developing and communicating ways for the general public to minimize exposure to PBDEs. Again, the Penta and Octa products will not be in commercial production after the end of 2004, and the US EPA will issue a SNUR covering any future use of these products. These efforts should effectively minimize future exposures to the general public. Given that current levels of the congeners associated with the Penta and Octa products are far below any known effect level, there appears no public health crisis that warrants further measures. Exposures to Deca are far below that predicted by the U.S. National Academy of Sciences as causing no harm whatsoever. Thus, no action is needed with respect to Deca.

Another recommendation would ask the Washington State legislature to prohibit manufacture, distribution and sale of new consumer electronics or upholstery fabric containing Deca. This recommendation has no merit. Deca has a NOAEL of at least 1000 mg/kg/d in repeated dose studies. The fact that it presents essentially no risk to human health has been concluded in numerous evaluations. Electronics and upholstery fabric have been shown to be a negligible source of Deca the consumer. Deca's current use in these products reduces the numbers of fires, saves lives, reduces injuries, and prevents property loss. A recommendation to prevent Deca's use serves no useful purpose in terms of protecting public health, and could have the opposite effect if no, or a less effective or less thoroughly tested, flame retardant were substituted.

I. Introduction

The Introduction relates that considerable effort went into developing a "broad understanding of PBDEs". This broad understanding, with little depth, is reflected in the generic nature of the Draft Document. The Draft does not distinguish between the composition, applications, toxicology, and environmental dispersion of the three commercial products. The shallow review conducted in the course of developing the Draft Document has resulted in inappropriate

recommendations for Deca, with the result that a valuable and nonhazardous material may no longer be available to the citizens of Washington State. This in turn may have unintended consequences for public health.

While the Draft Document's section on human health and toxicity was reviewed, only one of the 7 reviewers is a toxicologist. Our comments re the reviewers of Appendix A (Photolysis) are provided later in this document.

II. PBDEs: Intended Purpose and Applications

The 3rd paragraph, page 3, states that because additive BFRs do not form chemical bonds with the resins in which they are used, they are "much more likely to leach out of goods and products" compared to reactive FRs. This comment is frequently made where BFRs are discussed, and is true in the absolute sense. Whether the amount 'leached' is meaningful is another discussion. For example, few of the many different substances commonly added to the resins used in consumer goods are covalently bound. These additives include but are not limited to colorants, antioxidants, UV inhibitors, plasticizers, and mold releasing agents. What is critical about these additives is not whether they are covalently bound, but their hazard and rate and extent of migration such that meaningful exposures occur. Exposure to a non-hazardous substance does not equal risk. In the same manner, exposure to a hazardous substance at levels below those causing toxicity also does not equal risk.

The 4th paragraph states that PBDEs do not exist naturally. However, hydroxyl and methoxy-PBDEs are known to be synthesized by various marine organisms. A more correct statement would be that unsubstituted PBDEs are not known to exist naturally.

The statement in the Draft Document that the commercial products contain fewer congeners than the 209 possible due to a lack of stability and a tendency to debrominate is untrue. The three PBDE products are actually quite stable under normal conditions of storage. A more correct description of PBDE manufacture and their resultant composition would be: "PBDEs are manufactured in a closed system by the chemical reaction of bromine with diphenyl ether/oxide. The amount of bromine added to the system and the time allowed for the reaction controls the extent of bromination on the diphenyl ether molecule. Far fewer than the potential 209 possible PBDE congeners are found in the commercial Penta, Octa and Deca products. This is because the oxygen bridge has a strong directing influence on the addition of the bromine atoms to the phenyl rings. Basic principles of organic chemistry are responsible for the fewer number of congeners in the commercial PBDE products compared to the former PCB products."

Various publications, including the Draft Document, emphasize that there are potentially 209 PBDE congeners. The significance and reason for this emphasis is unclear, but may relate to the former PCB products. PCBs also have the potential for 209 different congeners, and the former PCB products were highly complex mixtures with many different levels of chlorination. This is very different from the PBDE products which are composed of a relatively few number of congeners. The reason that the PCB products were composed of so many different congeners is

that the biphenyl structure does not create a strong directing influence. This is in contrast to the diphenyl ether molecule where the ether bridge directs where on the rings that the halogen will be added.

Page 4. Table 1. We recommend this table be deleted. Its contents are inaccurate. Table 2, page 5, is accurate, but incomplete. Table 2 should include that the Penta product's composition is mainly BDE 99, 47, 100, 153 and 154, that order.

Page 4. Last para. The description of the 3 commercial PBDE products is inaccurate. We suggest: "There are three commercial PBDE products. The three products are known generically as Penta, Octa and Deca."

Page 5. The second paragraph should also include that over 80% of the global production of "PBDEs" consists of Deca, and that after the end of 2004; Deca will represent all of the PBDE production. As the paragraph is written now, it gives a skewed view of the relative volumes of the 3 commercial products. Given that the Draft Document was produced over the summer of 2004, after the announcement that production of Penta and Octa would end, Deca should have been the focus of the Draft Document. Instead, the Draft Document addresses 'PBDEs' generically while presenting information that is largely related to the Penta product.

Page 5. How PBDEs work. The three sentences in this section do not address these flame retardants' mechanism of action. The three sentences simply say that flame retardants provide ignition resistance and slow flame spread, but do not address HOW they do this. We believe a basic understanding of how flame retardants act is important to understanding why flame retardants, including PBDEs, are used.

We suggest replacing the 3 sentences with the following, and moving the section "Purpose of PBDEs" on page 6 to immediately before the section on "How PBDEs work":

"The PBDEs are used solely as flame retardants for the purpose of preventing or delaying ignition in combustible materials. Their flame retardant activity is derived from their bromine content. Bromine is one of the few elements able to provide flame retardancy in the gas phase. The ability of a flame retardant to act in the gas phase of a fire can be critical, because of the way certain plastics burn. Some plastics form gaseous compounds when they burn, and, thus require a flame retardant that exerts its action in the gas phase. Because of this, for a given resin in a given application, the most suitable substitute for one BFR is another BFR. If a BFR cannot be used, then typically the resin must also be substituted which often is an expensive proposition.

The chemical elements primarily responsible for flame retardance in thermoplastics are phosphorus, bromine and chlorine. Phosphorus compounds affect char formation, that is, condensed phase reactions, and typically are not as effective in plastics that form gaseous compounds when they burn. Bromine and chlorine form gaseous species that react in the gas phase with high energy radicals to terminate the combustion reaction, e.g. gas phase reactions. Bromine is unique in its efficacy as a flame retarding species and its compatibility with certain

plastics (especially engineering thermoplastics). No other chemical element provides equivalent flammability protection for materials requiring gas phase flame retardancy.

Fire is an exothermic, gas-phase reaction. A plastic will burn as long as the heat supplied is enough to sustain thermal degradation. The combustion reaction is maintained by free radicals and radiant heat. The reaction proceeds at an increasing rate until flashover as long as the available free radicals and heat exceed the energy required for combustion. Conversely, the rate of combustion will decrease to extinction if the available energy is less than required to maintain equilibrium. Flame retardants take advantage of this and reduce the heat supplied to the polymer to below the critical level needed to maintain combustion. They do this by three basic methods: scavenging the free radicals that propagate combustion, limiting the heat and mass transfer across the solid-gas phase boundary, or by creating a heat sink.

Scavenging Radicals In The Gas Phase. Gas phase flame retardants out-compete oxygen for the free radicals generated in the combustion process to terminate the reaction. The flame retardant must form a gaseous component in order to do this, and must produce the gaseous component at the same temperature at which the polymer decomposes. Very few elements have the ability to form gaseous compounds. Halogens are some of the few chemical elements with this ability and there are very few halogens that are effective flame retardants. The order of reactivity of the halogens as radical scavengers is I > Br > Cl > F. Iodine is the most effective scavenger, but is very expensive and lacks the thermal and photolytic stability required for most thermoplastic applications. Bromine is the next most effective radical scavenger and is the element most widely used by gas phase flame retardants. Chlorine is considerably less effective than bromine because it only marginally competes with oxygen for hydrogen radicals and the aromatic C-Cl bond is too stable. Fluorine has virtually no effect as a flame retardant due to the stability of C-F and H-F bonds. Because of these limitations, there are very few gas phase flame retardants. To function as a gas phase flame retardant, the compound must: (1) decompose to form a gaseous radical-scavenging species at the temperature the polymer begins to burn, and (2) successfully compete with oxygen for high energy free radicals to terminate the combustion reaction. The bottom line for plastics is that no other gas phase flame retarding species is as efficient or effective as bromine.

Limiting Transfer Across Solid-Gas Phase Boundary. Some polymers form a carbonaceous char when decomposed by heat. This char increases ignition resistance by reducing the amount of available fuel and by providing a heat barrier. Phosphorus is the principal condensed phase flame retardant. Phosphorus's mechanism of action in oxygen-containing polymers is through thermal decomposition to phosphoric acid. Phosphoric acid extracts water from the burning substrate and thereby increases the amount of char.

Phosphorus compounds are effective condensed phase flame retardants for polymers with char-forming tendencies, such as polycarbonate and polypheneylene oxide. In general, polymers that do not inherently form char as they burn cannot utilize condensed phase chemistry for flame retardancy.

Physical Action As A Heat Sink. Physical action flame retardants act as heat sinks. These flame retardants are inorganic compounds that give off nonflammable gases such as water and carbon dioxide in endothermic reactions and cool the burning substrate. Aluminum trihydrate (ATH) and magnesium hydroxide are two examples of physical action flame retardants.

Polymers begin to burn at temperatures between 150 and 400°C. In order to be effective, the flame retardant must decompose in the temperature range of the decomposing polymer. ATH begins to decompose at about 230°C, which is too low to function as flame retardant in engineering thermoplastics. It also requires very high loadings (between 40-80% by weight) that are detrimental to the performance properties of the thermoplastics. ATH is primarily used in polyesters and latexes, and it is the largest volume flame retardant in the world. Magnesium hydroxide decomposes at about 300°C and it is used primarily in polypropylene and polyethylene terephthalate."

Page 6. Purpose of PBDEs. We suggest moving this section prior to "How PBDEs work". It seems more logical to discuss why flame retardants are used prior to discussing their mechanism of action.

The six sentences in this section do not convey why PBDEs, and other flame retardants, are used. We believe the serious health risk and environmental pollution created by fires has not been adequately considered by the Departments of Health and Ecology. As a consequence, the threat to human and the environment presented by fires, and the benefits derived by preventing them, have not received the scrutiny deserved.

The last sentence that "Strict U.S. fire safety regulations may be a reason that flame retardants are used more here than in other countries" is incorrect. The U.S. has NO requirement mandating use of flame retardants, and in fact, lacks any regulation at all with respect to flammability in key areas, such as residential furniture and consumer electronics. Residential furniture, except in California, relies on voluntary measures of the furniture manufacturers. Most consumer electronics, e.g. TVs, computers, stereos, etc., rely on voluntary compliance with UL standards. UL has no statutory mandate. The U.S. does, however, have a serious problem with fires – we have one of the highest fire incidence and mortality rates of all developed countries.

We suggest replacing the six sentences with the following:

"Flame retardants are an important component of fire safety. Despite the combined efforts of fire departments, building codes, fire drills, fire alarms, smoke detectors, fire sprinklers, fire extinguishers, UL ratings and flame retardants, fires continue to be a serious public health problem, and drain on resources (see the Table: The US Fire Problem - 2001). The U.S. has one of the highest fire incidence and mortality rates of all developed countries according to the National Fire Protection Association (NFPA).

The most recent statistics indicate someone dies in a fire in the U.S. about every two and half hours. Most of those deaths are in the home. Those dying are typically the very young, the very old, and the economically challenged. The total cost of fire in the U.S. is estimated at $186-305 billion depending on whether the events of September 11 are included. According to NFPA (Hall J. 2003. The Total Cost of Fire in the United States. June. National Fire Protection Association, Quincy, MA): "The conclusion that fire has a tremendous impact on the way the U.S. uses its scarce resources is indisputable." And "It also is clear that we have a dual interest in reducing U.S. fire losses – which include human losses that are among the highest per capita in the industrial world – and in seeking ways to achieve equivalent fire safety at lower costs, since the growth in total cost of fire has been led not by the fire losses but by the other cost components. This provides a clear indication of need for product innovations or other programs (e.g., educational) that can improve fire safety at the same or lower costs. It also shows the need for improved methods (e.g. models) for calculating fire performance and costs, so the implications of different choices can be considered and judged more comprehensively."

Fires represent a serious risk in Washington State; the State Fire Marshall's 2003 *Fire in Washington* reported "Tragically, fire claimed the lives of 43 victims last year. It is imperative that the fire safety message continue to be shared so residents know how to prevent fire from occurring in places where they live, work, and play." Fire incidents resulted in more than $135 million in dollar loss in 2003. Residential properties accounted for 35% of the total reported fire incidents and approximately 55% of the total dollar loss, more than $64 million. The top three heat source categories involved in fire incidents were related to electrical equipment. Deadly fires occurred most frequently in places where people sleep, e.g. single-family dwellings; 55% of the fire victims were between the age of 35 and 64 years of age.

The National Fire Protection Association (Karter, Jr. M. 2003. Fire Loss in the United States During 2002. September 2003. National Fire Protection Association, Quincy, MA) reports that in the year 2002:

- Every 19 seconds, a fire department responded to a fire somewhere in the United States.
- Public fire departments attended 1,687,500 fires, of which 519,000 occurred in structures, 329,500 occurred in vehicles, and 839,000 occurred in outside properties.
- Nationwide, there was a civilian (non-firefighter) fire death every 156 minutes. There were 3,380 fire deaths, a decrease of 9.8% from the previous year, excluding the events of 9/11/01.
- About 79% of all fire deaths occurred in home fires. There were 2,670 deaths from fires in the home, a decrease of 14.1% from the previous year.
- Nationwide, there was a civilian fire injury every 28 minutes. There were an estimated 18,425 civilian fire injuries, of which 14,050 occurred in homes.

NFPA concluded that fire safety initiatives targeted at the home are key to any reductions in overall fire death toll, because 79% of all civilian fire deaths there (Karter 2003). One of the five recommendations made by NFPA to reduce these deaths was to seek additional ways to make home products more fire safe. The wider use of upholstered furniture and mattresses that are

The U.S. Fire Problem – 2001

Reported to Fire Departments	2001	Compared To 2000	1991	1981
Fire Incidents	1,734,500	Up 2%	Down 15%	Down 40%
Civilian Deaths				
Including 9/11/01	6,196	Up 53%	Up 39%	Down 8%
Excluding 9/11/01	3,745	Down 7%	Down 16%	Down 44%
Firefighter Deaths				
Including 9/11/01	439	Up 326%	Up 306%	Up 223%
Excluding 9/11/01	99	Down 4%	Down 5%	Down 24%
Civilian Injuries				
Including 9/11/01	21,100	Down 6%	Down 28%	Down 31%
Excluding 9/11/01	20,300	Down 7%	Down 29%	Down 32%
Firefighter Injuries	82,250	Down 3%	Down 20%	Down 20%
Direct Property Damage				
Including 9/11/01	$44,023,000,000	Up 293%	Up 365%	Up 559%
Excluding 9/11/01	$10,583,000,000	Down 6%	Up 12%	Up 59%
Adjusted for Inflation				
Including 9/11/01		Up 282%	Up 258%	Up 239%
Excluding 9/11/01		Down 8%	Down 14 %	Down 18%
Civilian Deaths per Thousand Fires				
Including 9/11/01	3.6	Up 35%	Up 39%	Up 34%
Excluding 9/11/01	2.2	Down 7%	Up 1%	Up 10%
Civilian Deaths per Million Population				
Including 9/11/01	22.1	Up 51%	Up 63%	Down 54%
Excluding 9/11/01	13.4	Down 9%	Down 1%	Down 7%
Property Damage per Fire				
Including 9/11/01	$25,381	Up 287%	Up 447%	Up 1000%
Excluding 9/11/01	$6,101	Down 7%	Up 32%	Up 164%
Adjusted for Inflation				
Including 9/11/01		Up 276%	Up 321%	Up 466%
Excluding 9/11/01		Down 10%	Up 1%	Up 36%

Sources: *Fire Loss in the United States,* (1981, 1991, 2000 and 2001), by Michael J. Karter, Jr., Fire Incident Data Organization (FIDO), and U. S. Census Bureau. Inflation calculations derived from a custom table created from purchasing power of the dollar for all urban consumers at www.bls.gov/cpi on April 24, 2003.

more resistant to cigarette ignitions was cited as an example of change that has "already accomplished much and will continue to do more".

Basic Fire Concepts Important to Understanding the Need for Flame Retardancy. Fire is dark. In television and movies, fire is often portrayed as a bright light, but the fire environment is actually pitch black due to the dense smoke produced. Escape plans must be memorized (USFA. 2002. This Is Fire! A Fact sheet on the nature of fire. United States Fire Administration http://www.usfa.fema.gov/dhtml/public/safety.cfm; Education World. 2002. Lesson Planning Article. Fire Safety: Activities to Spark Learning! http://www.education-world.com/a_lesson/lesson026.shtml).

Smoke from fire kills. Fire victims typically succumb to smoke inhalation before flames reach them. More fire deaths occur when people are sleeping—between 2 a.m. and 6 a.m.

Many people believe – falsely - that they would awaken in a fire. But toxic gases, typically carbon monoxide, actually put people into a deeper sleep.

Fire is intensely hot. This might seem obvious, but few understand that fire can cause the temperature to rise *several hundred degrees* in seconds. That degree of heat can prompt the human body to stop functioning and lose consciousness, making escape impossible.

Fire is fast. A home can be completely consumed by fire in less than five minutes. In less than 30 seconds a small flame can get completely out of control and turn into a major fire. It takes only minutes for thick black smoke to fill a house. Time is the biggest enemy and every second counts.

Flame retardants prevent or delay ignition, reduce the rate of heat release, reduce the quantity of toxic gases generated, and increase the time available for escape. Studies have shown that flame retardants can increase escape time by a factor of 15. In a fire where every second counts, this can literally mean the difference between life and death. (Babrauskas et al. 1988. Fire Hazard Comparison of Fire-Retarded and Non-Fire-Retarded Products. U.S. Department of Commerce, National Bureau of Standards, NBS Special Publication 749. Available From National Technical Information Service (NTIS), Technology Administration, U.S. Department of Commerce, Springfield, VA 22161. Website: http://www.ntis.gov. Order number: PB88-249966)

Groups at High Risk of Death and Injury in Fires. Populations at high-risk of death, injury or burns in fires are the very young, the elderly, and the economically disadvantaged (NFPA 2000. National Fire Protection Association. Http://www.nfpa.org; National SAFE KIDS Campaign (NSKC). Residential Fire Injury Fact Sheet. Washington (DC): NSKC, 2004; Stevens G and Mann A. 1999. Risks and Benefits in the Use of Flame Retardants in Consumer Products. URN 98/1026. Polymer Research Centre, School of Physical Sciences and School of Biological Sciences, University of Surrey, Guildford, Surrey GU25XH, UK, DTI). Children ages 5 and under, who represent 9 percent of the population but more than 17 percent of all fire-related deaths in the home, are more than twice as likely to die in a fire as the rest of the population. A child's risk of dying in a fire is twice the national average. Adults 65 and older also face a risk

twice the average, while people 85 and older had a risk that is almost four-and-a-half times more than average. Home fires and home fire-related deaths are more likely to occur during cold-weather months, December through March. The South has the highest fire-related death rate in the country, 21 percent higher than the national rate. Home cooking equipment is the leading cause of residential fires and fire-related injuries. However, residential fires caused by smoking materials (e.g., cigarettes) are the leading cause of fire-related death and the third leading cause of fire-related injury.

Children Are at Special Risk in Fires. Fires and burns are the fifth leading cause of unintentional injury-related death among children ages 14 and under. Children, especially those ages 5 and under, are at the greatest risk from home fire-related death and injury, with a fire death rate more than twice the national average. A less acute perception of danger, less control over their environment, and a limited ability to react promptly and properly to a fire contribute to this excess risk (NSKC 2004). In 2001, 493 children ages 14 and under died in residential fires. Nearly 54 percent of these children were ages 4 and under. Each year, nearly 40,000 children ages 14 and under are injured by fires in the home. More than 70 percent of all fire-related deaths are from smoke inhalation, caused by toxic gases produced as fires develop and spread. Burns are responsible for an additional 25 percent of fire-related deaths. Smoke inhalation alone accounts for more than half of all fire-related injuries to children ages 9 and under.

The majority of fire deaths and injuries occur in homes without a working fire alarm while the residents are asleep. A working smoke alarm is not present in two-thirds of the residential fires in which a child is injured or killed. Children in homes without smoke alarms are at greater risk of fires and fire-related death and injury. Almost 55 percent of children ages 5 and under who die from home fires are asleep at the time, while nearly one-third of these children are too young to react appropriately.

Children playing with fire account for 5 percent of residential fires, yet cause 40 percent of residential fire-related deaths among children. More than half of all child-play home fires begin in a bedroom, often while children have been left alone to play. Roughly children playing with matches or lighters start three out of five of these fires.

The number of candle-related fire deaths, most caused by candles left unattended or inadequately controlled increased 20 percent between 1998 and 1999, hitting a 20-year peak. A child playing with or near a candle is one of the leading contributors to candle-related fires.

Male children have a higher rate of fire-related death and injury than female children. Studies indicate that by age 12, half of all children have played with fire. Males are nearly twice as likely as females to have played with fire. Children from low-income families are at greater risk for fire-related death and injury, due to factors such as a lack of working smoke alarms, substandard housing, use of alternative heating sources and economic constraints on providing adequate adult supervision. Children living in rural areas have a dramatically higher risk of dying in a residential fire. Death rates in rural communities are more than twice the rates in large cities and more than three times higher than rates in large towns and small cities. Black children are more than twice as likely as white children to die in a fire. More than 43 percent of residential fire-related deaths among children ages 9 and under occur when the child is attempting to escape,

unable to act or acting irrationally. Although an escape plan may help to reduce these deaths, only 25 percent of households have developed and practiced a plan. People with a physical or cognitive disability are more than twice as likely to die in a house fire. Limited mobility may interfere with a child's ability to escape, and cognitive impairments may interfere with a child's awareness of imminent danger.

The total annual cost of fire- and burn-related deaths and injuries among children ages 14 and under is more than $11.9 billion. Children ages 4 and under account for more than $4.1 billion of these costs.

Flame Retardants – Protection Through Prevention. Years ago, most combustible building contents were made of cellulosic materials commonly found in nature (Leihbacher D. 1999. Search in the Modern Environment. Fire Engineering, July, 65-76). Chairs and tables were made of wood, sofas and bedding with cotton batting and jute, carpeting with wool and cotton fibers, and draperies with linen and other natural materials. Rapidly spreading fires were uncommon and generally indicated the use of a petroleum-based accelerant like gasoline. Today, the furnishings in homes and businesses include those constructed of petrochemicals such as polyurethane foams and rigid polystyrene plastic. These materials can behave in a fire as if they have built-in-accelerant, and can produce quantities of heat exceeding those of ordinary combustibles.

Another change from the past is that today's buildings and homes have more contents. The fire load in residential structures has more than doubled in the past 50 years on a pound per square foot basis (Leihbacher 1999). Flashover, when the room bursts into flame and the most dangerous time of a fire, has become more common as a result of the greater fire load and the use of synthetic furnishings. Synthetics, especially foams and plastics, produce more heat than natural products - the heat produced by burning foams and plastics can approach that of highly volatile flammable liquids. This contributes to the development of flashover[2] so that flashover now occurs rapidly - generally within 3-10 minutes after ignition. Flashover signals the change from a contents to a structure fire and the beginning of the structural collapse danger.

Another change in modern buildings and homes is increased energy efficiency (Leihbacher 1999). Buildings are designed to hold heat inside in the winter and exclude heat in the summer. Over the last 20 years new energy-efficiency standards have come into effect, and better and more insulation of walls, floors, ceilings, roofs, and windows has occurred. This higher energy efficiency influences the building's behavior during a fire. Energy efficient upper walls and ceilings are less able to conduct heat away from the fire room, resulting in a higher temperature fire in the room of origin. Energy efficient thermal pane windows are less likely to break and vent the fire's heat outdoors than older window types. In the event of a fire, the net result of enhanced energy efficiency is rooms that burn hotter and hold heat better.

[2] Flashover is caused by the radiation feedback of heat. Heat from the growing fire is absorbed into the upper walls and contents of the room, heating combustible gases and furnishings to their auto-ignition temperature. This build up of heat in the room triggers flashover. Flashover signals the end of an effective search and rescue in a room; it means the death of any person trapped in the blazing room — either civilians or firefighters.

The combination of higher energy efficiency and a greater quantity of synthetic materials increases the potential for a serious fire if ignition occurs (Leihbacher 1999). Thus, the extensive use of synthetic polymers has intensified the need and concern for flame retardancy in many applications. Flame retardants are especially useful in flammable foams and plastics where they act to delay ignition and slow flame spread. Flame retarded products, once ignited, generate a lower rate of heat release, which slows development of flashover. A slower rate of heat release also lowers the quantity of toxic gases produced. These factors translate into longer escape times for occupants - the use of flame retardants can increase escape times by a factor of 15 (Brabrauskas et al. 1988; FRCA 1987. Fire Retardant Chemicals Association: Reduction of Fire Hazard Using Fire Retardant Chemicals. Belles and Associates, Madison, TN) – and save lives (Clarke F. 1997. The life safety benefits of brominated flame retardants in the United States. Final Report to the Chemical Manufacturers Association Brominated Flame Retardant Industry Panel. Benjamin/Clarke Associates).

The benefits of brominated flame retardants (BFRs) in the U.S., in terms of lives saved, were determined using fire data from the National Fire Protection Association (NFPA) (Clarke 1997). Four product classes were identified in which BFRs are widely used and which could be directly associated with fire data: television/appliances, wire/cable insulation, curtains/draperies and upholstered furniture. Based on this data, an estimated 190 lives are saved annually through the use of BFRs (e.g. Deca) in television cabinets. For electrical insulation and draperies, less product and fire data were available, but 80 and 10 lives, respectively, were estimated saved annually through the use of BFRs in these products. Again, Deca is a major flame retardant used in electrical insulation and in draperies. Thus, an estimated 280 deaths are avoided each year in the U.S. due to the use of BFRs. A large portion of these lives saved are likely attributable to Deca. Another 140-220 fire deaths per year could be avoided if upholstered furniture fabrics were backcoated with BFR-latex as is now done to meet California standards for upholstered furniture.

Sources of Additional Information on Fires and Their Impact. Additional information on fires in the U.S. can be found on the following websites. This is only a partial list and there are many other excellent sources of information on this topic. The State Fire Marshall can also provide information on the local situation as well as educational tools and services.

The United States Fire Administration (USFA): www.usfa.fema.gov.
The USFA's Kid's Page: www.usfa.fema.gov/kids.
National Fire Protection Association (NFPA): www.nfpa.org.
Consumer Product Safety Commission (CPSC): www.cpsc.gov.
International Association of Fire Chiefs: www.iafc.org.
Education World, Lesson Planning, Fire Safety Activities: www.education-world.com.
National Safe Kids Campaign: www.safekids.org."

Page 6 and 7. PBDE Applications

We believe it is important to highlight that the applications of the 3 PBDE products, Penta, Octa,

and Deca, do not overlap. Each product and its applications should be listed individually.

There are several errors in this section. Penta is NOT used in "other upholstered furniture". The draft should clearly state that Penta is used in flexible polyurethane foam that is used as cushioning in upholstered furniture.

Octa is used almost exclusively in acrylonitrile-butadiene-styrene (ABS). Kitchen appliance housings are not routinely flame retarded; a fact that has resulted in a number of fires in these appliances.

We recommend clarifying that Deca's main use is in high impact polystyrene (HIPS), which is in turn used in television cabinet backs, and in wire and cable insulation. About 80% of Deca's volume goes into these applications. The remainder of Deca's volume goes into a flame retardant backcoat for upholstery textiles. The majority of flame retarded upholstery textiles in the U.S. is used in commercial applications and autos. Except in California, residential furniture is not required to meet any fire safety standard and is not routinely flame retarded. Deca's safety in upholstery textiles was reviewed by the U.S. National Academy of Sciences at the request of Congress in response to a potential Consumer Product Safety Commission rule. The U.S. National Academy of Sciences found Deca's use in upholstery textiles did not present a risk to the consumer, including children, and calculated an oral reference dose (RfD) of 4 mg/kg/d.

We also recommend adding the following re Deca:

"According to U.S. EPA Toxic Inventory Release (TRI) records, only one manufacturer of consumer electronics operates in Washington State and uses Deca in its processes. According to these records, this manufacturer is doing an exemplarily job handing Deca. This manufacturer reports no on-site releases or off-site disposal of Deca, and all Deca waste is handled via recycling. Deca's properties make it extremely suitable for recycling – it maintains its flame retardant properties through repeated recycling cycles. This is one of the advantages of Deca that may not be shared other FRs – its ease of recycling. In comparison, the 2002 total releases (off-site for disposal or other) for all TRI-listed chemicals in Washington State were 21,982,283 pounds, with an additional 18,606,434 pounds sent for further waste management."

IIII. Unintended Consequences: PBDEs, Human Health and the Environment (page 8)

PBDEs and Human Health

Human exposure to PBDEs

Page 8-11. PBDES in human tissues. This section requires extensive clarification. The generic term "PBDEs" is used throughout with no explanation of which individual PBDE congeners were measured in each study and whether the studies cited are sufficiently similar in

terms of analytical methods, study design, congeners measured, group sampled, year of sample collection, etc. such that it is valid to draw conclusions. For example, the statement is made that "The highest levels of PBDEs in human tissues collected from the general public have been found in the U.S. and Canada." Industry recognizes that several authors have made similar statements in the published literature. However, we can find that none of these authors attempted to determine that the various studies were sufficiently similar or representative such that comparison of the levels reported in each study is valid. We can find no evidence that the Department of Health attempted to do so, either. We believe a governmental agency, such as Washington's Department of Health, should be held to a higher standard when it comes to public health.

In addition, the total amount of PBDEs reported in human tissues in the various studies is seldom mentioned in the Draft Document. Readers have no opportunity to determine for themselves whether the levels detected are meaningful in term of health effects.

We find no evidence that the Department of Health critically evaluated the references cited. For example, page 10, says that some people have "very high tissue levels (high end) compared to the average tissue levels of all people tested" and cites a 2004 abstract by McDonald as the basis for this conclusion. McDonald's 4-page abstract cited 6 studies reporting PBDE levels (Which congeners? Same congeners measured in each of the 6 studies?) in women (Not all people; Similar ages?) in various tissues (Adipose, serum, or milk; Same congeners measured in all tissues from each individual?). The total number of individuals included in the 6 studies was 170, which is hardly representative of the U.S. population. The assumption that adipose, serum and milk PBDE levels are similar for each PBDE included in each of the different totals has not been validated.

This section should clearly report that the "PBDEs" typically detected in tissues are those associated with the Penta product which will be phased out by the end of 2004. These "PBDEs" are BDE47, 99, 100, 153, and 154. A single PBDE, BDE-47, typically makes up 50-70% of the total. Deca (BDE-209) is uncommonly detected, and when detected typically makes up only a very small fraction of the total.

Page 11. First paragraph. This paragraph discusses BDE-209 detection in breast milk, blood, sewage sludge, dust samples, etc. Not once are the levels detected mentioned; yet the level detected is a critical determinant of risk. Unless the Department of Health intends to recommend bans on all man-made substances detected in any matrix associated with human activity, the actual levels detected and the hazard associated with the substance at those levels must be determined.

This section also has errors. Butt et al. (Environ. Sci. Technol. 2004. 38, 724-731) did not report BDE-209 as the main PBDE congener in indoor air. Air concentrations were not measured in that study. Estimates were calculated for 12 congeners, not including BDE-209. BDE-209's level was not estimated because "reliable Koa values are not available and this compound is not expected to attain significant concentrations in the gas-phase". The major congener in air was BDE-47 (~60% in both indoor and outdoor air) followed by BDE-99 (15%).

Human Exposures to PBDEs – General Population, Pages 11-14.
This section again states that "PBDEs" have been detected in various matrixes, but does not report which congeners were detected, the amounts of each, or even the total amount (sum of congeners analyzed) detected. Without such information, no reasoned judgment can be made and the sole point conveyed by the section is simply that analytical methods are capable of detecting these compounds.

The three commercial PBDE products, Deca, Octa, and Penta, are included in the U.S. EPA Voluntary Children's Chemical Evaluation Program (VCCEP). Each of the three has gone through the VCCEP submission and peer consultation process. Potential exposures, using accepted principles, were estimated for each product. The resulting estimates of infants' and children's exposure to Deca in the U.S reported in a leading journal (Hays et al. 2003. Exposure of Infants and Children in the U.S. to the Flame Retardant Decabromodiphenyl Oxide (DBDPO). Journal of Children's Health, Vol 1, No. 4, pp.449-475). Deca's estimated exposures from this work are reproduced in the Table below. Deca's exposure estimates clearly show that this flame retardant presents no risk to infants or children. This exposure information should be included in the Draft Document, and the failure to do so implies bias or a less than thorough evaluation on the part of the Department of Health.

DBDPO exposure estimates and hazard quotient based on a RfD of 4 mg/kg/day.

Daily Intakes	Exposure Duration (yrs)	Exposure Estimate (mg/kg/d)		Hazard Quotient (RfD = 4 mg/kg/d[e])	
		Reasonable	Upper	Reasonable Estimate	Upper Estimate
Pathway-specific					
Ingestion, breast milk-manufacturer	0–2	1.9E-02[a]	3.4E-01	0.005	0.09
Ingestion, breast milk-disassembler	0–2	3.3E-06[a]	2.5E-05	8E-07	6E-06
Ingestion, consumer electronics	0–2	4.3E-06	2.5E-04	1E-06	6E-05
Ingestion, mouthing fabric (NAS)	0–2	2.6E-02	2.6E-02	0.007	0.007
General exposures	0–70	1.2E-03	3.9E-01	0.0003	0.1
Aggregate					
Infant, manufacturer[b]	--	0.046[b]	0.76[b]	0.01	0.2
Infant, disassembler[c]	--	0.027[c]	0.41[c]	0.007	0.1
Lifetime (0–70)[d]	--	0.0012[d]	0.39[d]	0.0003	0.1

[a] Assumes a shorter duration for nursing (0–3 months), based on Collaborative Group on Hormonal Factors in Breast Cancer 2002.
[b] This value incorporates the intakes for ingestion of breast milk from a mother who is a manufacturer, plus ingestion from consumer electronic products, ingestion from mouthing fabric, and general exposures.
[c] This value incorporates the intakes for ingestion of breast milk from a mother who is a disassembler, plus ingestion from consumer electronic products, ingestion from mouthing fabric, and general exposures.
[d] This value incorporates the intake from general exposures. See text for details.
[e] The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data. The RfD for DBDPO, 4 mg/kg/d, was calculated by the U.S.

National Academy of Sciences instead of using the current 1999 IRIS Rfd (0.01 mg/kg/d). NAS calculated a revised RfD for DBDPO using the NTP 2 year bioassay results, which were not available at the time of the IRIS derivation (1984-1985).

Human Exposures to PBDEs – Workers, Pages 14-15.

This section also dwells on mere detection, and does not report measured levels. The closest this section comes to reporting air levels is when it states that the Deca levels detected in the air (value not given) at a Swedish recycling plant were 25,000 times below the AIHA WEEL of 5 mg/m3. The actual value of those air levels, 0.0002 mg/m^3, was not mentioned.

This section also says that BDE-209 was detected in "high levels in the blood of the electronics dismantlers"; yet doesn't report what those levels were. In actuality, the BDE-209 levels were nearly non-detectable - ~5 pmol/gm blood lipid. These levels were exactly what were predicted based on the measured workplace air level of 0.0002 mg/m^3. Hardy in *Assessment Of Reported Decabromodiphenyl Oxide Blood And Air Levels In Swedish Workers And Their Workplace,* 2001 ACC BFRIP Brominated Flame Retardants Workshop November 13, 2001, Washington D.C., reported: "The DBDPO blood levels reported in Swedish electronics dismantling workers (5 pmol/g lipid) and computer technicians (1.6 pmol/g lipid) were extremely small and are representative of our increasing ability to detect minute amounts of chemicals in various media. The DBDPO blood levels were far below those of PCB 153 (dismantlers, 760 pmol/g lipid; technicians, 290 pmol/g lipid) measured in the same workers. Further, the electronics dismantling workers' internal DBDPO dose (1.2 ng/kg body weight) based on their measured blood level was comparable to the level expected (0.57 ng/kg body weight) calculated from the measured air levels. A similar comparison was not possible for the computer technicians because air values were not reported for that workplace." A full copy of Hardy's paper is attached.

We can compare the measured air levels in the Swedish recycling plant to the AIHA WEEL in more detail. The measured DBDPO air level at the Swedish electronic recycling plant was 0.0002 mg/m.. The American Industrial Hygiene Association (AIHA) evaluated Deca's toxicology and set a Workplace Environmental Exposure Level (WEEL) of 5 mg/m^3, e.g. that of a nuisance dust. Thus, the measured DBDPO air level at the electronics dismantling plant was 25,000 times below the AIHA level to which workers could be exposed every day with the expectation of no adverse effects. Further, using the equation $A_{dose} = A_c TVA_{bs}$ and a maximum absorption of 2%, the estimated internal DBDPO dose from an 8-hr exposure at the AIHA WEEL of 5 mg/m^3 would be 0.11 mg/kg body weight. The internal dose of the electronic recycling workers was 1.2 ng/kg or 0.001% of the internal dose that could be received at a DBDPO exposure equal to the AIHA WEEL. Finally, in the event that DBDPO absorption from the respiratory tract was greater than 2%, the internal dose of the electronic recycling workers at a measured DBDPO air level of 0.0002 mg/m^3 would remain substantially below that achievable at the AIHA WEEL. For example, if DBDPO absorption equaled 100%, the internal dose due to a workplace air level of 0.0002 mg/m^3 would be 0.004% of that dose which could be received at a DBDPO exposure equal to the AIHA WEEL.

Estimates of human daily intake of PBDEs, Pages 16-17.

This section, a recitation of information in the published literature, is incomplete. Table 5

requires modification to include Hayes et al. (2003) and the Deca VCCEP submission. The exposure estimates conducted for the U.S. EPA Voluntary Children's Chemical Evaluation Program (VCCEP) for the Deca and Octa products were not included. Those exposure estimates were performed using accepted methodologies and were peer reviewed. The exposure estimates for Deca were published (Hays et al. 2003. Journal of Children's Health, Vol 1, No. 4, pp.449-475), and are far below the oral RfD, 4 mg/kg/d, established by the U.S. National Academy of Sciences. Washington State's Department of Health apparently made no attempt to perform its own exposure estimate, nor is there any indication that Department of Health attempted to verify whether the published information related in this section was appropriate for use.

Toxicity of PBDEs

This section is inadequate. The available toxicology data on the three commercial PBDE products is much more extensive than provided in this limited review.

The 2 review articles recommended for additional information are inadequate. One was written by authors with no personal experience in the conduct of toxicology studies on these products. The second article's author's toxicology experience was limited to BDE-47 or the Penta product; this author had no experience with the Deca or Octa products. At a minimum, the review by Hardy (*The toxicology of the three commercial polybrominated diphenyl oxide (ether) flame retardants.* Chemosphere 2002 46:757-777), and the VCCEP submissions for the Deca, Octa, and Penta should be consulted by Washington State Department of Health, and included as references. The VCCEP submission for Deca is attached.

Page 19. DecaBDE.
The single paragraph on Deca's toxicology is inadequate. It fails to convey the exemplary characteristics of this product. Deca has a very large toxicology database with studies ranging from acute to 2 year carcinogenicity and covering virtually all endpoints. Its NOAEL in repeated dose studies is at least 1000 mg/kg/d (Hardy M. Chemosphere 2002 46:757-777). It's NOEL for developmental toxicity is 1000 mg/kg/d (Hardy et al. *Prenatal oral (gavage) developmental toxicity study of decabromodiphenyl oxide in rats.* International Journal of Toxicology 2002 21:83-91). For a complete review, see the ACC BFRIP submission on Deca under the EPA's VCCEP. Also see the U.S. NAS's review of Deca's toxicology in Toxicological Risks of Selected Flame Retardants. The Washington Department of Health should also consult Hardy M. Chemosphere 2002 46:757-77; and Hardy et al. International Journal of Toxicology 2002 21:83-91.

The statement to the effect that Deca's large size would prevent it from being absorbed needs clarification. Industry has always maintained that Deca's absorption is low (e.g. 0.3-2-3% of an oral dose). This is based on the work of the U.S. National Toxicology Program (Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No.1163-19-5) in F344/N Rats and B6C3F1 Mice (Feed Studies). 1986. National Toxicology Program Technical Report Series No.398. U.S. Department of Health and Human Sciences. Public Health Service. National Institutes of Health. Research Triangle Park, NC.) and El Dareer et al. (*Disposition of decabromodiphenyl ether in rats dosed intravenously or by feeding.* J Toxicol Environ Health,

1987 22:405-415). There are some that have interpreted 'low absorption' to mean 'absolutely no absorption', and who now claim detection of Deca in human tissues disproves previously assumptions.

Table 6 requires correction. The Deca LOEL reported for "thyroid changes, liver and kidney effects and fetal death" and "cancer" is wrong. In deriving the LOEL values, the Department of Health relied on 2 review articles by the same group of authors mentioned above. The Department of Health did not consult the original citations on which these reviews were based. Further, the Department of Health did not consult more recent publications on these endpoints; for example a recent prenatal oral developmental study found a NOEL of 1000 mg/kg/d administered to pregnant rats from day 0-19 of gestation (Hardy et al. International Journal of Toxicology 2002 21:83-91.) The LOEL of 80 mg/kg due to "thyroid changes, liver and kidney effects and fetal death" was based on a study performed in the 1970's (Norris et al. 1973, 1974, 1975) using an old formulation of the Deca product which contained only 77% Deca. The remainder of the old product was nona and octaBDE congeners. That product is no longer manufactured. Its toxicology does not reflect that of the current commercial Deca product (>=97% Deca). This is amply demonstrated by the 14 day, 13 week and 2 year studies in rats and mice performed by the U.S. National Toxicology Program (1986). The NTP used the commercial Deca product then in production, which was at least 96% in purity. No evidence of toxicity was seen in rats or mice feed up to 10% Deca in the diet for 14 days. No evidence of toxicity was seen in rats or mice feed up to 5% of the diet for 13 weeks. No mortality or changes in body weight and only minimal organ effects were seen in rats or mice feed up to 5% of the diet for 2 years! NTP attributed these minimal organ effects to the presence of low levels of the nona and octa congeners in the test article, and not to the Deca molecule itself. More recently, a prenatal oral developmental toxicity study on the current Deca commercial product (>=97% purity) produced a NOEL of 1000 mg/kg/d in rats (Hardy et al. 2002). **Taken as a whole, the data from repeated dose studies indicates a NOAEL for Deca of at least 1000 mg/kg/d (Hardy M 2002, Chemosphere 46, 757-777).** This is reflected in the oral reference dose (RfD) of 4 mg/kg/d determined by the U.S. National Academy of Sciences (Toxicological Risks of Selected Flame-Retardant Chemicals. 2000. National Academy Press. Washington, D.C. http://www.nap.edu.). The RfD is that dose to which a population, including the most sensitive, could be exposed for a lifetime with the expectation of no adverse effects.

Table 6 reports "cancer" as an endpoint for Deca, with a LOEL of 1120-3200 mg/kg, and cites Darnerud et al. 2001 as the reference. The Department of Health should have consulted the original U.S. National Toxicology Program report (NTP 1986) on the Deca cancer bioassay in rats and mice. If it had, "cancer" would not have appeared as endpoint in Table 6. The NTP reported "some, equivocal and no evidence of cancer" in male/female rats and mice after 2 years continual exposure of up to 5% Deca in the diet. The finding of "some evidence" was based on hepatic 'neoplastic nodules', which is not frank evidence of cancer. Deca is nonmutagenic in the Ames, Chromosome Aberration, Sister Chromatid Exchange or Mouse Lymphoma tests. Deca is not listed as a carcinogen by the U.S National Toxicology Program, the International Agency for Cancer Research, or the U.S. Occupational Health and Safety Administration. Thus, it is improper the Department of Health to designate 'cancer' as an

endpoint. See the Deca VCCEP submission for additional discussion of the NTP bioassays.

Build Up of PBDEs in the Body

Page 20-21. This section does not conform to accepted toxicological principals, and we recommend deleting it. For example, the paragraph comparing rodent studies to duration of human exposure doesn't consider that a rodent's lifespan (maximum ~2 years) is significantly less than humans so that studies in rodents of days or weeks duration cover a proportionally longer portion of the rodent's life span than it would in humans. It also fails to recognize that lifetime studies in two species have been performed on Deca (NTP 1986). The NTP studies exposed rats and mice to enormous levels of Deca (5% of the diet) every day for two years! The NTP 14 day, 13 week and 2 year studies were rigorous tests of Deca's potential to induce toxicity, and provided ample opportunity for Deca to 'build up in the body' and produce adverse effects as a consequence, if it were going to do so. Given the high NOAELs in these studies, at least 1000 mg/kg/d, there is no concern regarding Deca's potential to build up to toxic levels. See the Deca VCCEP submission for additional details.

The final sentence in this section cites a 4-page abstract by McDonald, "Distribution of PBDE levels among U.S. women: estimates of daily intake and risk of developmental effects", presented at BFR2004, Toronto Canada. This sentence states that based on McDonald's abstract "high-end exposures appear to be approaching toxic effects levels observed in animal studies, mainly for Penta associated congeners". The one correct portion of this sentence is that McDonald's abstract related to Penta-associated congeners. McDonald's abstract has serious flaws that need to be recognized before accepting his conclusions. McDonald takes 6 small studies (total number of individuals = 170), which measured differing PBDE congeners in differing tissues using different analytical methods, and attempts to estimate 'PBDE' levels in the entire U.S. population (some 300,000,000 individuals) while admitting the levels "exhibited wide variability among individuals". Then, he takes estimated 'PBDE' (which congeners comprise the sum?) levels and back calculates daily intake using assumed half-lives. Next, he compares this guess of highest human 'PBDE' levels that might be found in 5% of the population to estimated tissue concentrations in 10 rodent studies. Only 2 of the 10 rodent studies actually measured tissue (Which?) levels. How the rodent tissue concentrations were estimated was not described, thus we have no concept of whether the estimated concentrations are valid. Not considered (see Table 4 in the abstract) is that the more rigorous studies, e.g. those administering the test article over multiple days and performed by experienced research laboratories, have estimated C_{rodent}/C_{human} of 170, 330, and 33,000. In 2 of these 3 studies, actual measured data were available from rodents, and the ratio was 98 and 1316, rather than the estimated values of 170 and 330, respectively. (McDonald incorrectly reported the measured ratios were 90 and 1300). It is only the more questionable rodent studies that have lower C_{rodent}/C_{human} ratios. Also not considered is the large difference in NOELs reported for the same endpoint in Table 4. The more rigorous study, administration from GD6-PN21, reports a behavioral NOEL of 100 mg/kg whereas NOELs of < 1 mg/kg were reported in studies where only a single dose was administered on PND 3 or 10! Clearly, McDonald's conclusions cannot be taken seriously given the underlying problems with his methodology.

Products Containing PBDEs at End-of-Life

Page 22 contains a generic schematic depicting potential PBDE pathways into the environment via electronic goods. Please note that the Penta product is not used in electronics, and the congeners associated with the Penta product that constitute the majority of "PBDEs" detected in the environment. In fact, one congener, BDE-47, typically makes up 50-70% of the total PBDEs detected, and BDE-47 is associated with the Penta product. The Penta product will be phased out of production by the end of 2004. The Penta product is used in flexible polyurethane foam, not electronic goods.

The PBDE products used in electronics are Deca and Octa. Octa's volume is, and always has been, only a fraction of Deca's. Deca is the predominant PBDE used in electronics, and will be the only PBDE in production after the end of 2004. Thus, we recommend that the generic schematic be revised accordingly. Also, please note that Deca is not used in circuit boards or computer chips. (Penta and Octa are not used in circuit boards or computer chips, either.)

Deca's electronic applications are dominated by incorporation in hard dense plastics (e.g. television cabinet backs made of high impact polystyrene (HIPS)). It is unreasonable to assume that a substance with negligible water solubility and vapor pressure, such as Deca, will migrate out a matrix such as HIPS to any significant extent. Further, laboratory studies have shown Deca's migration to be minimal (see the Deca VCCEP submission for details). If released to the environment, models used by EPA predict Deca will distribute predominantly to sediment and soil with negligible distribution to air or water. Further, U.S. TRI data indicates Deca's environmental releases from the plastics industries during formulation operations are highly controlled, and that most waste is recycled. The plastics industries do not represent a major environmental source of Deca. These facts are not represented in the schematic on page 22.

The schematics on pages 23 and 24 are also too generic. The applications of the Deca, Octa and Penta products do not overlap, but overlapping applications are strongly implied by these schematics. Further, the error re use in computer chips is repeated.

The schematics on page 23 should be corrected to show that only the Penta product is used in flexible polyurethane foam. Only the Deca product is used as a backcoat on upholstery textiles. Only those components made of ABS may have utilized Octa as a flame retardant in the past - neither Deca nor Penta are used in ABS and Octa's use was virtually exclusively in that resin. Deca is used in the insulation of wire and cables – it is not used in wires themselves. Similar comments apply to the schematic on page 24.

Page 25. Electronics Recycling

None of the measured levels are reported, and instead this section focuses on detection. Without knowing the actual levels, it is impossible to determine if the measured PBDE levels outside a recycling facility (4-22 times higher than ambient) were meaningful or simply represent highly sensitive analytical methods. Given that the levels detected, 38.7 and 755 ng/m3 of 'total' PBDEs on outdoor and indoor window film at an electronics recycling facility, it is likely the later.

Also, the PBDEs congeners represented in the total are also important in determining hazard and risk. Using the reference cited in the Draft Document, Burns et al. 2004, as an example, the majority of the PBDE content on both indoor and outdoor window film was composed of BDE-209. BDE-209 contributes essentially nothing to human health hazard.

In referencing PBDE blood levels reported by Sjodin et al. (1999), the draft document again fails to report the actual levels, the individual congeners detected or the fact that the PBDE blood levels were <<< smaller than PCB levels concurrently detected in those workers.

Page 25. Landfills

While the fate of the PBDEs contained in consumer products and disposed of in landfills may not be exactly known, an educated guess can be made based on the properties of the individual PBDE products and their end uses. Deca is mainly used in hard dense plastics and has negligible water solubility and vapor pressure. There is no driving force compelling Deca to migrate out of the plastic, and migration will be negligible (see Deca VCCEP submission for details). Free Deca is expected to adsorb to particulate matter in water, soil, and air, and to not move in the water column. Thus, consumer products are not expected to be significant sources of Deca in landfills.

The Penta product, however, is different from Deca in both its applications and physical properties. It is used in an application, flexible polyurethane foam, which has a tremendous surface area. Thus, the potential loss from polyurethane foam of any additive will be higher than that from a hard dense plastic such as HIPS. The congeners in the Penta product also have water solubilties and vapor pressures, which while very low, are higher than that of Deca. For example, BDE-47's water solubility is 10.9 ug/L and its vapor pressure is 2.5×10^{-4} Pa, compared to Deca at <0.1 ug/l and 4.63 x 10-6 Pa. BDE-47 will also not bind to particulate matter as extensively as Deca. Polyurethane foam, when exposed to sunlight, becomes friable and crumbles, in contrast to HIPS, which is highly resistant to degradation. Thus, in a landfill, there may be potential for migration of the Penta product out of its end use. Whether this migration then serves as an environmental source depends on the management of the landfill. For example, the practice of using auto fluff for the daily cover at the Tacoma landfill may not be the best management practice if the fluff contains significant amounts of Penta and is subject to dispersal by the wind. On the other hand, landfills that are covered daily with soil would present less opportunity for environmental dispersal.

Finally, while there has been much speculation in the literature that landfills may be an environmental source of PBDEs, there is no evidence substantiating this. Further, chemically secure landfills, where the majority of free Deca (e.g. that not encapsulated in plastic) is disposed, are designed to contain wastes and prevent entry into the environment.

Formation of Polybrominated Dioxins and Furans

Page 25. Industry has performed a great deal of research on the potential formation of PBDD/Fs on incineration. That research shows that properly operating incinerators do not emit

PBDD/F even when the levels of waste electronic equipment are artificially elevated. Dioxin/furan generation in waste incinerators is a function of how the incinerator is operated; not the composition of the waste feed. Further, the chlorine content of municipal waste far outweighs that of bromine, such that chlorinated dioxins and furans dominate. Emission controls for chlorinated dioxins and furans will be effective for the brominated derivatives.

A synopsis of industry's research into incineration as a disposal option for waste plastics is as follows. The Association of Plastics Manufacturers in Europe (APME) has sponsored research utilizing working waste incinerators and not laboratory models. Their studies include determining the composition of MSW in Europe and the impact of added plastic waste on incineration. (Freisleben, W. "Plastics in Municipal Incineration." A technical paper by European Centre for Plastics in the Environment, Brussels. 1992; Mark, F. "Energy recovery through co-combustion of mixed plastics waste and municipal solid waste." A technical paper by the Association of Plastics Manufacturers in Europe (APME), Brussels. 1994; Mark, F. "The Role of Plastics in Municipal Solid Waste Combustion." A technical paper by European Centre for Plastics in the Environment, Brussels. 1993).

The impact of plastic waste on incineration was studied by adding plastic waste to typical MSW feedstock (Mark, F., Kayen, A., Lescuyer, J.L. "MSW Combustion. Effects of Mixed Plastics Waste Addition on Solid Residues and Chlorinated Organic Compounds." A technical paper from a series produced by Association of Plastics Manufacturers in Europe. 1994). Plastic waste in the feedstock ranged from the base level of 8-12% total polymer in the feedstock up to the highest level of 24-27%. The added levels of plastic waste improved the incinerator's combustion efficiency, which in turn produced beneficial effects on emissions. The plastic waste acted as an excellent fuel source, improved the burn of the incinerator, decreased total CO output, eliminated the need for added sulfur-containing fossil fuels and decreased the SO2 output. The added plastic waste also did not increased PCDD/PCDF emissions.

Work with polyurethane (PUR) or (expanded polystyrene) foam board was performed to investigate these materials on incineration (Vehlow, J. and Mark, F. "Co-combustion of Building Insulation Foams with Municipal Solid Waste." A technical paper from the Association of Plastics Manufacturers in Europe, European extruded Polystyrene Insulation Board Association, and European Isocyanate Producers Association. 1995). PUR and XPS foam are commonly flame retarded. XPS foam typically uses a brominated flame retardant, hexabromocyclododecane while PUR may contain phosphourus, chlorine, or bromine flame retardants. Adding PUR and XPS foam at several levels to the typical MSW feedstock increased the base load of bromine from 2 wt% to > 12 wt%. The added foam acted as an excellent fuel source and improved incinerator burn out. PCDD/PCDF raw gas levels were not increased. PBDD/PBDF were detected at very low levels near their detection limits in the raw gas (PBDD in pg/m3; PBDF at < 1 ng/m3). The PBDD/PBDF levels were far below the PCDD/PCDF levels and showed no clear correlation with Br input. Mono- and diBr mixed halogenated D/F were detected at very low concentrations in the raw gas. These mixed halogenated DD/DF were present at only 20% or 50% of the PCDD or PCDF levels, respectively, when Br was increased to the maximum of 6x the base load. Formation of the

mixed halogenated DD/DF appeared correlated with Br input at low Br levels, but not at high Br levels. A plateau or saturation in their formation was observed at > 5-10% Br in the fly ash. This saturation phenomena is known to occur with PCDD/PCDF; i.e. the Cl concentration on fly ash controls formation of PCDD/PCDF up to the saturation point but beyond this point Cl concentration has no influence on PCDD/PCDF formation. The saturation effect observed with Cl concentration and PCDD/PCDF formation appears to hold true for bromine and PBDD/PBDF or PHDD/PHDF formation.

APME's work with PUR or XPS foam showed that Br and mixed halogenated D/F do not add substantially to MSWI raw gas or emission levels. An apparent plateau in formation of Br and mixed halogenated D/F was observed. No increase in PCDD/PCDF levels was found when Br was increased in the feedstock. Total DD/DF emissions remained within the incinerator's typical range.

APME also sponsored work on the incineration of electrical and electronic waste plastic (Vehlow, J. and Mark, F. "E+E Polymer Waste Co-Combustion in the TAMARA Pilot Plant." Presented: Brominated Flame Retardants Workshop. November 14, 1995, Hyatt Regency Hotel, Crystal City, VA. Sponsor: CMA Brominated Flame Retardant Industry Panel). Waste plastic from these products may contain brominated, as well as other types, of flame retardants. Results from the incineration of electrical and electronic waste plastic compare very favorably with that on PUR and XPS foam. The waste plastic improved the overall efficiency of the incinerator. A moderate increase of Cl and a substantial increase in Br was observed, but the increased Cl and Br levels did not cause a significant increase in PCDD or PCDF content in the raw gas. Mixed bromo/chloro D/F were detected; mainly homologues carrying one Br atom. Total DD/DF emissions remained within the incinerator's typical range.

Regarding content of PBDD/F in either the flame retardant or products containing the flame retardant, the US EPA required testing the Deca, Octa and Penta products for 15 2,3,7,8-substituted PBDD/F. The DBDPO commercial product has been analyzed for trace quantities of 15 2,3,7,8-substituted polybrominated-p-dibenzodioxins (PBDD) and dibenzofurans (PBDF) under a U.S. Environmental Protection Agency (EPA) test rule. None of the analytes were present at or above the quantitation limits established by the agency (Ranken et al. *1*994. *Definitive study of the determination of polybrominated dibenzo-p-dioxins and polybrominated dibenzofurans in decabromodiphenyl oxide and tetrabromobisphenol A.* Bull. Soc. Chim. Belg. EUROPEAN SECTION. 1994 103/n 5-6:219-233). Resins containing DBDPO have also been analyzed for PBDD/PBDF content. A high impact polystyrene (HIPS) resin containing antimony trioxide and DBDPO was molded using normal (215-220°C; 30 sec.), abusive (235-245°C; 5 min.) or extreme (265-270°C, 7 min.) processing conditions (McAllister et al. *Analysis of polymers containing brominated diphenyl ethers as flame retardants after molding under various conditions.* Chemosphere 1990 20(10-12):1537-1541). The molded resin was cryogenically ground and analyzed for six 2,3,7,8-substituted PBDD/PBDFs. None were detected. Polybutyleneterephthalate (PBT) resin containing antimony trioxide and DBDPO was also molded under similar conditions, and analyzed. No 2,3,7,8-substituted PBDD/PBDFs were detected. Donnelly et al. (Biomedical and Environmental Mass Spectrometry 1987 18(10):884-96) also analyzed molded HIPS/DBDPO/Sb2O3 and molded

PBT/DBDPO/Sb2O3, and detected no 2,3,7,8-TBDF and no 1,2,37,8-PeBDF. Brenner and Knies (*Formation of polybrominated dibenzofurans (PBDE's) and –Dioxins (PBDD's) during extrusion production of a polybutyleneterephthlate (PBTP)/glassfibre resin blended with decabromodiphenyl ether (DBDPE)/Sb2O3: product and workplace analysis.* Organohalogen Compounds, 1990 2:319-324) also reported no PBDDs in their analysis of an extruded PBT/DBDPO blend. Virgin molded HIPS/DBDPO/Sb2O3 and repeatedly ground and injection molded (e.g. "recycled") HIPS/DBDPO/Sb2O3 resins meet the requirements of the German Chemicals Banning Ordinance with respect to 2,3,7,8-substituted PBDD/F content (Hamm S. 1999. *Analysis of a decabromodiphenyloxide blend, a HIPS plastics, the HIPS plastic containing the DecaBDPO and Sb2O3 and the repeatedly recycled HIPS/Sb2O3/DecaBDPO plastic for partially brominated diphenylethers and 8 polybrominated dibenzo(p)dioxin and dibenzofuran congeners.* Report 60425-001 B01. GfA. Munster, Germany; Hamm et al. *Determination of polybrominated diphenyl ethers and PBDD/Fs during the recycling of high impact polystryrene containing decabromodiphenyl ether and antimony oxide.* Chemosphere 2001 44(6):1353-1360). The concentrations of relevant PBDD/F congeners were at least one order of magnitude below the regulated limit values for PBDD/F (1 ppb for the sum of four congeners, 5 ppb for the sum of all eight regulated congeners).

Municipal Waste Combustors

Deca decomposes at temperatures above 300 degrees C. The minimum operating temperature of the sole waste combustor in Washington state (871 degree C) is nearly three times Deca's decomposition temperature.

Biosolids and Sewage Sludge

Deca's physical properties are such that it is expected to be extensively removed in sewage treatment plants via adsorption to sludge. Thus, its detection in that matrix is expected. The amounts reported to date in US sludge are highly variable and higher levels likely reflect local use by textile formulators/applicators. The textile backcoating operation uses water in its process. These operations may have outflows to publicly operated sewage treatment plants. No textile formulators/applicators report using Deca in Washington State under the US EPA TRI.

Episodic Fires

Fires are known to produce many substances including PCDD/Fs and polycyclic aromatic hydrocarbons (PAH). Typically, PAH content dominates the hazard of fire residue. In discussing hazardous substances produced by fires, the Departments of Health and Ecology should take into consideration that flame retardants actually prevent fires. Thus, their use actually decreases formation of hazardous substances associated with fires by reducing the incidence of fire. A life cycle study of the environmental impact of flame retarded versus non-flame retarded televisions demonstrates that flame retarded televisions have a lower environmental impact than their non-flame retarded counterparts do to the reduction in fires (Simonson et al. *LCA Study of TV Sets with V0 and HB Enclosure Material.* Organohalogen Compounds 2000 47:245-248).

Ash Reuse

This section speculates that incinerator ash reuse (in what way is it reused?) could be a pathway for release to the environment of PBDD/Fs. There are several problems with this. The manner in which the ash is reused will determine whether it is an environmental source for any substance. Is the ash spread out over agricultural soil? Is it incorporated somehow into a solid matrix that is then used for other purposes? Heavy metals distribute to incinerator ash, and this should be taken into consideration in the method of reuse. Finally, halogenated dioxins and furans are formed post-combustion while gaseous products are cooling, and associated with the gas phase of the process and not the bottom ash. *De novo* synthesis is the dominant mechanism of dioxin/furan formation in actual combustion systems, including waste incinerators (Huang, H. and Buekens, A. *On the Mechanisms of Dioxin Formation in Combustion Processes.* Chemosphere 1995 31(9):4099-4117).

PBDEs and the Environment

Page 27. The introductory paragraph is generic and oversimplifies a complex subject. The type of PBDE commonly detected in different matrixes varies. In biological systems, the predominant PBDEs are 47, 99, 100, 153, and 154. These are the congeners associated with the Penta product. Deca's most common site of detection is typically in sediments near point sources.

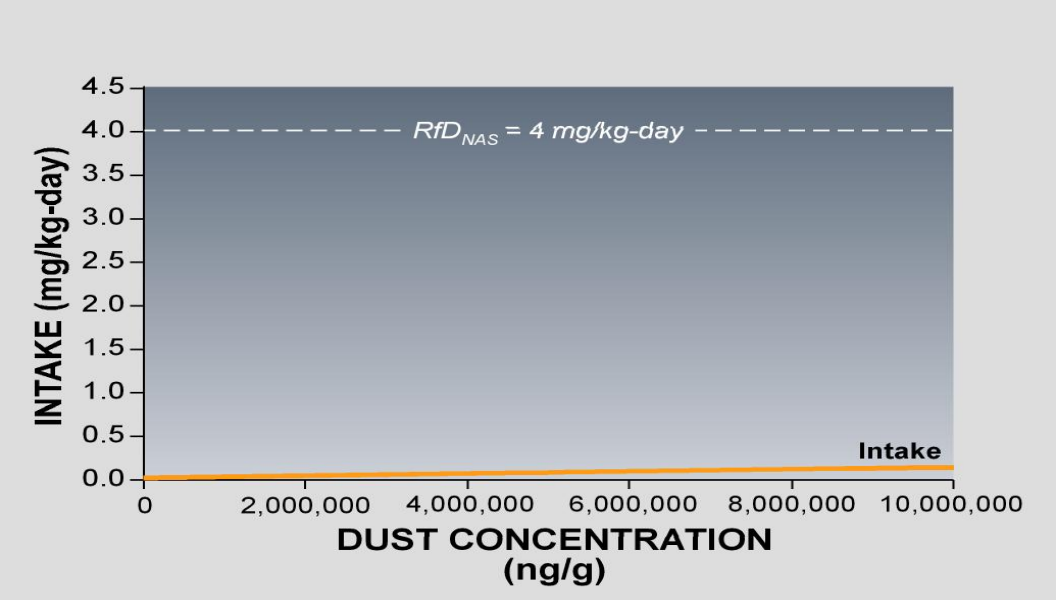
Air

Again, this section is a generic oversimplification. Environmental modeling predicts negligible distribution of Deca to air (see the Deca VCCEP submission). Deca is not typically detected in air (its vapor pressure is so low that it is not found in the gaseous phase), nor is it expected to undergo long range transport. Deca is expected to adsorb to particulate matter; this is borne out by its detection in house dust and on the surface dust of electronics housings. See Burt et al. (2004) and Wania and Dugani (*Assessing the long range transport potential of polybrominated diphenyl ethers: a comparison of four multimedia models.* 2002. Final Report. University of Toronto at Scarborough, Scarborough, Ontario). Hayes et al. (2003) estimated infants' and children's' exposure to Deca from house dust. House dust was an insignificant contributor to children's' exposures, and any contribution from house dust was far below the RfD of 4 mg/kg/d (see figure on the next page).

Sediment

Environmental modeling predicts, based on its physical/chemical properties that Deca will predominantly distribute in the environment to sediment and soil. Environmental monitoring indicates that sediments near point sources are the primary sites for Deca partitioning. Sediment organisms are not, however, adversely affected by Deca (Krueger et al. Decabromodiphenyl ether: a prolonged sediment toxicity test with *Lumbriculus variegates* using spiked sediment with 2% total organic carbon. Final Report. 2001. Project Number: 439A-113. Wildlife International, LTD., Easton, MD; Krueger et al. Decabromodiphenyl ether: a prolonged sediment toxicity test with *Lumbriculus variegates* using spiked sediment with 5% total organic carbon. Final Report. 2001. Project Number: 439A-114. Wildlife International, LTD., Easton, MD).

Estimated children's intake of Deca from house dust. From Hayes et al. 2003.



Biota

Pages 28-30 and Table 7. This section is largely generic and refers to simply 'PBDEs' with few descriptions of which congeners were detected in which species. Table 7 is entirely generic with no mention of which PBDEs were included in the analysis, detected, and used in deriving the 'total'. The predominant PBDEs detected in biota are those associated with the Penta product: BDE-47, 99, 100, 153, and 154. Deca is only rarely detected and generally makes up only a very small percentage of the total. These variations in the PBDE congener content of biological samples correspond to their relative potentials for absorption and elimination and use patterns as well as between species differences. We recommend revising this section to include the congeners detected and their amounts, with a focus on those studies particularly relevant to Washington State

Environmental Fate and Pathways

Long range transport

Wania and Dugani (*Assessing the long-range transport potential of polybrominated diphenyl ethers: a comparison of four multimedia models.* Environ Toxicol Chem. 2003 22(6):1252-61) used 4 different computer models to predict the potential of polybrominated diphenyl ethers to undergo long range transport. They found that the heavy congeners, e.g. those in the Deca and Octa products, had little potential to undergo long range transport. "A comparison of the LRTP estimates for the PBDEs with those of benchmark chemicals (polychlorinated biphenyls [PCBs]) suggest that the lower-brominated congeners have a LRTP comparable to that of PCBs

known to be subject to significant LRT, whereas the highly brominated congeners have a very low potential to reach remote areas." Wania and Dugani concluded that that their modeling results were in good agreement with remote indicating the presence of the lower brominated diphenyl ethers.

The paper cited in the first paragraph, Ter Schure et al. (Environ. Sci Technol 2004 38: 1282-1287) relates atmospheric transport, **not** long range transport. The distinction is key. Long range transport is defined as transport to remote regions in the Arctic via the atmosphere. The Ter Schure paper is not evidence of long range transport.

The paper cited as providing evidence of 'PBDE' detection in polar cod, ringed seal, polar bear and beluga whale, Wolkers et al. 2004, did not detect Deca. The paper cited as indicating that PBDEs are increasing in marine mammals (National Marine Fisheries Service 2004) actually included a single sentence to this effect and referred to Ikonomou et al. 2002. Ikonomou et al. did not find that the levels of Deca were increasing. In fact, Deca levels were either non-detectable or so low that they did not figure into the total PBDEs reported.

Environmental breakdown of PBDEs

Please see out detailed comments on Appendix A for a discussion of Deca's potential to breakdown in the environment. The weight of the evidence indicates that Deca is not a significant, if any, source of the lower brominated diphenyl ethers in the environment or that Deca breakdowns in the environment to PBDFs. The evidence cited in the Draft Document rests with laboratory studies performed in organic solvents. These studies are not accurate predictors of Deca's behavior in the environment.

IV. PBDES and the Regulatory Environment

Three additional programs were not included. First is the EPA Voluntary Children's Chemical Evaluation Program (VCCEP). The VCCEP program is specifically aimed at evaluating the hazard, exposure and risk presented to children by chemicals. The 3 PBDE commercial products were included separately in the program and were among the first to undergo peer consultation. Their VCCEP submissions should be consulted by Washington Department of Health for information on hazard, exposure and risk.

The second is EPA's High Production Volume (HPV) program. Again, all three PBDE commercial products are included. Deca's submission is expected by the end of this year; industry focused on the VCCEP submission due to primary concern with children.

The third is U.S. National Academy of Sciences review of Deca's use in upholstery textiles. NAS reviewed 16 flame retardants with potential for use in meeting a proposed CPSC regulation at the request of Congress. The NAS concluded Deca's use in upholstery textiles did not present a health hazard to the consumer and calculated an oral reference dose (RfD) for Deca of 4 mg/kg/d. The RfD is that dose to which a population, including its most sensitive members, could be exposed for a lifetime without expectation of adverse effects.

V. Alternatives and Market Changes

Alternatives to PBDEs

Table 8 provides no information with respect to application. The most useful comparison of alternatives is made on an application basis, and this is not addressed. The flame retardants listed may be appropriate for one resin in a specific application, but not one is suitable substitute for all uses of the Deca, Octa, or the Penta products, and several are incapable of being used in Deca, Octa or Penta's applications at all. Most of the FR listed in Table 8 are not currently used in Deca's, Octa's, and Penta's applications and are not viable alternatives. For example, tetrabromobisphenol A (TBBPA) is listed. TBBPA has largely replaced Octa in ABS, but finds no use in either HIPS (e.g. a Deca application) or flexible polyurethane foam (a Penta application). TBBPA's properties simply do not lend itself to use in these applications.

Table 8 also contains no information as to whether the alternative FR could achieve the same level of fire safety as the Deca, Octa or Penta products. One example is hexabromocyclododecane (HBCD). HBCD is incapable of attaining V-0 fire safety rating in HIPS, and therefore is not used in TV cabinet backs. HBCD can achieve the lower fire safety rating of V-2, and is suitable for use in HIPS stereo cabinets, which need a lesser level of fire protection. Also, the NAS recommendations pertained to flame retardants used in upholstery textiles, not polyurethane foam or electronics.

Appendix A: Degradation of PBDEs

Page 73. The first paragraph states that 7 individuals reviewed the section on photolytic degradation and the conclusions on photolytic degradation in the body of the report. Not one of these individuals is considered an expert in the field of photolysis. The only individual mentioned with specialized knowledge in this field was C. Jafvert of Purdue. Dr. Jafvert was not one of the reviewers. Thus, while this section gives the appearance of having been reviewed by experts in the field, this is not the case.

Photolytic degradation, Pages 73-77

This section is virtually the only portion of the Draft Document that addresses individual PBDE congeners. It is also one of the few portions of the Draft Document that provides more than sketchy summaries of the studies reviewed. In that respect, this section is one of the better ones in the Draft Document. However, this section does not consider all available data, including environmental monitoring results and what they tell us about the sources of the 'PBDEs' detected, nor does it recognize that the majority of the studies cited as demonstrating degradation were not performed under conditions representative of the environment. Most of the studies cited were performed in organic solvents, and as Norris et al. discussed, photolysis of halogenated organics proceeds via different routes in organic or aqueous environments. Reductive debromination is observed in organic solvents capable of donating a proton. In aqueous systems, addition of a hydroxyl group (-OH), rather than simple addition of a hydrogen atom (-H), occurs. Thus, the degradation products produced are different in the two systems, and the degradation products detected in laboratory studies performed in organic solvents are not directly transferable to breakdown in the environment.

The thrust of this section is the implication that Deca degrades in the environment via light or microbes and is a substantial source BDE-47 and BDE-99. However, the weight of the evidence from laboratory and monitoring studies indicate that environmental levels of BDE-47 and BDE-99 are attributable to the Penta product. That evidence is described in terms of Deca's environmental partitioning, routes of photodegradation, identity of degradants, and congeners detected in the environment compared to those in the commercial Deca, Octa and Penta products.

Environmental Partitioning's Impact on Photolysis as Route of Environmental Degradation. Deca's propensity to partition in air or water is important in determining if photolysis could be a significant route of environmental degradation. Air and water are the two environmental matrixes where photolysis can be expected to occur to any significant extent. Modeling programs developed by EPA predict that Deca will partition primarily to sediment (57%) and soil (42%), with negligible amounts to air (0.12%) or water (1.09%) (See Deca VCCEP Submission, page 43). That amount partitioning to air is predicted to be associated with particulate matter, rather than be present in air in the gaseous phase. Association with particulate matter is expected to decease and slow photolysis. This point was recognized in the Draft Document.

Air. Actual measurements indicate that Deca is not detected in the gaseous phase in indoor or outdoor air (Wilford et al. *Passive Sampling Survey of Polybrominated Diphenyl Ether Flame Retardants in Indoor and Outdoor Air in Ottawa, Canada: Implications for Sources and Exposures, Environ.* Sci. Technol. 2004, 38:5312-5318), and where detected in homes or offices, it is associated with dust particles. There will be only negligible amounts of Deca in air and the majority will be associated with particulate matter and shielded to a certain extent from photolysis. In air, Deca is expected to be associated with particulate matter, rather than in the gaseous phase, because of its low vapor pressure (4.63×10^{-6} Pa) and high adsorption coefficient (1.8×10^{6}). Thus, photolysis in air will not be an environmentally significant route of degradation for Deca due to the limited amount of the substance in that media.

Deca deposited on dust (silica particles), suspended in dry air, and irradiated with artificial sunlight was found to be photo inert; no measurable degradation to PBDEs occurred (C. Zetzsch, University of Bayreuth, Germany. 2003. Observations on 'UV spectra, photolysis and photochemistry of polybrominated diphenyl ethers in organic solvent, absorbed on particles in air and in aqueous suspension. www.bsef.com).

Just how little Deca could potentially contribute to BDE-47 or BDE-99 through photolyis in air can be estimated, and compared to that of the Penta product. Deca's 2001 global production was 56,000,000 kg compared to Penta's 7,500,000 kg. As a worst case, assume all 56,000,000 kg's of Deca were released to the environment, 0.12% of that amount (16,800 kg) was distributed to air, and 100% of that distributed to air was photylized to BDE-99 only. (BDE-99 is only one of the 5 major PBDEs detected in the environment and is the major component of the Penta product). Also assume that all 7,500,000 kg's of Penta's production were released to

the environment, and no degradation of any of its components occurred. Under these condition, the Penta product would contribute 3,6000,000 kg of BDE-99 to the environment whereas Deca's photolysis would contribute a maximum of 16,800 kg. Deca's maximum contribution of BDE-99 would be 0.46% of the amount derived from the Penta product. Deca's actual contribution would be even lower, because a) the majority of Deca's global production would be encapsulated in products and not available to undergo photolysis, and b) the very conservative assumption that all Deca in air would be converted solely to BDE-99 and nothing else. Cleary, even if Deca undergoes photolysis in air, its contribution to the lower brominated DPEs detected in the environment is negligible.

Water. Deca's water solubility is <0.1 ug/L. In water, it is not expected to be in solution, but to be bound to particulates, and only limited partitioning to water is predicted (1.09%). Light penetration in natural waters is typically only a few centimeters, such that not all of the substance would be exposed to radiation intense enough to induce photolysis. Using the same assumptions as for air: 100% release to the environment and 100% photolysis to BDE-99 but 1% distribution to water, Deca could potentially contribute a maximum of 15% of the BDE-99 derived from the Penta product. This is a highly conservative estimate, and assumes 100% of Deca in water was photolyzed, and that the sole degradation product was BDE-99. Deca's actual contribution would be substantially lower. The majority of Deca's global production would be incorporated in products and not released to the environment where it would be available to undergo photolysis. Like in air, Deca is expected to be associated with particulate matter in water, which will slow photolysis and rapidly remove Deca from the water column via settling to sediment (predicted by the 57% partitioning to sediment). Further, laboratory studies have found either no evidence for Deca's photolysis in water (Eriksson et al. 2004) or only an extremely small amount of degradation, about 0.57% after 98 days (Norris et al. *Toxicology of octabromobiphenyl and decabromodiphenyl oxide.* Environ Health Perspect 1975 11:153-161).

Photolyis: Different Routes and Products in Organic Solvents or Water. Many studies have reported the photolysis of Deca in organic solvents (Wantanabe, Eriksson et al., Benzares-Cruz, etc). Norris et al. (1975) first reported that Deca (7 ppm) in octanol decomposed with a half-life of 4 h. In xylene, a strong absorber of UV light, Deca photodegraded by reductive debromination with a half-life of 15 h on exposure to a 125 watt Hg lamp. However, none of these studies, until the Bezares-Cruz et al. publication of 2004 identified BDE-47 and 99 as degradation products of Deca, and even Benzares-Cruz et al. (2004) found that BDE-47 and 99 were just 2 of 43 different PBDEs detected after photolysis in hexane. Thus, not even in the matrix where there is persuasive evidence for Deca's photolysis, has significant production of BDE-47 and 99 been reported.

Yet, photolysis studies performed in organic solvents are unlikely to be applicable to Deca's environmental fate. Early in its development as a commercial product, it was recognized, based on other halogenated aromatics, that photolysis of Deca would likely proceed by different routes in water and organic solvents (Norris et al. 1975). In solvents capable of proton transfer, halogenated aromatics typically degrade by reductive dehalogenation; however, in water, oxidation led to the formation of phenolic compounds. Further, once

photohydroxylation was initiated in water, its rate was expected to accelerate as electron-withdrawing halogens were replaced by electron releasing hydroxyl groups. The resulting hydroxylated species were expected to adsorb light more strongly and this ultimately could result in rupture of the aromatic ring. Norris et al.'s laboratory results on Deca correlated with these predictions. Minimal evidence of Deca's (98% purity) aqueous photodegradation was found over a 3-month exposure to natural sunlight; degradants were not lower brominated diphenyl oxides. Evidence for degradation of only 0.57% of the amount initially present (10 g/8 l water) was detected after 98 days of exposure to sunlight. Erikkson et al. (2004) were unable to detect any degradation products of Deca in water, and suggested the disappearance of the compound from the solution may have been due to adsorption to the glass walls of the vessel.

Photolysis on Silica, Soil, Sediment, or Sand. In the environment, Deca is predicted to partition primarily to sediment (52%) and soil (42%). Thus, sediment and soil are the media where the largest portion of Deca in the environment could potentially undergo degradation

Deca deposited on dust (silica particles), suspended in dry air, and irradiated with artificial sunlight was found to be photoinert; no measurable degradation to PBDEs occurred (Zetzsch 2003).

Soderstrom et al. (2004) reported that irradiation of Deca deposited on moist sand, silica gel, sediment or soil resulted in photodegradation with the slow formation of unidentified products as well as PBDEs of differing composition from those commonly found in the environment. However, BDE 47, 99 and 100 were not detected after irradiation of soil, sand or sediment, and these Swedish researchers concluded that Deca was not the source of the tetra and pentaBDEs typically detected in the environment. In their concluding paragraph, they said "In this investigation the most commonly found PBDEs in environmental samples (BDE 47, BDE 99 and BDE 100) were only formed to a minor degree from the photolysis of DecaBDE and only in toluene and/or on silica gel. BDE 153 was formed in toluene, on sand outdoors and on sediment. The origin of these congeners in the environment is probably primarily from emission of technical PentaBDE products and possibly from other degradation pathways of DecaBDE. To further investigate the degradation pathways of decaBDE, combined photolytic/bacterial degradation pathways should be examined."

Source of PBDEs Detected in the Environment. Environmental monitoring indicates that the BDE-47 and BDE-99 detected originate from the Penta product, and not degradation of Deca. Zegers et al. (*Levels of Polybrominated Diphenyl Ether Flame Retardants in Sediment Cores from Western Europe.* Environ. Sci. Technol. 2003 37:3803-3807) reported that the PBDE congener pattern detected in Western Europe sediment cores showed a "high resemblance to their pattern in the industrial penta-BDE mixtures" and found no evidence of a contribution from degradation of Deca, which was also detected in these sediments. Rayne and Ikonomou (*Reconstructing source polybrominated diphenyl ether congener patterns from semipermeable membrane devices in the Fraser River, British Columbia, Canada: comparison to commercial mixtures.* Environ Toxicol Chem. 2002 21(11):2292-300) used pattern analysis in a source reconstruction of PBDEs detected in the Fraser River in British Columbia and

concluded that the lower brominated diphenyl ethers detected originated from the Penta and Octa products, not degradation of Deca. They further determined that the most likely source was inefficient rural septic tanks with direct outflows to the river. Song et. al. (*Polybrominated diphenyl ethers in the sediments of the Great Lakes. 1. Lake Superior*, Environ. Sci. Technol. 2004 *38:* 3286-3293) reported that the PBDEs detected in Lake Superior sediment resembled the commercial Penta product and concluded that the lower brominated PBDEs detected originated from that commercial product. North (*Tracking polybrominated diphenyl ether releases in a wastewater treatment plant effluent, Palo Alto, California.* Environ Sci. Techol. 2004 38:4484-4488) collected and analyzed effluent and sludge at a wastewater treatment plant in Northern California and reported the following: "The total concentration of PBDEs ranged from 61 to 1440 microg/kg dry wt in the sludge and from 4 to 29,000 pg/L in discharged effluent. The congeners with the highest abundance in sludge were BDE-47, BDE-99, and BDE-209, while in treated effluent BDE-47 and BDE-99 were the most abundant. BDE-47 and BDE-99 are major congeners of the penta-formulation, while BDE-209 composes the deca-formulation. The sum of the major congeners in the penta-formulation (BDE-47, 99, 100, 153, and 154) comprises 88% of the total PBDEs in the effluent, while BDE-209 is only 6%. Based on the loading analysis, the total PBDE concentrations loaded to the San Francisco Estuary through effluent discharge from this wastewater treatment plant is 2 lb/year (0.9 kg/year)." These result indicated the Penta-mixture was the source of lower brominated diphenylethers detected, not Deca microbial degradation. Ter Schure et al. (*Atmospheric Transport of Polybrominated Diphenyl Ethers and Polychlorinated Biphenyls to the Baltic Sea.* Environ. Sci. Technol. 2004 38(5)1282-1287) concluded that in environmental samples, "BDE47 and BE99 are markers for the commercial penta-BDE mixture" and that BDE47, BDE100 and BDE99 "originate from the commercial penta-BDE formulations" .

In conclusion, the weight of the evidence indicates that degradation of Deca is not responsible for the BDE-47 and 99 levels detected in the environment. The evidence indicates that these congeners were derived from the commercial Penta product.

Biological transformation of PBDEs, pages 77-78

This section reports 2 studies where Deca-treated food was fed to fish (Kierkegaard et al. 1999; Stapleton et al. 2004). No other information on biological transformation was provided.

Biota. In the Draft Document, Kierkegaard et al. (1999) and Stapleton et al. (2004) are briefly described and said to "indicate the potential for PBDEs to break down as a result of biological processes." However, the section doesn't include a key fact crucial to interpreting the results – the amount of the test article absorbed. Both studies fed Deca-treated food to fish. Kierkegaard et al. reported that, over a 120-day period, trout absorbed approximately 0.005% of the 7.5 or 10 mg Deca/kg/d administered in their food. Stapleton et al. was unable to detect any Deca in juvenile carp fed food containing Deca at a dose 940 ng/fish/d for 90 days. Nevertheless, Deca's bioavailability in carp was estimated to be 0.4% of the dose based on detection of presumed metabolites. At an uptake of 0.005 or 0.4%, the products of biological transformation of Deca (if any) are immaterial.

This very low uptake from food is consistent with results obtained from a standard fish bioconcentration test in water. Deca's fish bioconcentration factor (BCF) was 50 (Biodegradation and Bioaccumulation Data of Existing Chemicals Based on the CSCL Japan. Compiled under the supervision of Chemical Products Safety Division, Basic Industries Bureau, Ministry of International Trade & Industry, Japan. Edited by Chemicals Inspection & Testing Institute, Japan. 1992. Published by Japan Chemical Industry Ecology-Toxicology & Information Center).

The very low uptake from food by fish is also consistent with the results of Norris et al. (1974, 1975), NTP (1986) and El Dareer et al. (1987) showing very low bioavailability of Deca.

A very low uptake in fish is consistent with environmental monitoring. Detection of Deca is uncommon, and where found typically represents only a small fraction of the total PBDEs.

Microbial. Deca's potential for transformation by sediment microbes has been studied. Deca is expected to partition in the environment mainly to sediment, and sediment is therefore an important matrix for study. No evidence of Deca degradation was detected over a 32 week anaerobic sediment study (Schaefer E and Flaggs R. 2001. Potential for biotransformation of radiolabelled decabromodiphenyl oxide (DBDPO) in anaerobic sediment. Final Report. Project No: 439E-104. Wildlife International, Ltd. Easton, MD). de Wit (Brominated flame retardants. Report 5065. 2000. Swedish Environmental Protection Agency. Stockholm, Sweden.) reported that no degradation of Deca in sediment was observed during a 2-year study. Thus, Deca appears resistant to microbial degradation in its preferred site of distribution in the environment. See the Deca VCCEP submission for details.

VOLUNTARY CHILDRENS CHEMICAL EVALUATION PROGRAM (VCCEP)

DATA SUMMARY

DECABROMODIPHENYL ETHER

(A.K.A. DECABROMODIPHENYL OXIDE, DBDPO)

CAS # 1163-19-5

Prepared by

American Chemistry Council's
Brominated Flame Retardant Industry Panel (BFRIP)
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DBDPO SUMMARY FOR TECHNICAL AUDIENCES

The American Chemistry Council's Brominated Flame Retardant Industry Panel (BFRIP) volunteered under the U.S. EPA's Voluntary Children's Chemical Evaluation Program (VCCEP) pilot to prepare the Data Summary for decabromodiphenyl oxide (DBDPO). This compound (CAS No. 1163-19-5) is also known as decabromodiphenyl ether. DBDPO is a data-rich chemical having valid guideline studies or other information for all VCCEP Tiers I, II and III and Screening Information Data Set (SIDS) endpoints.

The Environmental Protection Agency (EPA) included DBDPO in the VCCEP pilot on the basis of its detection in human milk as reported by Noren et al. 1998 (see Table 1, page 81704, Federal Register, Vol. 65, No. 248, December 26, 2000). However, Noren et al. did not report DBDPO in human breast milk, and DBDPO has not been reported in human breast milk in publications appearing prior to or since 1998. Noren et al.'s, and other authors, use of the terminology "PBDEs" rather than specifying the isomers or congeners detected is likely the cause for this error.

The DBDPO product is one of three commercial polybrominated diphenyl oxide (a.k.a. ether) products manufactured, and accounts for approximately 83% of all polybrominated diphenyl oxide/ether (e.g. "PBDE") production worldwide. DBDPO is used solely as a flame retardant to prevent or delay ignition of combustible materials. DBDPO's flame retardant activity is derived from the bromine atoms on the diphenyl oxide molecule. Bromine is one of the few elements able to provide flame retardancy in the gas phase; a property needed by some plastic resins as a result of the way the plastic burns. DBDPO's high bromine content makes it a very effective flame retardant which in turn makes DBDPO extremely cost-effective. This combination has resulted in DBDPO's becoming the second largest volume brominated flame retardant in production and use. DBDPO's main application is in high impact polystyrene (HIPS) used for electronic enclosures, e.g. television set cabinet backs. A comparatively minor, but important, use of DBDPO is to flame retard upholstery fabric where it is applied as a fabric back coat encapsulated in latex. Two companies manufacture DBDPO in the U.S.

Flame retardants such as DBDPO are a component of efforts to control and reduce the risk of fires. Despite the best efforts of fire departments, building codes, sprinklers and fire alarms, fires in the United States are a serious problem and the United States has one of the highest fire incidence and mortality rates of developed nations. The National Fire Protection Association reports that in the year 2000:

- A fire department responded to a fire somewhere in the United States every 18 seconds.
- Public fire departments attended 1,708,000 fires, of which 505,500 occurred in structures, 348,500 occurred in vehicles, and 854,000 occurred in outside properties.
- Nationwide, there was a civilian (non-firefighter) fire death every 130 minutes. There were 4,045 fire deaths, a significant increase of 13.3% from the previous year.
- The majority of fire deaths (85%) occur in home fires. There were 3,420 deaths from fires in the home, an increase of 18.1% from the previous year.
- The civilian fire death rate in the United States was 14.8 deaths per million people.

- Nationwide, there was a civilian fire injury every 23 minutes. There were an estimated 22,350 civilian fire injuries, of which 16,975 occurred in homes.
- Smoking materials were the leading cause of civilian deaths, accounting for roughly one-fourth of the total.

Populations at high-risk of death, injury or burns in fires are the very young, the elderly, and the poor. Based on annual averages for the five-year period from 1994 through 1998, children five and under made up about 9% of the country's population, but accounted for 17% of the home fire deaths. A child's risk of dying in a fire is twice the national average. Adults 65 and older also face a risk twice the average, while people 85 and older had a risk that is almost four-and-a-half times more than average.

The life safety benefits derived from brominated flame retardants (BFRs) in the U.S. were determined (Clarke 1997). Four product classes were identified in which BFRs are widely used and which could be directly associated with fire data: television/appliances, wire/cable insulation, curtains/draperies and upholstered furniture. Using fire data from the National Fire Protection Association (NFPA), BFRs' use in television cabinets (primarily DBDPO) were estimated to save 190 lives annually. For electrical insulation and draperies, less product and fire data were available, but 80 and 10 lives, respectively, were estimated saved annually through the use of BFRs in these products. Again, DBDPO is a major flame retardant used in electrical insulation and in draperies. In total, BFRs are estimated to avoid 280 deaths in the U.S. annually, at a minimum. A large portion of these lives saved are likely attributable to DBDPO. Clarke also found that another 140-220 fire deaths per year could be avoided if upholstered furniture fabrics were backcoated with fire retardant latex as is now done to meet California standards for upholstered furniture.

DBDPO, a solid at room temperature, is a fully brominated (e.g. 10 bromine atoms) diphenyl oxide with a molecular weight of 959.17. The composition of the commercial product is typically ≥ 97% DBDPO with the remainder composed of nonabromodiphenyl oxide. DBDPO's measured water solubility (<0.1 ug/L) and vapor pressure (4.63×10^{-6} Pa) are negligible. DBDPO's solubility in organic solvents is also extremely low: acetone 0.05%, benzene 0.48%, methylene bromide 0.42%, xylene 0.87%, and 0.2% in toluene. DBDPO is often assumed to be lipophilic due its presumed similarity to PCBs. However, no formal fat solubility study has been performed, and pharmacokinetic studies show no appreciable affinity of DBDPO for adipose tissue. DBDPO's blood:liver:adipose ratio in the rat was 1:7:2 compared to Arochlor 1254's ratio of 1:22:359. DBDPO's measured octanol/water partition coefficient (Log K_{ow}) was 6.265, but its Log K_{ow} estimated by EPIwin, v3.4, was 12.61. It is apparent that DBDPO has a very low solubility coefficient in water and most organic solvents. Further, DBDPO's estimated K_{ow} appears to be a better predictor of it behavior in biological systems than its measured value, based on mammalian pharmacokinetic studies and fish bioconcentration data. This is likely due to its very poor solubility in both water and octanol so that any small change in concentration produces a large change in the measured K_{ow} value.

DBDPO has been extensively tested in acute through two year studies. DBDPO was not acutely toxic, was not irritating to the skin or eye, and did not induce skin sensitization in a human patch

test. Repeated dermal application to rabbits' ears did not induce a chloracne-like response. The soot and char combustion products from a high impact polystyrene/DBDPO/antimony trioxide matrix also were not acutely toxic and did not induce a chloracne-like response. Gavage administration of DBDPO (0.1 nmol/kg/day) to rats over 14 days did not induce hepatic cytochrome P450, cytochrome P450 reductase, UDP-glucuronyl-transferase, benzo[a]pyrene hydroxylase, p-nitroanisole demethylase, or EPN detoxification. Taken as a whole, the no-observable-adverse-effect-level (NOAEL) for DBDPO in repeated dose studies is at least 1,000 mg/kg body weight. DBDPO's low toxicity is likely related to its poor absorption and rapid elimination. Pharmacokinetic studies have shown that DBDPO is poorly absorbed (0.3 -2% of an oral dose), has a short half-life (24 hr) compared to PCB 153 (<2% of an oral dose was eliminated by rats in 21 days), can be metabolized, and is rapidly eliminated in the feces (>99% in 72 hr). No adverse effects in either parent or F1 animals were noted in a dietary one-generation reproduction test utilizing doses up to and including 100 mg of a 77% DBDPO mixture/kg body weight. No evidence of maternal or fetal toxicity or developmental effects was detected in a developmental test in the rat (n=25 pregnant females/dose) at 1,000 mg/kg body weight utilizing a composite of today's commercial DBDPO product produced by three manufacturers and administered from days 0 - 19 of gestation. The test article composition was 97.34% DBDPO, 2.66% nona- and octabromodiphenyl oxide. No evidence of a genotoxic effect was detected in the Ames Salmonella, chromosome aberration, mouse lymphoma, or sister chromatid exchange tests. No cytogenic changes were observed in the bone marrow of rats (parents and offspring) undergoing a one-generation reproduction test using a former DBDPO-commercial mixture of 77% purity (Dow FR-BA-300). No evidence of carcinogenicity was observed in female mice receiving 2.5 or 5% DBDPO in the diet (~3,760 or 7,780 mg/kg/d). Equivocal evidence of carcinogenicity was observed in male mice by an increase in the combined incidence of hepatocellular adenomas or carcinomas in both dose groups (~3,200 or 6,650 mg/kg/d); however, this finding may have been influenced by the larger number of early deaths in control male mice compared to the treated male mice. The large number of early deaths in the control males may have decreased expression of hepatocellular adenomas or carcinomas in this group. The combined incidence of hepatocellular adenomas and carcinomas in male mice treated with DBDPO was well within the historical range. Some evidence of carcinogenicity in male and female rats was observed by increased incidences of neoplastic nodules of the liver in low dose (2.5%, ~1,120 mg/kg/d) males and high (5%, ~2,240 mg/kg/d -males, ~2,550 mg/kg/d - females) dose groups of each sex. (The term "neoplastic nodule" is no longer used by NTP to describe hepatoproliferative lesions in rats. This change in nomenclature was made subsequent to a peer review of representative hepatoproliferative lesions from two-year carcinogenicity studies. The peer review found the use of this poorly defined and understood term had permitted some potentially useful drugs and chemicals to be unfairly categorized as carcinogens.) DBDPO is not listed as a carcinogen by NTP, the International Agency for Research on Cancer (IARC) or the U.S. Occupational Safety and Health Administration (OSHA).

Exposure scenarios considered are occupational, the general population through use of consumer goods, and the general population via food or breast milk. Reasonable occupational exposure routes/scenarios are a) inhalation of dust and/or dermal contact at manufacture and b) at formulation prior to encapsulation in polymer or inclusion in the textile dispersion. The most likely point at which exposure could occur during manufacture is when the flame retardant is

transferred into bags for shipping. Likewise, the point at which worker exposure is most likely during formulation into the polymer dispersion is when the bags of DBDPO are emptied into a hopper prior to mixing the dispersion. Once formulated into the polymer dispersion, DBDPO is encased in the polymer matrix and the potential for worker exposure is negligible.

Theoretically, workplace exposure could occur via the dermal or inhalation routes. DBDPO's physical and chemical properties make the probability of systemic absorption following dermal or inhalation exposure very low. DBDPO is a large molecule of high molecular weight (959.17) with negligible water solubility (<0.1 ug/L), and is likely to diffuse through biological membranes only with great difficulty. This assumption is borne out with pharmacokinetic studies that demonstrate DBDPO's poor oral bioavailability (0.3-2% of an oral dose). DBDPO's negligible water solubility and high molecular weight effectively preclude significant skin absorption, and DBDPO's skin absorption is estimated at <<0.03% of a dermally applied dose. DBDPO's vapor pressure (4.63×10^{-6} Pa) is such that volatilization is not expected to be a source of inhalation exposure. Occupational exposures to dusts may occur; however, DBDPO is a large poorly absorbed molecule that exhibits little toxicity, and for which the American Industrial Hygiene Association assigned a Workplace Environmental Exposure Level of 5 mg/kg/d. The combined effects of poor absorption and minimal toxicity (NOAEL ≥ 1,000 mg/kg/d) indicate adverse effects should not occur as a result of occupational exposure. Nonetheless, workplace controls should focus on points where fine-particle-size-DBDPO may become airborne to limit inhalation exposure. This would be during bagging at manufacture and at formulation prior to inclusion in the resin or polymer dispersion.

Theoretically, the flame retardant textile backcoat could crumble during fabrication of upholstered furniture. Any particles generated would likely be too large to be respirable. In addition, for systemic absorption to occur, not only would the particles need to be inhaled or ingested, but also DBDPO would have to diffuse out of the polymer prior to its absorption. Systemic absorption of significant amounts as a result of crumbling of the backcoat is highly unlikely.

An additional occupational exposure scenario explored in the published literature is electronics recycling and computer repair. A graduate student's research reports the detection of DBDPO, and other polybrominated diphenyl oxide (a.k.a. ether) isomers, in Swedish workers engaged in dismantling electronic equipment and in Swedish computer technicians. The mean DBDPO blood levels, characterized by the original author as "high", were 5 pmol/g lipid in the Swedish electronics recycling workers, and 1.6 pmol/g lipid in the Swedish computer technicians. DBDPO air levels in the recycling workplace were 0.0002 mg/m^3. The DBDPO blood levels were substantially below those of PCB 153 (dismantlers, 760 pmol/g lipid; technicians, 290 pmol/g lipid) measured in the same workers. The electronics dismantling workers' internal DBDPO dose (1.2 ng/kg body weight) based on their measured blood level was comparable to the level expected (0.57 ng/kg body weight) calculated from the measured air levels. A similar comparison was not possible for the computer technicians because air values were not reported for that workplace. The DBDPO air level (0.0002 mg/m^3) measured in the electronics recycling plant was approximately 25,000 times below the AIHA WEEL of 5 mg/m^3. No impact on

human health from DBDPO is expected in either the electronics dismantlers or computer technicians based on available data.

DBDPO is not sold directly to the public, but may be present in various consumer goods. A typical U.S. example is in the cabinet backs of television sets where DBDPO is used at a level of approximately 12% (weight). Upholstered furniture in commercial settings in the U.S. is required to met federal flammability standards and may utilize upholstery textiles that are flame retarded with a backcoating containing DBDPO at ~5 mg/m^2. Residential furnishings, except in the state of California, are not required to met a comparable standard, although the Consumer Product Safety Committee (CPSC) is considering implementing such a standard. CPSC is also considering a standard for mattresses.

Potential consumer exposure could theoretically occur via the dermal or inhalation routes (e.g. from dermal contact with the television cabinet back or upholstery textile or via inhalation of a vapor given off by the appliance). DBDPO's physical/chemical properties make these unlikely exposure scenarios. In infants or small children, another route could be oral through chewing or sucking on the upholstery textile. In addition, exposure to the general population could occur if DBDPO were present in food or in breast milk.

DBDPO's potential risk to the consumer, including children, in the upholstery application was recently reviewed by the National Academy of Sciences (NAS). The NAS evaluated the potential risk to the consumer posed by DBDPO-treated upholstery textiles. In all scenarios evaluated, dermal, oral or inhalation exposure for carcinogenic or non-carcinogenic risks, DBDPO was determined not to present a risk of adverse health effects to the consumer, including children.

A similar conclusion is reached for DBDPO's use in electrical and electronic applications. DBDPO is a large poorly absorbed molecule that exhibits little toxicity. These features coupled with DBDPO's low potential for migration out of plastic resin indicate this use also would not be expected to present a risk of adverse effects to the consumer. Further, the protection provided by DBDPO in terms of enhanced fire safety reduces the very real risk of death or injury that consumers face in the home from fires.

Laboratory studies have shown DBDPO is not bioconcentrated in fish, probably due to its poor solubility and large molecular weight. DBDPO has not been detected in limited sampling of fish and poultry in the U.S., and based on its properties, is not anticipated to be present in these food items or in meat or dairy products. Likewise, leafy vegetables and root crops are not expected to be a source of DBDPO exposure to the general public, and a risk of adverse health effects is not anticipated.

DBDPO transfer to breast milk is likely to be slow and limited, if at all. The combination of low absorption from the gut, rapid elimination in the feces, poor and/or slow diffusion into breast milk should effectively preclude DBDPO in milk. Build-up of concentrations in breast milk is not expected due to its slow diffusion into milk and periodic emptying of breast milk. A risk to the nursing infant is not anticipated based on current information.

Data is available on DBDPO for essentially all VCCEP Tier I, II and III endpoints. The NAS concluded no additional information was needed to evaluate DBDPO's risk to the consumer through the use of flame-retarded upholstery textiles. BFRIP concurs with that assessment, and extend it to DBDPO's use in electrical and electronic equipment as well.

DBDPO SUMMARY FOR NON-TECHNICAL AUDIENCES

The American Chemistry Council's Brominated Flame Retardant Industry Panel (BFRIP) volunteered under the U.S. EPA's Voluntary Children's Chemical Evaluation Program (VCCEP) pilot to prepare the Data Summary for decabromodiphenyl oxide (DBDPO). This compound (CAS No. 1163-19-5) is also known as decabromodiphenyl ether. DBDPO's toxicology has been extensively investigated and information is available on virtually all end-points listed in VCCEP's Tiers I, II and III.

DBDPO was identified for the VCCEP pilot on its assumed detection in human milk (see Table 1, page 81704, Federal Register, Vol. 65, No. 248, December 26, 2000 citing Noren et al. 1998). However, Noren et al. did not report DBDPO in human breast milk, and DBDPO has not been reported in human breast milk in publications appearing prior to or since 1998. Noren et al.'s, and other authors, use of the terminology "PBDEs" rather than specifying the isomers or congeners detected is likely the cause for this error.

DBDPO has been extensively tested for toxicity and exhibits minimal effects. These tests have shown that DBDPO is not toxic in a single large dose nor does it induce gene mutations. Tests have also shown DBDPO is not toxic to the developing embryo and fetus and does not interfere with reproduction. No harmful effects were seen in studies where DBDPO was repeatedly administered to rats and mice in doses of at least 1,000 mg/kg every day for several months. This is roughly equivalent to someone weighing ~150 pounds swallowing ~2.5 ounces of DBDPO every day for many years throughout his or her life or to a 44 pound child consuming about 0.7 ounces of DBDPO every day.

One reason that DBDPO has such little toxicity is that it is minimally absorbed into the body. Studies in rats show they absorb only 0.3 – 2% of the DBDPO added to their feed. DBDPO's low absorption leads to its quick elimination in the feces. More than 99% of a given dose exits the body in the feces within 72 hours. Because of this, DBDPO does not accumulate, or build-up, in the body.

DBDPO is used solely as a flame retardant to prevent or delay ignition in burnable materials. DBDPO's main application is in high impact polystyrene (HIPS) used for electronic enclosures, e.g. television set cabinet backs. A comparatively minor, but important, use of DBDPO is to flame retard upholstery fabric where it is applied to the back of the fabric encapsulated in latex. At a minimum, an estimated 280 deaths are avoided in the U.S. every year because of the use of brominated flame retardants in the applications where DBDPO is used.

Fires in the United States are a serious problem with our country having one of the highest fire incidence and mortality rates in developed nations. The National Fire Protection Association reports that in the year 2000:

- A fire department responded to a fire somewhere in the United States every 18 seconds.
- Public fire departments attended 1,708,000 fires, of which 505,500 occurred in structures, 348,500 occurred in vehicles, and 854,000 occurred in outside properties.

- Someone died in a fire in the United States every 130 minutes. There were 4,045 fire deaths, a significant increase of 13.3% from the previous year. This death rate was for civilians only and did not include firefighters.
- People are at greatest risk of death and injury from a fire in their homes – 85% of fire deaths occur at home. There were 3,420 deaths from fires in the home, an increase of 18.1% from the previous year.
- The civilian fire death rate in the United States was 14.8 deaths per million people.
- Someone was injured in a fire in the United States every 23 minutes. There were an estimated 22,350 civilian fire injuries, of which 16,975 occurred in homes. This injury rate was for civilians only and did not include firefighters.
- Smoking materials were the leading cause of civilian deaths, accounting for roughly one-fourth of the total.

Those at high-risk of death, injury or burns in fires are children, the elderly, and the poor. Fires are a leading cause of unintentional injury-related death among children in the United States. Each year, more than 600 children ages 14 and under die, and nearly 47,000 are injured, in fires. Based on annual averages for the five-year period from 1994 through 1998, children five and under made up about 9% of the country's population, but accounted for 17% of the home fire deaths. A child's risk of dying in a fire is twice the national average. Adults 65 and older also face a risk twice the average, while people 85 and older had a risk that is almost four-and-a-half times more than average.

The circumstances surrounding the potential for a deadly fire have changed in the last few decades. Today's homes and businesses store more contents than in the past - the fire load in a typical home has more than doubled in the past 50 years on a pound per square foot basis. Furnishings are often constructed of synthetics that are made from petrochemicals and that can actually enhance a fire's growth. Homes and offices are also more energy efficient and hold heat better than in the past and this also can enhance the seriousness of a fire. This combination of more synthetic materials and higher energy efficiency increases the risk of a serious fire, if a fire starts.

These factors have intensified the need for flame retardancy in many applications, especially electrical and electronic products such as television sets, computers, and wire and cable that combine a potentially flammable plastic with a source of ignition (e.g. electricity). Flame retardants can reduce the risk of death or injury in fires by preventing or delaying ignition, reducing the rate the fire releases heat, reducing the quantity of toxic gases produced, and increasing the time available to leave the burning building. Studies have shown that flame retardants can increase the time available to escape a burning building by a factor of 15. In a fire where every second counts, this can literally mean the difference between life and death.

The U.S. National Academy of Sciences (NAS) concluded DBDPO did not present a health risk to consumers, including children, when used in upholstery textiles. NAS did not review DBDPO's use in electrical and electronic products, but one can draw a similar conclusion about these uses by considering DBDPO's properties and toxicology. DBDPO is a large poorly absorbed molecule that has been shown to cause little toxicity. When used in television cabinet

backs, it is part of a dense hard plastic with minimal possibility of exposure to the user. The combination of these factors indicates DBDPO's use in electrical and electrical products would not be expected to be a health risk to consumers, including children. Further, the protection provided by DBDPO in terms of enhanced fire safety reduces the very real risk of death or injury one faces in the home.

Two other exposure possibilities are food and breast milk. Laboratory studies have shown DBDPO is not absorbed to any significant amount in fish. This is probably due to its poor solubility and large molecular weight. DBDPO has not been detected in limited sampling of fish and poultry in the U.S., and based on its properties, is not anticipated to be present in these foods or in meat or dairy products. Likewise, vegetables and root crops like lettuce or potatoes are not expected to be an exposure source because plants would not absorb DBDPO. This, coupled with DBDPO's very limited toxicity, indicates negligible health risk due to food exposure.

DBDPO's very poor absorption means that the nursing mother would have negligible amounts to pass on to her infant. Also, DBDPO is such a large molecule that transfer to breast milk, if it occurs at all, will be slow and limited and build-up of concentrations in breast milk is not expected. Taken together, the combination of poor absorption by the nursing mother, and poor and/or slow movement into breast milk should effectively preclude DBDPO in milk. A risk to the nursing infant is not anticipated.

Occupational exposure to DBDPO dust could occur when DBDPO is bagged at the manufacturer or when the user empties the bags. The American Industrial Hygiene Association (AIHA) established a Workplace Environmental Exposure Level (WEEL) of 5 mg/m^3. This was based on DBDPO's toxicology and is equivalent to a nuisance dust. A WEEL is the level that workers could be exposed to every day with the expectation of no harmful effects. Inhalation of vapor and absorption through the skin are not realistic sources of occupational exposure to DBDPO due to its negligible vapor pressure and predicted skin absorption. Theoretically, the flame retardant textile backcoat could crumble during fabrication of upholstered furniture, but absorption of significant amounts as a result of crumbling of the backcoat is highly unlikely.

In conclusion, DBDPO has undergone extensive testing and shows minimal toxicity. No additional tests are proposed. DBDPO's toxicology is such that harmful effects to workers, the general public or children are not anticipated. DBDPO provides significant benefits to consumers and their children by lessening the very real danger presented by fires in the home.

1.0 INTRODUCTION

The Brominated Flame Retardant Industry Panel (BFRIP) was formed in the 1980s to address issues related to the brominated flame retardants that its members manufacture in common, conduct research, and interact with regulatory agencies and other interested parties. Its members, who are global manufacturers of brominated flame retardants, are Albemarle Corporation, Ameribrom Inc. (a subsidiary of Dead Sea Bromine Group), and Great Lakes Chemical Corporation. Akzo-Nobel is an associate member. BFRIP, organized under the American Chemistry Council, volunteered under the U.S. EPA's Voluntary Children's Chemical Evaluation Program (VCCEP) pilot to prepare the Data Summary for decabromodiphenyl oxide (DBDPO). This compound (CAS No. 1163-19-5) is also known as decabromodiphenyl ether. As discussed below, DBDPO is a data-rich chemical having valid guideline studies or other information for all Screening Informational Data Set (SIDS) endpoints.

DBDPO was included in the VCCEP pilot on the basis of its detection in human milk (see Table 1, page 81704, Federal Register, Vol. 65, No. 248, December 26, 2000 citing Noren et al. 1998). However, Noren et al. did not report DBDPO in human breast milk, and DBDPO has not been reported in human breast milk in publications appearing prior to or since 1998. Noren et al.'s, and other authors, use of the terminology "PBDEs" rather than specifying the isomers or congeners detected is likely the cause for this error. BFRIP volunteered to sponsor DBDPO under the VCCEP pilot in an effort to rectify this, and to provide a publicly accessible summary of DBDPO's toxicology.

2.0 STRUCTURE AND PROPERTIES

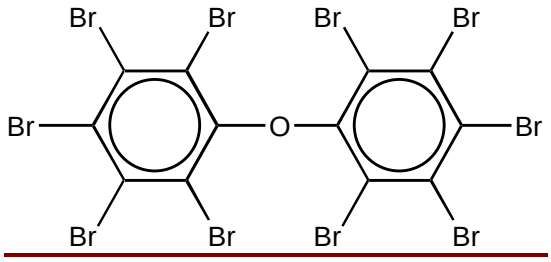

Figure 1. Decabromodiphenyl oxide (DBDPO).

DBDPO, a solid at room temperature, is a fully brominated (e.g. 10 bromine atoms) diphenyl oxide with a molecular weight of 959.17 (Figure 1). The composition of the commercial product is typically ≥ 97% DBDPO with the remainder composed of nonabromodiphenyl oxide. DBDPO's measured water solubility (<0.1 ug/L) (Stenzel and Markley 1997) and vapor pressure (4.63×10^{-6} Pa) (Stenzel and Nixon 1997) are negligible. DBDPO's solubility in organic solvents is also extremely low: acetone 0.05%, benzene 0.48%, methylene bromide 0.42%, xylene 0.87%, and 0.2% in toluene (WHO 1994; Norris et al. 1973). DBDPO is often assumed to be lipophilic due its presumed similarity to PCBs (Hardy 2002a). However, no formal fat solubility study has been performed, and pharmacokinetic studies show no appreciable affinity of DBDPO for

adipose tissue. Using NTP's (1986) pharamacokinetic data, DBDPO's blood:liver:adipose ratio in the rat was 1:7:2 compared to Arochlor 1254's ratio of 1:22:359 (Kodavanti et al. 1998). DBDPO's measured octanol/water partition coefficient (Log K_{ow}) was 6.265 (Macgregor and Nixon 1997), but its Log K_{ow} estimated by EPIwin, v3.4, was 12.61 (Meyland and Howard 1999). It is apparent that DBDPO has a very low solubility coefficient in water and most organic solvents. Further, DBDPO's estimated K_{ow} appears to be a better predictor of it behavior in biological systems than its measured value, based on mammalian pharmacokinetic studies and fish bioconcentration data. This is likely due to its very poor solubility in both water and octanol so that any small change in concentration produces a large change in the measured K_{ow} value.

The DBDPO commercial product has been analyzed for trace quantities of 15 2,3,7,8-substituted polybrominated-p-dibenzodioxins (PBDD) and dibenzofurans (PBDF) under a U.S. Environmental Protection Agency (EPA) test rule. None of the analytes were present at or above the quantitation limits established by the agency (Ranken et al. 1994). Resins containing DBDPO have also been analyzed for PBDD/PBDF content. A high impact polystyrene (HIPS) resin containing antimony trioxide and DBDPO was molded using normal (215-220°C; 30 sec.), abusive (235-245°C; 5 min.) or extreme (265-270°C, 7 min.) processing conditions (McAllister et al. 1990). The molded resin was cryogenically ground and analyzed for six 2,3,7,8-substituted PBDD/PBDFs. None were detected. Polybutyleneterephthalate (PBT) resin containing antimony trioxide and DBDPO was also molded under similar conditions, and analyzed. No 2,3,7,8-substituted PBDD/PBDFs were detected. Donnelly et al. also analyzed molded HIPS/DBDPO/Sb2O3 and molded PBT/DBDPO/Sb2O3, and detected no 2,3,7,8-TBDF and no 1,2,37,8-PeBDF. Brenner and Knies (1990) also reported no PBDDs in their analysis of an extruded PBT/DBDPO blend. Virgin molded HIPS/DBDPO/Sb2O3 and repeatedly ground and injection molded (e.g. "recycled") HIPS/DBDPO/Sb2O3 resins meet the requirements of the German Chemicals Banning Ordinance with respect to 2,3,7,8-substituted PBDD/F content (Hamm 1999; Hamm et al. 2001). The concentrations of relevant PBDD/F congeners were at least one order of magnitude below the regulated limit values for PBDD/F (1 ppb for the sum of four congeners, 5 ppb for the sum of all eight regulated congeners).

The DBDPO product is one of three commercial polybrominated diphenyl oxide (a.k.a. ether) products manufactured, and accounts for approximately 83% of all polybrominated diphenyl oxide/ether (e.g. "PBDE") production. The other two commercial polybrominated diphenyl oxide/ether products are known as octabromodiphenyl oxide/ether (OBDPO, CAS# 32536-52-0) and pentabromodiphenyl oxide/ether (PeBDPO, CAS# 32534-81-9) and are listed in the VCCEP's pilot. OBDPO, a mixture of brominated diphenyl oxide congeners ranging from nona- to hexa-, is used to flame retard business equipment constructed of acrylonitrile-butadiene-styrene (ABS) plastic. PeBDPO, a highly viscous liquid composed of tetra-, penta- and hexaBDPO congeners, is used to flame retard polyurethane foam that is used as cushioning in upholstery.

3.0 APPLICATIONS

DBDPO is used solely as a flame retardant for the purpose of preventing or delaying ignition in combustible materials (See Sections 3.1-3.6 for information on the fire hazard in the U.S. and Apppendix I for information on the importance of flame retardants in today's plastics). DBDPO's flame retardant activity is derived from its bromine content. Bromine is one of the few elements able to provide flame retardancy in the gas phase; certain plastics require a flame retardant active in the gas phase due to the way they burn. DBDPO's high bromine content makes it very effective as a flame retardant that in turn makes it extremely cost-effective. As a result, DBDPO is the second largest volume brominated flame retardant in production and use. Global market demand in 1999 for DBDPO was estimated at 54,800 metric tons (BSEF 2001). Market demand, 1999, for DBDPO in the regions of the America's, Europe and Asia was 24,300, 7,500 and 23,000 metric tons, respectively (BSEF 2001). These regional differences reflect differences in the location of end product manufacture. Two companies manufacture DBDPO in the U.S. Production facilities of both manufacturers are located in Arkansas to take advantage of the underground brine fields as a source of bromine.

DBDPO's main application is in high impact polystyrene (HIPS) used for electronic enclosures, e.g. television set cabinet backs (Hardy 2002b). A comparatively minor, but important, use of DBDPO is to flame retard upholstery fabric where it is applied as a fabric back coat encapsulated in latex (Hardy 2002b). DBDPO's potential risk to the consumer, including children, in the upholstery application was recently reviewed by the United States National Academy of Sciences (NAS 2000). DBDPO is not used to flame retard children's clothing or sleepwear.

3.1 U.S. Fire Risks

Fires in the United States are a serious problem. The U.S. has one of the highest fire incidence and mortality rates of all developed countries. This is despite all our modern efforts including fire departments, building codes, fire drills, fire alarms, smoke detectors, fire sprinklers, fire extinguishers, UL ratings, and flame retardants.

The National Fire Protection Association (NFPA 2001) reports that in the year 2000:

- Every 18 seconds, a fire department responded to a fire somewhere in the United States.
- Public fire departments attended 1,708,000 fires, of which 505,500 occurred in structures, 348,500 occurred in vehicles, and 854,000 occurred in outside properties.
- Nationwide, there was a civilian (non-firefighter) fire death every 130 minutes. There were 4,045 fire deaths, a significant increase of 13.3% from the previous year.
- About 85% of all fire deaths occurred in home fires. There were 3,420 deaths from fires in the home, an increase of 18.1% from the previous year.
- The civilian fire death rate in the United States was 14.8 deaths per million people.
- Nationwide, there was a civilian fire injury every 23 minutes. There were an estimated 22,350 civilian fire injuries, of which 16,975 occurred in homes.
- Smoking materials were the leading cause of civilian deaths, accounting for roughly one-fourth of the total.

3.2 Basic Fire Concepts

Fire is dark. In television and movies, fire is often portrayed as a bright light, but the fire environment is actually pitch black due to the dense smoke produced. Escape plans must be memorized (USFA 2002; Education World 2002).

Smoke from fire kills. Fire victims typically succumb to smoke inhalation before flames reach them. More fire deaths occur when people are sleeping—between 2 a.m. and 6 a.m (USFA 2002; Education World 2002).

Many people believe – falsely - that they would awaken in a fire. But toxic gases, typically carbon monoxide, actually put people into a deeper sleep (USFA 2002; Education World 2002).

Fire is intensely hot. This might seem obvious, but few understand that fire can cause the temperature to rise *several hundred degrees* in seconds. That degree of heat can prompt the human body to stop functioning and lose consciousness, making escape impossible (USFA 2002; Education World 2002).

Fire is fast. A home can be completely consumed by fire in less than five minutes. In less than 30 seconds a small flame can get completely out of control and turn into a major fire. It takes only minutes for thick black smoke to fill a house. Time is the biggest enemy and every second counts (USFA 2002; Education World 2002).

Flame retardants prevent or delay ignition, reduce the rate of heat release, reduce the quantity of toxic gases generated, and increase the time available for escape. Studies have shown that flame retardants can increase escape time by a factor of 15. In a fire where every second counts, this can literally mean the difference between life and death. See Section 3.5 for additional information.

3.3 Children and Elderly at High Risk of Death and Injury in Fires

Populations at high-risk of death, injury or burns in fires are the very young, the elderly, and the poor (NSKC 2002; Stevens and Mann 1999). Based on annual averages for the five-year period from 1994 through 1998, children five and under made up about 9% of the country's population, but accounted for 17% of the home fire deaths. A child's risk of dying in a fire is twice the national average. Adults 65 and older also face a risk twice the average, while people 85 and older had a risk that is almost four-and-a-half times more than average.

3.4 Why Children Are at Special Risk in Fires

Fires are a leading cause of unintentional injury-related death among children in the United States. Each year, more than 600 children ages 14 and under die, and nearly 47,000 are injured, in fires (NSKC 2002).

Picture a fire from a child's point of view: smoke and flames suddenly sweep through his room. It is dark, hot, loud and scary. A large stranger comes in, wearing equipment that makes

him look like a monster or an alien – or worse. Children's first instincts are often to hide from things that frighten them. But in the case of a fire, those instincts can be deadly (NSKC 2002).

Kids are at grave risk of injury and death from residential fires because they have less control of their environment than adults and limited ability to react appropriately. More than 40 percent of residential fire-related deaths among children ages 9 and under occur when the child is attempting to escape, is unable to act or is acting irrationally. Although an escape plan may help to reduce these deaths, only 26 percent of households have developed and practiced a plan (NSKC 2002).

The youngest children are at greatest risk. Children ages 5 and under are more than twice as likely to die in a fire as the rest of the population. More than half of the children in this age group who die are asleep at the time of the fire, and another one-third of them are too young to react appropriately (NSKC 2002).

Older children are often at risk due to their own curiosity. Studies indicate that an estimated 38 percent of children ages 6 to 14 have played with fire at least once. Child-play home fires tend to begin in a bedroom where children are left alone. Children playing with matches or lighters start 80 percent of these. Boys are nearly twice as likely as girls to play with fire (NSKC 2002).

Other risk factors especially related to children include the following. Children in homes without working smoke alarms are at the greatest risk. Households without working smoke alarms are approximately two and a half times more likely to experience a fire in their homes (NSKC 2002).

Home cooking equipment is the leading cause of residential fires and fire-related injuries. However, residential fires caused by smoking materials (i.e. cigarettes) are the leading cause of fire-related death, accounting for nearly 23 percent of all fatalities (NSKC 2002).

Home fires and fire-related deaths are more likely to occur during the cold weather months December through February, when there is a significant rise in the use of portable or area heating equipment such as fireplaces, space heaters and wood stoves (NSKC 2002).

Children living in rural areas have a dramatically higher risk of dying in a residential fire. Death rates in rural communities are more than two times higher than in large cities, and more than three times higher than in large towns and small cities (NSKC 2002).

3.5 Flame Retardants – Protection Through Prevention

Years ago, most combustible building contents were made of cellulosic materials commonly found in nature (Leihbacher 1999). Chairs and tables were made of wood, sofas and bedding with cotton batting and jute, carpeting with wool and cotton fibers, and draperies with linen and other natural materials. Rapidly spreading fires were uncommon and generally indicated the use of a petroleum-based accelerant like gasoline. Today, the furnishings in homes and businesses include those constructed of petrochemicals such as polyurethane foams and rigid polystyrene

plastic. These materials can behave in a fire as if they have built-in-accelerant, and can produce quantities of heat exceeding those of ordinary combustibles.

Another change from the past is that today's buildings and homes have more contents. The fire load in residential structures has more than doubled in the past 50 years on a pound per square foot basis (Leihbacher 1999). Flashover, when the room bursts into flame and the most dangerous time of a fire, has become more common as a result of the greater fire load and the use of synthetic furnishings. Synthetics, especially foams and plastics, produce more heat than natural products - the heat produced by burning foams and plastics can approach that of highly volatile flammable liquids. This contributes to the development of flashover so that flashover now occurs rapidly - generally within 3-10 minutes after ignition. Flashover is caused by the radiation feedback of heat. Heat from the growing fire is absorbed into the upper walls and contents of the room, heating combustible gases and furnishings to their auto-ignition temperature. This build up of heat in the room triggers flashover. Flashover signals the end of an effective search and rescue in a room; it means the death of any person trapped in the blazing room — either civilians or firefighters. Flashover signals the change from a contents to a structure fire and the beginning of the structural collapse danger.

Another change in modern buildings and homes is increased energy efficiency (Leihbacher 1999). Buildings are designed to hold heat inside in the winter and exclude heat in the summer. Over the last 20 years new energy-efficiency standards have come into effect, and better and more insulation of walls, floors, ceilings, roofs, and windows has occurred. This higher energy efficiency influences the building's behavior in the event of a fire. Energy efficient upper walls and ceilings are less able to conduct heat away from the fire room, resulting in a higher temperature fire in the room of origin. Energy efficient thermal pane windows are more break resistant than older window types, and are less likely to break and vent the fire's heat outdoors. The net result of enhanced energy efficiency, in the event of a fire, is rooms that burn hotter and hold heat better.

The combination of higher energy efficiency and a greater quantity of synthetic materials increases the potential for a serious fire if ignition occurs (Leihbacher 1999). Thus, the extensive use of synthetic polymers has intensified the need and concern for flame retardancy in many applications. Flame retardants are especially useful for flammable foams and plastics where they act to delay ignition and slow flame spread. Flame retarded products also generate a lower rate of heat release once ignited which in turn influences the development of flashover. A slower rate of heat release also lowers the quantity of toxic gases produced. These factors all translate into longer escape times for occupants - the use of flame retardants can increase escape times by a factor of 15 (FRCA 1987; Babrauskas et al. 1988) – and provide life safety benefits (Clarke 1997).

The life safety benefits derived from the use of brominated flame retardants (BFRs) in the U.S. were determined using fire data from the National Fire Protection Association (NFPA) (Clarke 1997). Four product classes were identified in which BFRs are widely used and which could be directly associated with fire data: television/appliances, wire/cable insulation, curtains/draperies and upholstered furniture. An estimated 190 lives are saved annually through the use of BFRs (e.g. DBDPO) in television cabinets. For electrical insulation and draperies, less product and fire

data were available, but 80 and 10 lives, respectively, were estimated saved annually through the use of BFRs in these products. Again, DBDPO is a major flame retardant used in electrical insulation and in draperies. Thus, an estimated 280 deaths are avoided each year in the U.S. due to the use of BFRs. A large portion of these lives saved are likely attributable to DBDPO. Another 140-220 fire deaths per year could be avoided if upholstered furniture fabrics were backcoated with BFR-latex as is now done to meet California standards for upholstered furniture.

3.5 Sources of Additional Information on Fires and Their Impact

Additional information on fires in the U.S. and their impact on children can be found on the following websites. This is only a partial list and there are many other excellent sources of information on this topic. The State Fire Marshall in each of the 50 states can also provide information on the local situation as well as educational tools and services.

The United States Fire Administration (USFA): www.usfa.fema.gov.

The USFA's Kid's Page: www.usfa.fema.gov/kids.

National Fire Protection Association (NFPA): www.nfpa.org.

Consumer Product Safety Commission (CPSC): www.cpsc.gov.

International Association of Fire Chiefs: www.iafc.org.

Education World, Lesson Planning, Fire Safety Activities: www.education-world.com.

National Safe Kids Campaign: www.safekids.org.

4.0 DBDPO HAZARD ASSESSMENT

4.1 Mammalian Toxicology (VCCEP Tiers I, II, and III)

DBDPO has undergone extensive testing in mammalian species (Table 1). All studies were performed using a commercial DBDPO product unless otherwise stated. In brief, the studies show that DBDPO is not acutely toxic or mutagenic, and is not a developmental or reproductive toxicant. The NOAEL for DBDPO in subchronic and/or chronic studies in the rat or mouse is at least 1,000 mg/kg/d. DBDPO's low toxicity is likely related to its poor absorption and rapid elimination (NTP 1986). Pharmacokinetic studies have shown that DBDPO is poorly absorbed (0.3 -2% of an oral dose), has a short half-life (24 hr) compared to PCB 153 (<2% of an oral dose was eliminated by rats in 21 days), can be metabolized, and is rapidly eliminated in the feces (>99% in 72 hr) (NTP 1986; Norris et al. 1973, 1975; El Dareer et al. 1987; Moreck and Klassen-Wheler 2001).

4.1.1 Acute Toxicology (Tier I)

TABLE 1. DBDPO Mammalian Toxicology Summary.

TEST	RESULTS
Water Solubility +	< 0.1 ug/L (Stenzel and Markley 1997)
Vapor Pressure+	4.63 x 10-6 Pa (Stenzel and Nixon 1997)
Octanol/Water Partition Coefficient +	6.265 (measured) (MacGregor and Nixon 1997)
Rat Oral LD50	> 2,000 mg/kg (Norris et al. 1973)
Rabbit Dermal LD50	> 2,000 mg/kg (Great Lakes 1974b)
Rat Inhalation LC50	> 48.2 mg/L (Great Lakes 1974c)
Rabbit Eye Irritation	Not an irritant (Great Lakes 1974e)
Rabbit Skin Irritation	Not an irritant (Norris et al. 1973, 1974)
Human Skin Sensitization	Not a skin sensitizer (Norris et al. 1973, Industrial Biotest 1975)
Ames+	Not mutagenic (Wagner and Klug 1998)
Mouse Lymphoma*	Not mutagenic (NTP 1986)
Sister Chromatid Exchange*	Did not induce (NTP 1986)
Chromosome Aberration*	Did not induce aberrations (NTP 1986)
14 Day Rat & Mice Oral (Diet)*	NOEL $\geq$ 100,000 ppm (10% of diet or ~ 10,000 mg/kg/d) (NTP 1986)
90 Day Rat & Mice Oral (Diet)*	NOEL $\geq$ 50,000 ppm (5% of diet or ~5,000 mg/kg/d) (NTP 1986)
30 Day Rat (Diet)**	NOEL = 0.01% (8 mg/kg/d) (Norris et al. 1973, 1974, 1975)
Rat 1 Generation Reproduction**	NOEL $\geq$ 100 mg/kg/d (highest dose tested) (Norris et al. 1975)
Rat Developmental, Days 0-19 Gestation* +	NOEL $\geq$ 1,000 mg/kg/d (maternal & fetal) (Hardy et al. 2002)
Rat Developmental, Days 6-15 Gestation**	NOEL ≥ 1,000 mg/kg/d (maternal) NOEL = 100 mg/kg/d (fetal) (Norris et al. 1973, 1974, 1975)
Rat & Mouse Carcinogenicity (Diet)*	25,000 (2.5%) or 50,000 (5%) ppm for 2 years (~3,200 – 7,780 mg/kg/d Mice; ~1,120 – 2,550 mg/kg/d Rats) Negative, equivocal or some evidence of carcinogenicity No effect body weight or mortality Minimal evidence of chronic toxicity (NTP 1986)
Rat Carcinogenicity (Diet)**	NOEL $\geq$ 1 mg/kg/d for 2 years (highest dose tested) (Kociba et al. 1975)
Rat Hepatic Enzyme Induction	Did not induce hepatic enzymes: cytochrome P450, cytochrome P450 reductase, UDP-glucuronyl-transferase, benzo[a]pyrene hydroxylase, p-nitroanisole demethylase, or EPN detoxification. (Carlson 1980)
Rabbit Skin Acnegenicity	Not acnegenic; Soot and char not acnegenic (Pinkerton et al 1989)
Rat Pharmacokinetics (Oral & IV)*	Poorly absorbed (<0.3-2%) from GI tract Rapidly Eliminated (>99% in 72 hours) Half life < 24 hours (NTP 1986; El Dareer et al 1987)

*Test article 94-99% DBDPO.
** Test article only 77% DBDPO.
+Studies Performed under Good Laboratory Practices and using today's commercial DBDPO product as test article.

DBDPO was not acutely toxic, was not irritating to the skin or eye of rabbits, and did not induce skin sensitization in a human patch test (Norris et al. 1973, 1974, 1975; NTP 1986). The $LD50_{oral,\ dermal}$ in rats and rabbits, respectively, was > 2,000 mg/kg. The rat 1 hr $LC50_{inhalation}$ was > 48.2 mg/L. The soot and char combustion products of a DBDPO plastic matrix were also not acutely toxic (Pinkerton et al. 1989).

4.1.1.1 Acute Studies

Intragastric intubation of a single dose of a 10% corn oil suspension of DBDPO (Dow FR-300-BA: 77.4% DBDPO, 21.8% NonaBDPO and 0.8% OBDPO) to female Sprague Dawley rats resulted in the survival of all rats at doses of 126, 252, 500, 1,000 or 2,000 mg/kg. No indication of toxicity after intubation or during the 14-day period was observed. No gross pathological changes were observed at necropsy carried out on one rat/dose level (Norris et al., 1973).

Groups of 2 male and 2 female New Zealand White rabbits were administered single doses of 200 or 2,000 mg/kg of DBDPO (DE-83) applied neat under occlusive wraps for 24 hours: all the animals survived. Animals were observed for 14 days. At the 2,000 mg/kg dosage level all rabbits exhibited normal body weight gains. Local and general signs of toxicity were not reported and necropsies not performed (Great Lakes 1974b).

Groups of 5 male and 5 female Spartan rats were exposed for one hour to 2 or 48.2 mg/l DBDPO (DE-83) in air and subsequently observed for 14 days. All rats survived. Dyspnea and ocular discharge were noted from 2 mg/l concentration (one animal); moreover, in the 48.2 mg/l group, eye squint and increasing motor activity were observed. All rats were normal at the end of 14-day-observation period. Necropsies were not performed (Great Lakes 1974c).

Norris et al. (1973 and 1974) reported that DBDPO applied as dry solid on shaved skin of New Zealand albino rabbits caused essentially no response on intact skin and a slight erythematous and edematous response on abraded skin after a single confined exposure of 24 hours. Repeated exposures to intact skin for five days/week for two weeks and to abraded skin for three days did not alter the responses observed following a single administration.

DBDPO as dry solid (500 mg), cause no irritation on intact or abraded skin when applied to shaved skin under occlusion to 2 groups of 3 New Zealand White rabbits. No erythema or edema was observed after a single exposure for 24h and followed by an observation period of 72h (Great Lakes 1974d).

Studies with 3 male and 3 female New Zealand White rabbits showed that 100 mg DBDPO (93 - 98.5% purity) as dry solid caused transient (reversible in 48h) mild irritation of the conjunctival membranes. The cornea, iris and lens were unaffected (Great Lakes 1974e). This study was carried out in accordance with the GLP procedures.

4.1.1.2. Human Sensitization

In 50 human subjects, repeated application of a suspension of 5% DBDPO in petrolatum 3 times a week for 3 weeks and challenged two weeks subsequent to the last induction application did not result in skin sensitisation. Skin irritation was observed in 9 out of the 50 persons (Norris et al., 1974; WHO, 1994).

Human volunteers (80 males and 120 females) were treated with 9 induction patches of 2 batches of DBDPO. The first sample was evaluated as received, and the second as a 2% (w/v) aqueous solution. The patches were applied once every 2 days, allowed to contact the skin for 24h, and the skin was graded for irritation. Fifteen (15) subjects among the 200 volunteers showed some slight irritation reactions: very slight erythema - barely perceptible in 14/1,800 patches and mild – well defined erythema in 2/1,800 patches and very slight edema – barely perceptible in 1/1,800 patches. After a non-patching period of 12 days, the challenge patch was applied to detect sensitisation. No evidence of skin sensitisation with either of the test materials in any of the subjects tested was observed (Industrial Bio-Test Laboratories, 1975).

4.1.1.3 Soot and Char Combustion Products

The soot and char combustion products from a high impact polystyrene/DBDPO/antimony trioxide matrix also were not acutely toxic in rats (LD_{50} > 2,000 mg/kg) (Pinkerton et al. 1989). Six groups of 5 male and female Sprague-Dawley rats were treated with a single dose via gavage in 1% methylcellulose with 0, 0.5, 5, 50, 500 or 2,000 mg/kg of the combined soot and char generated from the combustion of a DBDPO/high impact polystyrene/antimony trioxide matrix and observed for 28 days. No animals died during the study and clinical signs of toxicity were observed. No histologic lesions were detected in the examined organs – thyroid, parathyroid, adrenal gland, spleen, gonads, heart, liver, lung, brain, kidneys and thymus. The $LD50_{oral}$ of the soot and char combustion products of a high impact polystyrene/DBDPO/antimony trioxide matrix was > 2,000 mg/kg body weight (Pinkerton et al. 1989). Based on these results, toxicologically significant amounts of polybrominated dioxins or polybrominated dibenzofurans were not present in the soot and char, or if present, were not biologically available.

4.1.2 Repeated Dose Toxicology (VCCEP Tiers I and II)

DBDPO administered at 10% and 5% of the diet for 14 and 90 days, respectively, produced no adverse effects in F344/N rats and $B6C3F_1$ mice (NTP 1986).

4.1.2.1 U.S. NTP 14-Day Repeated Dose Studies in Rats and Mice (1986) (Tier I)

Groups of five males and five females were fed diets containing 0, 5,000, 10,000, 20,000, 50,000 or 100,000 ppm DBDPO for 14 days. Male and female F344/N rats and B6C3F1 mice were obtained from Charles River Breeding Laboratories and held for approximately 3 weeks before the studies began. Animals were assigned to groups such that cage weights were approximately equal at initiation of the study. Animals were housed 5 per cage (polycarbonate) on heat-treated hardwood chips. Formulated or control diets and water were available ad libitum. The formulated diets were checked for homogeneity and correctness of concentration.

Rats and mice were observed daily for clinical signs of toxicity and were weighed on days 1, 7 and 14. A necropsy was performed on all animals in all doses. Organs examined at the gross necropsy included gross lesions, skin, mandibular lymph nodes, mammary glands, salivary glands, thigh muscle, sciatic nerve, sternebrae, femur or vetebrae including marrow, costochondral junction (rib), thymus, larynx, trachea, lungs and bronchi, heart, thyroid gland, parathyroids, esophagus, stomach, duodenum, jejunum, tissue masses, ileum, colon, cecum, rectum, mesenteric lymph nodes, liver, gallbladder (mice), pancreas, spleen, kidneys, adrenal glands, urinary bladder, seminal vesicles/prostate/testes or ovaries/uterus, nasal cavity, brain, pituitary gland, spinal cord and eyes.

DBDPO doses up to 10% (100,000 ppm) of the diet in F344/N rats and $B6C3F_1$ mice produced no mortality, no effect on body weight, and no compound-related clinical signs or gross pathologic effects (histopathology was not performed). The test article in the 14 day study was 99% pure DBDPO.

4.1.2.2 U.S. NTP 13-Week Repeated Dose Studies in Rats and Mice (1986) (Tier II)

In the 13-week study, DBDPO doses up to 5% of the diet in F344/N rats (n = 10 rats/sex/dose) and $B6C3F_1$ mice (n = 10 mice/sex/dose) produced no mortality, no effect on body weight, and no compound related gross or microscopic pathologic effects. The dietary dose levels were 0, 3, 100, 6,200, 12,500, 25,000 or 50,000 ppm DBDPO and were fed for 13 weeks.

Four-week-old male and female F344/N rats and 5-wek-old B6C3F1 mice were obtained from Charles River Breeding Laboratories, observed for 4 weeks, and assigned to cages according to a table of random numbers. The cages were then assigned to dosed and control groups according to another set of random numbers. Animals were housed five per cage (polycarbonate) on heat-treated hardwood chips. Formulated or control diets and water were available ad libitum. The formulated diets were checked for homogeneity and correctness of concentration. Animals were checked twice daily; moribund animals were sacrificed. Feed consumption was measured weekly by cage. Animal weights were recorded weekly. Clinical signs and behavior was recorded weekly. At the end of the 13-week studies, survivors were sacrificed and a necropsy was performed on all animals. Approximately 30 tissues were examined histologically in the control and high dose groups: gross lesions and tissue masses, mandibular or mesenteric lymph nodes, salivary gland, sternebrae, femur or vertebrae including marrow, thyroid, parathyroids, small intestine, colon, liver, gallbladder (mice), prostate/testes or ovaries/uterus, lung and mainstem bronchi, heart, esophagus, stomach, brain, thymus, trachea, pancreas, spleen, kidneys, adrenal glands, urinary bladder, pituitary gland, spinal cord (if neurologic signs present), eyes if grossly abnormal), and mammary gland. The test article used in this study consisted of two lots of DBDPO; one lot was that used in the 14 day study and the second was ~97% pure DBDPO.

4.1.2.3 U.S. NTP Two Year Studies in Rats and Mice (1986) (Tier III)

Doses of 2.5 or 5% DBDPO in the diet for two years (103 weeks) were also well tolerated by F344/N rats (n=50 rats/sex/dose) and $B6C3F_1$ mice (n=50 mice/sex/dose) with no effect on body

weight or mortality and only minimal evidence of organ effects (NTP 1986). The U.S. National Toxicology Program (NTP) estimated the average amount of DBDPO consumed per day in the two year study to be 1,120 mg/kg and 2,240 mg/kg for low and high dose male rats, respectively, and 1,200 mg/kg and 2,550 mg/kg for low and high dose female rats, respectively. Likewise, NTP estimated the average DPDPO consumed per day by mice in the two year study was 3,200 and 6,650 mg/kg for low and high dose male mice, respectively, and 3,760 and 7,780 mg/kg for low and high dose female mice, respectively. The test article used in this study consisted of two lots of DBDPO that were 96% or 94-97% pure DBDPO, respectively.

Animals used in the 2-year study were produced under strict barrier conditions at Charles River Breeding Laboratories. Animals were shipped to the test laboratory at 5-6 weeks of age, quarantined for 14 (rats) or 16 (mice) days, and placed on the study when 7-8 (rats) and 9 (mice) weeks old. Animals were housed in polycarbonate cages with heat-treated hardwood chips. Rats and female mice were housed 5/cage, male mice 5/cage until month 8 and then 1/cage for intermittent periods, and 1/cage after 15 months. The animal room environment was 68-80 degrees F, 15-90% humidity, fluorescent lighting 12 hours/d, and with 10-12 room air changes/hour. Animals were randomized to groups by weight class and then to dose groups. Formulated or control diets and water were available ad libitum. The formulated diets were checked for homogeneity and correctness of concentration.

Animals were observed twice per day, weighed initially and then once/week for 12 weeks and monthly thereafter until wk 100 or 101 when observations were performed every 2 weeks. All animals were subjected to a necropsy and histologic examination of tissues. The tissues examined histologically were gross lesions, skin, mandibular lymph nodes, mammary glands, salivary glands, sternum (including bone marrow), thymus, trachea, lungs and bronchi, heart, thyroid gland, parathyroids, esophagus, stomach, pancreas, gallbladder (mice), small intestine, colon, mesenteric lymph nodes, liver, spleen, kidneys, adrenal glands, urinary bladder, prostate/testes or ovaries/uterus, brain, pituitary gland, tissue masses, and regional lymph nodes. Tissues were preserved in 10% neutral buffered formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin.

Organ effects reported in high dose male rats (~2,240 mg/kg/d) at the conclusion of NTP's two year study consisted of thrombosis and degeneration of the liver, fibrosis of the spleen, and lymphoid hyperplasia. Degeneration of the eye was observed in low dose female rats (~1,200 mg/kg/d). This later effect has been correlated with exposure to artificial light due to cage placement, and as a result, long term studies presently incorporate cage rotation into the study design. The DBDPO two-year study was conducted prior to NTP instituting cage rotation as a part of their experimental protocols. In mice, granulomas in the liver of low dose males and hypertrophy in the liver of low (~3,200 mg/kg/d) and high (~6,650 mg/kg/d) dose males were observed. Follicular cell hyperplasia was observed in thyroid glands of dosed male mice. The U.S. NTP concluded " ... effects observed in these studies must be attributed to the approximately 95% pure preparation used rather than to pure decabromodiphenyl oxide" (NTP 1986).

4.1.2.4 30-Day Repeated Dose Study (1973) (Tier I)

An earlier repeated dose study using a DBDPO material of lower (77%) purity, Dow FR-BA-300 (Norris et al. 1973, 1974, 1975), produced somewhat different results from those of NTP which used a test article of ≥ 95% DBDPO (NTP 1986). This DBDPO mixture is no longer manufactured, and has not been manufactured since the mid-1980s.

In a 30-day feeding study 5 male Sprague-Dawley rats/group were administered the DBDPO mixture in the diet at 0, 0.01, 0.1 and 1.0%, which corresponded approximately 0, 8, 80 and 800 mg/kg body weight (Norris et al. 1973, 1974, 1975). No overt signs of toxicity were detected in any dose group. Liver weights were statistically increased in the 1.0 and 0.1 % dose groups compared to the control group. Gross pathologic changes were limited to hepatomegaly in 2 of 5 rats at the 1.0% dose level. Centrilobular cytoplasmic enlargement with minimal vacuolation was observed in 2 of 5 rats at the 1.0% dose level. Thyroid hyperplasia was detected in a non-dose-related manner: in 1 of 5 rats at the 1.0% dose level and in 3 of 5 rats at the 0.1% dose level. Hyaline droplet tubular cytoplasmic changes were detected in the kidneys of 4 of 5 rats at the 1.0% dose level. A dose of 8 mg/kg per day was established as a no-effect level and 80 mg/kg per day as a marginal-effect level. The 77% DBDPO commercial product is no longer manufactured and the results of the 1974 30-day study are not applicable to the ≥ 97% DBDPO commercial product in use today.

4.1.3 Reproductive and Developmental Toxicology (Tiers I and II)

4.1.3.1 One Generation Reproduction Study (1975) (Tier I)

No adverse effects in either parent or F1 offspring were noted in a dietary one-generation reproduction test in male and female Sprague-Dawley rats utilizing doses up to and including 100 mg of a 77% DBDPO mixture (FR-300 BA)/kg body weight (Norris et al. 1975). The test article was composed of 77.4% DBDPO, 21.8% nonabromodiphenyl oxide, and 0.8% octabrmodiphenyl oxide. This DBDPO mixture is no longer manufactured, and has not been manufactured since the mid-1980s.

Groups of male and female rats were maintained on diets containing sufficient test article to provide dose levels of 0, 3, 30 or 100 mg/kg/d for 60 days prior to mating, during mating, and subsequently throughout gestation and lactation. There were 10 males and 20 females at the 2 lower dose levels, and 15 and 30 males and females, respectively at the high dose level. Twenty male and 40 female rats served as controls. The additional males and females were included with the controls and the group receiving the high dose level for tissue analysis for content of DBDPO. After 60 days on the test diet, each male was placed with 2 female from the same treatment regimen for 15 days (3 estrus cycles). After the 15-day mating period, the males and females were separated and maintained on the appropriate treatment diets. The females continued to receive the test diets throughout gestation and for 21 days following parturition. After 21 days of lactation, the females and their young were killed and necropsied. The brain, heart, liver, kidneys, and testes of 10 adult males and females in each group were removed and weighed. Microscopic examination of approximately 30 tissues was performed on 5 animals/sex in the control and high dose groups. Serum chemistries (BUN, alkaline phosphatase, and serum

glutamic pyruvic transaminase) and urinalysis were performed on the control and high dose animals at termination (~ day 120). Sections of brain, liver, kidney, pancreas, spleen, heart, lung, testes/ovaries, adrenal gland, small intestine, large intestine, urinary bladder, and uterus were preserved from one male and one female of each litter for microscopic examination. After gross exam, the remaining weanlings of each litter were prepared for skeletal exam. Bone marrow was saved from 5 male and 5 female adults and weanling animals/dose level at termination of the study for cytogenic evaluation. Statistical evaluation of the indices of reproduction was made by the Fisher exact probability test. Analysis of the neonatal and maternal body weights and organs weights were made by an analysis of variance and the means were compared to control values by Dunnett's test. The level of significance chosen for all was $P<0.05$.

The results of this study indicate that incorporation of the DBDPO mixture in the diet of rats for 60 days prior to mating, and subsequently throughout mating, gestation and lactation had no effect on reproductive parameters. No signs of toxicity were observed in the adult rats or the neonates during the study or at necropsy. Unaffected parameters included body weight gain and food consumption by adults, reproductive parameters (the percent pregnant and neonatal growth, survival and development), pre-terminal urinalyses and clinical chemistry measures in adult rats, gross examination of all adult and weanling animals and microscopic examination of selected tissues from both age groups. Cytogenic aberrations were not detected in bone marrow collected from the femurs of adults or weanlings. Thus, no toxicological manifestations were associated with ingestion of the DBDPO mixture at the highest dose level tested, 100 mg/kg/d.

4.1.3.2 Developmental Toxicity (Tier II)

4.1.3.2.1 Rat Developmental Toxicity Study (2002) (Tier II)

No evidence of maternal or fetal toxicity or developmental effects was detected in a developmental test in the Sprague Dawley rat (CD [Crl:CD(SD)GS BR) (n = 25 pregnant females/dose) at 1,000 mg/kg body weight utilizing a composite of today's commercial DBDPO product produced by three manufacturers and administered from days 0 - 19 of gestation (Hardy et al. 2002 (APPENDIX II); Schroeder 2000). The test article composition was 97.34% DBDPO, 2.66% nona- and octabromodiphenyl oxide congeners. This study was performed according to current EPA and GLP guidelines.

In this study, female rats (25 mated females/group) received 0, 100, 300 or 1,000 mg DBPDO/kg/day via gavage in corn oil from Gestation Day 0-19. All dams survived until scheduled sacrifice. No clinical signs of toxicity were observed. Pregnancy rates in the control and treated groups ranged from 96-100% and provided 23 or more litters in each group for evaluation on Gestation Day 20. No effect of treatment was detected in maternal gestational parameters (body weight, body weight gain and food consumption), uterine implantation data, liver weight or necropsy findings. Likewise, no treatment-related effect was detected in fetal body weights, fetal sex distribution, or from the fetal external, visceral, or skeletal examinations. The NOEL (No Observable Effect Level) for maternal and developmental toxicity was 1,000 mg DBPDO/kg/day, the highest dose level tested.

4.1.3.2.2 Rat Developmental Toxicity Study (1973) (Tier II)

An earlier developmental study, using the former commercial product of only 77% DBDPO purity (Dow FR-BA-300) and administered to female Sprague-Dawley rats (n=20/treatment group and 30/control) on gestation days 6-15 at doses of 0, 10, 100, or 1,000 mg/kg/day, also was negative for maternal toxicity and developmental effects (Norris et al. 1973; 1974; 1975). The test article used by Norris et al., FR-300 BA, was a product composed of 77.4% DBPDO, 21.8% NBDPO, and 0.8% OBDPO, and is no longer manufactured.

No maternal toxicity or mortality was observed, and the mean maternal liver weights of the treated groups were statistically comparable to the control mean. No statistical differences between the control and treated groups were observed for the position and number of fetuses *in utero*, number of corpora lutea/dam, individual pup weight, crown rump ratio, sex ratio, number of litters, implantation sites/litter, live fetuses/litter, litters totally resorbed, or resorptions/litters with resorptions. The numbers of resorptions/implantation sites and the number of litters with resorptions was statistically significantly increased in the treated groups compared to control.

The statistical increase in resorption rate was secondary to an unusually low control value, showed no dose-response relationship, and was comparable to historical control values. Soft tissue variations detected in higher incidence in the 1,000 mg/kg dose group, but not in the 100 or 10 mg/kg groups, compared to control group were subcutaneous edema and delayed ossification of the interparietal bones of the skull.

4.1.4 Genotoxicity (Tiers I and II)

No evidence of a genotoxic effect was detected in the Ames Salmonella, chromosome aberration, mouse lymphoma, or sister chromatid exchange tests (Wagner and Klug 1998; WHO 1994; NTP 1986; McGregor et al. 1988). No cytogenic changes were observed in the bone marrow of rats (parents and offspring) undergoing a one-generation reproduction test using a former DBDPO-commercial mixture of 77% purity (Dow FR-BA-300) (Norris et al. 1975).

4.1.4.1 Ames Test (Tier I)

DBDPO (>98% purity) was tested in the bacterial reverse mutation assay using S. typhimurium tester strains TA98, TA100, TA 1535 and TA 1637 and E. coli tester strain WP2 uvrA in the presence and absence of Arochlor-induced rat liver S9 (Wagner and Klug 1998). The assay was performed in two phases, using the plate incorporation method. The first phase, the preliminary toxicity-mutation assay, was used to establish the dose range for the mutagenicity assay and to provide a preliminary mutagenicity evaluation. The second phase, the mutagenicity assay, was used to evaluate and confirm the mutagenic potential of the test material. Positive controls plated concurrently were 2-aminoantracene, 2-nitrofluorene, sodium azide, 9-aminoacridine, and methyl methanesulfonate.

Dimethyl sulfoxide was selected as the solvent based on solubility of the test article and compatibility with the target cells. Concentrations from 50 to 250 mg/ml were workable suspensions.

In the preliminary assay, the maximum dose tested was 5,000 ug/plate; this dose was achieved using a concentration of 100 mg/ml and a 50 uL plating aliquot. The test article was soluble but cloudy in dimethyl sulfoxide at $\leq$ 3.0 mg/ml and soluble and clear at $\leq$ 0.3 mg/ml. Precipitate was generally observed at $\geq$ 500 ug/plate but no appreciable toxicity was observed. Based on the findings of the toxicity-mutation assay, the maximum dose plated in the mutagenicity assay was 5,000 ug/plate.

In the mutagenicity assay, no positive response was observed (Table 4-2). Precipitate was generally observed at >500 ug/plate but no appreciable toxicity was observed.

Under the conditions of this study, DBDPO was concluded to be negative in the Bacterial Reverse Mutation Assay. This study was conducted according to US EPA and OECD guidelines and Good Laboratory Practices.

Similar results were reported by the U.S. NTP in their own tests (NTP 1986).

TABLE 4-2. DBDPO Ames test results.

S9 Activation	Overall Evaluation[a] and Dose Range Tested (ug/plate)									
	TA98		TA100		TA1535		TA1537		WP2 uvrA	
	Low	High	Low	High	Low	High	Low	High	Low	High
None	-	-	-	-	-	-	-	-	-	-
	15	5000	15	5000	15	5000	15	5000	15	5000
Rat Liver	-	-	-	-	-	-	-	-	-	-
	15	5000	15	5000	15	5000	15	5000	15	5000

[a] - = negative; + = positive (maximum fold increase)

4.1.4.2 Mouse Lymphoma (In excess of Tier III)

DBDPO (the test article used in the NTP 2 carcinogenicity studies) was tested for muagenicity in L5178Y/TK$^{+/-}$ mouse lymphoma cells in the presence and absence of S9 (NTP 1986). Experiments were performed twice, and all doses were tested in duplicate, except the solvent control (DMSO), which was tested in triplicate. Cells (6 x 10^5/ml) were treated for 4 hours at 37 degrees C in medium, washed, resuspended in medium, and incubated fro 48 hrs at 37 $^{\circ}$C. After expression, 3 x 10^6 cells were plated in medium supplemented with trifluorothymidine for selection of cells that were mutant at the thymidine kinase (TK) locus, and 600 cells were plated in nonselective medium to determine the percentage of viable cells. DBDPO did not induce mutations in this mouse lymphoma assay (Tables 4-3 and 4-4).

TABLE 4-3. DBDPO Mouse lymphoma results in presence of S9.

Compound	Dose (ug/ml)	Total Mutant Clones	Cloning Efficiency (%)	Relative Total Growth (%)	Mutation Frequency (mutants/10^6 clonable cells)
DMSO		117	78	100	50
		90	77	100	39
		90	66	100	45
		87	68	100	43
Ethylmethanesulfonate	2.5	544	53	35	344
		474	36	31	437
DBDPO	7	75	57	74	44
		91	53	84	57
	8	58	64	84	30
		97	124	158	26
	9	51	55	70	31
		85	60	76	48
	10	114	83	104	46
		94	52	72	61

TABLE 4-4. DBDPO mouse lymphoma results in absence of S9.

Compound	Dose (ug/ml)	Total Mutant Clones	Cloning Efficiency (%)	Relative Total Growth (%)	Mutation Frequency (mutants/10^6 clonable cells)
DMSO		134	98	100	45
		102	105	100	33
		140	115	100	41
		178	100	100	59
Ethylmethanesulfonate	15	750	57	32	436
		762	70	36	365
DBDPO	7	77	89	87	29
		143	90	88	53
	8	97	81	85	40
		180	118	125	51
	9	49	90	86	35
		130	99	93	44
	10	115	97	91	40
		152	104	99	49

4.1.4.3 *In vitro* Sister-Chromatid Exchange (In excess of Tier III)

DBDPO (the test article used in the NTP 2 year carcinogenicity studies) was tested for the induction of sister-chromatid exchanges in Chinese hamster ovary cells in the presence or absence of S9 (NTP 1986). In the absence of S9, Chinese hamster ovary cells were incubated with DBDPO or solvent for 2 hr at 37 degrees C. BrdU was added, and incubation was continued for 24 hr. Cells were washed, fresh medium containing BrdU (10 uM) and colcemid (0.1 ug/ml) was added, and incubation was continued for 2-3 hrs. Cells were collected by mitotic shake-off, treated for 3 minutes with potassium chloride (75 mM), washed twice with

fixative, dropped onto slides and air dried. Staining was by a modified technique (after Perry and Wolff, 1974; Goto el al., 1978). In the presence of S9, cells were incubated with DBPDO or solvent for 2 hrs at 37 degrees C. Cells were washed, and medium containing 10 uM BrdU was added. Cells were incubated for a further 26 hrs, with colcemid (0.1 ug/ml) for the final 2-3 hrs. S9 was derived from the livers of Arochlor 1254-induced male Sprague-Dawley rats. DBDPO did not induce sister-chromatid exchanges in Chinese Hamster Ovary cells when tested with or without metabolic activation (Table 4-5).

TABLE 4-5. DBDPO sister-chromatid exchange results in Chinese hamster ovary cells.

Without S9		With S9	
Dose (ug/ml)	SCE/cell	Dose (ug/ml)	SCE/Cell
DMSO (10 ul)	8.5	DMSO (10 ul)	9.3
DBDPO		DBDPO	
50	8.1	50	8.6
100	7.9	100	9.3
250	8.1	250	8.4
500	7.6	500	8.8
Mitomycin C		Cyclophosphamide	
0.001	11.1	0.3	12.9
0.01	49	2.0	35.6

4.1.5.4 *In vitro* Chromosome Aberration (Tier I)

DBDPO (the test article used in the NTP 2-year carcinogenicity studies) was tested for induction of chromosome aberrations in Chinese hamster ovary cells with and without metabolic activation (NTP 1986). In the absence of S9, Chinese hamster ovary cells were incubated with DBDPO or solvent of 8-10 hrs at 37 degrees C. Cells were washed and fresh medium containing colcemid (0.1 ug/ml) was added. After a further 2-3 hr incubation, cells were harvested by mitotic shake-off, fixed, and stained with 6% Giemsa. In the presence of S9, cells were incubated with DBDPO or solvent for 2 hrs at 37 degrees C. Cells were washed, medium added and incubation continued for 8-10 hrs. Colcemid (0.1 ug/ml) was added for the last 2-3 hrs of incubation. Cells were harvested and fixed as described for the sister-chromatid exchange test. S9 was derived from the livers of Arochlor 1254-induced male Sprague-Dawley rats. DBDPO did not induce chromosome aberrations in Chinese hamster ovary cells when tested with or without metabolic activation (Table 4-6).

TABLE 4-6. DBDPO chromosome aberration results in Chinese hamster ovary cells.

Without S9		With S9	
Dose (ug/ml)	Abs/100 Cells	Dose (ug/ml)	Abs/ 100 Cells
DMSO (10 ul)	1	DMSO (10 ul)	1
DBDPO		DBDPO	
50	0	50	0
100	0	100	2
250	1	250	0
500	0	500	1
Mitomycin C		Cyclophosphamide	
0.150	16	15	28
0.250	22	30	40

4.1.4.5 *In vivo* Bone Marrow Cytogenetics (Tier II)

No cytogenic changes were observed in the bone marrow of rats (parents and offspring) undergoing a one-generation reproduction test using a former DBDPO-commercial mixture of 77% purity (Dow FR-BA-300) (Norris et al. 1975). This one-generation study was described in the section 4.1.3.1.

In the one-generation study, bone marrow, obtained from the femur, was saved from 5 male and female adults and weanling animals per dose level at termination of the study for cytogenetic evaluation. The DBDPO mixture did not induce cytogenetic aberrations in the treated animals.

4.1.5 Hepatic Enzyme Induction (In excess of Tier III)

Gavage administration of DBDPO (0.1 nmol/kg/day) to rats over 14 days did not induce hepatic cytochrome P450, cytochrome P450 reductase, UDP-glucuronyl-transferase, benzo[a]pyrene hydroxylase, p-nitroanisole demethylase, or EPN detoxification (Carlson 1980).

4.1.6 Chloracne Potential (In excess of Tier III)

Repeated dermal application of DBDPO did not induce a chloracne-like response (Naismith and Matthews 1981; WHO 1994). Chloracnegenic activity was studied by applying the test article on the ear of each of 4 New Zealand White male and female rabbits. The test material (Saytech® 102) was administered once daily at 0.1 ml/d 5 times/wk for 4 weeks, at 1, 10, 100 or 1,000 g/kg in chloroform to rabbits' ears. Observations were recorded prior to the initial dose and at 7, 14, 21 and 28 days of dosing. No chloracne was observed.

Between 1971-1974, pilot plant samples of DBDPO were studied for chloracnegenic activity. The samples (0.1 ml) were applied as a 5 or 10% solution in chloroform on the rabbit ear 5 days per week for 4 weeks. No chloracne was observed (WHO 1994).

The soot and char combustion products from a high impact polystyrene/DBDPO/antimony trioxide matrix also did not induce a chloracne-like response (Pinkerton et al. 1989). Soot and char generated from the combustion of high impact polystyrene flame retarded with and without DBDPO and antimony trioxide were tested in New Zealand rabbits. The dose levels were 0.001, 0.003, 0.005, 0.008, 0.01, 0.3, 0.05, 0.08 and 0.1 grams. The materials were applied in 0.1 ml of water for 5 consecutive levels. Ears were examined on day –1, 0, and daily during dosing and one day post-dosing. Erythema was observed in the nonflame retarded and flame retarded groups.

Two groups of four male and female New Zealand White rabbits were used to test a mixture of soot and char generated from the combustion of high impact polystyrene or high impact polystyrene/DBDPO/antimony trioxide resin in a rabbit ear comedogenicity bioassay. Daily doses of 2, 5, 8, 20 or 50 mg were administered. Each daily dose was rubbed with 0.1 ml of water on the inner surface of the pinna of one ear of each rabbit. The animals were dosed 5 days per week for a total of 4 weeks. The total cumulative dose levels were 40, 100, 160, 400 and

1,000 mg. The ears were graded for irritation (Draize test) and for hyperkeratosis (Adams test). Dermal irritation was observed in all groups. No comedogenic responses were observed. A slight increase in hyperkeratosis of the sebaceous follicles was observed on histopathological examination of the skin at the 2 highest dose levels of the high impact polystyrene groups. No evidence of overt toxicity was seen, and results from the high impact polystyrene/DBDPO/antimony trioxide soot and char groups were comparable.

4.1.7 Carcinogenicity (Tier III)

Two two-year carcinogenicity bioassays have been conducted on DBDPO (Kociba et al. 1975; NTP 1986).

The first, a single species study performed at a top dose level of 1 mg/kg using a DBDPO material of only 77% purity, produced no evidence of carcinogenicity or toxicity in rats (Kociba et al. 1975).

The second, conducted at 2.5 and 5% of the diet in F344/N rats and $B6C3F_1$ mice using a DBDPO material more closely resembling today's commercial product, produced no, equivocal and some evidence of carcinogenicity depending on genus and sex (NTP 1986).

4.1.7.1 Two Year Carcinogenicity Studies in Rats (1975) (Tier III)

Groups of 25 male and 25 female Sprague-Dawley rats were fed 0, 0.01, 0.1 or 1 mg/kg body weight/day of a DBDPO-mixture (Dow FR BA-300: DBDPO 77.4%, NBDPO 21.8%, OBDPO 0.8%) in the diet for 100 to 105 weeks. Ingestion of up to 1 mg/kg/day of the DBDPO mixture did not influence survival rates; appearance, mean body weights, feed consumption, hematology, urinalysis, clinical chemistry (blood urea nitrogen, alkaline phosphatase and glutamic pyruvic transaminase activities) and organ weights of treated groups were similar to those of controls. Gross and microscopic examinations performed on all rats killed or dying during the course of the study, did not reveal any significant finding, all the observed changes or variations from normal occurred with similar frequency and severity in the treated and control groups of rats. All these changes were considered spontaneous in nature and unrelated to ingestion of the test article. No significant difference in the number of rats developing tumours, the total number of tumours or the specific type of tumours was observed between treated and control groups (Kociba et al. 1975).

4.1.7.2 U.S. NTP Two Year Carcinogenicity Study in Rats and Mice (1986) (Tier III)

Three groups of F344/N rats (n=50 rats/sex/dose) and $B6C3F_1$ mice (n=50 mice/sex/dose) were fed diets containing 0, 2.5% or 5% DBDPO for 2 years. The test article consisted of two lots of DBDPO that were of 96% and 94-97% pure, respectively. Doses up to 5% of the diet for two years were well tolerated by F344/N rats and $B6C3F_1$ mice with no effect on body weight or mortality and only minimal evidence of organ effects (NTP 1986). The U.S. National Toxicology Program (NTP) estimated the average amount of DBDPO consumed per day in the two year

study was 1,120 mg/kg and 2,240 mg/kg for low and high dose male rats, respectively, and 1,200 mg/kg and 2,550 mg/kg for low and high dose female rats, respectively. Likewise, NTP estimated the average DPDPO consumed per day by mice in the two year study was 3,200 and 6,650 mg/kg for low and high dose male mice, respectively, and 3,760 and 7,780 mg/kg for low and high dose female mice, respectively.

No evidence of carcinogenicity was observed in female mice receiving 2.5 or 5% DBDPO in the diet (~3,760 or 7,780 mg/kg/d). Equivocal evidence of carcinogenicity was observed in male mice by an increase in the combined incidence of hepatocellular adenomas or carcinomas in both dose groups (~3,200 or 6,650 mg/kg/d); however, this finding may have been influenced by the larger number of early deaths in control male mice compared to the treated male mice. The large number of early deaths in the control males may have decreased expression of hepatocellular adenomas or carcinomas in this group. The combined incidence of hepatocellular adenomas and carcinomas in male mice treated with DBDPO was well within the historical range.

Some evidence of carcinogenicity in male and female rats was observed by increased incidences of neoplastic nodules of the liver in low dose (2.5%, ~1,120 mg/kg/d) males and high (5%, ~2,240 mg/kg/d - males, ~2,550 mg/kg/d - females) dose groups of each sex. (The term "neoplastic nodule" is no longer used by NTP to describe hepatoproliferative lesions in rats. This change in nomenclature was made subsequent to a peer review of representative hepatoproliferative lesions from two-year carcinogenicity studies. The peer review found the use of this poorly defined and understood term had permitted some potentially useful drugs and chemicals to be unfairly categorized as carcinogens (Maronpot et al., 1986). DBDPO is not listed as a carcinogen by NTP (NTP 2001), the International Agency for Research on Cancer (IARC 1990) or the U.S. Occupational Safety and Health Administration (OSHA 1990).

The abstract from the NTP final report on this study is attached in Appendix V.

4.1.8 DBDPO Absorption, Distribution, Metabolism, Elimination (Tier II)

The uptake, distribution and elimination of DBDPO after oral or intravenous (IV) dosing in the rat have been evaluated in several studies (NTP 1986; Norris et al. 1973, 1974; El Dareer et al. 1987; Moreck and Klassen-Wheler 2001). These processes were monitored by following total ^{14}C-radioactivity after administration of labeled-DBDPO or by following total bromine content via neutron activation after administration of DBDPO. NTP evaluated the uptake and disposition of DBDPO in the rat as part of the two-year bioassay. Four studies were performed and the results were reported in the 1986 NTP report (NTP, 1986) and in the publication of El Dareer et al. (1987). Earlier studies are reported in Norris et al. (1974, 1975). Similar work was recently performed by Morck and Klassen Wheeler (2001).

In the dietary NTP-sponsored studies conducted by El Dareer et al. (1987; NTP 1986), DBDPO treatment for 7 days at varying dose levels preceded treatment with the radiolabeled compound. Pretreatment dose levels were 51,000, 25,400, 4,730,2,510, 496 and 238 ppm in the diet. Test articles used for pretreatment in the ^{14}C-DBDPO studies (NTP 1986; El Dareer et al. 1987) closely resembled today's commercial product which is ≥ 97% DBDPO. In the studies conducted

by Norris et al. (1973; 1975), a single dose of ^{14}C-DBDPO was administered orally or bromine tissue levels were monitored by neutron activation after repeated administration of DBDPO for 3, 6 or 12 months. The test article for the neutron activation experiments was the former low purity product "Dow FR-300-BA" composed of 77.4% DBDPO, 21.8% nonabromodiphenyl oxide and 0.8% octabromodiphenyl oxide. In the Morck and Kassen Wheeler (2001) study, ^{14}C-DBDPO was synthesized in the laboratory.

All studies showed similar results. The NTP studies by El Dareer et al. (NTP 1986; El Dareer et al. 1987) showed that DBDPO was poorly absorbed (2-0.28% of the oral dose) from the gastrointestinal tract at all pretreatment doses (277-50,000 ppm in the diet, respectively) and rapidly eliminated. The whole body half-life was < 24 hr. Excretion in the urine accounted for ≤ ~0.01% of the dose. Feces was the major route of elimination and > 99% of the dose was recovered in the feces by 72 hr post-dosing. At all oral doses tested (277 - 50,000 ppm in the diet), the majority of the test article (~98 - 70%, respectively) was eliminated as the parent molecule. Three metabolites were detected in the feces and ranged from ~2 to 30%, respectively, of the total recovered ^{14}C-label. The highest percentage of metabolites (~30% of the dose) was present in the feces of animals pretreated with higher doses of DBDPO (25,000 and 50,000 ppm) in the diet. The lowest percentage of metabolites (~2% of the dose) was present in the feces of animals pretreated with lower levels of DBDPO (277 ppm). The identity of the metabolites was not determined.

Only trace levels of the ^{14}C-label were detected in any organ or tissue at any time point (24, 48 or 72 hr post dosing with the radiolabel) (NTP 1986; El Dareer et al. 1987). The maximum total ^{14}C-activity detected in the body at any time was only ~1% of the oral dose. The maximum ^{14}C-activity, calculated as the sum of the radioactivity in liver, kidneys, lungs, spleen, brain, muscle, skin, fat, and blood, was detected in the 277 ppm treatment group 24 hr post-dosing. Studies utilizing intravenous (IV) administration of 1 mg ^{14}C-DBDPO/kg and bile duct canulation showed that the ^{14}C-label was excreted in the bile as the parent molecule and 3 metabolites. Approximately 60% of the dose was eliminated as metabolites after IV administration. The bile contained 7.17% of the IV dose within 4 hr post-doing, and 2.2% of the dose was excreted in the bile per hr.

The above results are consistent with earlier reports by Norris et al. (1973, 1975). Norris et al. (1975) administered 1 mg/kg ^{14}C-DBDPO orally to 3 male and 3 female rats. The level of radioactivity found in the expired air and urine, measured at 24 hr intervals over a 16-day period, was < 1 %. The principal route of excretion was the feces. The rate of excretion was the same for both sexes. Within the first 24 hr post-dosing, 90.6% of the administered dose was detected in the feces, and 99% of the ^{14}C-activity was accounted for by day 2. Tissues (adipose, heart, skin, adrenals, spleen, liver, pancreas) taken on day 16 post-dosing showed no ^{14}C-label with the exception of the adrenal (0.01% of the dose) and spleen (0.06% of the dose). The ^{14}C-activity in these two tissues was at the limit of detection. The half-life of the disappearance of ^{14}C-activity from the body of DBDPO-treated rats was < 24 hours.

Norris et al. (1973, 1975) also measured bromine concentrations (via neutron activation analysis) in the kidney, skeletal muscle, serum testes, liver and adipose tissue in male and female rats

maintained on diets providing 1, 0.1, 0.01 and 0 mg DBDPO mixture/kg/day for 6 or 12 months. The composition of the DBDPO mixture (Dow FR-300-BA) was 77.4% DBDPO, 21.8% nonabromodiphenyl oxide and 0.8% octabromodiphenyl oxide. After 180 days of treatment, mean bromine levels in the control and treatment groups in liver, kidney, skeletal muscle, serum and testes were statistically comparable. The mean bromine level in adipose tissue from the 0.1 mg/kg/day dose group (~3.3 ug/g) was statistically greater than the control mean (~1.7 ug/g). After 12 months on treatment, bromine concentrations in both the liver and adipose tissue were statistically comparable to controls.

Norris et al. (1975) evaluated the elimination of bromine from liver and adipose tissue. Male rats were maintained for 90 days on diets providing a dose of 1 mg DBDPO mixture (Dow FR-300-BA)/kg/day and then placed on control diet. Kidney, serum, adipose tissue, and liver were analyzed for bromine by neutron activation analysis. On recovery day 0 there was no difference in bromine content in kidney or serum between the control and treated rats. After 10 days on the control diet, bromine concentrations in the liver of treated rats were comparable to controls. Adipose bromine levels in the treated group (~2.5 - 4 ug/g) were higher than the controls (~0 - 2 ug/g) during the recovery period.

Morck and Klassen Wehler (2001) reported similar results. Male rats were gavaged with a single dose of ^{14}C-DBDPO (3 umol/kg; 0.00288 ug/kg). Feces were the predominant excretory route and contained ~90% of the dose within 3 days. Only trace amounts were eliminated in the urine (<0.5% of the dose). Approximately 9.5% of the dose was recovered in the bile within 3 days. Approximately 3% of the dose remained in tissues at 72 hr post-dosing. The majority of the ^{14}C-activity was detected in the liver followed in declining amount in the muscle, skin, adipose tissue and colon wall plus contents. Eight phenolic metabolites were reported in the feces, and included di-substituted penta- to octaBDPOs. Trace amounts of 3 nona-BDDPOs were also reported.

Based on the findings of NTP, El Dareer et al. and Norris et al. (NTP 1986; Norris et al. 1973, 1975; El Dareer et al. 1987), DBDPO is poorly absorbed from the gastrointestinal tract as would be expected for a molecule of this size, weight and poor solubility. Following oral administration of ^{14}C-DBDPO, only trace levels of radioactivity were found in organs/tissues at any time point. The parent molecule (and all metabolites) was rapidly eliminated - > 99% of the dose was recovered in the feces and gut contents within 72 hours of oral dosing. The overwhelming route and form of elimination was by fecal excretion as the parent molecule. Less than 0.01% of the oral dose was excreted in the urine. DBDPO was capable of being metabolized; the parent molecule and 3 metabolites were detected in feces following oral or IV dosing of rats. The lower the dietary dose the lower the percent eliminated as metabolites, e.g. at a pretreatment dose of 277 ppm in the feed, approximately 2% of the dose was eliminated as metabolites. Recent studies by Morck and Klassen Wheeler (2001) performed at a substantially lower dose reported similar findings to that of NTP and El Dareer.

4.1.9 Immunotoxicology (Tier II)

DBDPO has not been evaluated for immunotoxicity using the OPTS 870.7800 guideline that is intended to provide information on suppression of the immune system that might occur as a result of repeated exposure to a test chemical. However, data available from long-term studies conducted in two species at high doses indicate DBDPO is not immunotoxic. DBDPO at 2.5 and 5% of the diet and administered for two years to rats and mice did not affect mortality or body weight (NTP 1986). If DBDPO was toxic to the immune system, deaths, decreased body weights and histologic evidence of infections would be expected. This was not the case. Routine histopathology of organs/tissues of the immune system also provide no evidence of toxicity. Organs of the immune system examined histologically in the NTP studies were the mandibular lymph nodes, sternum including bone marrow, mesenteric lymph nodes, spleen, and regional lymph nodes. Complete blood counts in the 30-D and 2-year studies (Norris et al. 1973, 1974; Kociba et al. 1975) were considered normal, and no histologic evidence of immunotoxicity was observed in the mesenteric and thoracic lymph nodes or sternal bone marrow. DBDPO's poor bioavailability reinforces a low potential for an adverse effect on the immune system. No additional testing on this endpoint is proposed.

4.1.10 Neurotoxicity Screening Battery (Tier III)

The neurotoxicity screening battery (OPPTS 870.6200) consists of a functional observational battery, motor activity, and neuropathology. The functional observational battery consists of noninvasive procedures designed to detect gross functional deficits in animals and to better quantify behavioral or neurological effects detected in other studies. The motor activity test uses an automated device that measures the level of activity of an individual animal. The neuropathological techniques are designed to provide data to detect and characterize histopathological changes in the central and peripheral nervous system. This battery is designed to be used in conjunction with general toxicity studies and changes should be evaluated in the context of both the concordance between functional neurological and neuropatholgical effects, and with respect to any other toxicological effects seen. This test battery is not intended to provide a complete evaluation of neurotoxicity, and additional functional and morphological evaluation may be necessary to assess completely the neurotoxic potential of a chemical.

DBDPO has not been specifically tested according to OPPTS 870.6200. However, no indication of neurotoxicity was observed in the NTP lifetime studies in rats and mice at exceptionally high doses (2.5 and 5% of the diet for two years) or in any of the other tests performed on DBDPO. These studies all included frequent observations for clinical signs of toxicity or effects on behavior that are essential components of the functional observational battery. Histopathology of the nervous system was normal in all studies.

Considering the high doses administered in the NTP 14-D, 13-Wk and 2-Yr studies to two species, ample opportunity was provided for induction and/or development of neurotoxicity. The fact that no evidence was detected indicates DBDPO is not neurotoxic. DBDPO's poor bioavailability reinforces a low potential for an adverse effect on the nervous system. No additional testing on this endpoint is proposed.

4.1.11 Developmental Neurotoxicity (Tier III)

The developmental neurotoxicity study (OPPTS 870.6300) is designed to develop data on the potential functional and morphological hazards to the nervous system that may arise in the offspring from exposure of the mother during pregnancy and lactation. The test substance is administered to several groups of pregnant animals during gestation and early lactation, one dose level being used per group. Offspring are randomly selected from within litters for neurotoxicity evaluation. The evaluation includes observations to detect gross neurologic and behavioral abnormalities, determination of motor activity, response to auditory startle, assessment of learning, neuropathological evaluation, and brain weights. This protocol may be used as a separate study, as a follow-up to a standard developmental toxicity and/or adult neurotoxicity study, or as part of a two-generation reproduction study, with assessment of the offspring conducted on the second (F2) generation. Testing should be performed in the rat. Because of its differences in timing of developmental events compared to strains that are more commonly tested in other developmental and reproductive toxicity studies, it is preferred that the Fischer 344 strain not be used. If a sponsor wishes to use the Fischer 344 rat or a mammalian species other than the rat, ample justification reasoning for this selection must be provided.

While DBDPO has not undergone testing via OPPTS 870.6300, none of the repeated dose toxicology studies, including those administering DBDPO over the animals' lifetime, indicate an impact on the nervous system or on the developing embryo/fetus. The NOEL of DBDPO in a rat developmental toxicity study was 1,000 mg/kg/d administered on gestation days 0-19 (Hardy et al. 2002).

A non-guideline developmental neurotoxicity study of DBDPO in the mouse was briefly reported in 2001. DBDPO was reported to disrupt habituation in adult mice which were exposed on postnatal day 3 to a single oral dose of 20.1 mg lab-synthesized DBDPO/kg (Viberg et al. 2001; Appendix IV). Animals exposed on neonatal day 3 to 2.3 mg/kg were not similarly affected nor were animals treated with either dose on neonatal day 19 or on neonatal day 10 with 1.34, 13.4 or 20.1 mg/kg. No data was reported in the 4-page abstract, and much of the details relating to the performance of the study were not reported. The composition of the test article was not specified. According to the human health portion of the draft DBDPO EU risk assessment, the toxicological significance of these findings is unclear. The authors declined to provide data or specific details under the EU risk assessment process.

The neonatal mouse study was performed using an experimental design developed by P. Eriksson (Uppsala University, Sweden), and reported by Proff. Eriksson's graduate student. The design is not that typically used to investigate developmental neurotoxicity (e.g. is not equivalent to OPPTS 870.6300), and appears to be used exclusively in that laboratory. The probability is very low that DBDPO would produce an adverse effect in humans because of the very high dose administered in the Viberg et al. study, the lengthy exposure period required to cover a corresponding period in humans, DBDPO's poor oral absorption (less than 2% in the rat), rapid elimination (>99 % after 72 hours with a half-life less than 24 hours), poor solubility, and lack of bioaccumulation. These concepts are more fully developed below.

In several publications (Eriksson 1992; Eriksson and Talts, 2000; Eriksson 1997), Eriksson cites Davison and Dobbing (1968) as the source of information regarding the brain growth spurt, a key concept in the postnatal timing of dose administration in Eriksson's design. Davison and Dobbing (1968) state that the brain growth spurt occurs after birth in rats and mice, is almost complete at birth in guinea pigs, and occurs prior to birth in humans and primates: "The main fact which emerges is the very different timing of the brain growth spurt in relation to birth in different species, and it follows from this that such expression as 'foetal brain' or 'neo-natal' or 'post-natal brain' are quite meaningless unless one knows both the species being considered and the growth characteristics of its brain. Such an observation, which seems almost too obvious to mention, is very frequently ignored when interspecies extrapolations are being considered, especially when these are between man and other species." Eriksson and Talts (2000) state "The BGS does not take place at the same time point in all mammalian species. In the human, this period begins during the third trimester of pregnancy and continues throughout the first 2 years of life. In mouse and rat the BGS is neonatal, spanning the first 3-4 weeks of life."

Based on the timing of brain growth in humans, exposure would have occur during the last trimester of pregnancy and be followed by continued exposure during the first 2 years of the child's life in order to mimic exposure on neonatal mouse day 10. Assuming equal susceptibility in the child and mouse, absorption between 0.3-2% of an oral dose, and 100% transfer of the absorbed dose to the fetus, a 50 kg woman have to receive a total dose of 50 to 1,000 mg DBDPO every day during the later stages of pregnancy followed by additional exposure to the child during the first two years of its life to reach a dose equivalent to that administered to neonatal mice.

A similar calculation can be made with respect to mice. In terms of the dose a lactating mouse would have to receive in order to pass on an equivalent dose to her nursing offspring, neonatal day 3 is of interest with respect to Viberg's findings. On day 3 of life, the pup's total nutrition is received via nursing. Therefore, oral exposure to the pup at this age would be via milk. However, DBDPO's high molecular weight, its physical/chemical properties, and its pharmacokinetics, make it highly unlikely that DBDPO would be eliminated in the milk (see Section 5.3.7). Therefore, neonatal exposure via this route is not expected. Nonetheless, doses that a lactating mouse would have to receive in order to transmit in her milk doses equivalent to Viberg's are estimated below. The following conservative assumptions were used in calculating the dose received:

Weight of the female mouse = 20 g,
Weight of the day 3 neonate = 2.5 g based on an average birth weight of 1.5 g,
6 pups/litter (average litters range from 1-12 pups; Viberg did not provide the number pups/litter),
Female mouse produces 10 % of her body weight/day in milk,
3% absorption of an oral DBDPO dose by the lactating mouse,
100% transfer of the dose to milk and 100% absorption of the dose by the pup.

Based on these assumptions, each pup would consume 0.33 g of milk, and the 2.2 and 20.1 mg/kg dose administered to the day 3 neonates would be equivalent to a total dose of 0.005 or 0.05 mg/pup, respectively. To achieve a total dose of 0.005 or 0.5 mg, the milk would have to

contain 0.015 or 0.15 mg/g milk. The total day's milk production (2 g) would thus contain 0.03 or 0.3 mg total. Assuming the dam absorbed 3% of an oral dose, she would have to be exposed to doses of 50 or 500 mg/kg body weight in order to generate the estimated milk content. To achieve a dose of this amount, the dam would have to be exposed to 415.9 or 4,159 mg DBDPO/kg food. It is highly unlikely that lactating female mouse (or another mammalian species) could be exposed to a dose of 415.9 or 4,159 mg DBDPO/kg food except under laboratory conditions.

Using the results of the NTP mouse 2-year study, a dose of 25,000 ppm food, and assessment factor of 100, the oral predicted no effect concentration for a lifetime exposure would be 250 mg/kg food. The food exposure to a female mouse, 415.9 or 4,159 mg DBDPO/kg food, in order to generate doses in a day 3 neonate equivalent to those administered by Viberg (2001), are higher than the oral predicted no effect concentration calculated from the NTP two year mouse study.

4.2 Environmental Fate and Toxicology (Not included in Tiers I, II, or III)

4.2.1 Environmental Fate

DBDPO's measured and predicted environmental fate parameters are shown in Table 4-7. DBDPO is predicted to partition in the environment to soil and sediment (~99%) where it will bind extensively to organic carbon (estimated Koc_{soil} = 1.67 x 10^{12}) and to be essentially immobile in soil. Based on a release of 1,000 kg/hr to air, water and soil, the predicted partitioning is: air 0.12%, water 1.09%, soil 41.8% and sediment 57% (Level III Fugacity Model, EPIwin V3.04). DBDPO is not expected to volatilize from water based on its river and lake volatilization half-lives and air-water partition coefficient. DBDPO is expected to partition from water to organic carbon. Sewage treatment plants are predicted to remove DBDPO from the influent to a high degree (94%), but biodegradation in the treatment plant is not expected. Removal in the treatment plant is via partitioning to sludge. DBDPO leaching from polymers was insignificant (Norris et al., 1973,1974) as expected for a molecule of negligible water solubility and vapor pressure. DBDPO is not expected to undergo long range transport (Wania and Dugani 2002).

TABLE 4-7. Environmental fate parameters for DBDPO.

Property	Method	Result
Predicted Movement in the Environment: Expected to partition to sediment and soil and be essentially immobile.		
Water Solubility	Measured	< 0.1 ug/L
Vapor Pressure	Measured	4.63 x 10-6 Pa
Henry's Law Constant	Estimated	1.9 x 10-8 atm-m3/mole at 25°C (EPIwin, V.3.04)
		7.9 x 10-7 unitless at 25°C (EPIwin, V.3.04)
Soil Koc	Estimated	1.8 x 10+6 (EPIwin, V.3.04)
Log Octanol Water Partition Coefficient	Estimated	12.61 (EPIwin, V.3.04)
	Measured*	5.625 (MacGregor and Nixon 1997)
Air to Water Partition Coefficient	Estimated	7.9 x 10-7 (EPIwin, V.3.04)
Biomass to Water Partition Coefficient	Estimated	8.1 x 10+11 (EPIwin, V.3.04)
Volatilization from Water	Estimated	Half life: 10.7 years (river), 117 years (lake) (EPIwin, V.3.04)
Sewage Treatment Plant Fugacity Model	Estimated	Total Removal: 94%, Total Biodegradation: 0.78%, Primary Sludge: 60%, Waste Sludge: 33%; Final Water Effluent: 6% (EPIwin, V.3.04)
Level III Fugacity Model	Estimated	At Emissions to Air, Water, Soil and Sediment of 1,000, 1,000, 1,000 and 0 kg/hr, respectively (EPIwin, V.3.04):
		Distribution: Air 0.12%, Water 1.09%, Soil 42%, Sediment 57%
		Fugacity (atm): 3.1 x 10-16, Water 8.4 x 10-21, Soil 2.4 x 10-22, Sediment 1.5 x10-20.
		Reaction (kg/hr): Air 4.2, Water 43.4, Soil 1.7 x10+3, Sediment 570.
		Advection (kg/hr): Air 249, Water 226, Soil 0, Sediment 237.
		Reaction (%): Air 0.1, Water 1.5, Soil 56, Sediment 19.
		Advection (%): Air 8, Water 7.5, Soil 0, Sediment 8.
Long Range Transport Potential	Computer Modeling	Not expected to undergo long range transport (Wania and Dugani 2002)
Biodegradation: No evidence of biodegradation.		
Ready Biodegradation	MITI	Not readily degradable in a 2 week study (CITI 1992)

Sludge Respiration	OECD 209; EU67/548;EEC, Annex V, C.11; GLP	Not inhibitory to activated sewage sludge (limit dose = 15 mg/L) (Schaefer and Siddiqui 2001)
Anaerobic Sediment Degradation	Other; GLP* Other	Not degraded after 32 weeks (Schaeffer and Flaggs 2001) Not degraded after 2 years (de Wit 2000)

Abiotic degradation: Not likely a significant route of environmental degradation due to negligible vapor pressure & water solubility and expected environmental partitioning.

Aqueous Photodegradation	Other**	Half-life >> 90 days; Products not lower BDPOs (Norris et al. 1974, 1975)
Organic Solvent Photodegradation	Other	Half-life < 15 minutes; Sequential reductive debromination; PBDFs formed from degradants (Norris et al. 1974, 1975; Watanabe and Tatsukawa 1987; Eriksson et al. 2001)
Solid Surface Photodegradation	Other*	6 different exposure scenarios investigated; Less than 10% of the DBDPO decayed in a worst-case exposure scenario for inducing solar photochemical transformation in a model aqueous environment (e.g. DBDPO precipitated on humic acid-coated sand particles & exposed to 12 days of summer sunlight); No evidence for production of Tetra or PeBDPO congeners (Jafvert and Hua 2001)
	Other	Toluene half-life< 15 minutes; Sand half-life ~35 hr (rooftop sunlight); Sediment half-life ~ 100 hr (rooftop sunlight); Soil half-life ~200 hrs (rooftop sunlight); Some evidence of sequential reductive debromination but not as pronounced in sand/sediment/soil as in organic solvents; No evidence of 2,2',4,4'-TeBDPO formation (Sellstrom et al 1998; Tysklind et al. 2001)
Hydrolysis	Estimated	Not likely to be a significant route of environmental degradation due to low water solubility
Atmospheric Oxidation	Estimated	Overall OH Rate Constant = 0.6 x 10-12 cm3/molecule-sec; Half-Life = 169 Days (12-hr day; 1.5 x 10+6 OH/cm3 (EPIwin V3.04)

*Studies Performed under Good Laboratory Practices and using today's commercial DBDPO product (≥97%) as test article.
**Test article only 77% DBDPO.

4.2.1.1 Abiotic Degradation

Abiotic degradation may occur via hydrolysis or photolysis. DBDPO is not expected to undergo hydrolysis based on its chemical structure. DBDPO's negligible water solubility (< 0.1 ug/L) also does not lend hydrolysis to being a significant route of environmental degradation. Photodegradation requires exposure of the molecule to light, and is not expected to be a significant route of environmental degradation due to DBDPO's negligible vapor pressure that precludes substantial levels in air. Further, a Level III fugacity model predicts that only minimal amounts would partition to air. Nonetheless, questions regarding DBDPO's potential to undergo photolysis to lower brominated diphenyl oxides have been raised.

Norris et al. (1973, 1975) investigated the photolysis of DBDPO by sunlight in organic solvent or water (Norris et al. 1974, 1975), and predicted different routes/mechanisms of photodegradation for the DBDPO molecule in water or organic solvents based on the behavior of other halogenated aromatic compounds. Norris et al. found that halogenated aromatics photodegraded by reductive dehalogenation when dissolved in solvents capable of proton transfer. However, in water, photodegradation proceeded via an oxidative process of hydroxylation leading to the formation of phenolic compounds. Once photohydroxylation was initiated, its rate was expected to accelerate as electron-withdrawing halogens were replaced by electron releasing hydroxyl groups. The resulting hydroxylated species were expected to adsorb light more strongly and this ultimately could result in rupture of the aromatic ring.

Norris et al.'s laboratory findings correlated with the predictions. Minimal evidence of DBDPO (98% purity) aqueous photodegradation was found over a 3-month exposure to natural sunlight; degradants were not lower brominated diphenyl oxides. Evidence for degradation of only 0.57% of the amount initially present (10 g/8 l water) was detected after 98 days of exposure to sunlight. The minimal degradation was likely related to DBDPO 's extremely poor water solubility (<0.l ug/L) and its stability. However, Norris et al. also found that DBDPO (7 ppm) in octanol decomposed with a half-life of 4 h. In xylene (a strong absorber of UV light), DBDPO photodegraded by reductive debromination with a half-life of 15 h on exposure to a 125 watt Hg lamp. In comparison, neither Arochlor 1242 nor 1260 showed any evidence of degradation after 350 h.

DBDPO degradation via reductive debromination in organic solvents (hexane, toluene, methanol/water) to lower brominated diphenyl oxides was also reported by Wantanabe and Tatsukawa (1987) and Eriksson et al. (2001). A further stepwise formation of polybrominated dibenzofurans was also observed (Watanabe and Tatsukawa, 1987; Eriksson et al. 2001). However, organic solvent photodegradation of DBDPO is not anticipated to be an environmentally relevant degradation mechanism (WHO 1994; Existing Substances Regulation 793/93/EEC, 2000a).

The potential photodegradation of DBDPO adsorbed to sand, soil or sediment was also investigated (test article composition unknown but contained nonaBDPO and trace levels of octaBDPO) (Sellstrom et al. 1998). Sellstrom et al. reported a DBDPO half-life in sand of 37 h in natural sunlight. Evidence of reductive debromination was reported. However, the amounts

of nona-, octa- and heptaBDPO were not nearly as pronounced as in the toluene experiments carried out by this group. No 2,2',4,4'–TeBDPO was detected. Although small amounts of nonaBDPO formed, octaBDPO was a small fraction of this and heptaBDPO a small fraction of this. These results indicate that either a stepwise reductive debromination pathway was less significant in environmental media (e.g. sand, soil or sediment) or that in these media the lower brominated products themselves degrade at a faster rate than in toluene. Thus, although it appears possible for reductive debromination of DBDPO to occur under certain circumstances, the amounts of lower brominated diphenyl oxides formed would be very small and would also undergo similar degradation. Further, 2,2',4,4'-TeBDPO, the primary PBDPO detected in the environment, does not appear to be produced from DBDPO.

4.2.1.2 Biodegradation

DBDPO was not readily biodegradable (CITI 1992) nor was DBDPO degraded by anaerobic sediment over a 32 week (Schroeder 2001) or 2 year time frame (de Wit 2000).

Aerobic Biodegradation. DBDPO (100 mg/l) was incubated with activated sludge (30 mg/l) from mixed sources in Japan over a 2-week period (equivalent to MITI I test). No degradation (as measured by BOD) was observed; therefore DBDPO is not readily biodegradable (CITI, 1992). This result indicates that DBPDO is unlikely to biodegrade rapidly in the environment under aerobic conditions.

Anaerobic Biodegradation. Based on other halogenated aromatic substances, reductive dehalogenation of DBDPO may possibly occur under some conditions and anaerobic degradation studies were performed DBDPO and 2,2',4,4'-TetraBDE

KEMI (1999) and de Wit (2000) reported that no degradation/transformation of DBDPO was seen after four months incubation in sediment samples under anaerobic conditions. The inoculum used was an enrichment culture from a polybrominated diphenyl oxide-contaminated sediment. The incubation of one of the anaerobic cultures was extended to two years, but no degradation of decabromodiphenyl ether was seen. De Wit (2002) stated, "A study of anaerobic microorganisms' ability to break down DeBDE to lower brominated PBDE in sediment was carried out during 1994. DeBDE was applied to anaerobic sediment which was then inoculated with micro-organisms enriched from a PBDE-contaminated sediment. The sediment was then divided into smaller samples and allowed to gently shake. Samples were analyzed at different time points but no breakdown of DeBDE was seen during the experimental time of four months. The experiment was extended by letting one aliquot of sediment continue incubation. Subsamples were analyzed at several time points but no breakdown could be seen after an incubation of 2 years (unpublished results, Ulla Sellström; de Wit,1995; 1997)."

The anaerobic biodegradation of ^{14}C-DBDPO was also studied in a sediment-water system over 32 weeks at 5 or 500 mg/kg sediment (Schaefer and Flaggs, 2001a). ^{14}C-Glucose served as a positive control. The test article was a mixture of unlabelled substance (supplied as a composite sample from three manufacturers; purity 97.4% DBDPO, 2.5% NonaBDPO and 0.04%

OctaBDPO) with ^{14}C-DBDPO (radiochemical purity 96.8%). This study was conducted according to Good Laboratory Practices.

The sediment and accompanying overlying surface water used was collected from the Schuykill River, Valley Forge, Pennsylvania, USA. The redox potential of the sediment was -284 mV. The average moisture content of the sediment was 26%, its pH was 6.3, and the organic matter content was 1.4%. A 0.2 mg/l resazurin solution was prepared using the collected overlying surface water.

The test chambers consisted of 500 ml bottles containing 300 ml of the sediment and were prepared in an anaerobic chamber. The sediment was carefully added to the bottles in order to maintain the sediment column structure. Three replicate chambers were used at each concentration. In addition, a further six treatment groups at 5 mg/kg and 500 mg/kg were run to allow the concentrations of the test material and any metabolites to be determined at the start and end of the test. The test chambers were incubated in the dark at ambient room temperature (22^{o}C) in an anaerobic chamber. At the end of the incubation period, samples from each treatment group were analysed for DBDPO and the presence of any degradation products by a HPLC method using both UV and radiometric detection.

The mass balance results from the experiment are shown in Table 4-8.

TABLE 4-8. Mass balance results from a 32-week anaerobic sediment degradation of ^{14}C-DBDPO.

Nominal concentration	Mass balance at week 32			
	% as $^{14}CO_2$	% as $^{14}CH_4$	% ^{14}C in solids	Total % recovery of ^{14}C
5 mg/kg	0.4±0.04	0.4±0.04	129.9±24.1	130.9±24.1
500 mg/kg	0.4±0.03	0.4±0.06	122.5±7.9	123.3±7.9
Positive control (glucose, 5 mg/kg)	67.2±2.1	18.1±1.1	9.5±4.9	94.9±1.8

For the positive control, an average of 95% of the total radioactivity added as glucose was recovered with 85% converted to $^{14}CO_2$ and $^{14}CH_4$ and 10% associated with the sediment-phase. The degradation seen in the positive control indicated that the sample pre-treatment methods (e.g. use of tetrahydrofuran solvent) appeared to have had little effect on the viability of the microbial community present.

For DBDPO, <1% of the total radioactivity added was found as $^{14}CO_2$ and $^{14}CH_4$ indicating that essentially no mineralisation occurred. Parent compound analysis (mean of seven replicate samples) indicated that the concentrations of DBDPO in the nominal 5 mg/kg treatment were 6.64 ± 0.70 mg/kg at day 0 and 6.51 ± 2.15 mg/kg at week 32. Similarly, the measured concentrations of DBDPO in the nominal 500 mg/kg treatment were 543 ± 77 mg/kg at day 0 and 612 ± 158 mg/kg at week 32. The differences in concentration between day 0 and week 32

were not statistically significant. The composition of the sediment cores were found to account for some of the variability seen in the measured concentrations, with sediments containing a greater number of gravel/stones leading to a higher variability between replicate measurements of concentration.

The HPLC chromatographic profiles also indicated that traces of some ^{14}C-labelled components with shorter retention times than DBDPO were present in some of the 32-week samples in the 5 mg/kg treatment group. Similar components also were present in the stock solution of the ^{14}C-DBDPO. A more detailed GC-MS analysis was carried out on Day 0 and Week 32 sediment samples. No evidence for the formation of lower brominated congeners was found.

A similar anaerobic degradation study was performed with 2,2',4,4'-TetraBDE (Schaefer and Flaggs, 2001c). The substance tested was a mixture of ^{14}C-2,2',4,4'-TetaBPE (radiochemical purity 96.5%) and unlabelled 2,2',4,4'-TetraBDE (purity ~99%). The test concentrations were 5 and 500 mg/kg dry sediment. A positive control (^{14}C-Glucose) was also run. The test was carried out using the same sample preparation method, a similar sediment and the same test system as used for DBDPO. The mass balance results from the experiment are shown in Table 4-9.

TABLE 4-9. Mass balance results from a 32-week anaerobic sediment degradation study of ^{14}C-2,2',4,4'-TetraBDE.

Nominal concentration	Mass balance at week 32			
	% as $^{14}CO_2$	% as $^{14}CH_4$	% ^{14}C in solids	Total % recovery of ^{14}C
5 mg/kg	0.5±0.34	0.01±0.01	134.3±5.0	134.8±5.2
500 mg/kg	0.2±0.02	0.01±0.02	124.8±7.7	125.0±7.7
Positive control (glucose at 5 mg/kg)	73.4±8.5	7.8±4.7	19.6±4.0	100.9±0.25

The total recovery of ^{14}C from the positive control was 101%, with 81.2% converted to $^{14}CO_2$ and ^{14}C, and 19.6% associated with the sediment-phase. The degradation seen in the positive control indicates that the sample pre-treatment methods using tetrahydrofuran solvent appear to have had little effect on the viability of the microbial community present.

4.2.1.3 Transport (Fugacity)

If released in equal amounts to air, water and soil, DBDPO is predicted to partition to soil and sediment. Based on a release of 1,000 kg/hr to air, water and soil, the predicted partitioning is: air 0.12%, water 1.09%, soil 41.8% and sediment 57% (Level III Fugacity Model, EPIwin V3.04). The majority (73%) would be reacted in soil and sediment, with only 23% of the total undergoing advection.

A preliminary evaluation of DBDPO's potential for long-range transport in the atmosphere indicated that this was unlikely (Hardy and Smith 1999). Wania and Dugani (2002) recently concluded extensive computer modeling of the long-range transport potential of DBDPO. Four

multimedia models were used: Characteristic Travel Distance, Spatial Range, Arctic Accumulation Potential, and Globo-POP. All four models produced similar results, and Wania and Dugani concluded that DBDPO was unlikely undergo long-range transport. Instead, DBDPO released to the environment would deposit near the point of release. This behavior was very different from that predicted by the models for brominated diphenyl oxides having 2-4 bromine atoms/molecule. The Di- to TetraBDE molecules were predicted to have a long range transport potential similar to chlorinated biphenyls with 4-6 chlorine atoms/molecule known to undergo significant long-range transport.

4.2.1.4 Leaching from Polymers

The potential leaching of a DBDPO mixture (Dow FR-300-BA; 77.4% deca-, 21.8% nona- and 0.8% octabromodiphenyl oxide) from pellets of acrylonitrile-butadiene-styrene (ABS) polymer and polystyrene was studied. The pellets of either plastic contained 10% of the DBDPO mixture. The pellets were placed in 2 L of water and shaken mechanically. The results, expressed as the concentration of bromine in water, are shown in Table 4-10. The lack of increase of the bromine concentration with time and the erratic results are best explained by assuming that extraction of DBDPO was mainly due to erosion of surface particles (Norris et al., 1973 and 1974).

Little or no leaching into water, acetic acid or cottonseed oil at elevated temperature also occurred from ABS pellets containing 4.25% of DBPDO mixture (Dow FR-300-BA) (Table 4-11). No DBDPO was detected in water or acetic acid, and only about 0.03% of the total was extracted by cottonseed oil over 7 days at elevated temperatures (Norris et al., 1973 and 1974).

TABLE 4-10. Extraction of a DBDPO mixture from ABS or Polystyrene by water.

Time (hours)	Concentration (mg bromine/L water)	
	ABS	Polystyrene
3	1.8	<1
19	1.3	<1
27	1.0	<1
43	3.7	<1
51	<0.5 (not detected)	<0.5 (not detected)
187	<0.5 (not detected)	<0.5 (not detected)

TABLE 4-11. Solvent extraction of a DBDPO mixture from ABS.

Solvent	Time (days)	Temperature (°C)	Concentration of DBDPO in solvent (mg/l)
Water	1	48.9	<0.075 (not detected)
3% Acetic acid	1	48.9	<0.075 (not detected)
3% Acetic acid	7	48.9	<0.075 (not detected)
Cottonseed oil	7	57.2	1

4.2.2 Environmental Toxicology

DBDPO was not acutely toxic to fish (CITI 1992) or marine algae (Walsh et al.1987), and is not expected to be chronically toxic in aquatic species due to its large molecular weight, negligible water solubility, and the lack of toxicity exhibited by the OBDPO commercial product. DBDPO also was not toxic to the sediment oligochaete, *Lumbriculus variegatus* (Krueger et al. 2001), or to six species of terrestrial plants. DBDPO also did not bioconcentrate in fish, and no evidence of its degradation in fish to 2,2',4,4'-TeBDPO or 2,2',4,4',5-PeBDPO has been found.

4.2.2.1 Aquatic Organisms

A 48h-LC_{50} for orange-red killifish (*Oryzias latipes*) has been determined for DBDPO as part of a six-week bioconcentration study. The LC_{50} was >500 mg/l (CITI 1992).

Walsh et al. (1987) studied the toxicity of DBDPO to the marine unicellular algae *Skeletonema costatum*, *Thalassiosira pseudonana* and *Chlorella* sp. The tests were carried out at a salinity of $30^{o}/_{oo}$ for either 72 hours (*S. costatum* and *T. pseudonana*) or 96 hours (*Chlorella* sp.). The end-point measured was the EC_{50} for growth based on cell numbers. The exposure concentrations in the test solutions were verified by analysis. In the tests, the DBDPO was added as a solution in acetone (final acetone concentration around 1 ml/l). Six different growth media were used in the test, one natural seawater and five synthetic seawater formulations. The natural seawater had a salinity of 32‰ and was diluted to give a final test salinity of 30‰ to be comparable with that of the synthetic media. The pHs of the various test media were in the range 7.6-8.2. The EC50 for all three species was greater than the highest concentration tested (1 mg/l).

DBDPO is not expected to be chronically toxic to aquatic organisms owing to its lack of acute toxicity, negligible water solubility, and tests on the commercial OBDPO product. A long-term *Daphnia* test has been performed on the commercial OBDPO product, and no effects on survival, reproduction or growth were seen over 21-days at concentrations up to 2 µg/l (solubility limit). Taken as a whole, it is clear that the aquatic toxicity and bioaccumulation potential of the PBDPO products (penta-, octa- and decabromodiphenyl oxide) decreases with increasing bromination and therefore it is unlikely that DBDPO will show any toxic effects to invertebrates at concentrations below its solubility limit.

4.2.2.2 Fish Bioconcentation/Bioaccumulation

The bioconcentration of ^{14}C-DBDPO (20 ug/L) in rainbow trout under static conditions over a 48-hour period was compared to a known bioaccumulative substance, 2,2',4,4'-tetrachlorobiphenyl (TCBP) (16 ug/L). Little change in the DBDPO water concentration was seen in the water (initial concentration was 20 µg/l), indicating minimal uptake by the trout and insignificant losses by other means (e.g. volatilisation, adsorption onto surfaces etc.). DBDPO's lack of bioconcentration was confirmed by analysis of ^{14}C-residues in fish samples at intervals during the experiment (Table 4-12). Little or no uptake of DBDPO

occurred. The positive control, TCBP, was found to bioconcentrate at least 50 times over the initial exposure levels within 4 hours (Norris et al., 1973 and 1974).

TABLE 4-12. Concentrations of ^{14}C-DBPDO and TCBP (ppb) in fish on exposure to water concentrations of 20 or 16 ug/L, respectively.

Time (Hr)	14C-DBDPO (ppb)	TCBP (ppb)
0	-	<100
0.5	-7*	150
1	1	330
2	1	520
4	3	1,000
6	1	1,200
12	-2*	1,300
24	3	1,200
48	6	1,000

*Below background.

The bioconcentration of DBDPO in carp was studied over a six-week period in a study performed according to Japan's "Bioaccumulation test of chemical substance in fish and shellfish" (CITI 1992). The 48 hr LC50 was first determined in orange-red killifish (*Orizias latipes*), and the value was used along with the analytical detection limit of the test substance to select two test concentrations for the bioconcentration test in Japanese carp (*Cyprinus carpio*). Concentrations used in this design are typically 1/100, 1/1000 or 1/10,000 of the 48 hr LC50. The highest exposure concentration was 10 times that of the low exposure concentration. For DBDPO, the test concentrations were 6 and 60 ug/L. The control and test groups consisted of 15-20 fish each. The duration of exposure was 6-8 wks until equilibrium was reached in the fish. Test article concentrations in the aquaria and fish were determined twice/wk, and in 2-3 treated fish/exposure concentration every 2 weeks. The control fish were analyzed before test initiation and at termination of exposure for the test substance. The whole body of each fish was homogenized and extracted using an analytical method suitable for the test substance. Test article concentrations in fish and water were corrected for analytical recovery rates. Analytical method blanks were also performed. The BCFs measured at the end of the experiment were <5 at an initial concentration of 60 µg/l and <50 at an initial concentration of 6 µg/l (the two values are consistent if no DBDPO was detected in the fish, and the detection limit in fish was around 300 µg/kg, and indicate that little or no bioconcentration is occurring) (CITI, 1992).

Kierkegaard et al. (1997, 1999) investigated the uptake in trout of Dow FR-300-BA following administration in food. The Dow product has not been manufactured since the 1980s, contained only 77.4% of the DBDPO isomer with the remainder being nona- (21.8%) and octaBDPO isomers (0.8%) (Norris et al 1973, 1974, 1975). However, Kierkegaard et al. did not provide the composition of the mixture tested. Rainbow trout were force-fed homogenized cod containing the suspended test article for a period of 16, 49 and 120 d. Doses ranged between 7.5 and 10 mg/kg/d. Only a very small amount of the test material was taken up during the 120-day exposure phase. Uptake was estimated to be 0.02 –0.13% of the dose after 120 days of exposure

based on the muscle concentrations of the total hexa-to DBDPO isomers. Uptake of the DBDPO component was estimated at only 0.005% of the dose, and declined significantly during depuration. No evidence of debromination of the test article to 2,2',4,4'-TeBDPO, 2,2',4,4',5-PeBDPO or 2,2',4,4',6-PeBDPO was found, and the authors concluded "… possible metabolism seem not to be the major sources of tetra- and pentabromodiphenyl ethers found in wild fish".

Some hexa-, hepta-, octa- and nonaBDPO congeners' concentrations increased with exposure in liver and muscle. Some of these congeners were not detectable in the test article and Kierkegaard et al. speculated that their presence might be the result of a metabolic process or a more efficient absorption of trace amounts initially present in the food/test article. Kierkegaard et al. was not able to distinguish between these two possibilities. A third possibility, not considered in Kierkegaard et al., is that these hexa-, hepta-, octa- and nonaBDPO congeners were present in the test article but not detected, and slowly increased in fish tissue over time to detectable levels over the 120 d test period as a result of slow metabolism/elimination.

The results of this bioaccumulation study are consistent with previous work showing insignificant bioconcentration of DBDPO in fish, do not provide evidence that DBDPO is debrominated metabolically, and indicate that metabolic debromination of DBPDO is not the source of tetra- and pentaBDPO congeners detected in wild-caught fish.

4.2.2.3 Sediment Organisms

Prolonged sediment toxicity tests (28-D) on DBDPO were preformed with the oligochaete *Lumbriculus variegatus* using a flow-through test system with sediments of either 2.4% or 5.9% organic carbon content (Krueger et al. 2001a,b). The test was based on the ASTM E 1706-95b Guideline and USEPA Series 850 Ecological Effects Test Guidelines (OPPTS No. 850.1736) and performed according to Good Laboratory Practices.

The test substance was a composite sample from three manufacturers and had a purity of 97.9%. The total exposure period was 28 days. The nominal concentrations tested in the studies were 0, 313, 625, 1,250, 2,500 and 5,000 mg/kg dry weight. Each treatment and control group was replicated eight times with ten oligochaetes/replicate. Additional replicates were also run in each treatment and control group for analytical sampling of water and sediment. The endpoints were survival/reproduction (as measured by the total number of organisms present which is a combination of parent survival and reproduction) and growth (as determined by dry weight of organism).

In both the 2.4 and 5.9% organic carbon sediment, the NOEC for survival and growth ≥ 5,000 mg/kg dry sediment (nominal). Based on the measured sediment concentrations, the NOECs were 4,536 and 3,841 mg/kg dry weight for the 2.4 and 5.9% organic carbon sediments, respectively.

4.2.2.4 Sludge Microorganisms

An activated sludge respiration inhibition (OECD 209) test was performed on a composite sample of commercial DBDPO products from three manufacturers (Schaefer and Siddiqui, 2001). The purity of the test substance was 97.9% DBDPO. The substance was tested in triplicate at a concentration of 15 mg/l. The inoculum used in the test was activated sludge from a waste water treatment plant that received predominantly domestic waste. The test was carried out at 20-22^{o}C and the respiration rate of the activated sludge over 3 hours was determined. Two controls and a positive control (3,5-dichlorophenol at concentrations of 5, 15 and 50 mg/l) were also run. The respiration rates in the two controls were both 41.6 mg O_2/l/hour. The mean respiration rate in the DBDPO treatments was 41.1 mg O_2/l/hour and so no inhibition of respiration was seen at the concentration tested. The EC_{50} for the positive control was determined as 9.8 mg/l, which was within the normal range of 5 to 30 mg/l for this test. The NOEC for DBDPO from this test was therefore ≥ 15 mg/l. This indicates DBDPO's lack of ready biodegradation is not due to inhibition of the microorganisms present in sewage sludge.

4.3 Potential Degradation of DBDPO

There has been speculation that DBDPO may degrade in the environment or in biological systems to lower brominated diphenyl oxide/ether congeners. The apparent reasoning for this speculation is as follows: The commercial DBDPO product represents approximately 82% of the global commercial polybrominated diphenyl oxide/ether (PBDE) usage. Approximately 50-70% of "PBDEs" detected in biological samples is composed of a single isomer, 2,2',4,4'-TetraBDE, whereas DBDPO is rarely detected. 2,2',4,4'-TetraBDE is a component of only one PBDE product – the commercial pentabromodiphenyl oxide product – and the total content of tetra-substituted congeners in the pentabromodiphenyl oxide product is ~34%. Hence, the speculation that DBDPO may degrade to tetra and/or pentaBDE congeners either in the environment or in biological systems.

The above speculation does not take into consideration the impact that the differences in physical properties, potential for bioaccumulation, or the potential for environmental release between DBDPO and the tetra- and pentaBDE congeners. In addition, the available monitoring and experimental data do not support degradation of DBPDO to lower brominated diphenyl oxide congeners.

4.3.1 Differences Between DBDPO and Lesser Brominated Diphenyl Ether Isomers

The measured water solubility and vapor pressure of DBDPO are negligible (<0.1 ug/L and 4.63 x 10^{-6} Pa). Although small, the measured water solubility of 2,2',4,4'-TetraBDE (10.9 ug/L) is greater than that of DBDPO (Hardy 2002a). The vapor pressure of 2,2',4,4'-TetraBDE (2.5 x 10^{-4} Pa) (Wong et al. 2001), although small, is greater than that of DBDPO. Likewise, there are major differences in the potential bioaccumulation of DBDPO and the 2,2',4,4'-TetraBDE isomer. DBDPO has been shown not to bioconcentrate in fish (BCF < 50), is very poorly absorbed in rats (0.3-<2% oral dose) and rapidly eliminated (>99% in 72 hrs) and as a consequence does not bioaccumulate. The 2,2',4,4'-TetraBDE isomer, however, bioconcentrates in fish (BCF > 10,000) is readily absorbed (> 95% of an oral dose) by the rat and slowly

eliminated (14% in 5 days). DBDPO's predominant use is in hard dense plastics (e.g. television cabinets), which limit its potential for release. The 2,2',4,4'-TetraBPE isomer is a component in certain flame retarded flexible polyurethane foams. This foam has an open cell structure, which presents a large surface area to the environment and therefore the potential for release is likely to be greater than the hard dense plastics where DBPDO is used. In addition, this foam may become friable and crumble with age. Thus, small particles could thereby released could move into the environment and disperse its components. All of these factors working together likely contribute to the differences in the environmental behavior and detection of DBDPO and 2,2',4,4'-TeBDE.

4.3.2 Potential for Environmental Degradation of DBDPO

DBDPO's potential for degradation in the environment can be examined by reviewing environmental monitoring data and laboratory study results. In the environment, DBDPO is expected to partition predominantly (~99%) to soil and sediments where it will undergo extensive binding to particulate matter. DBDPO has been detected in sediments near point sources, and thus sediments are a logical matrix in which to look for evidence of degradation. The monitoring data does not support degradation of DBDPO to lower brominated diphenyl ether congeners.

European sediments have been monitored over a 20-year period (de Boer 2001), and do not support degradation of DBDPO. de Boer et al. concluded that significant amounts of lesser brominated diphenyl ethers were unlikely to be formed from DBDPO in sediment based on the results of a detailed survey of the levels of PBDPOs in various European sediment cores. Although the sediment concentration of DBDPO increased in recent years, no parallel increase in the concentrations of lesser brominated diphenyl ethers (e.g. tetra- to penta- congeners) occurred and there was no indication of increasing levels of nona- and octabromodiphenyl ethers.

The Mersey River estuary was used as a disposal site for U.K. sewage sludge for a number of years. (This practice has been discontinued.) DBDPO was used by industries in the area, and DBDPO was detected in sediment collected from this estuary. Samples of the Mersey River sediment were analyzed via HRGCMS for mono- to decabromodipnenyl oxide congeners. Hexa- to nona congeners would be expected if DBDPO were undergoing reductive debromination. However, only DBDPO and tetra/pentaBDPE congeners were detected. Thus, the monitoring data does not support the degradation of DBDPO to lower brominated diphenyl oxides.

With respect to laboratory degradation studies, the results indicate DBDPO is not biodegradable (see section 4.2.1.3). Abiotic degradation, e.g. hydrolysis or photolysis, is also not expected to be a significant route of environmental degradation. Hydrolysis is unlikely to occur based on DBDPO's chemical structure and negligible water solubility. Photolysis is not expected, because of DBPDO's negligible vapor pressure, negligible partitioning to air (~0.1%), and experimental photodegradation work showing little or no degradation under environmentally relevant conditions. Furthermore, in aqueous systems, halogenated organic compounds are expected to degrade via substitution of a Br atom with a hydroxyl group. Replacement of a Br

atom by a hydrogen atom (e.g. reductive debromination) is only anticipated in organic solvents (Norris et al 1975). Thus, if DBDPO were to photodegrade in the environment, reductive debromination would not be the expected pathway. Finally, in the environment, DBDPO is likely to be adsorbed to bulk matrices, and only a small fraction of that present (i.e. that near the exposed surface) would be exposed to light and therefore available for photodegradation to occur.

Studies carried out using organic solvents indicate that products such as lower brominated diphenyl ether congeners (which are potentially more toxic and accumulative than the parent compound) and in some cases polybrominated dibenzofurans are formed from DBDPO under UV or natural sunlight (Watanabe and Tatsukawa 1987). However, organic solvents act as hydrogen donors in these reactions and affect the products formed. Degradation of DBDPO under more environmentally relevant conditions using solid matrices in contact with water and either natural or artificial sunlight, provide little if any evidence for the photolytic degradation of DBPDO (Jaffvert and Hua 2001). Little or no evidence of photolysis of DBDPO was detected when the molecule was adsorbed to sand or humic acid coated sand and exposed to sunlight. Further, no evidence for the formation of 2,2',4,4'-TetraBDE was found (Jaffvert and Hua 2001, Sellstrom 1998).

4.3.3 Potential for Biological Degradation of DBDPO

No study has reported metabolism of DBDPO to lower brominated diphenyl ethers.

DBDPO is very poorly absorbed by the rat or fish. Oral absorption in the rat has been reported as 0.3-2% of the dose. The uptake of DBDPO from a DBDPO mixture (Dow FR BA 300) when force-fed to fish was <0.005% of the dose (Kierkgaard et al. 1999). DBDPO BCF in fish is < 50. Due to the very poor uptake, minimal levels of DBDPO would be available for systemic metabolism. Further, Kierkegaard et al. (1999) concluded that there was no evidence that DBDPO was debrominated in wild fish to the major PBDEs detected in fish, 2,2',4,4'-TetraBDE, 2,2',4,4',5-PentaBDE, or 2,2',4,4',5,6-PentaBDE, and that DBDPO was not a source of the Te/PeBDEs detected in wild fish.

Studies performed by NTP (1986) indicate at low levels in the diet (277 ppm pre-treatment) only about 2% of the dose was eliminated as DBDPO metabolites, and that much of this metabolism apparently took place in the gut. The percentage eliminated as metabolites increased with increasing dose so that at 50,000 ppm, about 30% of the dose was eliminated as metabolites. Based on this work, at environmental levels $\geq$ 98% of DBDPO is expected eliminated as the parent molecule.

NTP's work also demonstrated that DBDPO, and any metabolites, were rapidly cleared from the body. This rapid elimination of ^{14}C-DBDPO-associated activity argues against accumulation of metabolites. NTP's work, conducted at very high dose levels, also indicates a lack of toxicity associated with DBDPO and any metabolites.

5.0 EXPOSURE ASSESSMENT

The objective of the exposure assessment component of the VCCEP is to quantify the levels of exposure to DBDPO experienced by children. The U.S. Environmental Protection Agency (EPA) has suggested that this will involve quantification of the following:

- What are the sources of exposure (e.g., environmental releases, consumer products)?
- What are the pathways of exposure (e.g., breathing air, drinking water, eating food, contact with skin)?
- What are the chemical concentrations in the various media?
- What are the frequency and duration of chemical exposures?
- Who and how many children are exposed?

The VCCEP guidance suggests that a Tier I exposure assessment should contain "at a minimum…screening level (or, if available, better) information on exposure from manufacturing supplemented with relevant screening level data on downstream processing and use activities and specific information on children's exposures, if available." In addition, the VCCEP guidance suggests that the Tier I exposure assessment should "generate conservative, quantitative estimates of exposure." With regard to the availability of data, the VCCEP guidance suggests, "The screening approach generally involves using readily available measured data, existing release and exposure estimates and other exposure-related information."

There have been at least two recent evaluations conducted to address the potential health risks due to DBDPO (NAS 2000; ECB 2002) and one thorough review of the toxicology and exposures to DBDPO (WHO 1994). The National Academy of Sciences (NAS) evaluated the use of DBDPO in textiles, including child-specific activities that might result in their increased exposure potential (e.g. mouthing textiles). The European Chemicals Bureau (ECB) evaluated exposures to DBDPO via the general environment. Both evaluations concluded that exposures via these two pathways did not pose a health risk to the general population. The World Health Organization (WHO) (1994) concluded that exposures to DBDPO could occur in the course of manufacture and formulation into polymers, and that exposure of the general population to DBDPO was insignificant (WHO 1994). These three evaluations indicate that exposures to DBDPO are minimal and not likely to pose a health risk, but with the exception of NAS's assessment of upholstery textiles, did not explicitly address a child's exposure to DBDPO. The following outlines the approach to calculating a child-specific exposure assessment for DBDPO, per the requirements of the VCCEP program.

Based on DBDPO's applications, the following are plausible scenarios by which children might be exposed to DBDPO: exposures secondary to manufacturing of DBDPO, exposures related to consumer products containing DBDPO, and exposures from the general environment (food, water, air, soil, dust, etc.).

5.1 Occupational Exposure

The American Industrial Hygiene Association (AIHA) established a Workplace Environmental Exposure Level (WEEL) for DBDPO of 5 mg/m^3 based on DBDPO's toxicology data (AIHA 1996). A WEEL is the level at which at workers could be exposed every day for an 8-hour shift with the expectation of no adverse effects. The U.S. Occupational Safety and Health Agency (OSHA) has not set a Permissible Exposure Limit (PEL) for DBDPO. However, OSHA has set a PEL of 5 mg/m3 for the "respirable fraction of particulates not otherwise regulated", e.g. nuisance dusts. Thus, the AIHA WEEL for DBDPO is equivalent to that of a nuisance dust.

Workplace exposures to DBDPO may occur at a) manufacturing, and b) formulation into the resin or liquid polymer dispersion. DBDPO is manufactured in a closed system by the reaction of bromine with diphenyl oxide. The point at which exposure could occur during manufacture is when DBDPO is transferred into bags for shipping. Likewise, the point at which worker exposure is most likely during formulation is when the bags of flame retardant are emptied into a hopper prior to mixing. Once formulated, DBDPO is encased in the polymer matrix and the potential for worker exposure is negligible.

Theoretically, workplace exposure could occur via the dermal or inhalation routes. DBDPO's physical and chemical properties make the probability of systemic absorption following dermal (Section 5.1.1) or inhalation (Section 5.1.2) exposure very low. DBDPO is a large molecule of high molecular weight (959.17) with negligible water solubility (<0.1 ug/L), and is likely to diffuse through biological membranes only with great difficulty. This assumption is borne out with pharmacokinetic studies that demonstrate DBDPO's poor bioavailability. DBDPO's vapor pressure (4.63×10^{-6} Pa) is such that volatilization is not expected to be a source of inhalation exposure. Occupational exposure to dusts may occur, and the particle size of the DBDPO commercial product is within the inhalable and/or respirable range. The particle size used resin application is ~5 microns, whereas that used in textile applications is finely ground to ease its dispersion in latex coatings. Any DBDPO particles present in air are likely to be associated with larger dust particles due to DBDPO's affinity for adsorption (estimated $K_{oc} = 1.796 \times 10^6$) (Meyland and Howard 1999). It is likely that the primary routes of absorption in the workplace are via incidental ingestion resulting from inhalation (and mucociliary escalator effect) and contaminated clothing and surfaces.

Theoretically, the flame retardant textile backcoat could crumble during fabrication of upholstered furniture. Any particles generated would likely be too large to be respirable. In addition, for systemic absorption to occur, not only would the particles need to be inhaled or ingested, but also DBDPO would have to diffuse out of the polymer prior to its absorption. Systemic absorption of significant amounts as a result of crumbling of the backcoat is highly unlikely.

An additional occupational exposure scenario explored in the published literature is electronics recycling, computer repair and rubber manufacture. DBDPO, and other polybrominated diphenyl oxide (a.k.a. ether) isomers, was detected in Swedish workers engaged in dismantling electronic equipment (Sjodin et al. 1999; Sjodin 2000) and in Swedish computer technicians (Hagmar et al.

2000). This work was performed as a Ph.D. research project (Sjodin 2000). These findings are discussed in Section 5.1.2 where the measured workplace air levels (0.0002 mg/m^3) are compared to the AIHA WEEL for DBDPO of 5 mg/m^3. More recently, Thuresson et al. (2002a,b) also reported detection of DBDPO in Swedish workers engage in rubber manufacturing and electronic shredding operations. Occupational blood and air levels are summarized in Table 5-1.

Table 5-1. Measured DBDPO human serum and air concentrations in various occupations.

Type of Work	DBDPO Serum Levels (pmol/g lipid)		DBDPO Air Concentration	Reference
	Median	Number of Individuals		
U.S.				
Manufacture	N.D.*	39 (all male)	0.21-5.9 mg/m3	Bahn et al. 1980; Bialik 1982
Sweden				
Electronics Recycling	5	19 (15 males, 4 females)	36 ng/m3	Sjodin et al. 1999; Sjodin 2000
Computer Repair	2	19 (15 males, 4 females)	N.R.+	Hagmar et al. 2000
Rubber Manufacture	32	19 (all male)	7.6 $\pm$ 5.6 pmol/m3	Thuresson et al. 2002b
Electronics Shredding	3	5 **	13 pmol/m3	Thuresson et al. 2002a
Referents				
Hospital Cleaning	<0.7	20 (all female)	N.R.	Sjodin et al. 1999; Sodin 2000
Computer Clerks	<0.7	20 (all female)	N.R.	Sjodin et al. 1999; Sodin 2000
Abattoir Workers	3	18 (all male)	N.R.	Thuresson et al. 2002a,b

*N.D. = Not Detected (ng/ml serum)
**Gender distribution not provided.
+N.R. = Not Reported

Children's exposure to DBDPO could potentially occur during gestation or via ingestion of breast milk that might contain DBDPO resulting from lactating mothers who are exposed in the workplace. To assess the potential magnitude of exposure, we evaluated several occupational exposure scenarios for the working mother. These include workers involved in the actual manufacturing of DBDPO, or employees of companies using the chemical in specific processes (i.e., formulators). Some studies have reported detectable air concentrations and blood levels of DBDPO in workers at Swedish electronics recycling and computer repair facilities (Sjödin et al.

1999; Sjödin 2001a), and these occupations are included in this assessment. The estimated maternal occupational exposures were used to estimate an infant's exposure via breast milk.

5.1.1. Dermal

No *in vivo* dermal absorption studies of DBDPO have been performed. Nonetheless, the potential for absorption of DBDPO through the skin can be estimated based on known characteristics of dermal absorption, DBDPO's physical/chemical properties and pharmacokinetics, and the extent of dermal absorption of related compounds.

5.1.1.1.Characteristics of Dermal Absorption

The barrier function of the skin is the best that the human body possesses. "From an evolutionary standpoint, the skin did not develop as an epithelium through which absorption was intended. Quite the reverse; the architecture and biology of the skin are, in large part, directed towards the construction of a highly efficient barrier to the outward loss of water. The most superficial and least permeable skin layer, the stratum corneum, is a remarkable feat of bioengineering, both from a structural and compositional viewpoint, and provides a uniquely impressive resistance to molecular transport both from and into the body. This is the reason that transdermal delivery requires potent drugs - one simply cannot transfer very many micrograms of any compound across a small surface area in the period of a few hours. Because the principal function of the skin is to minimize transepidermal water loss, the stratum corneum is a predominantly lipophilic barrier that is particularly impermeable in a passive sense to hydrophilic drugs (including charged species)." (Guy 1996).

Dermal absorption is defined as penetration through the skin into capillary walls and the blood stream. Substances move through the skin via passive diffusion, and the anatomical structure of the skin limits absorption of most substances. Permeability is largely determined by the skin's least penetrable layer, the stratum corneum (SC). Penetration of high molecular weight substances, molecular aggregates, and particulate matter through the skin is virtually nil. In general, the criteria for significant skin absorption of foreign compounds include a molecular weight of < 500 and reasonable solubility in both water and lipid (Guy 1996). Water solubility is required because a prerequisite for absorption from any site, including the skin, is that the penetrant must be in aqueous (true) solution at the absorption site (Ritschel 1982) and is requirement for passage through the epidermis. The rate of penetration is limited more by penetration into the relatively water-rich viable epidermis than by penetration through the lipid-rich SC (Garner and Mathew 1998; Jackson et al. 1993). Nonetheless, diffusion into and through human skin is at least partially rate-limiting for all chemicals with a octanol water partition coefficient (K_{ow}) > 3 due to the lipid-rich SC intercellular space and the relatively aqueous epidermis. Dermal absorption actually decreases for those penetrants with high K_{ow}s because of this phenomena. Smaller molecules (i.e. those with relatively small molecular volumes) are more readily absorbed through the skin than larger ones. The rate of absorption is also less for large molecules than small molecules (Wester and Maibach 1983).

The relative impermeability of skin is much greater than other membranes in the body, and is much less permeable than the mucosal lining of the mouth cavity, gastrointestinal tract, rectum, and lung. In addition to being less permeable, the surface area of the skin is less than these other routes of entry into the body (Ritschel 1982). The surface area of the skin is only 1.73 m^2 in the Caucasian adult whereas the absorbing surface area of the lung is about 70 m^2. The gastrointestinal tract has an even larger surface area (120 m^2) due to small intestinal villi and microvilli. Thus, the total absorptive surface area of the skin is only ~1.4% of that of the gastrointestinal tract.

5.1.1.2 Potential for Dermal Absorption of DBDPO

DBDPO's potential for dermal absorption is low based on its physical and chemical properties and the known requirements for absorption of any compound through the skin.

DBDPO's negligible water solubility will reduce its skin absorption since a prerequisite for absorption is that the substance must be present in aqueous (true) solution at the absorption site. DBDPO's large molecular sizes and weight will also negatively impact its skin absorption since absorption of molecules weighting > 500 is severely limited. Because DBDPO's octanol water partition coefficient is > 3, diffusion into and through the epidermis is at least partially rate-limiting. This relative inability to move into and through the epidermis limits the rate at which systemic uptake could occur.

Passage through the SC and the viable epidermis into the systemic circulation requires both lipid and water solubility. DBDPO is severely limited in this respect. With measured and estimated octanol water partition coefficients > 6, DBDPO is often assumed to be highly soluble in lipid (e.g. that DBDPO is "lipophilic"). However, DBDPO's high K_{ow} is likely more reflective of its very low water solubility (hydrophobicity) than its absolute affinity for lipid (lipophilicity). DBDPO is only sparingly soluble in common organic solvents: < 0.01 wt % at 25 degrees C in acetone and methanol, and only 0.76 wt % in toluene. In fact, DBDPO is soluble to any extent in only a limited number of organic solvents.

Using polyhalogenated aromatic hydrocarbons as an example of lipophilic compounds, a main determinant of their dermal absorption is the partitioning of the highly lipophilic members of this class between the lipid-rich SC intercellular space and the relatively aqueous viable epidermis. Quantitative structure activity relationships between dermal absorption and octanol-water partition coefficient (K_{ow}) have shown that within structurally related groups of compounds, absorption increases with the K_{ow} up to a maximum since diffusion through the lipid matrix of the SC is rate-limiting. Dermal absorption then decreases with further increases in K_{ow} because diffusion out of the SC and into and through the viable epidermis becomes the rate-limiting step for highly hydrophobic penetrants (Jackson et al. 1993). Table 5-2 compares DBDPO's molecular weight and volume with two halogenated aromatic hydrocarbons, 2,3,7,8-TCDD and 2,3,7,8-TBDD, having measured dermal absorption data. TBDD has a larger molecular volume and weight than TCDD due to the presence of 4 bromine atoms versus 4 chlorine atoms. Even though both have K_{ow}'s > 6, their dermal absorption differs greatly. TBDD is poorly absorbed (~12%) while about 40% of a dermally applied TCDD dose was absorbed by the rat. In

comparison, DBDPO has a significantly greater molecular weight and molecular volume than either TCDD or TBDD. DBDPO's molecular weight is 49% greater than TBDD. Therefore, DBDPO's dermal absorption is expected to be correspondingly lower than that of TBDD, e.g. DBDPO's dermal absorption is expected to be < 12% of the dose based on this data.

TABLE 5-2. DBDPO: comparison of molecular volume and weight with 2,3,7,8-TCDD and 2,3,7,8- TBDD. Effect of molecular weight and volume on dermal absorption.

PROPERTY	TCDD[1]	TBDD[1]	DBDPO[2]
Molecular Weight (g/m)	316	492	959
Molecular Weight (vs. TCDD)	X	40% >X	67%>X
Molecular Volume (vs. TCDD)	Y	13% >Y	43%>Y
Dermal Absorption (%)	40	~12	N.M.
Water Solubility (ug/L)	N.R.	N.R.	< 0.1
K_{ow}	6.01	6.56	6.26

[1]Dilberto J et al., Toxicol. Appl. Pharmacol. 120, 315-326, 1993.
[2]Hardy, M. Proceedings of FRMP '97. Lille, France, Sept 1997.
N.M.= Not Measured
N.R.= Not Reported

Oral pharmacokinetic studies in the rat have shown DBDPO is sparingly absorbed from the GI tract (NTP 1986; El Dareer et al. 1987). Results of these studies demonstrate that after ^{14}C-DBDPO exposure, at all doses tested (250 - 50,000 ppm in the diet), greater than 99% of the radioactivity recovered was excreted in the feces within 72 hours. Concentrations of the radiolabel in all major organs and tissues were near the detection limits. Estimates of DBDPO absorption from the GI tract were calculated by comparing tissue levels after oral exposure versus intravenous administration. Absorption from the GI tract was calculated to be 0.33% $\pm$ 0.19% of the 50,000 ppm dose. Given that the GI tract has a greater absorptive surface and is more permeable than the skin, it is reasonable to conclude the amount of DBDPO absorbed from the GI tract is greater than that which could be absorbed through the skin. Skin absorption of DBDPO, if it occurs at all, should be significantly less than that absorbed orally. Therefore, DBDPO skin absorption is expected to be << 0.33% of a dermally applied dose. Thus, skin absorption is not expected to be a source of DBDPO exposure in the workplace.

DBDPO's no adverse effect level (NOAEL) in repeated dose studies is $\geq$ 1000 mg/kg/d. This NOAEL encompasses studies of prenatal developmental toxicity and assessment of the reproductive organs in subchronic/chronic studies. This high NOAEL, negligible skin absorption, the small amount of total skin surface area exposed in the workplace and the possible use of personal protective equipment indicate a high margin of safety with respect to dermal exposure to DBDPO in the workplace.

5.1.2 Inhalation

AIHA established a WEEL of 5 mg/m^3 for DBDPO. This WEEL is essentially that of a nuisance dust. Occupational exposure to dusts containing DBDPO may occur at the manufacturing site

during bagging operations or when the bags are emptied into hoppers at the processing site. The particle size used resin applications is ~5 microns, whereas that used in textile applications is finely ground to ease its dispersion in latex coatings. Any DBDPO particles present in air are likely to be associated with larger dust particles due to adsorption (estimated $K_{oc} = 1.796 \times 10^6$). DBDPO is a large molecule of high molecular weight (959.17) with negligible water solubility (<0.1 ug/L), and is likely to diffuse through biological membranes only with great difficulty based on oral pharmacokinetic studies. This, coupled with DBDPO's high no-adverse-effect level of 1,000 mg/kg/d in chronic studies, indicates the worker is not at risk of adverse effects due to dust exposure.

DBDPO oral absorption is minimal (<0.3 to 2% of an oral dose), but no data on its pulmonary absorption is available. Although the absorptive processes in the lung and gastrointestinal (GI) tract are similar, DBDPO absorption from the respiratory tract is expected to be less than from the GI tract. The respiratory membrane has a surface area of 160 m^2 versus 250 m^2 for the intestinal mucosal villi (Ritschel 1982), and the lung's absorptive surface is therefore ~64% of that of the small intestine. DBDPO has negligible solubility, and thus inhaled particle-bound DBDPO can be expected to behave similar to other inert insoluble particles deposited in the respiratory tract. Insoluble particles deposited within the ciliated airways of the respiratory tract (e.g., the nasal passages and tracheobronchial tree) undergo passive transport via the mucuciliary escalator to the pharynx and are subsequently swallowed (Lippman 1980). Insoluble particles reaching the alveoli are predominantly cleared by alveolar macrophages that phagocytize the particles and transport them proximally on the bronchial tree to be swallowed. Absorption of insoluble particles from the alveoli directly into the bloodstream is low and exceedingly slow. Thus, it appears unlikely that absorption of DBDPO from the respiratory tract is greater than that of the gastrointestinal tract.

5.1.2.1 Electronics Recycling or Computer Repair

Recent publications report detection of DBDPO in the blood of Swedish workers engaged in electronics recycling or computer repair (Sjodin et al. 1999; Sjodin 2000; Hagmar et al. 2000; Thuresson et al. 2002a) and in rubber manufacturing (Thuresson et al. 2002b). The studies in electronic recycling workers are the best documented of these papers and is discussed further in the following paragraphs.

The mean DBDPO blood levels reported were 5 pmol/g lipid in the Swedish electronics recycling workers, and 1.6 pmol/g lipid in the Swedish computer technicians. DBDPO air levels in the recycling workplace were 0.0002 mg/m^3. The PCB 153 blood levels measured in the same workers was 760 pmol/g lipid in the dismantlers and 290 pmol/g lipid in the technicians. Greater than or equal to 99% of the DBDPO detected in air at the electronics dismantling plant was associated with particulate matter (Sjodin 2000).

The amount of a substance absorbed (A_{dose}) through the respiratory tract over a given period of exposure can be calculated (Patty 1994) using the concentration in air in mg/m^3 (A_c), the duration of exposure in hours (T), the ventilation rate in m^3/hour (V), and the absorption rate (A_{bs}):

$$A_{dose} = A_c TVA_{bs.}$$

A theoretical DBDPO blood concentration can be calculated using the percent oral absorption as an indicator of respiratory uptake and the equation above. Using a maximum absorption of 2% of the dose, a ventilation rate of 10 m^3/8 hr work shift and an exposure equivalent to the AIHA WEEL (5 mg/m^3), the amount of DBDPO absorbed would be 1 mg/70 kg man or 0.014 mg/kg body weight. This is orders of magnitude less than DBDPO's reference dose (RfD) of 4 mg/kg-d calculated by NAS (see Appendix V). In the event that DBDPO's absorption was equal to 100%, the absorbed dose would still remain less than NAS's RfD. At 100% absorption and an exposure concentration of 5 mg/m^3, the internal dose would be 0.71 mg/kg body weight.

Using the equation $A_{dose} = A_c TVA_{bs,}$ a maximum absorption of 2% of the dose, a ventilation rate of 10 m^3/8 hr work shift and at maximum measured DBDPO air concentration of 0.2 ug/m^3 in the electronics dismantling plant (Sjodin et al. 1999), the absorbed dose would be 0.04 ug DBDPO/70 kg man or 0.57 ng DBDPO/kg body weight.

At a measured DBDPO serum lipid level of 4.8 ng/g lipid in the electronics dismantling workers (Sjodin et al. 1999), the DBDPO plasma level would be 0.0288 ng/ml plasma. Assuming 3,000 ml plasma in a 70 kg man and a normal plasma lipid concentration of 0.6% (Guyton 1986), the 0.0288 ng DBDPO/ml plasma represents a total blood volume content of 86.4 ng DBDPO/70 kg man or 1.2 ng/kg body weight. Thus, the theoretical DBDPO internal dose (0.57 ng/kg body weight) due to a measured air concentration of 0.2 ug/m^3 compares favorably with the actual dose of 1.2 ng/kg body weight in the electronics dismantling workers calculated from their measured blood values. The theoretical and measured values are well within the variation expected due to the assumptions used in calculating the expected values and the collection and analytical methods.

The measured DBDPO air level at the electronic recycling plant was 0.0002 mg/m (Sjodin et al. 1999). The American Industrial Hygiene Association (AIHA) evaluated DBDPO's toxicology and set a Workplace Environmental Exposure Level (WEEL) of 5 mg/m^3, e.g. that of a nuisance dust (AIHA 1996). Thus, the measured DBDPO air level at the electronics dismantling plant was 25,000 times below the AIHA level to which workers could be exposed every day with the expectation of no adverse effects. Further, using the equation $A_{dose} = A_c TVA_{bs}$ and a maximum absorption of 2%, the estimated internal DBDPO dose from an 8-hr exposure at the AIHA WEEL of 5 mg/m^3 would be 0.014 mg/kg body weight. The internal dose of the electronic recycling workers was 1.2 ng/kg or 0.01% of the internal dose that could be received at a DBDPO exposure equal to the AIHA WEEL. Finally, in the event that DBDPO absorption from the respiratory tract was greater than 2%, the internal dose of the electronic recycling workers at a measured DBDPO air level of 0.0002 mg/m^3 would remain substantially below that achievable at the AIHA WEEL. For example, if DBDPO absorption equaled 100%, the internal dose due to a workplace air level of 0.0002 mg/m^3 would be 0.004% of that dose which could be received at a DBDPO exposure equal to the AIHA WEEL.

5.1.2.2 Discussion of Blood and Air Levels

The DBDPO blood levels reported in Swedish electronics dismantling workers (5 pmol/g lipid) and computer technicians (1.6 pmol/g lipid) were extremely small and are representative of our increasing ability to detect minute amounts of chemicals in various media. Further, these values should be viewed as tentative given the difficulty of DBDPO analysis, the extremely low levels reported, and the problem of laboratory contamination contributing to measured values. The DBDPO blood levels were far below those of PCB 153 (dismantlers, 760 pmol/g lipid; technicians, 290 pmol/g lipid) measured in the same workers. Further, the electronics dismantling workers' internal DBDPO dose (1.2 ng/kg body weight) based on their measured blood level was comparable to the level expected (0.57 ng/kg body weight) calculated from the measured air levels. A similar comparison was not possible for the computer technicians because air values were not reported for that workplace. In addition, the DBDPO measured air level (0.2 ug/m^3) in the electronics recycling plant was approximately 25,000 times below the acceptable DBDPO workplace exposure level of 5 mg/m^3. This acceptable workplace exposure level, set by the AIHA, was based on an evaluation of DBDPO toxicology data. Thus, no impact on human health from DBDPO is expected in either the electronics dismantlers or computer technicians.

5.1.3 Occupational Exposure Conclusions

DBDPO is used to flame retard synthetic polymers used in electrical and electronic equipment and upholstery. Once encapsulated in a polymer matrix, DBDPO will be essentially unavailable. Therefore, reasonable exposure routes/scenarios are as follows: a) inhalation of dust and/or dermal contact at manufacture and b) at formulation prior to encapsulation in polymer or inclusion in the textile dispersion.

The most likely point at which exposure could occur during manufacture is when the flame retardant is transferred into bags for shipping. Likewise, the point at which worker exposure is most likely during formulation into the polymer dispersion is when the bags of DBDPO are emptied into a hopper prior to mixing the dispersion. Once formulated into the polymer dispersion, DBDPO is encased in the polymer matrix and the potential for worker exposure is negligible.

Theoretically, workplace exposure could occur via the dermal or inhalation routes. DBDPO's low vapor pressure makes vapor inhalation an unrealistic exposure scenario. DBDPO's potential for dermal absorption is low based on its physical and chemical properties and the known requirements for absorption of any compound through the skin. DBDPO's very low water solubility and very high molecular weight effectively precludes any significant skin absorption, and DBDPO's skin absorption is estimated at $<<0.03\%$ of a dermally applied dose. Occupational exposures to dusts may occur; however, DBDPO is a very large poorly absorbed molecule that exhibits little toxicity, and for which AIHA has assigned a WEEL of 5 mg/kg/d. The combined effects of poor absorption and minimal toxicity (NOAEL $\geq$ 1,000 mg/kg/d) indicate adverse effects should not occur as a results of occupational exposure. Nonetheless, workplace controls should focus on points where fine-particle-size-DBDPO may become airborne to limit inhalation exposure. This would be during bagging at manufacture and at formulation prior to inclusion in the resin or polymer dispersion.

5.2 General Population

DBDPO is not sold directly to the public, but may be present in various consumer goods; e.g. electrical and electronic equipment (primary use) and upholstery textiles (secondary use). A typical U.S. example is in the cabinet backs of television sets where DBDPO is used at a level of approximately 12%. Upholstered furniture in commercial settings in the U.S. is required to met federal flammability standards and may utilize upholstery textiles that are flame retarded with a backcoating containing DBDPO at ~5 mg/m^2. Residential furnishings, except in the state of California, are not required to met a comparable standard, although the Consumer Product Safety Committee (CPSC) is considering implementing such a standard. CPSC is also considering a standard for mattresses.

DBDPO exposure to a child could potentially occur via direct contact with consumer products containing DBDPO found in the typical U.S. home. Such exposures include direct dermal contact with fabric, inhalation of vapors and particles that are derived from fabric and electronic equipment, and ingestion following oral contact with both types of products. DBDPO's physical/chemical properties make these unlikely exposure scenarios. In infants or young children, another route could be oral through chewing or sucking on the upholstery textile.

5.2.1. Upholstery Textiles

The U.S. National Academy of Sciences (NAS) was asked by the Congress to evaluate the consumer risk of flame retardants that could be used to meet CPSC's proposed standard for upholstered furniture. The evaluation was published in the document "Toxicological Risks of Selected Flame-Retardant Chemicals" which is available on-line at www.nap.edu (NAS, 2000). DBDPO was one of the flame retardants evaluated (See pages 72-98 of that report), and the NAS's quantitative toxicity assessment of DBDPO is provided in Appendix V.

5.2.2 Electrical and Electronic Equipment

DBDPO has an extremely low vapor pressure, thus vaporization with subsequent inhalation will not occur to any significant extent once incorporated in a polymer matrix such as HIPS or a latex fabric backcoatings. Once encapsulated in a polymer matrix, DBDPO will be essentially unavailable. This prediction is borne out by the barely detectable DBDPO air level, 0.0002 mg/m^3, reported in the Swedish electronics recycling plant (Sjodin et al 1999).

Because of the reasons cited here and in the earlier discussion of dermal absorption, dermal absorption of DBDPO in this context is not anticipated.

5.3 U.S. Monitoring Data

A third potential exposure source is via environmental media (foods, water, air, soil, sediment, dust, etc.). Exposures among the general population are considered to be insignificant (WHO 1994). Nonetheless, for the purposes of this VCCEP program, this potential exposure source is evaluated using the available data. To supplement the monitoring data, DBDPO's predicted

behavior in the environment is also briefly reviewed. DBDPO is expected to partition to soil and sediment (~99%) if released to the environment. DBDPO is expected to bind to particulate matter in soil and sediment and be essentially immobile. DBPDO is not expected to volatilize from water. Waste-water treatment plants are predicted to remove the majority of the DBDPO in the effluent and to do so via sludge adsorption.

Concentrations of polybrominated diphenyl oxides/ethers (PBDPO, PBDEs) have been reported in many environmental and biological samples. However, the majority of studies reported only total PBDPOs or measured only the lower brominated congeners. DBDPO, specifically, has been measured in a smaller subset of studies, most of which were conducted in Europe and Japan. The European Union included a comprehensive compilation of the European and Japanese data (ECB 2002), and these data are also summarized in papers by Hardy (2000) and de Wit (2002). The available data for the U.S. are discussed below.

5.3.1 Sediment

Zweidinger et al. (1978) analyzed sediment near a DBDPO manufacturing site. Levels ranged from N.D. to 14,000 ug/kg. The detection limit was ~ 100 ug/kg. DBDPO was detected at 13 μg/kg in a sample of surface sediment from a sediment core collected from the western basin of Lake Ontario (Alaee 2001).

5.3.2 Sewage Sludge

Hale et al. (2001) reported detectable levels of DBDPO in some sewage sludge samples collected from four different regions in the U.S. DBDPO concentrations ranged from < 75 to 9,160 ug/kg dry wt. DBDPO was also detected in sewage sludge (1,470 ug/kg dry wt) collected from a sewage treatment plant located in a region of the U.S. where DBDPO-treated upholstery textiles are manufactured (Hale et al. 2002).

In some parts of the U.S., sewage sludge undergoes further treatment and is then applied to agricultural soils as a fertilizer. Thus, DBDPO could be present in soils used for agricultural purposes and the potential for its uptake into food crops or by grazing farm animals is considered. No studies have evaluated the potential for uptake of DBDPO by plants, but studies have demonstrated that DBDPO is not toxic to 6 species of terrestrial plants (Porch and Krueger 2001) or to earthworm survival or reproduction (Aufderheide et al. 2001). DBDPO's potential for uptake by plants can be evaluated based on its physical/chemical properties, data on related compounds, and plant physiology.

Although DBDPO data is not available, information is available on polybrominated biphenyl (PBB) plant uptake and translocation. No detectable PBB was found in plants collected from the 10 most highly contaminated fields in Michigan, U.S. (Jacobs et al. 1978). Autoradiograms of corn and soybean seedlings grown in the presence of ^{14}C-PBB showed no translocation (Chou et al. 1978). PBB was found associated with the roots of these plants, but due to the insolubility of PBB in water, the PBB was primarily associated with the root surface (e.g. physically adsorbed to the root's surface). The amount of PBB associated with (not absorbed by) three root crops

(onions, radishes, carrots) grown in two PBB-contaminated soils of differing organic matter and clay content ranged from 0 to a maximum of 0.5% of the soil concentration (Chou et al. 1978).

Three root crops, radishes, carrots, and onions, were grown in two PBB-contaminated soils of differing organic matter and clay content (Chou et al. 1978). No PBB uptake was found, but trace amounts of PBB were associated with the edible portions of each crop (Table 5-3). No PBB were associated with the roots of radishes, carrots or onions grown in high organic carbon soil contaminated with 100 ppb PBB. A maximum of 0.5% of the soil concentration was found in carrots grown on low organic carbon soils contaminated with 100,000 ppb PBB; high organic soil reduced the association to 0.1% of soil concentration. The authors concluded these trace amounts were probably associated with root surfaces, because Iwata et al. (1974) found 97% of polychlorinated biphenyl (PCB) residues in carrot roots in the peel and similar results were reported previously for DDT and other organochlorine pesticides in the soil in which carrots were grown.

Radishes grown in a garden (estimated PBB concentration = 500-1000 ppb) located in a heavily contaminated field (500-1000 ppb) did not contain PBB. Chou et al. concluded: "From these results plus our previous results of greenhouse and field studies in which we found no PBB in plant tops, we conclude that little if any PBB will be transferred from contaminated soil to plant tops. Thus, recontamination of animals from feeds grown in contaminated soil will likely not occur. Although some root crops from very highly contaminated soil might contain traces of PBB, much of this PBB could probably be removed by peeling."

TABLE 5-3. PBB found associated with radish, carrot, and onion roots after 6, 9, and 10 weeks, respectively, of growth in PBB contaminated soil. Detection limit = 0.3 ppb.

Soil Type	PBB Added to Soil (ppb)	PBB in plant roots (ppb)		
		Radishes	Carrots	Onions
Loamy Sand	100	7	20	ND
(Low Carbon)	100,000	49	535	63
Clay Loam	100	ND	ND	ND
(High Carbon)	100,000	44	117	34

Plants may be a source of exogenous chemicals via retention by root surfaces, root uptake and translocation, and foliar uptake. Transfer to animal tissues can occur via soil and herbage ingestion (Wild and Jones 1992). "Assuming degradation of the compound does not occur within the plant, and plant root uptake and translocation of organic chemicals from the soil is passive, plant uptake can be described as a series of consecutive partition reactions. Partitioning occurs between soil solids and soil water, soil water and plant roots, plant roots and transpiration stream, and transpiration stream and plant stem. This partitioning can be related to the octanol:water partition coefficient, such that compounds with high log K_{ow} values (e.g. PAHs, PCBs, PCDD/Fs) are most likely to be sorbed by the soil and/or plant root. Chemicals with lower Kow values are likely to be translocated within the plant and may reach the above ground portions of the plant."

Wild and Jones (1992) go on to state "Relatively few studies have investigated the plant uptake of organic compounds from sludge-amended soils. However, some general comments can be made from the studies: (a) to date studies have been confined to relatively few groups of compounds, namely PCB, PAHs and some other organochlorines; (b) these compounds are generally not taken up into the above-ground portion of crop plants; (c) there is some evidence of slight enrichment of some compounds in some root crops, but the transfers are very inefficient, and consequently the BCFs are very low. Generally enrichments are confined to the root peels which are normally removed before consumption; (d) it is worth noting that the studies to date have focused on compounds which, because of their physico-chemical properties, are thought less likely to be transferred efficiently into crop plants. Future studies should focus on semi-volatile compounds of intermediate log Kow, and some polar compounds."

Based on the screening approach of Wild and Jones (1992), which uses log K_{ow} to predict plant uptake, DBDPO is predicted to have high adsorption to soil, low volatilization from soil, low degradation in soil, low potential for leaching, high retention by root surfaces, low potential for root uptake and translocation, low potential for foliar uptake, high potential for transfer to animal tissues by soil ingestion and low potential for transfer to animal tissues by foliage ingestion.

This screening approach identifies two possible routes of exposure to DBDPO following application of sewage sludge to agricultural soil: retention on root surfaces and transfer to animal tissues by soil ingestion. Based on DBDPO's log K_{ow}, adsorption to root surfaces appears likely. Although it seems likely that DBDPO could adsorb to root surfaces and thereby be ingested, DBDPO's known poor absorption from the gastrointestinal tract (<0.3-2% of the oral dose), makes potential for systemic exposure very low. Similarly, the potential for transfer to animal tissues by soil ingestion is based on the soil half life and log K_{ow}, and does not does not take into account actual animal absorption data. Since DBDPO is known to poorly absorbed from the gastrointestinal tract (<0.3-2% of the oral dose), the potential transfer of DBDPO to animal tissues by soil ingestion is therefore low.

In summary, based on the screening approach of Wild and Jones (1992) and PBB plant uptake data (Chou et al. 1976; Jacobs et al. 1978; Iwata et al. 1974) DBDPO is expected to sorb to root surfaces if present in soil, but not to be transferred into the interior of the root. The amount of DBDPO available for adsorption to roots is expected to be some fraction of the total soil content due to extensive binding to soil particles (K_{oc} = 1.796 x 10^6). DBDPO is not expected to be absorbed into the root nor is it expected to be transferred to foliage. Based on pharmacokinetic data, mammalian uptake of DBDPO after ingesting root crops would be < 0.3-2% of the oral dose. Thus, exposure to DBDPO as a result of its presence in agricultural soils due to the application of sewage sludge is expected to be insignificant.

5.3.3 Air

Sampling over a 3 year period (1997-1999) at 4 locations on the shores of the Great Lakes detected DBDPO only at trace levels in the Chicago filter samples. The average concentration over the three years in the Chicago area was 0.3 pg/m^3 and ranged from 2.0×10^{-7} μg/m^3 to

3.5×10^{-7} μg/m³ (Strandberg et al. 2001). DBDPO was not detected in any of the three years in samples collected from the shores of Lake Superior and Lake Erie, and a site on Lake Michigan farther north than Chicago (D.L. = 0.1 pg/m^3).

Samples of dust and smoke aerosols that settled east of the World Trade Center (WTC) in lower Manhattan after the collapse of the WTC were collected 5 or 6 days after September 11, 2001 were analyzed for a wide variety of components including volatile/semivolatile organic compounds, metals, polychlorinated dioxins and furans, polychlorinated biphenyls and PBDEs. DBDPO was detected in all three samples ranging in concentrations from 1,330 to 2,660 ug/kg dry weight (Lioy et al 2002).

5.3.4 Poultry, Meat and Dairy Products

Due to its poor bioavailability, poultry, meat and dairy products are not expected to be a human exposure source of DBDPO. Only limited monitoring data is available. DBDPO was not elevated above background levels (0.87 ng) in most samples of chicken fat (n=13) collected from four different areas of the U.S.; matrix and laboratory blanks contained low, but detectable, levels of DBDPO. Tetra- (0.56-10.58 ng/g), penta- (0.42-16.97 ng/g), and hexaBDE (0.02-4.63 ng/g) congeners were generally present at 3-100 times the background. Mono- to deca-brominated congeners were not detected in chicken feed or its ball clay additive (Huwe et al. 2002).

5.3.5 Fish

Fish consumption is not expected to be a source of human exposure to DBDPO. DBDPO has not been detected in fish collected in the U.S. (Table 5-4). DBDPO has negligible water solubility (<0.1 ug/L) and will preferentially partition to soil and sediment in the environment. Thus, any exposure to fish via water will be low. Further, DBDPO has been shown not to bioconcentrate in fish (CITI 1992; Kierkegaard et al. 1997, 1999). Bottom feeding species could conceivably be exposed to DBDPO-containing sediment. However, limited uptake by these species is expected due to DBDPO-sediment binding and DBDPO's large molecular weight and size. Thus, any exposure to humans would be extremely limited.

TABLE 5-4. Analytical results of freshwater fish collected in U.S. waters for DBDPO content.

Species	Location & Year Collected	DBDPO Level	Number of Samples	Reference
Various Fish spp.	3 Lakes, US, 2000	N.D. (D.L. = 1.5 ng/g wet wt)	20	Dodder et al. 2001. Environ. Sci. Technol., 36:146-151.
Carp	River, US, 1991	N.D. (D.L. = 0.1 ug/kg wet wt)	48	Loganathan, B et al. 1995. Environ. Sci. Technol., 29:1832-1838.
Salmon	Alaska, 2000*	N.D. (D.L. = 0.65 pg/g wet wt)	2	Easton et al. 2002. Chemosphere, 46:1053-1074.

*DBDPO was also not detected in 6 samples of wild or farmed salmon or fish-food collected in Canada in 2000. D.L. = 0.65 pg/g wet wt.

5.3.6 Human Tissues

A few studies have analyzed U.S. human adipose tissue, serum, and hair for the presence of DBDPO.

Responses were noted that corresponded to qualitative criteria for hexa- through octabromodiphenyl oxide congeners in adipose tissue collected from the general U.S. population in fiscal year 1987. This adipose tissues was collected as a part of the National Human Adipose Tissue Survey (NHATS 1990). A subsequent study analyzed selected composites from the 1987 NHATS repository (Cramer et al. 1990; Stanley et al. 1991). The presence of hexa- through octabromo congeners was confirmed, and nonabromo- and DBPDO were also identified. DBDPO was detected in 3 of the five extracts analyzed. The concentrations ranged from N.D. to 700 pg/g adipose.

Twelve samples collected in 1988 from a general population of U.S. blood donors in the Midwest were analyzed approximately 10 years later for DBDPO content. DBDPO concentrations in the serum ranged from <1 pmol/g lipid to 35 pmol/g lipid (equivalent to < 0.96 ng/g lipid to 33.6 ng/g lipid) (Sjödin et al. 2001b). Only five of the twelve samples were above the limit of quantification (LOQ = 1 pmol/g lipid).

Out of three composite hair samples collected in a barbershop located in the vicinity of DBDPO manufacture, one composite had a DBDPO concentration of 5 μg/kg, and a second had a low level of DBDPO detected, but not quantified (DeCarlo 1979).

5.3.7 Breast Milk

As discussed in Section 1, DBDPO has not been reported in breast milk. DBDPO is not expected to be transferred to breast milk based on its physical/chemical properties, pharmacokinetics and the physiology of milk production. These factors, along with a summary of polybrominated diphenyl oxide/ether (PBDPO; PBDE) isomers/congeners reported in breast milk are discussed below.

5.3.7.1 Transfer into Breast Milk

Published information on the physiology of xenobiotic excretion into breast milk is generally limited to pharmaceuticals; however, this information is relevant to all xenobiotics (Anderson 1991; Pons et al. 1994; Loebstein et al. 1997; Bailey and Ito 1997; Wilson 1983). Excretion into breast milk depends mainly on the passive diffusion of the unionized unbound drug from the bloodstream into milk along a concentration gradient. Although active or facilitated transport has been described for some endogenous substances across some membranes in the body, no drugs are known to enter human milk by these mechanisms. Because most drugs are excreted into milk by passive diffusion, the drug concentration in milk is directly proportional to the corresponding concentration in maternal plasma. Diffusion into milk is a minor route of drug elimination (usually <1% of a maternal dose), and, generally, drugs given to nursing mothers

reach infants in much smaller amounts than drugs given to pregnant women. For most drugs the amount ingested by the infant rarely attains therapeutic levels.

Drugs pass across the mammary epithelium by passive diffusion down a concentration gradient formed by the nonionized free drug on each side of the cell membrane. The membrane acts as a semipermeable lipid barrier, similar to other membranes in the body. Transit through membranes is via the lipid portion (for unionized drugs with high lipid solubility) or via water filled pores surrounded by proteins (for water soluble presumably low molecular weight drugs). Once inside the mammary alveolar cell, a drug may be metabolized. Drug in the alveolar cell may be expelled into the milk-containing lumen concomitantly with secretion of fat droplets and protein granules. Drug reuptake from the milk into plasma occurs and there is rapid bi-directional diffusion and rapid equilibration of drug between plasma and milk for the majority of drugs.

Passive diffusion of xenobiotics into breast milk is affected mainly by the drug's disposition in lactating mothers, by the physicochemical properties of the molecule and by the protein and lipid contents of breast milk. Drugs with molecular weights (< 200 D) diffuse more readily than drugs with larger molecular weights. Small, e.g. <200 D, highly lipid-soluble, unionized drugs are expected to diffuse more rapidly than other drugs. Lipid soluble drugs concentrate in milk lipids, and their milk to plasma concentration ratio is dependent on the lipid concentration of milk. Protein content is lower in milk (8-9 g/L) than in plasma (75 g/L), and protein binding in milk is thus lower than protein binding in plasma.

The complexity of the fluids on both sides of the mammary epithelium results in several simultaneous equilibration processes: ion trapping, protein binding, and lipid partitioning.

5.3.7.2 Ion Trapping

The most important equilibration process occurs across the mammary epithelium between the unbound, nonionized drug in the bloodstream and the aqueous phase of the milk. Because the pH of milk is typically slightly acid relative to that of plasma, the pH partition theory (Henderson-Haslebach equation) predicts that the ionized form of a weak base concentrates in milk in a process commonly called "ion trapping". Conversely, a weak acid is "trapped" in plasma because of the relatively greater concentration of the ionized form there. Ion trapping affects weak acids with a pKa of ~ 8 or less and weak bases with a pKa of 6 or greater. Weaker acids and bases act as noneletrolytes and do not undergo ion trapping.

Ion trapping will not affect DBDPO concentrations since it has no ionizable groups.

5.3.7.3 Protein Binding

Both plasma and milk contain proteins that can bind drugs. The total plasma protein concentration is ~ 75 g/L, whereas milk contains ~ 8-9 g/L. Of the plasma proteins, 45 g/L is albumin, a major drug-binding protein. In contrast, the albumin concentration in milk is only ~0.4 mg/L, and the major proteins in milk are casein, alpha-lactalbumin, lactoferrin, and

immunoglobulin A. Casein is apparently the major drug-binding protein, but none of these proteins binds drugs well and quantitatively important binding of drugs to milk proteins does not occur except in the case of drugs that are also extensively bound to plasma proteins. The net effect of protein binding is that highly protein-bound drugs tend to remain in the plasma and pass into the milk in low concentrations.

Protein binding is not expected to affect DBDPO concentrations. DBDPO is not known to be protein bound, but protein binding would serve to decrease DBDPO available for transfer into milk.

5.3.7.4 Lipid Partitioning

Unlike plasma, milk contains emulsified fat, ranging from 3-5%. Milk fat has the potential to concentrate lipid-soluble drugs, causing the total amount of drug in milk to increase. For highly lipid-soluble drugs such as diazepam and chlorpromazine well over half of the total amount of drug in milk is found in milk fat. Nevertheless, because the amount of fat in milk is low compared with the total volume of milk, the net effect of lipid partitioning on the total amount of drug reaching the infant is usually relatively small.

Lipid partitioning is not expected to affect DBDPO concentrations. DBDPO does not show an appreciable affinity for lipids.

5.3.7.5 Non-Steady State Conditions

The previous discussion relates to constant, steady state conditions in which plasma and milk drug concentrations have come to equilibrium. Because constant plasma concentrations are the exception, other factors must be taken into account during intermittent drug administration to the mother. The shorter the half-life of a drug, the greater the fluctuations in plasma concentrations during intermittent administration. A drug that enters the milk rapidly will achieve a greater initial concentration in milk relative to the plasma concentration than a drug that enters slowly. Because milk is produced and periodically emptied from the breast during nursing, slowly equilibrating drugs may never achieve high concentrations in milk. The physicochemical factors that determine the rate of passage into milk are the drug's lipid solubility and its molecular weight. Lipid solubility is important because the drug must dissolve in the lipid mammary epithelial cell membrane (on both entering and exiting the cell), whereas low molecular weight favors rapid diffusion across the aqueous interior of the cell. Another factor that comes into play during intermittent drug administration is retrograde diffusion of drugs from the milk back into plasma. The rate and extent of retrograde diffusion are determined by the same physicochemical factors governing passage from the plasma into milk. Many have the impression that once a drug has passed into milk, it will remain until the breast is emptied. However, because of retrograde diffusion, this is not the case.

5.3.7.6 Impact of Disposition on PBDE, PBB, PCB Content in Milk

DBDPO has not been reported in breast milk. Other brominated aromatics have been detected in milk, and include polybrominated diphenyl oxides/ethers (PBDPO, PBDE), polybrominated biphenyls (PBB) and polychlorinated biphenyls (PCB). Information on the pharmacokinetics and milk concentrations of these compounds can be of value in understanding the potential for DBDPO to be eliminated in milk.

Passive diffusion of substances into breast milk is affected by the substance's disposition in the lactating mother. Halogen content affects the absorption of PBDE/PBDPO, PBB and PCB congeners from the maternal gut and their subsequent transfer from plasma to depot fat. Studies with PCB have shown that the number of chlorines on the biphenyl molecule generally affects absorption, excretion, and toxicity. In addition, the behavior and toxicity of some chlorinated biphenyls is influenced by the chlorine atoms' position on the biphenyl molecule. Similar differences have been observed with the commercial PBDE/PBDPO products, individual PBDE/PBDPO isomers, and individual PBB congeners.

Maternal Oral Absorption. As a general rule, the percent absorption of PBBs and PBDE/PBDPOs declines with increasing halogenation (Fig 5.1). DBDPO, with 10 bromine atoms, is very poorly absorbed (<0.3-2% of an oral dose) (El Dareer et al. 1987). Only ~ 35% of octabromobiphenyl (OBB; 8 bromine atoms/molecules) was absorbed (Norris et al. 1973). In contrast, approximately 90% of an oral dose of 2,2',4,4',5,5'-HexaBB (the major component of the former PBB product known as FireMaster BP-6) was absorbed from the intestine (Matthews et al. 1977). When FireMaster BP-6 was fed to dairy cattle, a large proportion of its HeptaBB content was apparently excreted in feces without absorption (Willet and Durst 1978). The opposite occurred with the PentaBB component of FireMaster BP-6. PentaBB was more efficiently absorbed, and obtained equilibrium between plasma and tissues at a higher relative concentration than its concentration in the FireMaster BP-6 test article. A high absorption of ^{14}C-2,2',4,4'-TetraBDE and ^{14}C-2,2',4,4',5-PentaBDE was also reported (Orn and Klassen-Wheler, 1998; Hakk et al., 1999).

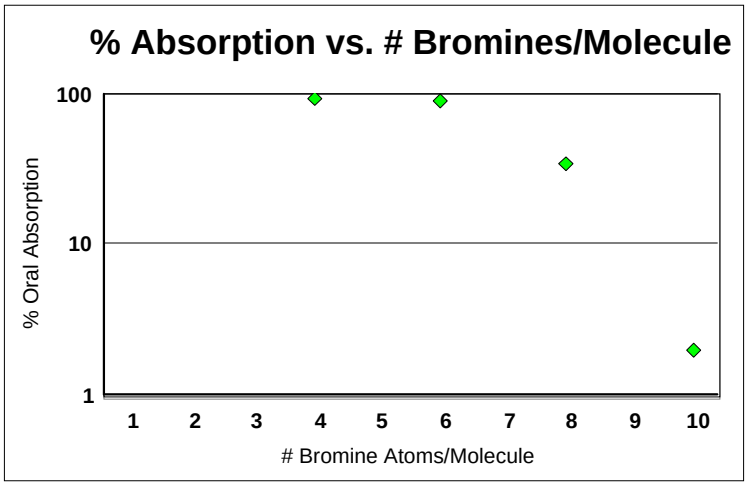

Figure 5-1. Decline in the percent of the oral absorption of PBB or PBDE/PBDPO congeners with increasing bromine content in the molecule.

Maternal Fecal Elimination. Elimination is influenced by both the degree and position of halogenation on the biphenyl or diphenyl oxide molecule, and as a general rule, increases as the number of bromines increases on the biphenyl or diphenyl oxide molecule (Figure 5.2). DBDPO was rapidly eliminated in the feces (>99% of the dose in 72 hr) with a half-life of ~ 24 hours (NTP 1986; El Dareer et al. 1987). Rats also eliminated OBB rapidly. After a single oral dose of ^{14}C-OBB, 65% of the isotope appeared in the feces in 1 day and a total of 73% was excreted in feces in 16 days (Norris et al. 1973 as cited by Di Carlo et al. 1978). In contrast, excretion of ^{14}C-2,2',4,4',5,'5-HexaBB by rats was extremely slow. After intravenous administration of a single dose, only 6.6% of the label was excreted in feces over a period of 6 weeks and the total urinary excretion was less than 0.1%. Mathematical extrapolation of the excretion data "indicates that only 9.5% of the total PBB dose would ever be excreted in the feces." Thus, elimination of 2,2'4,4',5,5'-HexaBB appeared to depend both on the number of bromines and their position (Matthews 1977 as cited by Di Carlo et al. 1978). A similar pattern was found for 2,2'4,4',5',5-HexaCB: only 2% of this hexa-chlorinated biphenyl was eliminated in the feces in 21 days. In contrast, 2,2',3,3',6,6'-HexaCB was readily metabolized and cleared (~93% of the dose). Thus, for hexa-halogenated biphenyls, the position of the halogens appears to be a very important determinant of clearance. 2,2',4,4',5-PentaBDE was poorly metabolized in the rat, but 43% of the oral dose was excreted within 3 days (Hakk et al. 1999). 2,2',4,4'-TetraBDE was readily absorbed, poorly metabolized and slowly eliminated by the rat with less than 0.5% of the oral dose eliminated in 5 days (Orn and Klassen-Wheler 1998).

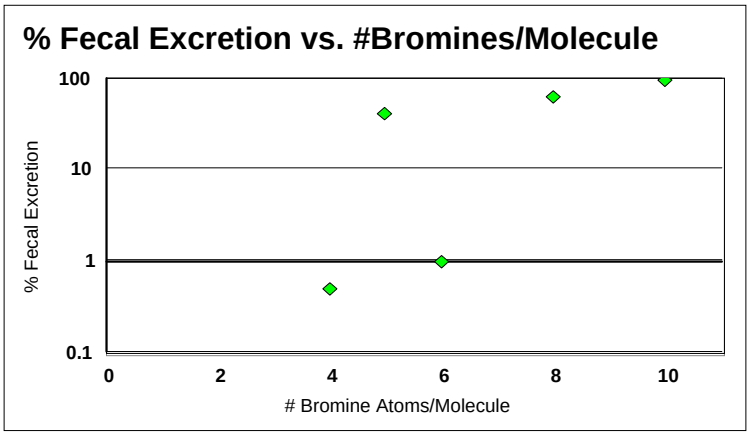

Figure 5-2. Increase in the percent excretion of PBB or PBDE/PBDPO congeners in the feces within 24-72 hr of dosing with increasing bromine content in the molecule.

Elimination in Milk. Following feeding FireMaster BP-6 (250 mg/kg/day for 60 days) to dairy cattle, fecal clearance of the HexaBB component was 0.7 mg/day in non-lactating cows whereas 2 mg/day were cleared in milk by lactating dairy cows. Thus, 2,2',4,4',5,5'-HexaBB was eliminated via milk at 3 times the rate in feces, although its total elimination was very low. Further, high relative concentrations of the PentaBB component present in the FireMaster product were detected in milk fat, but its HeptaBB component was virtually undetectable in milk

(Willet and Durst 1978). Relative to intake, five times more HexaBB than HeptaBB was transferred to milk when their concentrations were normalized to equal intakes of each. A similar relationship was found with hen eggs for PBB and milk for PCB: the more highly halogenated components were less efficiently transferred to milk and eggs. The less halogenated components of PBB and PCB more readily diffuse across biological membranes than the more halogenated compounds (Fries et al. 1978).

Thus, halogen content influences the absorption of PBDE/PBDPOs, PBBs and PCBs from the gut and their subsequent elimination. With the brominated compounds, a breakpoint in absorption and elimination (via feces and/or milk) occurs at 5-6 bromines/molecule. Congeners containing 4 to 5-6 bromines appear relatively well absorbed and slowly eliminated from the body. The major excretory route for these congeners is via the feces, with an added route being milk in lactating females. Congeners containing $\geq$7 bromines appear poorly absorbed and rapidly eliminated via the feces. Excretion in milk is not an important elimination pathway for these congeners.

5.3.7.7 Potential for Transfer of DBDPO into Breast Milk

Prior to transfer to breast milk, a substance must first be absorbed into the mother's bloodstream and presented to the mammary epithelium. DBDPO is very poorly absorbed (< 0.3-2% of an oral dose) and rapidly eliminated (> 99% of the dose within 72 hr). Low absorption coupled with rapid elimination will effectively limit the amount of DBDPO in the mother's bloodstream and available for transfer to breast milk.

Further, DBDPO is a large bulky molecule so that transfer to breast milk is likely to be slow and limited, if at all. Its concentration in breast milk is not expected to be affected by ion trapping, protein binding or lipid partitioning in the milk. DBDPO has no ionizable groups, is not known to undergo protein binding, and shows no preferential partitioning to lipids. Build-up of concentrations in breast milk over extended periods of time is not expected due to DBDPO's predicted slow diffusion into milk and periodic emptying of breast milk. This combination of low absorption from the maternal gut, rapid elimination in the maternal feces, and poor and/or slow diffusion into breast milk will effectively preclude DBDPO in breast milk. Thus, a risk to the nursing infant is not anticipated.

5.3.7.8 Measured PBDPO/PBDE Levels in Breast Milk

DBDPO has not been reported in breast milk. Other polybrominated diphenyl oxide/ether (PBDPO/PBDE) congeners have been reported, and to avoid confusion, publications (through 2001) are summarized here.

The congeners reported in human breast milk include tetrabromodiphenyl ether (TetraBDE), pentabromodiphenyl ether (PentaBDE) and hexabromodiphenyl ether (HexaBDE). Most of the data is derived from Europe, and only limited U.S. data is available. Unfortunately, problems with sampling and analysis methodology, incomplete reporting, non-representative sampling, sampling duration, and small sample size typically limit the value of these studies. The results

are generally reported as the total PBDE content derived as the sum of the congeners/isomers detected.

Analytical results of breast milk collected from Swedish, German, Finnish, Japanese, Canadian, and American women have been reported. The PBDE congeners detected in breast milk include the tri- to HeptaBDEs (Figure 5.3). PBDE congeners with higher levels of bromination (e.g. $\geq$ 7 bromine atoms) have not been reported, and are not expected based on their high molecular weights and their limited potential for absorption into the body and subsequent diffusion into breast milk. The predominant isomer reported in breast milk is 2,2',4,4'-TetraBDE, and generally accounts for 50-70% of the total PBDE content. The next most common isomer detected is 2,2',4,4',5-PentaBDE, followed by 2,2',4,4',6-PentaBDE or 2,2'4,4',5,5'/6-HexaBDE.

Mean levels in breast milk, reported as the total of all PBDEs detected as of 2000, are variable (Table 5.5). Mean levels reported in Canada (Ryan and Patry 2001), Germany (Furst 2001), Finland (Strandman et al. 2001), Sweden (Lind et al. 2001; Meironyte et al. 1998; Darnerud et al. 1998)

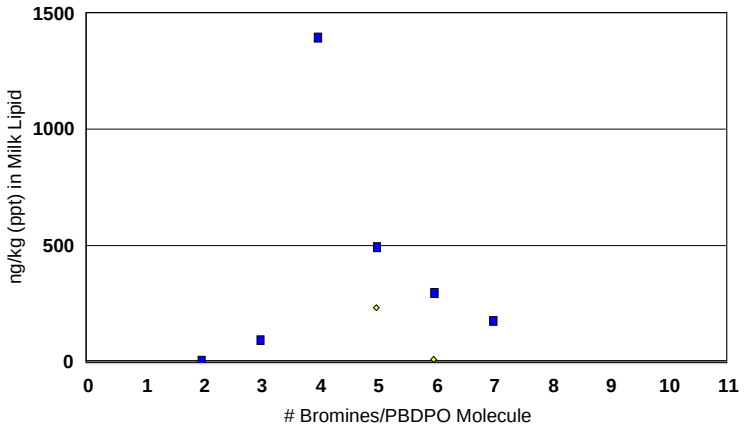

Figure 5-3. PBDE/PBDPO congener content in Canadian breast milk samples collected in 1992 (n=72). The congener content in milk declines with increasing bromine content in the congener.

and Japan (Ohta et al. 1998) range from 1.1-4.4 ng/g lipid (ppb). Levels recently reported for breast milk collected in Austin, TX and Denver, CO in 2000 are 204 ng/g lipid (Papke 2001), and is obviously outside the range of that reported in other countries. The cause of this outlier is unknown but could be related to exposure, diet, age, analytical methodology, unequal sample size, sample contamination or other factors. These breast milk samples were collected for a PCB survey and a subsample analyzed as an afterthought for PBDE content.

Recent results from Sweden (Table 5.5) indicate total PBDE concentrations in breast milk are declining (Lind et al. 2001). This is a reversal of the trend seen from 1975-1997 in which a

doubling of the levels every 5 years was reported (Meironyte et al. 1998; Darnerud et al. 1998). Levels in breast milk collected in Germany in 1992 and 2000 were similar (e.g. did not increase with time) and represent the lowest level reported in these 6 countries (Furst 2001). Thus, based on this very limited sample, total PBDE levels in breast milk in Europe do not appear to be increasing.

In Swedish breast milk, the highest concentration of PBDEs, reported for the year 1997, was approximately 0.36 % of that of the highest PCB level ever measured in Swedish milk samples (Meironyte et al. 1998; Darnerud et al. 1998). The 1997 total PBDE content in breast milk was ~100 times less than that of PCB. The authors concluded the PBDE content was unlikely to alter the sum of the toxicity equivalents (TEQs) represented in milk by the PCDDs, PCDFs, and PCBs. The authors initially found no correlation between the levels in Swedish breast milk and the mother's age, computer usage frequency, consumption of fish (total or specifically fatty Baltic fish), consumption of alcohol, place of residence during the mother's childhood and adolescence (in fishing village or not) or the birth weight of the child (Darnerud et al. 1998). A correlation was found between the total PBDPO levels in breast milk and the mothers' smoking habits and body mass - an increase in smoking correlated with an increase in total amount of PBDE. An update indicated that the major source of PBDE was from fish consumption with a weak association to smoking (Lind et al. 2001). Age, body mass index and alcohol showed no correlation with PBDE levels in breast milk. In contrast, Ryan and Patry (2001) calculated that most of the exposure to PBDEs (~80%) subsequently detected in Canadian breast milk was through meat consumption. Whether these differences are due to patterns of food consumption in Sweden versus Canada or some other factor is not known.

TABLE 5-5. Mean total PBDE/PBDPO content in breast milk (ng/g (ppb) milk fat).

Year Collected	Canada (n=72)	US (n=?)	Germany (n=?)	Finland (n=11)	Sweden (n=20)*	Japan (n=6)
1981-2						
1992	2.4		1.1			
1997					4.4+	
1998				2.25	3.88	
1999					3.46	
2000		205	1.1		2.79	
?						1.12

*1998, 1999, 2000
+n = 39

In summary, analytical results of breast milk collected from Swedish, German, Finnish, Japanese, Canadian, and American women have been reported. The PBDE congeners detected in breast milk include the tri- to HeptaBDEs. The predominant isomer reported in breast milk is 2,2',4,4'-TetraBDE, and generally accounts for 50-70% of the total PBDE content. Levels of total "PBDE" content in breast are generally far below that of total PCB content measured in the same samples. DBDPO, which makes up approximately 82% of "PBDEs" sold worldwide, has not been reported in breast milk, and is not expected to be present based on its physical/chemical

properties and limited potential for absorption into the body and subsequent diffusion into breast milk.

Except in unusual situations, breast feeding remains the preferred nutrition for the infant and a better understanding of the levels of environmental chemicals in breast milk, particularly in the United States, would be of value in predicting infant exposures (LaKind and Berlin 2001). A carefully planned and executed program of breast milk sampling and analysis would provide such information. An Expert Panel on Breast Milk Monitoring for Chemicals in Human Milk was convened in 2002 to develop harmonized guidelines for the surveillance and study of human milk for environmental chemicals in the U.S. The results are reported in a special issue of the Journal of Toxicology and Environmental Health ("Technical Workshop on Human Milk Surveillance and Research on Environmental Chemicals in the United States", Volume 65, Number 22, November 22, 2002). BFRIP contributed financial support to this panel.

5.3.8 Occupational

The American Industrial Hygiene Association (AIHA) set a Workplace Environmental Exposure Level (WEEL) for DBDPO of 5 mg/m³. The U.S. Occupational Safety and Health Administration (OSHA) has not set a Permissible Exposure Level (PEL) for this specific chemical, but has a "nuisance dust" limit of 5 mg/m³. Most industrial hygiene surveys determined employee 8-hour time-weighted average (TWA) exposures to DBDPO to be in the 1–4 mg/m³ range, with possible excursions as high as 42 mg/m³ during certain tasks (AIHA 1981). During operations involving dumping the material into hoppers, airborne concentrations reached as high as 400 mg/m³ (AIHA 1981). During these operations, workers were required to wear respirators.

5.4 U.S. Toxic Inventory Release Data

The Toxics Release Inventory (TRI) is a publicly available EPA database that contains information on chemical releases and other waste management activities reported annually by certain covered industry groups as well as federal facilities (http://www.epa.gov.tri). This inventory was established under the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA) and expanded by the Pollution Prevention Act of 1990. TRI stores the information that is self-reported annually from industries that conduct manufacturing operations within certain specified standard industrial classification codes (SIC Codes 20-39) until the 1998 reports when additional sectors were added. In addition to industrial classification, facilities are only required to report if they manufacture or process more than 25,000 pounds of a listed chemical during a year, or otherwise used more than 10,000 pounds, and have the equivalent of more than 10 full-time employees. They must report the on-site releases of toxic chemicals into the air, water, and land; and quantities treated, combusted for energy recovery, and recycled on-site. They also report on transfers of wastes that are disposed, treated, combusted for energy recovery, or recycled at a separate facility. Approximately 650 chemicals have been designated for reports under TRI. In all about 73,000 reports are submitted annually by 21,000 manufacturing facilities and 200 Federal facilities in 1996.

According to the U.S. EPA, TRI data have certain limitations. TRI data reflect releases and other waste management of chemicals, and not exposures of the public to those chemicals. TRI data alone are not sufficient to determine exposure or to calculate potential adverse effects on human health and the environment. TRI data, in conjunction with other information, can be used as a starting point in evaluating exposures that may result from release and other waste management activities, which involve toxic chemicals.

Definitions provided by EPA for the terms used in describing releases are:

- Total Air Emissions: This is the sum of fugitive air and stack air release amounts (in pounds for all chemicals other than Dioxin and Dioxin-like compounds. The data for Dioxin and Dioxin-like compounds is in grams).
- Surface Water Discharges: Releases to water include discharges to streams, rivers, lakes, oceans, and other bodies of water. This includes releases from contained sources, such as industrial process outflow pipes or open trenches. Releases due to runoff, including storm water runoff are also reportable to TRI.
- Underground Injections: Underground injection is the subsurface emplacement of fluids through wells. TRI chemicals associated with manufacturing, the petroleum industry, mining, commercial and service industries, and Federal and municipal government related activities may be injected into class I, II, III, IV, or V wells, if they do not endanger underground sources of drinking water (USDW), public health or the environment. Class I wells are industrial, municipal, and manufacturing related wells which inject fluids into deep, confined and isolated formations below potable water supplies.
- Releases to Land: Disposal to land on site is the release of a chemical to land within the boundaries of the reporting facility. Releases to land include disposal of toxic chemicals in landfills (in which wastes are buried), land treatment/application farming (in which a waste containing a listed chemical is applied to or incorporated into soil), surface impoundments (which are uncovered holding areas used to volatilize and/or settle materials), and other land disposal methods (such as waste piles) or releases to land (such as spills or leaks).
- Total On-site Releases: This field is the sum of total air emissions, surface water discharges, underground injections, and releases to land.
- Total Off-site Releases: Off-site releases are from Section 6 (transfers off-site to disposal) of the Form R. Off-site releases include metals and metal compounds transferred off-site for solidification/stabilization and for waste water treatment, including to POTWs.
- Total On- and Off-site Releases: This field is the sum of total on-site release and total off-site release amounts (in pounds).

DBDPO was included on the original inventory created under TRI. Annual DBDPO releases reported from1988 through 2000 or for the year 2000 are shown in Tables 5-6, 5-7, and 5-8. Annual total on- and off-site releases for DBDPO's manufacture and use throughout the U.S. are ~1% of DBDPO's manufactured volume. As expected, air emissions make up only a small fraction of the total in any given year and are largely associated with DBDPO's manufacture (Chemical, SIC 29). On-site surface water discharges also make up only a small fraction of the total, and are nearly all associated with operations (Textiles, SIC 28) that apply DBDPO to

upholstery textiles. Again, this is as expected because textile operations utilize water in their processes whereas DBDPO manufacturing and plastics applications (Plastics, SIC 30) do not. Releases to land dominate on-site releases and are predominantly associated with disposal of DBDPO in either a manufacturer's on-site landfill or in a commercial chemical landfill. The manufacturer's landfill is built to hazardous waste standards with a double liner and leachate collection system. Off-site releases for disposal typically exceed on-site releases.

TABLE 5-6. TRI On-site and Off-site Reported Releases (in pounds), Trend Report for facilities in Original Industries (SIC codes 20-39), DBDPO, U.S., 1988-2000.

Year	Air Emissions	Surface Water Discharges	Underground Injections	Releases to Land	Total On-Site Releases	Total Off-Site Releases	Total On- and Off-Site Releases
1988	29,604	500	292	21,450	51,846	555,181	607,027
1989	50,207	3,450	52	9,394	63,103	749,567	812,670
1990	64,601	2,577	43	24,844	92,065	710,187	802,252
1991	50,235	3,817	38	220,075	274,165	839,031	1,113,196
1992	37,217	3,873	285	529,340	570,715	721,583	1,292,298
1993	203,168	2,176	39	506,785	712,168	856,809	1,568,977
1994	170,122	1,958	40	298,191	470,311	998,628	1,468,939
1995	39,283	3,846	11	204,248	247,388	716,245	963,633
1996	45,608	3,680	0	196,688	245,976	707,498	953,474
1997	29,549	2,499	.	869,294	901,342	726,500	1,627,842
1998	31,114	3,168	0	191,253	225,535	773,136	998,671
1999	116,241	2,701	0	396,169	515,111	916,182	1,431,293
2000	106,219	9,006	0	487,409	602,634	1,006,690	1,609,324

TABLE 5-7. TRI On-site and Off-site Reported Releases (in pounds), Trend Report for facilities in New Industries (SIC codes 10, 12, 4911, 4931, 4939, 5169, 5171, 4953, 7389), DBDPO, U.S., 1998-2000.

Year	Air Emissions	Surface Water Discharges	Underground Injections	Releases to Land	Total On-Site Releases	Total Off-Site Releases	Total On- and Off-Site Releases
1998	0	.	.	310,000	310,000	.	310,000
1999	0	.	0	350,000	350,000	.	350,000
2000	0	.	0	400,837	400,837	.	400,837

TABLE 5-8. TRI On-site and Off-site Reported Releases (in pounds), DBDPO, By Industry, U.S., 2000.

SIC-Industry	Air Emissions	Surface Water Discharges	Underground Injections	Releases to Land	Total On-Site Releases	Total Off-Site Releases	Total On- and Off-Site Releases
28 Chemicals	88,811	5	0	370,578	459,394	102,540	561,934
30 Plastics	8,417	34	.	15,653	24,104	470,632	494,736
4953/7389 RCRA/Solvent Recovery	0	.	0	400,837	400,837	.	400,837
22 Textiles	4,314	8,933	.	90,496	103,743	136,489	240,232
26 Paper	.	5	.	1,100	1,105	116,326	117,431
33 Primary Metals	59	0	.	.	59	68,948	69,007
34 Fabricated Metals	41	0	.	.	41	60,960	61,001
36 Electrical Equip.	27	.	.	.	27	17,798	17,825
35 Machinery	.	.	.	7,982	7,982	7,983	15,965
Multiple Codes 20-39	3,800	2	.	1,600	5,402	8,904	14,306
37 Transportation Equip.	.	.	.	.	.	13,000	13,000
32 Stone/Clay/Glass	750	27	.	.	777	3,110	3,887
Total	106,219	9,006	0	888,246	1,003,471	1,006,690	2,010,161

In 2000, total on- and off-site DBDPO releases in the 50 states were dominated in descending order by Arkansas, Louisiana, North Carolina, Pennsylvania, California and Ohio (data not shown). These 6 states reported ~ 76% of the total releases in the U.S. Arkansas, home to the 2 DBDPO manufacturing facilities in the U.S., reported the highest total releases with the majority going to land in one manufacturer's on-site landfill. Releases in Louisiana consisted of land releases to a commercial chemical landfill for disposal. Releases in Pennsylvania, California and Ohio were primarily off-site for disposal, a category that includes discharges to publicly owned treatment works (POTWs). North and South Carolina accounted for 92.5% of the nation's discharges to surface waters. These trends are generally applicable to previous years (data not shown).

EPA defines transfers off-site for further waste management as follows:

- Transfers to Recycling: The total amount (in pounds) of the chemical transferred from the facility to a off-site location during the calendar year (January 1 - December 31) for recycling. This refers to the ultimate disposition of the chemical, not the intermediate activities used for the waste stream.
- Transfers to Energy Recovery: The total amount (in pounds) of the chemical transferred from the facility to a off-site location during the calendar year (January 1 - December 31) for energy recovery. This refers to the ultimate disposition of the chemical, not the intermediate activities used for the waste stream.
- Transfers to Treatment: The total amount (in pounds) of the chemical transferred from the facility to a off-site location during the calendar year (January 1 - December 31) for treatment. This refers to the ultimate disposition of the chemical, not the intermediate activities used for the waste stream.
- Transfers to POTWs: The total amount (in pounds) of the chemical transferred from the facility to all Publicly Owned Treatment Works (POTWs) during the calendar year (January1 - December 31). POTW refers to a municipal sewage treatment plant. The most common transfers will be conveyances of the chemical in facility wastewater through underground sewage pipes, however, trucked or other direct shipments to a POTW are also included in this estimate.
- Other Off-site Transfers: In TRI Explorer, chemicals in waste that were reported as transferred off-site but for which the off-site activity (i.e., recycling, energy recovery, treatment, or disposal) was not specified or was not an accepted code has been classified as "other off-site transfers."
- Total Transfers Off-site for Further Waste Management: This field is the sum of transfers to recycling, transfers to energy recovery, transfers to treatment, transfers to POTWs, and other off-site transfers amounts (in pound).

Total waste transfers of DBDPO off-site for further waste management are generally dominated by transfers to POTWs (Table 5-9). Between 1991 and 2000, waste transferred off-site to POTWs ranged from 22-63% of the aggregate waste transfers. However, in 2000, a significant rise in transfers going to recycling occurred and these recycling transfers were slightly over twice that to POTWs. Connecticut, North Carolina and Washington accounted for 79% of this recycling, and of Chemicals (SIC 29), Textiles (SIC 28) and Plastics (SIC 30), Textiles

accounted for the largest share of the growth in recycling with a corresponding drop in that sent to POTWs. Transfers from New Industries between 1998 and 2000 were solely to treatment and did not account for a significant portion of each year's total. Textiles were responsible for the bulk of the waste transfer to POTWs between 1991-2000 (Tables 5-10, 5-11, 5-13), whereas Plastics released minimal amounts to POTWs (Tables 5-10 and 5-12). The amounts sent to POTWs by Textiles have declined over the past 3 years. The states with the largest amounts of DBDPO waste transferred to POTWs in 2000 were Maryland, South Carolina and North Carolina, in descending order. These three states accounted for ~71% of all DBDPO-waste transfers to POTWs in 2000.

In summary, land releases dominate DBDPO releases on-site from its manufacture and use, and are predominantly associated with disposal of DBDPO in either a manufacturer's on-site landfill in Arkansas or in a commercial chemical landfill in Louisiana. Air and water releases make up only a small fraction of on-site releases. DBDPO releases off-site for disposal from its manufacture and use are typically larger than that on-site. DBDPO-waste transfers for further waste management are dominated by transfers to POTWs. The largest releases of DBDPO or DBDPO-waste to water occur in North and South Carolina and result from its use in textile applications. These water releases occur as a result of discharge of DBDPO to surface water and DBDPO-waste to POTWs. DBDPO is expected to settle out of surface water or in a POTW to sediment or sludge, respectively, and bind extensively to organic carbon (see Section 4.2).

TABLE 5-9. TRI Transfers Off-site for Further Waste Management (in pounds), Trend Report for facilities in Original Industries (SIC codes 20-39), DBDPO, U.S., 1991-2000.

Year	Transfers to Recycling	Transfers to Energy Recovery	Transfers to Treatment	Transfers to POTWs	Other Off-site Transfers	Total Transfers Off-site for Further Waste Management
1991	68,313	8,551	71,567	44,914	0	193,345
1992	31,032	7,406	93,759	127,772	.	259,969
1993	35,105	8,129	73,725	203,871	.	320,830
1994	169,003	30,860	64,923	396,137	.	660,923
1995	141,971	18,826	64,977	249,108	.	474,882
1996	117,679	4,881	67,422	265,565	.	455,547
1997	61,842	6,338	75,079	313,967	.	457,226
1998	87,794	3,473	38,271	246,375	.	375,913
1999	95,188	6,040	71,478	162,496	.	335,202
2000	320,844	6,637	43,107	152,881	.	523,469

TABLE 5-10. TRI Transfers Off-site for Further Waste Management (in pounds), DBDPO, By Industry, U.S., 2000.

SIC – Industry	Tranfers to Recycling	Transfers to Energy Recovery	Transfers to Treatment	Transfers to POTWs	Other Off-site Transfers	Total Transfers Off-site for Further Waste Management
22 Textiles	91,496	3,000	5,139	144,376	.	244,011
26 Paper	.	.	6,084	.	.	6,084
28 Chemicals	.	.	2,273	2,865	.	5,138
30 Plastics	26,096	1,231	28,393	273	.	55,993
32 Stone/Clay/Glass	.	.	.	.	.	.
33 Primary Metals	117,867	.	.	10	.	117,877
34 Fabricated Metals	.	270	.	250	.	520
35 Machinery	.	.	.	4,822	.	4,822
36 Electrical Equip.	59,143	.	837	0	.	59,980
37 Transportation Equip.	.	.	.	.	.	.
Multiple Codes 20-39	26,242	2,136	381	285	.	29,044
Original industry subtotal:	320,844	6,637	43,107	152,881	.	523,469
4953/7389 RCRA/Solvent Recovery	.	.	19,500	.	.	19,500
New industry subtotal:	.	.	19,500	.	.	19,500
Total	320,844	6,637	62,607	152,881	.	542,969

TABLE 5-11. TRI Transfers Off-site for Further Waste Management (in pounds), Trend Report for facilities in Chemicals (SIC 28), DBDPO, U.S., 1991-2000.

Year	Tranfers to Recycling	Transfers to Energy Recovery	Transfers to Treatment	Transfers to POTWs	Other Off-site Transfers	Total Transfers Off-site for Further Waste Management
1991	999	.	12,241	4,846	0	18,086
1992	0	250	14,610	5,401	.	20,261
1993	.	250	10,555	6,986	.	17,791
1994	.	250	905	7,808	.	8,963
1995	.	.	4,490	1,445	.	5,935
1996	.	.	3,233	5,620	.	8,853
1997	.	.	2,481	3,532	.	6,013
1998	.	.	2,815	3,426	.	6,241
1999	.	.	2,319	2,466	.	4,785
2000	.	.	2,273	2,865	.	5,138

TABLE 5-12. TRI Transfers Off-site for Further Waste Management (in pounds), Trend Report for facilities in Plastics (SIC 30), DBDPO, U.S., 1991-2000.

Year	Transfers to Recycling	Transfers to Energy Recovery	Transfers to Treatment	Transfers to POTWs	Other Off-site Transfers	Total Transfers Off-site for Further Waste Management
1991	.	1	56,025	4,960	.	60,986
1992	4,235	2,606	62,660	355	.	69,856
1993	6,905	2,729	54,072	515	.	64,221
1994	81,711	3,450	41,000	601	.	126,762
1995	32,216	5,973	35,947	301	.	74,437
1996	19,042	890	31,055	284	.	51,271
1997	15,620	1,838	37,001	255	.	54,714
1998	7,448	1,120	28,512	520	.	37,600
1999	2,630	1,180	54,742	520	.	59,072
2000	26,096	1,231	28,393	273	.	55,993

TABLE 5-13. TRI Transfers Off-site for Further Waste Management (in pounds), Trend Report for facilities in Textiles (SIC 22), DBDPO, U.S., 1991-2000.

Year	Tranfers to Recycling	Transfers to Energy Recovery	Transfers to Treatment	Transfers to POTWs	Other Off-site Transfers	Total Transfers Off-site for Further Waste Management
1991	.	8,500	2,557	17,045	.	28,102
1992	.	4,500	1,016	112,656	.	118,172
1993	.	5,100	2,335	191,381	.	198,816
1994	.	25,010	6,773	385,436	.	417,219
1995	1,993	3,300	5,434	243,056	.	253,783
1996	5	3,741	3,337	257,651	.	264,734
1997	.	3,750	9,547	308,920	.	322,217
1998	250	505	3,738	240,839	.	245,332
1999	71,768	3,755	6,826	152,924	.	235,273
2000	91,496	3,000	5,139	144,376	.	244,011

5.5 Human Exposure Estimation (Developed by Exponent, Boulder, CO)

The studies regarding DBDPO in U.S. environmental media and human tissues, discussed above, preclude a direct assessment of children's exposure. Therefore, exposures were estimated based on hypothetical contact that might reasonably be expected to occur. These exposure estimates were derived in a manner that biases the derived values high, likely overestimating actual exposures. Therefore, this assessment provides upper-bound estimates of exposure, and thus risk, and actual risk would be expected to be lower. The following section presents the approach we used to calculate these hypothetical exposures.

5.5.1 Potential Exposure Scenarios

Exposures that can affect children are defined in the VCCEP guidance as, "those which would occur prior to conception (to either parent), during prenatal development, and postnatally to the age of sexual maturation, which is completed around 18–21 years of age" (U.S. EPA 2000a). As mentioned above in section 4.1.3, DBDPO has not caused any reproductive or developmental effects in any animals tested at doses up to 1,000 mg/kg/day (the highest dose tested). Because of this lack of reproductive and developmental effects associated with DBDPO exposures, there is no need to conduct exposure assessments for pregnant women or prospective parents (male or female). Therefore, the only exposures considered in this evaluation for DBDPO are for children exposed postnatally.

The exposure scenarios that are the most relevant to children's exposure to DBDPO are:

- Infant ingestion of breast milk from a mother who manufactures DBDPO or works in a formulation or molding facility
- Infant ingestion of breast milk from a mother who disassembles computer monitors

- Infant ingestion from mouthing DBDPO-containing plastic electronic products
- Children inhaling DBDPO particulates released from plastic electronic products
- Infants mouthing DBDPO-containing fabric
- Adult and child dermal exposure to DBDPO-containing fabric
- Exposure to DBDPO via the general environment (e.g., eating food, incidental ingestion of soil and dust, breathing ambient air, and drinking water).

Due to the lack of available information, the typical exposure calculation—in which a contact rate (consumption, drinking, mouthing, and inhalation rate) is multiplied by an exposure-point concentration (DBDPO concentration in food, water, air, consumer product, etc.)—is not currently possible for every possible pathway. However, the published literature does provide sufficient data to allow calculation of some hypothetical exposures. A large degree of uncertainty is associated with these estimates due to the paucity of reliable scientific information on levels of DBDPO. However, in each scenario, sufficient conservatism is built into the calculations such that these exposure estimates represent potential upper bounds.

Throughout these calculations, the approach used a plausible, yet conservative, estimate of potential exposure to DBDPO. These calculations are termed the Reasonable Estimate (RE) for the purposes of this report. A higher exposure value was also calculated for each scenario. These calculations are termed the Upper Estimate (UE) and should be viewed as so conservative that they represent the absolute worst-case exposures. This approach—calculating a "bounding estimate" of exposure that is probably beyond the realm of plausible exposures—was undertaken to determine whether any hypothetical exposure would warrant further evaluation. Using this approach, our analysis indicates that a significant health risk is not expected for children under any of the scenarios evaluated, even using extremely conservative assumptions. Therefore, no further, more detailed evaluation of DBDPO is warranted to ensure adequate health protection for young children.

The various scenarios and the exposure assumptions used for quantitative analysis of exposures are described in the sections below. For each scenario, a separate section is included describing the results of the analysis and uncertainties associated with the calculated values.

5.5.2 Infant Ingestion of Breast Milk from a Mother Who Manufactures DBDPO

Workplace exposure to DBDPO could potentially occur during manufacturing or formulation into resins or a liquid polymer dispersion. DBDPO is manufactured in a closed system by the reaction of bromine with diphenyl oxide, and the highest potential worker exposures are associated with the activities of packaging DBDPO for shipping, or in emptying bags of flame retardant into a hopper for product formulation. Once formulated, DBDPO is encased in a polymer matrix, significantly reducing the potential for worker exposure.

This assessment evaluates the potential exposures that might be incurred by a nursing infant of a working mother, and assumes that the mother packages DBDPO at its manufacturing site or empties bags of DBDPO into hoppers at formulating/compounding site.

Assumptions

It is assumed that the mother works at this task for 8 hours a day, 5 days per week. It is also assumed that during this task, the mother inhales DBDPO-containing dust from the workplace air, and the DBDPO that she inhales partitions into her breast milk. For the reasonable estimate (RE), an infant is assumed to breast feed from the exposed mother daily from birth through 3 months of age (Collaborative Group on Hormonal Factors in Breast Cancer 2002). For the upper estimate (UE), an infant was assumed to breast feed from the exposed mother daily from birth for 2 years based on professional judgment.

Ideally, a calculation to estimate intake from breast milk would begin with a measured concentration of the chemical of interest in breast milk. However, DBDPO has never been measured in breast milk in any country, and may not actually partition into breast milk, so a hypothetical intake is estimated indirectly. Because of DBDPO-specific data is not available, several steps were taken to estimate this intake, using the best available data. The concentrations of DBDPO that might be found in breast milk were estimated based on information regarding the relation between air concentrations of DBDPO and associated serum concentrations and on limited data regarding the partitioning of lower brominated DPO congeners to breast milk. Specifically, published measured workplace air concentrations and associated blood levels for Swedish workers were used to estimate an air-to-serum relation, which was then combined with estimates of air concentrations for a U.S. worker to derive a hypothetical serum level for a U.S. worker. Then, data regarding the partitioning of lower brominated DPO congeners from serum to breast milk were used to set an upper bound for the partitioning behavior of DBDPO, yielding an estimate of DBDPO in breast milk. The assumptions used almost certainly overestimate the possible intake of DBDPO through this pathway. The specific assumptions for this scenario are discussed below and shown in Table 5-5.

DBDPO concentration in workplace air (respirable). Upper Estimate (UE): Because workplace air data in the U.S. is not available, an upper value of 5 mg/m^3 was selected based on the Workplace Environmental Exposure Level (WEEL) (AIHA 1996). The WEEL is the level at which workers could be exposed every day for an 8-hour shift with the expectation of no adverse effects.

Reasonable Estimate (RE): An air concentration of 1 mg/m^3 was selected, because the EU risk assessment notes that the majority of workplace air levels (TWA) are below 1 mg/m^3 (ECB 2002).

Air-to-serum conversion factor. To yield a breast-milk concentration of DBDPO for a given inhalation exposure concentration, it was necessary to develop an estimate of internal dose/body burden or serum concentration for a given workplace exposure. Sjödin et al. (1999) measured workplace air concentration and serum levels of DBDPO for Swedish workers disassembling and

shredding used computer monitors and other plastic components containing DBDPO. Mean levels (mean of two samples) in the air were 175 ng/m³ (Hagmar et al. 2000), and the median serum concentration of DBDPO (n=19) among the workers was 4.8 ng/g lipid (Sjödin et al. 1999). This yields a ratio factor of 27.4 (μg DBDPO/g serum lipid)/(mg DBDPO/m³). This ratio of serum levels to workplace exposures is the best available. There are many simplifying assumptions in this approach that collectively make it quite conservative. It assumes that all the measured DBDPO in the serum of the workers is attributed to the workplace exposures. This is conservative, because food and ambient exposures may contribute to the body burden of DBDPO. Sjödin and coworkers did not provide enough detailed information to develop a reasonable range of estimates for this parameter. Only two air concentrations were reported in the study (Hagmar et al. 2000). Therefore, this single value is used for both the UE and the RE.

Breast-milk to blood-serum ratio. To yield an estimate of breast-milk concentrations of DBDPO associated with occupational exposure, the correlation between serum levels and breast-milk levels must be established. DBDPO has never been documented as being present in breast milk, so a transfer ratio was estimated based on information from other compounds. Efforts by Exponent, Judy LaKind, and Jake Ryan to determine this ratio for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) resulted in a rapid communication that is in press in the *Journal of Toxicology and Environmental Health*. The available data indicate that the ratios of breast milk to serum levels for TCDD and related dioxins, furans, and polychlorinated biphenyls (PCBs) are near 1.0 when calculated on the basis of lipid weight. For these highly lipophilic compounds, lipid partitioning is the key to their disposition in serum and breast milk. These molecules are one-half to one-third the molecular weight of DBDPO; thus, there is no size limitation on their passage into breast milk.

To establish a ratio between serum and breast milk levels for DBDPO, we calculated the ratios of breast-milk levels reported for various PBDPOs (in Ryan et al. 2001) to levels in serum for the same PBDPOs (in Sjödin et al. 2001b). We found that the ratio of breast milk to serum levels (both were reported on a lipid weight basis) was less than 1.0 for all congeners, and decreased with increasing molecular weight/bromination. The ratio of breast milk to serum levels for HeptaBDPO, the highest molecular weight congener in the series, was 0.54. There is some uncertainty in using this analysis. First, these were not paired samples: the PBDPO levels in breast milk were determined in samples collected in Canada in the late 1990s, whereas the serum levels were derived from U.S. donors whose blood was collected in the late 1980s. Second, they are from different countries. Thirdly, and most importantly, they are from different time periods. However, because the breast-milk levels are from later time points, and because levels of PBDPOs in humans have reportedly increased between the 1980s and late 1990s, the levels in breast milk in the late 1990s could be expected to be higher than that in the late 1980s. Therefore, this analysis would be expected to produce an overestimate of the ratio of breast milk to serum for these congeners. Further, the ratio for DBDPO would be lower than for HeptaBDPO. How much lower (if not zero) is uncertain. Because of this uncertainty, we used the following reasoning for the values used for these parameters.

- UE: A breast-milk to blood-serum ratio of 0.5 was selected (i.e., the concentration in breast milk is one-half the concentration in serum on a lipid weight basis), because this

was the ratio observed for HeptaBDPO. Because the higher brominated congeners appear to have a lower transfer rate to breast milk, using the ratio derived for HeptaBDPO is a conservative estimate that is likely to overestimate the actual relation.

- RE: A breast-milk to blood-serum ratio of 0.1 was selected, because it is likely that the larger molecular size and greater bromination of DBDPO would limit its ability to enter breast milk, and toxicologists have predicted that it would not transfer into breast milk at all, thereby eliminating breast milk as a route of exposure for infants (Hardy 2001). A prudent estimate is approximately 0.1 for the ratio of DBDPO in serum lipids that can partition into breast-milk lipids.

Fraction of breast milk that is lipid. UE and RE: A value of 4% was selected based on the value recommended in the *Child-Specific Exposure Factors Handbook* (U.S. EPA 2000).

Ingestion rate. UE: A UE ingestion rate of 980 mL/day was selected based on the upper percentile value for a 12-month average breast-milk ingestion rate for a child less than 1 year old (U.S. EPA 2000). The EPA does not present any ingestion rates for children between 12 and 24 months, but they do show that ingestion rates decrease after the age of 9 months (U.S. EPA 2000). Therefore, using the 12-month average value to represent the entire 2-year period is a conservative assumption that is likely to overestimate actual exposures.

RE: An RE ingestion rate of 742 mL/day was used, based on the mean value for children ages 1–6 months (U.S. EPA 2000).

Absorption. Although the gastrointestinal absorption of DBDPO is estimated to be less than 2% (Hardy 2002, NTP 1986, El Dareer et al. 1987), an absorption factor of 100% was used in these intake calculations, because the toxicity values are based on an ingested dose rather than an absorbed dose. This same parameter and value were used in the intake calculations shown in Tables 5-5 through 5-8.

Body weight. UE: As an upper estimate, it was assumed that the maximum duration over which an infant would be breast fed in the U.S. would be two years. Therefore, a UE body weight of 7.84 kg was derived from the 50th-percentile weights for children ages birth through 24 months, presented in Table 11-1 of the *Child-Specific Exposure Factors Handbook* (U.S. EPA 2000). This value was used in the intake calculations presented in Tables 5-2 through 5-5.

RE: Because the majority of children are breast fed only through the first three months of life (Collaborative Group on Hormonal Factors in Breast Cancer 2002), an RE body weight of 4.36 kg was derived from the 50th-percentile weights for children ages birth through 3 months, presented in Table 11-1 of the *Child-Specific Exposure Factors Handbook* (U.S. EPA 2000). This value was used in the intake calculations presented in Tables 5-5 and 5-6.

Results

In this scenario, the mother of a breast-feeding infant was assumed to work in the bagging operation at a DBDPO manufacturing site. The calculated daily intake for an infant exposed via

breast milk ranged from 1.9×10^{-2} to 3.4×10^{-1} mg/kg-day for the RE and UE, respectively (Table 5-14). As discussed in previous sections, DBDPO has not been reported in breast milk in any country, and is not expected to partition into breast milk, so a hypothetical intake was estimated indirectly.

There is a great deal of uncertainty surrounding the methods employed to calculate serum levels, as well as the percentage of DBDPO in the serum that would partition into the breast milk. Even for the RE value, a large degree of conservatism is built into these values. It is also unlikely that a worker would be exposed at the WEEL for an entire 8-hour shift. It should be noted that 5 mg/m³ is the PEL for nuisance dust, suggesting that AIHA's WEEL for DBDPO assumes no intrinsic toxicity to the inhalation of this compound. If DBDPO does not, in fact, partition into breast milk, then the true exposure from this scenario would be zero.

Table 5-14. Infant ingestion of breast milk from a mother who manufactures DBDPO.

Exposure Parameters	Reasonable Estimate (birth to 3 months) Value	Source/Comment	Upper Estimate (birth to 2 years) Value	Source/Comment
$C_{a:}$ DBDPO concentration in workplace air (respirable) (mg/m3)	1	BFRIP	5	WEEL (AIHA 1996)
$CF_{a\text{-}s:}$ Air-to-serum conversion factor; (μg DBDPO/g lipid serum) per (mg DBDPO/m^3 air)	27.4	Based on working 8 hour/day and 5 day/week (Sjödin et al. 1999). See text for details.	27.4	Based on working 8 hour/day and 5 day/week (Sjödin et al. 1999). See text for details.
$R_{b\text{-}m:}$ Breast milk to serum ratio (unitless)	0.1	Based on the fact that higher brominated DPO do not partition into milk as effectively as lower brominated DPO (see text)	0.5	Conservative assumption that DBDPO partitions into breast milk and serum on a lipid weight basis at the ratio that hepta-DPO does (BDE-183) (see text)
$F_{l:bm:}$ Fraction of breast milk that is lipid (g/mL)	0.04	4% expressed as g/mL (CS-EFH Table 2-12; U.S. EPA 2000)	0.04	4% expressed as g/mL (CS-EFH Table 2-12; U.S. EPA 2000)
CF: Conversion factor (mg/μg)	1E-03		1E-03	
IR: Ingestion rate (mL/day)	742	Mean for ages 1–6 months (CS-EFH, Table 2-12, p. 2-19, U.S. EPA 2000)	980	12-month average, upper percentile (CS-EFH, Table 2-12, p. 2-19, U.S. EPA 2000)
ABS: Absorption (percent)	100[a]	Necessary to use with toxicity value	100%[a]	Necessary to use with toxicity value
BW: Body weight (kg) 0–3 months (RE) and 0–2 years (UE)	4.36	Average of 50th percentile weights, birth through 3 months (CS-EFH, Table 11-1, U.S. EPA 2000)	7.84	Average of 50th percentile weights, birth through 24 months (CS-EFH, Table 11-1, U.S. EPA 2000)
Daily Intake (mg/kg-day)	1.9E-02	Calculated	3.4E-01	Calculated

$$\text{Intake} = \frac{C_a \times CF_{a\text{-}s} \times R_{b\text{-}m} \times F_{l:bm} \times CF \times IR \times ABS}{BW}$$

[a] Although the absorption of DBDPO is estimated to be less than 2%, an absorption of 100% is necessary in the intake calculations because the toxicity values are based on an ingested dose rather than an absorbed dose. See text for details.

5.5.3. Infant Ingestion of Breast Milk from a Mother Who Disassembles Electronics

In this scenario, the mother of a breast-feeding infant was assumed to be an electronics disassembly worker. DBDPO has not been reported in breast milk in any country, and is not expected to partition into breast milk, so a hypothetical intake was estimated indirectly. There are no U.S. data for either workplace air concentrations or serum levels for a disassembly worker. However, DBDPO and other polybrominated diphenyl ether isomers were detected in serum of Swedish workers engaged in dismantling electronic equipment (Sjödin et al. 1999, Sjödin 2000) and in Swedish computer technicians (Hagmar et al. 2000). Therefore, DBDPO serum concentrations for a U.S. worker were assumed to be the same as the levels measured in the computer disassembly workers in Sweden (Sjödin et al. 1999). Additionally, DBDPO in the serum was assumed to partition into breast milk, as discussed above, and consumed by an infant daily from birth through 3 months (RE) and from birth through 2 years (UE).

Assumptions

The assumptions made for this scenario are described below and shown in Table 5-6.

DBDPO concentration in mother's blood. UE: Based on the study by Sjödin et al. (1999), Swedish workers who disassembled computer monitors and worked near the shredding devices had detectable levels of DBDPO in their blood. As a conservative assumption, the maximum level reported by Sjödin was used for the UE (9.9 ng/g serum lipid).

RE: For the RE, the median level reported by Sjödin (1999) was used (4.8 ng/g serum lipid). The values used for the ratio of breast milk to serum, fraction of breast milk that is lipid, ingestion rate, absorption, and body weight are the same as in the previous scenario.

All other assumptions are identical to the assumptions used in the previous scenario.

Results

In this scenario, the mother of a breast-feeding infant was assumed to be a computer monitor disassembly worker. The calculated daily intake for the infant ranged from 3.3×10^{-6} to 2.5×10^{-5} mg/kg-day for the RE and UE, respectively (Table 5-15). For this pathway, there was less uncertainty regarding serum concentration than in the previous scenario, because the values were taken directly from the published literature for workers performing this activity. However, there is still a great deal of uncertainty and conservatism in the percentage of DBDPO in the serum that would partition into the breast milk. Even for the RE value, a large degree of conservatism is built into these values.

Table 5-15. Estimated intake of DBDPO by an Infant ingesting breast milk from a mother who disassembles electronics.

Exposure Parameters	Reasonable Estimate (birth to 3 months)		Upper Estimate (birth to 2 years)	
	Value	Source/Comment	Value	Source/Comment
$C_{b:}$ DBDPO concentration in mother's blood (ng/g lipid)	4.8	Median for computer disassembly workers in Sweden (Sjödin et al. 1999)	9.9	Highest level reported for computer disassembly workers in Sweden (Sjödin et al. 1999)
$R_{b\text{-}m:}$ Breast milk to serum ratio (unitless)	0.1	Based on the fact that higher brominated DPO do not partition into milk as effectively as lower brominated DPO (see text)	0.5	Conservative assumption that DBDPO partitions into breast milk and serum on a lipid weight basis at the ratio that hepta-DPO does (BDE-183) (see text)
$F_{l:bm:}$ Fraction of breast milk that is lipid (g/mL)	0.04	4% expressed as g/mL (CS-EFH Table 2-12; U.S. EPA 2000)	0.04	4% expressed as g/mL (CS-EFH Table 2-12; U.S. EPA 2000)
CF: Conversion factor (mg/ng)	1E-06		1E-06	
IR: Ingestion rate, breast milk (mL/day)	742	Mean for ages 1–6 months (CS-EFH, Table 2-12, p. 2-19, U.S. EPA 2000)	980	12-month average, upper percentile (CS-EFH, Table 2-12, p. 2-19, U.S. EPA 2000)
ABS: Absorption (percent)	100[a]	Necessary to use with toxicity value	100[a]	Necessary to use with toxicity value
BW: Body weight, (Kg) 0–3 months (RE) 0–2 years (UE)	4.36	Average of 50th percentile weights, birth through 3 months (CS-EFH, Table 11-1, U.S. EPA 2000)	7.84	Average of 50th percentile weights, birth through 24 months (CS-EFH, Table 11-1, U.S. EPA 2000)
Daily Intake (mg/kg-day)	3.3E-06	Calculated	2.5E-05	Calculated

$$\text{Intake} = \frac{C_b \times R_{b\text{-}m} \times F_{l:bm} \times CF \times IR \times ABS}{BW}$$

[a] Although the absorption of DBDPO is estimated to be less than 2%, an absorption of 100% is necessary in the intake calculations because the toxicity values are based on an ingested dose rather than an absorbed dose. See text for details.

5.5.4 Infant Ingestion from Mouthing DBDPO-Containing Electronics

DBDPO is used to flame-retard synthetic polymers used in electrical and electronic equipment. A typical example of DBDPO's use in the United States is in the cabinet backs of television sets, where DBDPO is used at a level of approximately 12% (WHO 1994). In this exposure scenario, an infant is assumed to mouth consumer electronic products (e.g., television, computer monitor) that contain DBDPO. The DBDPO is assumed to leach from the surface of the electronic product into the saliva of the infant. The infant is exposed via swallowing the DBDPO-containing saliva. This scenario is not likely to represent typical exposures, because children are unlikely to be mouthing television sets or computer monitors. However, to be conservative, it is assumed that the possibility exists.

Assumptions

The assumptions made for this scenario are described below and shown in Table 5-16.

DBDPO concentration leached from surface into liquid. DBDPO is unlikely to leach from electronic equipment based on its physical/chemical properties, the types of plastics it is used in (e.g. dense, hard high impact polystyrene), and laboratory study results (Norris et al. 1974).

Norris et al. (1974) demonstrated that a pellet of acrylonitrile butadiene-styrene (ABS) terpolymer containing 4.25% DBDPO, placed in 2 L of water for a full day at 120 °F, did not leach DBDPO into the water at levels that could be detected (detection limit of 0.075 mg/L). Similarly, a 3% acetic acid solution at 120 ^{o}F for 1 or 7 days also did not leach DBDPO from the ABS pellets. The only conditions in this experiment under which DBDPO leached into the solution were pellets being put into a solution of cottonseed oil for seven days at 135 ^{o}F, with a resulting DBDPO concentration of 1 mg/L (Norris et al. 1974).

For the calculations involved in estimating intake from this exposure pathway, the concentrations obtained in the experiment described above were converted into a mass-per-time value. The first step in doing this conversion entailed calculating the total mass leached from the pellet by multiplying the concentration (i.e., 1 mg/L) by the total volume used in the experiment (i.e., 2 L). Then, to obtain a mass-per-time rate, the total mass leached was divided by the total number of days in the experiment (i.e., 7 days).

Step 1: (1 mg/L) × (2 L) = 2 mg

Step 2: (2 mg) / 7 days = 0.29 mg/day

This conversion assumes that the rate of leaching would be constant over the entire 7-day period. It also assumes that a smaller volume of liquid would have leached a smaller mass of DBDPO. Either assumption may be incorrect; however, because DBDPO was not extracted by either water or acetic acid (at high temperatures and over a 7 day period), and may not leach from plastic at all when mouthed by an infant; these assumptions are likely to have a negligible impact on the final calculations.

UE: A value of 0.29 mg/day was used based on DBDPO leached by cottonseed oil at 135 ^{o}F in seven days in the Norris et al. (1974) experiment, and the conversions described above.

RE: For a more reasonable estimate, the limit of detection (0.075 mg/L) over a 1-day period was used in the conversion described above to derive a value of 0.15 mg/day. Because no DBDPO was actually detected in the water, this value is likely to overestimate actual exposures.

Mouthing time per day – all objects. UE: A value of 97.2 minutes per day of total mouthing time was selected based on an average of the maximum mouthing times for children ages 3–18 months presented in Table 6-1 of the *Child-Specific Exposure Factors Handbook* (U.S. EPA 2000). This mouthing time encompasses all objects mouthed, including fingers and toys.

RE: A value of 32.4 minutes per day of mouthing time was selected based on an average of mean mouthing times presented in the table described above for children ages 3–18 months (U.S. EPA 2000), again for all objects mouthed.

Fraction of objects with DBDPO. UE: A value of 10% was selected based on professional judgment. It is not known for certain what percentage of items in the normal household contain DBDPO flame retarded polymer casings. Tulve et al. (2002) reported that 90% of the time, items mouthed by children less than 24 months were hands, other areas of their body or toys. The items mouthed in the remaining 10% were not provided, but it is unlikely that this consisted entirely of electronic equipment or textiles containing DBDPO. To be conservative, however, 10% was assumed as an upper estimate of the fraction of items that contain DBDPO and are mouthed by a child.

RE: A value of 1% was chosen as a more reasonable estimate based on professional judgment. Again, it is unlikely children will mouth hard plastic electronic items, such as television set cabinets, that might contain DBDPO.

Results

The calculated daily intake for a child exposed via mouthing DBDPO-containing electronic products ranged from 4.3×10^{-6} to 2.5×10^{-4} mg/kg-day for the RE and UE, respectively (Table 5-16). There is a significant amount of uncertainty surrounding the amount of DBDPO that may actually leach out of treated plastics. Both values used in these calculations were produced by a method (i.e., Norris et al. 1974, see discussion above) of which the relevance to actual human contact is debatable. Other values with considerable variability are the total mouthing time and fraction of objects mouthed that contain DBDPO. Even with this level of uncertainty, a large degree of conservatism is built into both the UE and RE values. Despite this conservatism, the calculated exposures for this hypothetical scenario are very small.

Table 5-16. Estimated DBDPO intake by an infant mouthing DBDPO-containing electronics.

Exposure Parameters	Reasonable Estimate		Upper Estimate	
	Value	Source/Comment	Value	Source/Comment
C_L: Mass of DBDPO leached from surface into liquid per day (mg/day)	0.15	Norris et al. 1974. No DBDPO was extracted from ABS terpolymer in water for 1 day at 120 °F. This value is the limit of detection (0.075 mg/L) multiplied by the total volume (2 L) divided by the total number of days (1 day).	0.29	Norris et al. 1974. Extraction of DBDPO from ABS terpolymers in cottonseed oil at 135 °F for 7 days. This value is the concentration of DBDPO leached (1 mg/L) multiplied by the total volume (2 L) divided by the total number of days (7 days).
CF: Conversion factor (day/min)	6.9E-04	1 day has 1,440 minutes	6.9E-04	1 day has 1,440 minutes
MT: Mouthing time (min/day)	32.4	Total mouthing time, average of means for ages 3–18 months (CS-EFH, Table 6-1, p. 6-12, U.S. EPA 2000)	97.2	Total mouthing time, average of maximums for ages 3–18 months (CS-EFH, Table 6-1, p. 6-12, U.S. EPA 2000)
FS: Fraction of objects with DBDPO (percent)	1	Professional judgment (see text)	10	Professional judgment (see text)
ABS: Absorption (percent)	100[a]	Necessary to use with toxicity value	100[a]	Necessary to use with toxicity value
BW: Body weight, 0–2 years (kg)	7.84	Average of 50th percentile weights, birth through 24 months (CS-EFH, Table 11-1, U.S. EPA 2000)	7.84	Average of 50th percentile weights, birth through 24 months (CS-EFH, Table 11-1, U.S. EPA 2000)
Daily Intake (mg/kg-day)	4.3E-06	Calculated	2.5E-04	Calculated

$$\text{Intake} = \frac{C_L \times CF \times MT \times FS \times ABS}{BW}$$

[a]Although the absorption of DBDPO is estimated to be less than 2%, an absorption of 100% is necessary in the intake calculations because the toxicity values are based on an ingested dose rather than an absorbed dose. See text for details.

5.5.5 Child's Inhalation of DBDPO-containing Dust Originating from Electronics

In addition to the low likelihood of leaching from a hard plastic into a liquid, DBDPO's negligible vapor pressure indicates that it also would not volatilize out of the plastic components. DBDPO has been measured in air in a room full of computers, and therefore we calculated the exposures that a child might experience when exposed to DBDPO in indoor air.

Assumptions

The assumptions made for this scenario are outlined below and shown in Table 5-17.

DBDPO concentration in air (respirable); vapor attaches to dust particulates in air. UE: A respirable air concentration of 0.087 ng/m³ was selected as an upper estimate based on the highest value reported for an office with computers (Sjödin et al. 2001a). Sjödin et al. (2001a) reported that DBDPO in air was associated with particulates.

RE: An air concentration of 0.052 ng/m³ was selected, based on the mean of all samples reported for an office with computers, using one-half the detection limit for non-detected concentrations (Sjödin et al. 2001a).

Fraction of time spent in room with TV or computer. UE: As an upper estimate, it was assumed that an infant spends all of his or her time indoors, in a room with a TV or computer, yielding a fraction of 1.

RE: A fraction of 0.833 was selected based on the assumption that 20 hours in a 24-hour period were spent indoors in a room with a TV or computer monitor. There are no guidance values for this parameter from the EPA *Child-Specific Exposure Factors Handbook*, but it is reasonable to assume that infants spend a large portion of a day indoors, and that most of their time might be spent in a room that also contains a television or computer monitor.

Inhalation rate. UE and RE: An inhalation rate of 5.65 m³/day was used based on the average of the mean value for children less than 1 year old (4.5 m³/day) and the mean for children aged 1–2 years old (6.8 m³/day). No medians or high-end values were presented for this parameter, so the same value was used as both the UE and the RE (U.S. EPA 2000).

Results

The calculated daily intake for a child exposed via the inhalation of particulates from plastic electronic products ranges from 3.1×10^{-8} mg/kg-day to 6.3×10^{-8} mg/kg-day for the RE and UE, respectively (Table 5-17). Even using the conservative assumptions discussed above, the calculated intakes via this pathway are orders of magnitude less than the intakes estimated via other pathways. Therefore, inhalation of DBDPO in the household is estimated to contribute only minimally to total exposure.

Table 5-17. Estimated DBDPO intake of young children inhaling particulates released from electronics.

Exposure Parameters	Reasonable Estimate		Upper Estimate	
	Value	Source/Comment	Value	Source/Comment
$C_{a:}$ DBDPO concentration in air (respirable); vapor attaches to dust particulates in air (ng/m3)	0.052	Office w/computers (mean of all samples, using one-half the detection limit for non-detects) Sjödin et al. 2001a	0.087	Office w/computers (highest value reported) Sjödin et al. 2001a
CF: Conversion factor (mg/ng)	1.0E-06		1.0E-06	
FI: Fraction of time spent in room w/TV or computer (unitless)	0.83	20 hrs in 24-hr period; professional judgment	1	24 hrs in 24-hr period; professional judgment
IhR: Inhalation rate (m3/day)	5.65	Average of <1 yr (4.5) & 1–2 yrs (6.8), means [CS-EFH, Table 7-13, p. 7-20, U.S. EPA 2000][a]	5.65	Average of <1 yr (4.5) & 1–2 yrs (6.8), means [CS-EFH, Table 7-13, p. 7-20, U.S. EPA 2000][a]
ABS: Absorption (percent)	100[b]	Necessary to use with toxicity value	100[b]	Necessary to use with toxicity value
BW: Body weight, 0–2 years (kg)	7.84	Average of 50th percentile weights, birth through 24 months (CS-EFH, Table 11-1, U.S. EPA 2000)	7.84	Average of 50th percentile weights, birth through 24 months (CS-EFH, Table 11-1, U.S. EPA 2000)
Daily Intake (mg/kg-day)	3.1E-08	Calculated	6.3E-08	Calculated

$$\text{Intake} = \frac{C_a \times CF \times FI\ IhR \times ABS}{BW}$$

[a] Only means are reported

[b] Although the absorption of DBDPO is estimated to be less than 2%, an absorption of 100% is necessary in the intake calculations because the toxicity values are based on an ingested dose rather than an absorbed dose. See text for details.

5.5.6 Exposure via Mouthing, Dermal Contact with DBDPO-Containing Textiles, and Inhalation of DBDPO-Containing Dust Originating from the Textiles

The National Academy of Sciences (NAS) recently conducted a consumer exposure and risk assessment for flame retardants that may be used to flame retard upholstery textiles (see Table 5-18 for a summary of the NAS calculations, and Appendix V). The results from the NAS are reported in Table 5-6 without modifications.

Assumptions

A number of very conservative assumptions were used in NAS's exposure assessment calculations, including the following.

For dermal exposure:

- An adult spends one-fourth of every day sitting on furniture upholstery that is back-coated with DBDPO.
- One quarter of the receptor's upper torso is in contact with the upholstery.
- Receptor's skin and clothing, and the upholstery fabric, present no barrier to DBDPO movement.
- Sufficient water (e.g., from sweat) is present to allow dissolution of DBDPO in the water and transfer to the skin and into the body of the receptor.
- All of the DBDPO that dissolves is absorbed immediately by the receptor. An alternative iteration of dermal exposure assumed that DBDPO dissolves up to its solubility limit in water.
- Estimated upholstery application rate for DBDPO is 5 mg/cm^2.
- Estimated extraction rate by water for DBDPO (0.025/day) is based on extraction data for hexabromocyclododecane in polyester fiber (McIntyre et al. 1995).

For inhalation exposure:

- An adult spends one-fourth of a lifetime in a room with a low air-exchange rate (0.25/hour).
- The room contains a relatively large amount of fabric upholstery (30 m^2 in a 30-m^3 room) treated with DBDPO.
- The DBDPO treatment is gradually wearing away over 25% of its surface, to 50% of its initial quantity over the 15-year lifetime of the fabric.
- One percent of the worn-off DBDPO is released into indoor air as respirable particulates.
- For vapor inhalation, release of DBDPO by evaporation from the upholstery is assumed.

For oral exposure:

- A child, 0–2 years, mouths 50 cm^2 of fabric back-coated with DBDPO for 1 hour per day, daily, for 2 years.

- Extraction rate by saliva for DBDPO (0.025/day) is based on extraction data for hexabromocyclododecane in polyester fiber (McIntyre et al. 1995).

NAS noted uncertainty in these exposure estimates because dermal absorption data for DBDPO was available, the minimal solubility of DBDPO in water, the low vapor pressure of DBDPO, and the encapsulation of DBDPO in a polymer matrix.

Results

NAS's exposure estimates indicated that potential consumer exposure to DBDPO as a result of its use in upholstery fabrics, reported as a single-value for each exposure route, was minimal. The estimated exposures that NAS derived are higher than some of the intake estimates calculated here for other types of exposures. NAS stated that its results were extremely conservative, and that the estimated exposures did not warrant concern from a human health risk perspective. NAS also concluded that no further data was needed.

Table 5-18. Summary of NAS (2000) results re DBDPO exposures from upholstery textiles.

Exposure Pathway	Noncancer Intake	Noncancer Hazard Quotient	Comment/Assumptions
Dermal-Adult (mg/kg-day)	9.8E-01	0.25	▪ Adult spends 1/4 of every day sitting on furniture upholstery backcoated with DBDPO ▪ 1/4 of the adult's upper torso is in contact with upholstery ▪ Adult's skin and clothing and upholstery fabric present no barrier to DBDPO movement ▪ Sufficient sweat is present to allow dissolution of DBDPO and transfer to the skin and into the body ▪ All DBDPO that dissolves is absorbed immediately
Dermal-Adult (mg/kg-day)	1.33E-09	3.34E-10	Same as dermal scenario above except assumes that DBDPO only dissolves up to its solubility limit in water
Inhalation of Particulates-Adult (mg/m3)	4.8E-04	0.000034	▪ Adult spends 1/4 of life in a room with low air-exchange rates ▪ Room contains relatively large amount of fabric upholstery treated with DBDPO ▪ DBDPO treatment is gradually wearing away over 25% of its surface to 50% of its initial quantity over the 15-year lifetime of the fabric ▪ 1% of the worn-off DBDPO is released into indoor air as small particles that may be inhaled
Inhalation of Vapors-Adult (mg/m3)	3.8E-04	0.0000271	Same as particulate scenario above except assumes that DBDPO is released by evaporation
Oral-Child, 0-2 yrs (mg/kg-day)	2.6E-02	0.0065	Child mouths fabric backcoated with DBDPO for 1 hour per day, daily, for 2 years

5.5.7 Exposure to Children via the Environment

There are limited data on measured DBDPO concentrations in the U.S. environment or food items. Fish, chicken, air, sewage, sewage treatment plant (STP) sludge, and sediment have been sampled to quantify DBDPO in the U.S. (see Table 5-1), but the data are insufficient to calculate a reasonable exposure for the general population that might be exposed to DBDPO via food, water, air, and soil.

DBDPO has been detected in U.S. citizens, albeit at very low levels, with the majority of the results being non-detects. These detections indicate that at least some persons in the U.S. were exposed in some way to DBDPO. Quantifying exposures based on levels measured in humans is a reasonable approach, given the lack of alternative data on levels of DBDPO in the environment.

This intake calculation was assumed to represent the total amount of DBDPO that a child living in the U.S. might absorb via pathways other than breast milk ingestion or direct ingestion from electronic products or fabrics, including inhalation of ambient outside air; inhalation of indoor air; ingestion from food; incidental ingestion and dermal contact with soils or sediments; and ingestion, dermal contact, and inhalation from water.

Calculating the absorbed dose of DBDPO that would result in the measured serum DBDPO in humans requires information on the half-life of DBDPO in humans, and the volume distribution of DBDPO in humans. To calculate the ingested dose (which is required to compare to the reference dose, or RfD), requires knowledge of the bioavailability of DBDPO in humans (via all routes).

Assumptions

The following describes what is known about each of these parameters, and this information is summarized in Table 5-19.

Concentration of DBDPO in Humans. Sjodin et al. (2001) reported measuring DBDPO in the blood of U.S. blood donors collected in 1988. The median serum concentration of DBDPO in these blood donors was < 1 pmol/g lipid weight. The range was <1 – 35 pmol/g lipid weight.

UE: The highest level reported was 35 pmol/g lipid weight or 33.6 ng/g serum lipid.

RE: The median level was < 1 pmol/g lipid weight or < 0.96 ng/g serum lipid. For the purposes of this assessment, a median = 0.96 ng/g will be used.

Half-life. A study of workers who had detectable DBDPO levels in their serum showed that the levels of DBDPO declined rapidly when the workers went on vacation for an extended period of time (at least 30 days). Sjödin et al. (2000) reported that the average half-life of DBDPO in these workers was 6.8 days, with a confidence interval of 3–12 days.

UE: A half-life in the body of 3 days was used, based on lower bound value of the confidence interval report by Sjödin et al. (2000)[1]. This value may underestimate the half-life, because the confidence interval represents a statistical calculation (i.e., mean minus two times the standard deviation), rather than the lowest observed half-life, which was not reported. In another study, a half-life of approximately 15 days was observed in a worker who had detectable serum levels (Thuresson 2002a). However, because additional data regarding the possible lower-end values is unavailable, the conservative estimate of 3 days was used.

RE: A half-life in the body of 6.8 days was used, based on the median value reported by Sjödin et al. (2000).

Volume of distribution. This parameter represents the volume of the tissues in the body into which a chemical will distribute. Measures of DBDPO in serum report the compound in the lipid fraction of serum. When rats were exposed to DBDPO via oral ingestion, concentrations of DBDPO were higher in the liver than in adipose tissue, and levels in other tissues and muscle were much lower (El Dareer et al. 1987). Therefore, the following reasoning was used for the volume of distribution values.

UE: A value of 50% was used as an upper-bound estimate, because DBDPO does not partition in the lipid fraction exclusively. DBDPO does not partition into muscle (which accounts for approximately 50% of the body's volume) to any appreciable amount. In rats dosed with DBDPO, the concentration of DBDPO in the muscle tissue was one to two orders of magnitude lower than that in the adipose tissue. However, the lack of more detailed information precludes us from refining this estimate any further.

RE: A value of 25% adipose tissue in humans was used based on EPA's value used to calculate 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) body burdens. EPA reported this value to be on the high end of the range for the general population. It is probably a reasonable figure to use for DBDPO, which does not partition in the lipid fraction to the extent and affinity that TCDD does.

Absorption (bioavailability). The bioavailability of DBDPO in rats has been found to be less than 2% when administered in feed (El Dareer et al. 1987). Data on the bioavailability of DBDPO via the inhalation route is not available, and only limited data is available (via an *in vitro* assay) on the bioavailability via the dermal route (Hughes et al. 2001). An assessment of the DBDPO blood levels in Swedish workers suggests bioavailability via inhalation may be similar to that of oral (Hardy 2001).

UE: A value of 1% absorption was used. Intake is calculated from an "absorbed dose," so a lower absorption factor provides a higher intake estimate (i.e., more conservative assumption), because the absorption factor is in the denominator of the equation.

RE: A value of 2% absorption (bioavailability) was used based on the highest value report by El Dareer et al. (1987).

[1] In this equation, a lower half-life was chosen for the UE because a lower value will yield a higher estimate of intake since the half-life is in the denominator of the equation.

Results

The calculated intakes for exposures via the general environment were 1.2×10^{-3} and 3.9×10^{-1} mg/kg-day for the RE and UE, respectively (Table 5.19). These values are probably higher than the true intake. Given that the majority of serum samples tested had non-detectable levels of DBDPO, it is most likely that the majority of the U.S. population has very low, if not zero, exposure.

Table 5-19. Estimated children's exposures to DBDPO via the general environment.

Input Parameters	Reasonable Estimate		Upper Estimate	
	Value	Source/Comment	Value	Source/Comment
C_{ss}: Concentration in body, steady state (ng/g lipid)	0.96 [a]	Median (Sjödin et al. 2001b)	33.6[a]	Maximum (Sjödin et al. 2001b)
V_d: Volume of distribution = {BW × FL}				
BW: Body weight (3–6 yrs) (kg)[b]	18.7	Average of means (CS-EFH, Tables 11-3 & 11-4, U.S. EPA 2000)[b]	18.7	Average of means (CS-EFH, Table 11-3 & 11-4, U.S. EPA 2000)[b]
FL: Fraction of lipid per body weight (kg lipid/kg BW)	0.25	Used by U.S. EPA Dioxin Reassessment	0.5	Upper-end estimate
CF_1: Conversion factor 1 (g lipid/kg lipid)	1E+03		1E+03	
k: First order rate constant = {Ln(2) / $t_{1/2}$}				
Ln(2): Natural log of 2 (unitless)	0.693		0.693	
$t_{1/2}$: Half life of chemical (days)	6.8	Mean (Sjödin 2000)	3	Lower bound on calculated confidence interval (Sjödin 2000)
CF_2: Conversion factor 2 (mg/ng)	1E-06		1E-06	
ADD: Average daily dose (absorbed) (mg/day)	0.0005	Calculated	0.073	Calculated
BW: Body weight (3–6 yrs) (kg)	18.7	Average of means (CS-EFH, Tables 11-3 & 11-4, U.S. EPA 2000)	18.7	Average of means (CS-EFH, Tables 11-3 & 11-4, U.S. EPA 2000)
ABS: Absorption (percent)	2	El Dareer et al. 1987	1	In this equation, use of a lower ABS will result in a higer intake estimate.
Daily Intake (mg/kg-day)	1.2E-03	Calculated	3.9E-01	Calculated

$ADD = C_{ss} \times V_d \times CF_1 \times k \times CF_2$
$ADD = C_{ss} \times \{BW \times FL\} \times CF_1 \times \{Ln(2) / t_{1/2}\} \times CF_2$
Daily Intake = ADD / (BW × ABS)
Note: The daily intake must be converted from an absorbed dose to an ingested dose because the toxicity value (RfD) is calculated based on an ingested dose.
[a] Values converted from pmol/g lipid to ng/g lipid using the formula:
(pmol/g lipid) × (959 g/mol) × (1 mol/10^{12} pmol) × (10^9 ng/1 g) = ng/g lipid
[b] In this equation for daily intake, the value for body weight is in both the numerator and the denominator, and thus, will cancel out. Therefore, these calculated intakes are applicable to a receptor of any age.

5.5.8 Aggregate Exposure Estimate and Discussion of Uncertainties

The calculations presented here suggest that the potential exposures for each scenario evaluated are very small. Furthermore, the upper estimates (UEs) for each scenario are considerably larger than the reasonable estimates (REs)—several orders of magnitude larger for some of the scenarios. For each of the scenarios, it must be stressed that the RE is very likely to be an estimate of exposure that is greater than the actual exposure experienced by the mean of the U.S. population. For example, the prevailing opinion of experts in the field is that DBDPO does not partition into breast milk. However, given the uncertainties that still exist for this pathway (because of the lack of definitive proof that DBDPO does not exist in breast milk), a conservative assumption was made to calculate exposure. As can be seen from Figures 5-1 and 5-2, the breast-milk exposures calculated for the worker scenarios are among the highest exposure potentials calculated, if not the highest. If DBDPO is not present in breast milk, then exposure via this pathway would be zero for all populations.

The UE estimates of exposure almost certainly represent levels that no one would actually receive, and no exposures would be expected to be above that level. This approach of calculating an exposure that is unlikely to occur for anyone was undertaken to develop an upper bound on potential exposures. The actual upper bound may be less than that calculated here. Barring evidence that the U.S. general population has serum levels on the order of 100–300 ng/g lw DBDPO, this conclusion would not change. Preliminary results from recent surveys conducted by the Centers for Disease Control (CDC) indicate that DBDPO serum levels in U.S. citizens have not changed appreciably compared to the levels measured in the late 1980s (Patterson 2002, personal communication). Therefore, it is not likely that the results presented herein would need to be adjusted upward for the early 2000s, as opposed to the late 1980s.

These UEs for the general environment scenario should be placed into appropriate perspective. Assuming that all U.S. citizens are exposed to DBDPO in the environment at the UE levels, then humans in the U.S. ingest over 13% of the total volume of DBDPO produced in the U.S. each year. This is obviously not the case; therefore, the UE estimates should be considered upper bounds that are not likely to occur.

Table 5-20 summarizes the aggregate exposures experienced by the three populations evaluated in this exposure assessment—the infant of a mother working in the bagging operations at a DBDPO manufacturing site (infant, manufacturer), infant of a mother who disassembles computer monitors (infant, disassembler), and a child's average exposures associated with DBDPO in the environment. The infant of a mother in the bagging operation would experience exposures from drinking the mother's milk, ingesting DBDPO while mouthing electronic consumer products, ingesting DBDPO while mouthing furniture fabric, and from general environmental exposures. The infant of a mother who disassembles computer monitors would have the same exposures, except that the mother's breast milk would contain a different amount of DBDPO. Children through age 18 who do not breast feed would be exposed only via the general environment.

There is up to an order-of-magnitude difference between the RE and UE for the two infant scenarios, and a two order-of-magnitude difference between the RE and UE for the general environment scenario (Table 5-20). The highest estimated exposure (UE for the infant, manufacturer scenario) is 0.76 mg DBDPO/kg-day. The lowest estimated exposure (RE for the general environment scenario) is 0.0012 mg DBDPO/kg-day.

Table 5-20. Summary: Estimated pathway-specific and aggregate U.S. children's exposures to DBDPO.

Daily Intakes	Exposure Duration (yrs)	Reasonable Estimate	Upper Estimate
Pathway-specific			
Ingestion, breast milk (manufacturer); mg/kg-day	0–2	1.9E-02[a]	3.4E-01
Ingestion, breast milk (disassembler); mg/kg-day	0–2	3.3E-06[a]	2.5E-05
Ingestion, consumer electronic products; mg/kg-day	0–2	4.3E-06	2.5E-04
Ingestion, mouthing fabric (NAS); mg/kg-day	0–2	2.6E-02	2.6E-02
General exposures; mg/kg-day	0–18	1.2E-03	3.9E-01
Aggregate			
Infant, manufacturer[b]; mg/kg-day	--	0.046[b]	0.76[b]
Infant, disassembler[c]; mg/kg-day	--	0.027[c]	0.41[c]
Lifetime (0–70)[d]; mg/kg-day	--	0.0012[d]	0.39[d]

[a] Assumes a shorter duration for nursing (0–3 months), based on Collaborative Group on Hormonal Factors in Breast Cancer 2002.
[b] This value incorporates the intakes for ingestion of breast milk from a mother who is a manufacturer, plus ingestion from consumer electronic products, ingestion from mouthing fabric, and general exposures.
[c] This value incorporates the intakes for ingestion of breast milk from a mother who is a disassembler, plus ingestion from consumer electronic products, ingestion from mouthing fabric, and general exposures.
[d] This value incorporates the intake from general exposures. See text for details.

Some of the highest estimated exposures for both the RE and UE scenarios are associated with breast-milk ingestion by an infant whose mother works in the bagging operation at a DBDPO manufacturing site. There are only two facilities in the U.S. that manufacture DBDPO, the number of employees who might be exposed at these levels is less than 50, and no women are employed in the bagging operation (Personal communication, BFRIP). (Women are not excluded from this task, but the type of work does not attract female employees.) It is less certain how many employees might be involved in compounding operations where DBDPO bags are emptied into hoppers prior to incorporating into a polymer matrix. It is likely that fewer than 1,000 employees are engaged in this type of work. Of these potential 1,000 employees, very few, at any point in time, would be lactating mothers. Therefore, fewer than 10 infants would be estimated to be exposed to the highest levels predicted for the infant, manufacturing scenario.

The number of infants that might be exposed to breast milk from a mother who disassembles computer monitors, molds plastic casings, or handles flame-retarded upholstery textiles in the

workplace might be larger—perhaps several thousands. Therefore, the corresponding number of nursing infants that might have this exposure potential might number in the hundreds.

The remaining evaluations are for exposures that all U.S. infants have the potential to experience. Because residential furniture is required to meet fire safety standards (and thus might utilize DBDPO in the textiles) only in California, the number of infants that actually might be exposed to DBDPO via upholstery textiles is likely a fraction of the U.S. population. The calculations for the general-population exposures (child through age 18) also reflect the exposures experienced by the entire U.S. population, even though, again, only a fraction of the population might incur such exposures.

For all of these scenarios, it must be stressed that the exposures predicted in this evaluation are meant to be highly conservative (as a screening estimate), and the actual exposures experienced by nursing infants, infants that mouth fabrics, and the general population are very likely to be below the levels predicted for the RE, and far below the UE estimates.

6.0 RISK ASSESSMENT

DBDPO is used solely as a flame retardant, and in all applications is encapsulated in a polymer matrix with no direct consumer exposure. Its primary application is in electrical and electronic equipment with a secondary application in upholstery textiles. A typical U.S. application for DBDPO is in television cabinets composed of high impact polystyrene. DBDPO is not sold directly to the public.

DBDPO is a data rich chemical with virtually all VCCEP Tier I, II and III hazard endpoints fulfilled. It is a large poorly absorbed molecule that exhibits little toxicity. Testing has shown that DBDPO is not acutely toxic or mutagenic, and is not a developmental or reproductive toxicant. The NOAEL for DBDPO in subchronic and/or chronic studies in the rat or mouse is at least 1,000 mg/kg/d. DBDPO's low toxicity is likely related to its poor absorption and rapid elimination (NTP 1986). Pharmacokinetic studies have shown that DBDPO is poorly absorbed (0.3 -2% of an oral dose), has a short half-life (24 hr in rats) compared to PCB 153 (<2% of an oral dose was eliminated by rats in 21 days), and is rapidly eliminated in the feces (>99% in 72 hr in rats) (NTP 1986; Norris et al. 1973, 1975; El Dareer et al. 1987; Moreck and Klassen-Wheler 2001).

These features coupled with DBDPO's low potential for migration out of plastic resin are indicative of low risk. The U.S. National Academy of Sciences (NAS) evaluated the potential risk to the consumer posed by DBDPO-treated upholstery textiles. In all scenarios evaluated by NAS, dermal, oral or inhalation exposure to DBDPO was determined not to present a risk of adverse health effects to the consumer, including children mouthing upholstery textiles. A similar conclusion was reached in the current assessment with respect to exposures resulting from DBDPO's use in electrical and electronic applications. The WHO and the European Union also concluded the general population is at negligible risk from DBDPO.

Exposure to DBDPO could potentially occur through food or breast milk. However, DBDPO has not been detected in limited sampling of fish and poultry in the U.S., and based on its properties, is not anticipated to be present in these food items or in meat or dairy products. Likewise, leafy vegetables and root crops are not expected to be a source of DBDPO exposure to the general public, and a risk of adverse health effects is not anticipated.

DBDPO transfer to breast milk is likely to be slow and very limited, if at all. Protein binding, ion trapping and lipid partitioning are not expected to alter DBDPO concentrations in breast milk due to DBDPO's physical/chemical properties. Build-up of DBDPO concentrations in breast milk is not expected due to its anticipated slow diffusion into milk and periodic emptying of breast milk. This combination of low absorption from the gut, rapid elimination in the feces, poor and/or slow diffusion into breast milk should effectively preclude DBDPO in milk. Thus, a risk to the nursing infant is not anticipated.

Highly conservative estimates of U.S. DBDPO pathway-specific and aggregate exposures (Table 6-1) are substantially lower than DBDPO's NOAEL of 1,000 mg/kg/d and the reference dose (RfD), 4 mg/kg/d, calculated by NAS[2] (NAS 2000). These estimated exposures are intentionally biased to generate worst-case exposures; actual exposures in the U.S. are expected to be substantially lower. For noncancer health effects, quantitative risk estimates are typically provided in the form of Hazard Quotients (HQs). The HQ represents the estimated exposure for a specific chemical divided by the reference dose (RfD), expressed in mg/kg-day. As such, HQs indicate the calculated exposure estimates in comparison to an exposure level that is unlikely to result in adverse health effects. If an HQ value is less than one, then it can reasonably be assumed that the chemical exposure will not be associated with toxicity. As HQ values increase above one, the potential for toxicity increases. As shown in Table 6-1, all calculated HQs for DBDPO are significantly less than one, with the highest aggregate HQ of 0.2 being five-fold lower than one. Thus, these HQs indicate that even when using conservative, worst-case estimates of exposure to DBDPO, adverse health effects are not expected.

The protection provided by DBDPO in terms of enhanced fire safety reduces the very real risk of death or injury that consumers face in the home from fires. In the applications in which DBDPO is used, an estimated 280 lives are saved each year in the U.S. through the use of a brominated flame retardant. These estimated lives-saved are particularly relevant to the VCCEP program, because children are especially vulnerable to fire deaths and injuries. The benefits derived from the use of DBDPO in consumer products, particularly for children, far outweigh the insignificant potential for harm.

[2] NAS derived an oral RfD for DBDPO by using the chronic NOAEL of 1,120 mg/kg-d, based on liver thrombosis and degeneration observed in rats at the next higher dose (NTP 1986), and a composite uncertainty factor of 300, resulting in an RfD of 4 mg/kg-d (RfD = NOEL ÷ 300). In the IRIS Database, EPA provides a reference dose (RfD) of 1 x 10-2 mg/kg-d for DBDPO based on the 1 mg/kg-d NOAEL for histopathology and other toxicity endpoints in rats exposed via diet for 2 yr (Kociba et al. 1975). Doses higher than 1 mg/kg-d were not tested in this study, precluding identification of a LOAEL. The reason the NTP (1986) 2-yr toxicology/carcinogenesis bioassay for DBDPO was not considered in the current Risk summary in IRIS (EPA 1999) is because the NTP results were not available at the time of the Risk derivation (1984-1985).

TABLE 6-1. DBDPO exposure estimates and hazard quotient based on a RfD of 4 mg/kg/day.

Daily Intakes	Exposure Duration (yrs)	Exposure Estimate (mg/kg/d)		Hazard Quotient (RfD = 4 mg/kg/d[e])	
		Reasonable	Upper	Reasonable Estimate	Upper Estimate
Pathway-specific					
Ingestion, breast milk-manufacturer	0–2	1.9E-02[a]	3.4E-01	0.005	0.09
Ingestion, breast milk-disassembler	0–2	3.3E-06[a]	2.5E-05	8E-07	6E-06
Ingestion, consumer electronics	0–2	4.3E-06	2.5E-04	1E-06	6E-05
Ingestion, mouthing fabric (NAS)	0–2	2.6E-02	2.6E-02	0.007	0.007
General exposures	0–70	1.2E-03	3.9E-01	0.0003	0.1
Aggregate					
Infant, manufacturer[b]	--	0.046[b]	0.76[b]	0.01	0.2
Infant, disassembler[c]	--	0.027[c]	0.41[c]	0.007	0.1
Lifetime (0–70)[d]	--	0.0012[d]	0.39[d]	0.0003	0.1

[a] Assumes a shorter duration for nursing (0–3 months), based on Collaborative Group on Hormonal Factors in Breast Cancer 2002.
[b] This value incorporates the intakes for ingestion of breast milk from a mother who is a manufacturer, plus ingestion from consumer electronic products, ingestion from mouthing fabric, and general exposures.
[c] This value incorporates the intakes for ingestion of breast milk from a mother who is a disassembler, plus ingestion from consumer electronic products, ingestion from mouthing fabric, and general exposures.
[d] This value incorporates the intake from general exposures. See text for details.
[e] The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data. The RfD for DBDPO, 4 mg/kg/d, was calculated by the U.S. National Academy of Sciences instead of using the current 1999 IRIS Rfd (0.01 mg/kg/d). NAS calculated a revised RfD for DBDPO using the NTP 2 year bioassay results, which were not available at the time of the IRIS derivation (1984-1985).

7.0 DATA NEEDS ASSESSMENT

Data is available on DBDPO for essentially all Tier I, II and III hazard endpoints (Table 7-1). The U.S. National Academy of Sciences concluded its review of DBDPO with a finding that no additional information was needed to evaluate its risk to the consumer through the use of flame-retarded upholstery textiles. BFRIP concurs with the findings of that assessment, and believe they also apply to DBDPO's use in electrical and electronic equipment and for exposures from the diet and ambient environment.

While available data do not provide for an accurate estimation of children's actual exposures to DBDPO, the exposure assessment conducted as part of this VCCEP submission is so conservative in nature that it most likely vastly overestimates actual exposures that might be encountered by children. Despite their overly conservative nature, the exposure estimates are below the lifetime daily dose expected to result in no harmful effects (RfD). Therefore,

additional data to help refine our estimates of children's exposures to DBDPO appear unnecessary.

TABLE 7-1. A comparison of the data available on DBPDO to the studies listed in the VCCEP's Tiers I, II and III.

TESTS	DATA AVAILABLE?
Tier I	
Acute Oral	Yes
Acute Inhalation	Yes
In vitro Gene Mutation – Bacterial Reverse Mutation	Yes
Repeated Dose Oral Toxicity	Yes: In Two Species
Reproductive Toxicity (1-Generation)	Yes
In vitro Chromosome Aberrations	Yes. Additional mutagenicity results are available from mouse lymphoma and sister chromatid exchange studies
Tier II	
90-Day Subchronic Toxicity in Rodents	Yes: In Two Species
Reproduction and fertility effects (2-Generation)	Data is not available from a 2-generation study. Data from repeated dose, developmental and reproduction studies do not indicate effects on reproduction or fertility.
Prenatal Developmental Toxicity (Two Species)	Yes: One Species
In vivo mammalian bone marrow chromosome aberrations	Evaluated as a part of a 1-Generation study
Immunotoxicity	Data is not available from studies performed via the listed guideline. No indication of immunotoxicity was observed in two species tested at high dose levels administered over a two year time period.
Metabolism and Pharmacokinetics	Yes
Tier III	
Carcinogenicity	Yes: Two Species
Neurotoxicity Screening Battery	Data is not available from a study performed via the listed guideline. Data from repeated dose studies at very high dose levels in two species do not indicate an effect on the nervous system.
Developmental Neurotoxicity	Data is not available from a study performed via the listed guideline.

REFERENCES

AIHA. 1981. Workplace environmental exposure level guide: Decabromodiphenyl oxide. American Industrial Hygiene Association, American Industrial Hygiene Association, 2700 Prosperity Avenue, Suite 250, Fairfax, VA 22031.

AIHA. 1996. Workplace Environmental Exposure Level: Decabromodiphenyl Oxide. American Industrial Hygiene Association, 2700 Prosperity Avenue, Suite 250, Fairfax, VA 22031.

Alaee, M. 2001. Levels and trends of PBDEs in North American Environment. Second International Workshop on Brominated Flame Retardants, May 14–16, Stockholm University, Sweden, 131–134.

Anderson P. 1991. Drug use during breast-feeding. Clinical Pharmacy Vol. 10, Aug 594-624.

Aufderheide J, Kendall T and Nixon W. 2001. Effect of decabromodiphenyl oxide on the survival and reproduction of the earthworm, *Eisenia fetida*. Study No. 46540. ABC Laboratories, Inc., Columbia, Missouri.

Babrauskas V, Harris R Jr., Gann R, Levin B, Lee B, Peacock R, Paabo M, Twilley W, Yoklavich M and Clark H. 1988. Fire Hazard Comparison of Fire-Retarded and Non-Fire-Retarded Products. U.S. Department of Commerce, National Bureau of Standards, NBS Special Publication 749. Available From National Technical Information Service (NTIS), Technology Administration, U.S. Department of Commerce, Springfield, VA 22161. Website: http://www.ntis.gov. Order number: PB88-249966.

Bailey B and Ito S. 1997. Breast-feeding and maternal drug use. Pediatric Clinics of North America, Vol. 44, No1, February, 41- 54.

Brenner K and Knies H. 1990. Formation of polybrominated dibenzofurans (PBDE's) and –Dioxins (PBDD's) during extrusion production of a polybutyleneterephthlate (PBTP)/glassfibre resin blended with decabromodiphenyl ether (DBDPE)/Sb2O3: product and workplace analysis. Organohalogen Compounds, Vol 2, 319-324.

Bromine Science and Environmental Forum (BSEF) Web Site. http://www.bsef.com. 2001.

Burreau S, Axelman J, Erornan D and Jakobsson E. 1997. Dietary uptake in pike *(Esox lucius)* of some polychlorinated biphenyls, polychlorinated naphthalenes and polybrominated diphenyl ethers administered in natural diet. Environmental Toxicology and Chemistry 15, 2508-2513.

Carlson G. 1980. Induction of xenobiotic metabolism in rats by short-term administration of brominated diphenylethers. Toxicol Lett 5, 19-25.

Cramer P, Stanley J and Thronburg K. 1990. Mass Spectral Confirmation of Chlorinated and brominated Diphenyethers in Human Adipose Tissue. EPA-560/5-90-012. Washington, D.C.

Chou S, Jacobs L, Penner, D and Tiedje J. 1978. Absence of plant uptake and translocation of polybrominated biphenyls (PBBs). Environmental Health Perspectives, Vol. 23, 9-12.

Clarke F. 1997. The life safety benefits of brominated flame retardants in the United States. Final Report to the Chemical Manufacturers Association Brominated Flame Retardant Industry Panel. Benjamin/Clarke Associates.

CITI. 1992. Biodegradation and Bioaccumulation Data of Existing Chemicals Based on the CSCL Japan. Compiled under the supervision of Chemical Products Safety Division, Basic Industries Bureau, Ministry of International Trade & Industry, Japan. Edited by Chemicals Inspection & Testing Institute, Japan. Published by Japan Chemical Industry Ecology-Toxicology & Information Center.

Darnerud P, Atuma S, Aune M, Cnattingius S, Wernroth M and Wicklund-Glynn A. 1998. Polybrominated Diphenyl Ethers (PBDEs) in Breast Milk from Primiparous Women in Uppsala County, Sweden. Organohalogen Compounds, 35, 411-414.

Davison A and Dobbing J. 1968. The developing brain. In Davison AN, Dobbing J (eds): Applied Neurochemistry. Philadelphia: F.A. Davis, pp 253-286.

DeCarlo, V.J. 1979. Studies on brominated chemicals in the environment. Annals of the New York Academy of Sciences 320:678–681.

de Boer J, Aldridge J, Allchin C, Bennett M, Boon J, Brandsma S, van Hesselingen J, Law R, Lewis W, Morris S, Tjoen-A-Choy M and Zegers B. 2001. Polybrominated Diphenylethers in the Aquatic Environment. Netherlands Institute for Fisheries Research (RIVO) BV. RIVO report Number: C023/01.

de Wit, C.A. 2000. Brominated flame retardants. Report 5065. Swedish Environmental Protection Agency. Stockholm, Sweden.

Di Carlo F, Seifter J and DeCarlo V. 1978. Assessment of the hazards of polybrominated biphenyls. Environ Health Perspect **23**, pp. 351-365.

Donnelly J, Grange, A, Nunn N, Sovocool G, Brumley W and Mitchum R. 1987. Biomedical and Environmental Mass Spectrometry 18(10), 884-96.

ECB. 2002. European Union risk assessment report: bis(pentabromophenyl) ether. European Chemicals Bureau, Institute for Health and Consumer Protection. Vol. 17.

Education World. 2002. Lesson Planning Article. Fire Safety: Activities to Spark Learning! http://www.education-world.com/a_lesson/lesson026.shtml.

El Dareer S, Tillery K, Hill D, Kalin J. 1987. Disposition of decabromodiphenyl ether in rats dosed intravenously or by feeding. J Toxicol Environ Health, 22, 405-415.

Eriksson P, Ahlbom J, Fredriksson A. 1992. Exposure to DDT during a defined period in neonatal life induces permanent changes in brain muscarinic receptors and behavior in adult mice. Brain Research 582(2):277-81.

Eriksson P. 1997. Developmental neurotoxicity of environmental agents in the neonate. Neurotoxicology, 18(3):719-26.

Eriksson P, Jakobsson E and Fredriksson A. 1998. Developmental neurotoxicity of brominated flame-retardants, polybrominated diphenyl ethers and tetrabromo-bis-phenol A. Organohalogen Compounds 35, pp. 375-377.

Eriksson P and Talts U. 2000. Neonatal exposure to neurotoxic pesticides increases adult susceptibility: a review of current findings. Neurotoxicology 21(1-2):37-47.

Eriksson J, Jakobsson E, Marsh G, Bergman A. 2001. Photodecomposition of brominated diphenylethers in methanol/water. In: The Second International Workshop on Brominated Flame Retardants, May 14 –16.BFR 2001.Stockholm.Stockholm University,Sweden,pp.203 –206.

Ethyl Corporation, Baton Rouge, LA (1981). Assay of comedogenicity in the rabbit ear (Saytex 102). Unpublished Laboratory Report. Pharmakon Laboratories.

Ethyl Corporation, Baton Rouge, LA (1986). Toxicity data on DBDPO (Saytex 102). Primary Eye Irritation. Unpublished Laboratory Report. Pharmakon Research International Inc.

FRCA 1987. Fire Retardant Chemicals Association: Reduction of Fire Hazard Using Fire Retardant Chemicals. Belles and Associates, Madison, TN.

Fries G, Marrow G and Cook R. 1978. Distribution and kinetics of PBB residues in cattle. Environ Health Perspectives, Vol. 23, 43-50.

Furst P. 2001. Organochlorine Pesticides, Dioxins, PCB and Polybrominated Biphenylethers in Human Milk from Germany in the Course of Time. Organohalogen Compounds.

Garner C and Mathews H. 1998. The effect of chlorine substitution on the dermal absorption of polychlorinated biphenyls. Toxicol. Appl. Pharmacol. 149,2, 150-158.

Great Lakes Chemical Corporation, West Lafayette, IN (1974a). Toxicity data on DBDPO (DE-83). Acute oral toxicity in the Albino rat. Unpublished Laboratory Report, Intl. Res. & Dev. Corp.

Great Lakes Chemical Corporation, West Lafayette, IN (1974b). Toxicity data on DBDPO (DE-83). Acute dermal toxicity in the Albino rabbit. Unpublished Laboratory Report, Intl. Res. & Dev. Corp.

Great Lakes Chemical Corporation, West Lafayette, IN (1974c). Toxicity data on DBDPO (DE-83). Acute inhalation toxicity in the male Albino rat. Unpublished Laboratory Report, Intl. Res. & Dev. Corp.

Great Lakes Chemical Corporation, West Lafayette, IN (1974d). Toxicity data on DBDPO (DE-83). Primary skin irritation in Albino rabbits. Unpublished Laboratory Report, Intl. Res. & Dev. Corp.

Great Lakes Chemical Corporation, West Lafayette, IN (1974e). Toxicity data on DBDPO (DE-83). Primary eye irritation in Albino rabbits. Unpublished Laboratory Report, Intl. Res. & Dev. Corp.

Guy R.H. 1996. Review: Current Status and Future Prospects of Transdermal Drug Delivery. Pharmaceutical Research, 13, 12, 1765- 1769.

Guyton A. 1986. Textbook of Medical Physiology (W.B. Saunders Company, Philadelphia, ed. Seventh Edition).

Hakk H, Larsen G, Klasson-Wehler E, Orn U and Bergman A. 1999. Tissue Disposition, Excretion, and Metabolism of 2,2'4,4',5-Pentabromodiphenyl ether (BDE-99) in Male Sprague-Dawley Rats. Organohalogen Compounds, 40, 337-339.

Hale R, La Guardia M, Harvey E, Mainor T. 2002. Potential role of fire retardant-treated polyurethane foam as a source of brominated diphenyl ethers to the US environment. Chemosphere, Feb, 46(5):729-35.

Hale, R., M. La Guardia, E. Harvey, and M. Mainor. 2001. Brominated diphenyl ethers in land-applied sewage sludges in the US. Second International Workshop on Brominated Flame Retardants, May 14–16, Stockholm University, Sweden, pp149–152.

Hamm S. 1999. Analysis of a decabromodiphenyloxide blend, a HIPS plastics, the HIPS plastic containing the DecBDPO and Sb2O3 and the repeatedly recycled HIPS/Sb2O3/DecaBDPO plastic for partially brominated diphenylethers and 8 polybrominated dibenzo(p)dioxin and dibenzofuran congeners. Report 60425-001 B01. GfA. Munster, Germany.

Hamm S, Strikkeling M, Ranken P, Rothenbacher K. 2001. Determination of polybrominated diphenyl ethers and PBDD/Fs during the recycling of high impact polystryrene containing decabromodiphenyl ether and antimony oxide. Chemosphere Sep;44(6):1353-1360.

Hagmar L, Jakobsson K, Thuresson K, Rylander L, Sjodin A and Bergman A. 2000. Computer technicians are occupationally exposed to polybrominated diphenyl ethers and tetrabromobisphenol A. Organohalogen Compounds, 47, 202-205.

Hardy M, Schroeder R, Biesemeier J and Manor O. 2002. Prenatal oral (gavage) developmental toxicity study of decabromodiphenyl oxide in rats. International Journal of Toxicology, 21,83-91.

Hardy M. 2002a. The toxicology of the three commercial polybrominated diphenyl oxide (ether) flame retardants. Chemosphere 46, 757-777.

Hardy M. 2002b. Properties of the major commercial PBDPO flame retardant, DBDPO, in comparison to PBB and PCB. Chemosphere 46, 717-748.

Hardy M. 2000. Distribution of Decabromodiphenyl Oxide in the Environment. Organohalogen Compounds 47, 237-240.

Hardy M and Smith R. 1999. The potential of certain brominated flame retardants for persistence, bioaccumulation, or long range transport. Proceedings of the American Chemical Society Annual Meeting. Anaheim, CA.

Hughes, M.F., B.C. Edwards, C.T. Mitchell, and B. Bhooshan. 2001. In vitro dermal absorption of flame retardant chemicals. Food Chem. Toxicol. 39(12):1263–1270.

Huwe J., M. Lorentzsen, K. Thuresson, and Å. Bergman. 2002. Analysis of mono- to deca-brominated diphenyl ethers in chickens at the part per billion level. Chemosphere 46(5):635–640.

IARC. 1990. Monographs on the Evaluation of Carcinogenic Risks to Humans. Some Flame Retardants and Textile Chemicals, and Exposures in the Textile Manufacturing Industry. International Agency for Research on Cancer. Lyon, France. Vol 48:73-84.

Industrial Bio-Test Laboratories. 1975. Human repeated insult patch test with DBDO-1 and XD 8186.02. Report No. IBT 636-0654. Northbrook, IL.

Iwata Y, Gunther F and Westlake W. 1974. Uptake of PCB (Arochlor 1254) from soil by carrots under field conditions. Bulletin of Environmental Contamination 11:523.

Jackson J, Dilberto J and Birnbaum L. 1993. Estimation of octanol-water partition coefficients and correlation with dermal absorption for several polyhalogenated aromatic hydrocarbons. Fund and Appl. Toxicol. 21, 334-344.

Jacobs L, Chou S, and Tiedje M. 1976. Fate of polybrominated biphenyls (PBBs) in soils. Persistence and plant uptake. J. Agric. Food Chem., Vol. 24, 1198.

Jacobs L, Chou S, Tiedje J. 1978. Field concentrations and persistence of polybrominated biphenyls in soils and solubility of PBB in natural waters. Environmental Health Perspectives, Vol. 23, 1-8.

Jafvert C and Hua I. 2001. Photochemical reactions of decabromodiphenyl oxide and 2,2',4,4'-tetrabromodiphenyl oxide. Final Report. School of Civil Engineering. Purdue University. West Lafayette, IN.

Karter, Jr. M. 2001. NFPA's 2000 United States Fire Loss, September 2001.

Kierkegaard A, Balk L, Tjarnlund U and de Wit C. Uptake of decabromodiphenyl ether in the rainbow trout via administration in the diet. Presented at the SETAC Meeting 16-20 November 1997.

Kierkegaard A, Balk L, Tjarnlund U, de Wit C and Jansson B. 1999. Dietary uptake and biological effects of decabromodiphenyl ether in rainbow trout (Oncorhynchus mykiss). *Environ. Sci. Technol.* 33, 1612-17.

KEMI 1999. Risk assessment of PBDE. National Chemicals Inspectorate, Sweden. Letter dated 14th July 1999. As cited in ECB 2002.

Kociba R, Frauson L, Humiston C, Norris J, Wade C, Lisowe R, Quast J, Jersey G and Jewett G. 1975. Results of a two-year dietary feeding study with decabromodiphenyl oxide (DBDPO) in rats. *Combustion Toxicology* 2, pp. 267-285.

Kodavanti P, Ward T, Derr-Yellin E, Mundy E, Case A, Bush B and Trotter W. 1998. Congener- specific distribution of polychlorinated biphenyls in brain regions, blood, liver, and fat of adult rats following repeated exposure to Arochlor 1254, *Tox Applied Pharm* 153, pp. 199-210.

Kroger M. General environmental contaminants occurring in milk. In: Lactation, Nutrition and Biochemistry of Milk/Maintenance, Vol III (Larson BL and Smith VR, eds). New York: Academic Press, Inc.; 1974:135-158

Krueger H, Kendall T and Jaber M. (2001a) Decabromodiphenyl ether: a prolonged sediment toxicity test with *Lumbriculus variegates* using spiked sediment with 2% total organic carbon. Final Report. Project Number: 439A-113. Wildlife International, LTD., Easton, MD.

Krueger H, Kendall T and Jaber M. (2001b) Decabromodiphenyl ether: a prolonged sediment toxicity test with *Lumbriculus variegates* using spiked sediment with 5% total organic carbon. Final Report. Project Number: 439A-114. Wildlife International, LTD., Easton, MD.

LaKind J and Berlin C. 2001. PBDEs in Breast Milk: Where Do We Go From Here? Organohalogen Compounds 47, 241-244.

Lind Y, Aune, M, Bjerselius R, Darnerud P, Cnattingius S and Glynn A. 2001. Polybrominated diphenyl ethers (PDEs) in breast milk from Uppsala women – extension and up-dating of data. Proceedings, BFR2001 Stockholm, pages 117-120.

Lioy P, Weisel C, Millette J, Eisenreich S, Vallero D, Offenberg J, Buckley B, Turpin B, Zhong M, Cohen M, Prophete C, Yang I, Stiles R, Chee G, Johnson W, Porcja R, Alimokhtari S, Hale R, Weschler C and Chen L. 2002. Characterization of dust/smoke aerosol that settled east of the World Trade Center (WTC) in lower Manhattan after the collapse of the WTC 11 September 2001. Environmental Health Perspectives. 110,7:703-714.

Lippmann M, Yeates D and Albert R. 1980. Deposition, retention, and clearance of inhaled particles. British Journal of Industrial Medicine 37, 337-362.

Leihbacher D. 1999. Search in the Modern Environment. Fire Engineering, July, 65-76.

Loebstein R, Lalkin A, and Koren G. 1997. Pharmacokinetic changes during pregnancy and their clinical relevance. Clin Pharmacokinet Nov; 33(5):328-43.

MacGregor J and Nixon W. 1997. Decabromodiphenyl oxide (DBDPO): Determination of n-octanol water partition coefficient. Wildlife International Ltd., Easton, MD.

Maronpot R, Montgomery C, Boorman G and McConnell E. 1986. National Toxicology Program nomenclature for hepatoproliferative lesions of rats. Toxicol Pathol. 14, 263-273.

Matthews H, Fries G, Gardner A, Garthoff L, Goldstein J, Ku Y and Moore J. 1978. Metabolism and biochemical toxicity of PCBs and PBBs. Environmental Health Perspectives 24, 147-155.

Matthews H and Dedrick R. 1984. Pharmacokinetics of PCBs. Annual Reviews of Pharmacology and Toxicology 24, 85-103.

Matthews H, Kato S, Morales M, and Tuey D. 1977. Distribution and excretion of 2,4,5,2',4',5'-hexabromobiphenyl, the major component of Firemaster BP-6. J Toxicol Environm Health, Oct; 3(3):599-605.

McAllister D, Mazac C, Gorsich R, Freiberg M and Tondeur Y. 1990. Analysis of polymers containing brominated diphenyl ethers as flame retardants after molding under various conditions. Chemosphere, 20, 10-12:1537-1541.

McGregor D, Edwards I, Riach C, Cattanach P, Martin R, Mitchell A and Caspary W. 1988. Studies of an S9-based metabolic activation system used in the mouse lymphoma L5178Y cell mutation assay. Mutagenesis 3(6):485-90.

McIntyre, J.E., I. Holme, and O.K. Sunmonu. 1995. The desorption of model compounds from poly(ethylene terephthalate) fibre. Colourage 41(13):77–81.

Meironyte D, Bergman A and Noren K. 1998. Analysis of Polybrominated Diphenyl Ethers in Human Milk. Organohalogen Compounds, 35, 387-390.

Meyland W and Howard P. EPI – Estimation Programs Interface for Microsoft Windows. US EPA Version for Windows. June 1999. Syracuse Research Corporation, North Syracuse, NY.

Morck A and Klasson Wehler E. 2001. Metabolism of decabromodiphenyl ether (BDE-209) in the rat. BFR 2001 Stockholm. The Second International Workshop on Brominated Flame Retardants. May 14-16, 2001. Stockholm University, Sweden.

NAS 2000. Toxicological Risks of Selected Flame-Retardant Chemicals. National Academy Press. Washington, D.C. http://www.nap.edu.

Naismith R and Matthews R. 1981. Assay of comedogenicity in the rabbit ear of Saytex 102. PH 425-ET-001-81. Pharmakon Research Laboratories, Waverly, PA.

NFPA 2000. National Fire Protection Association. Http://www.nfpa.org.

NHATS 1990. Brominated Dioxins and Furans in Human Adipose Tissue. EPA 560/5-90-005. Washington, D.C.

NSKC 2002. Fire. National Safe Kids Campaign. http://www.safekids.org/tier2_rl.cfm?folder_

NTP 1986. Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No.1163-19-5) in F344/N Rats and B6C3F1 Mice (Feed Studies). National Toxicology Program Technical Report Series No.398. U.S. Department of Health and Human Sciences. Public Health Service. National Institutes of Health. Research Triangle Park, NC.

NTP. 2000. National Toxicology Program. 9th Report on Carcinogens. http://ehis.niehs nih.gov/roc/toc9.htm1.

Norris J, Ehrmantraut J, Gibbons C, Kociba R, Schwetz B, Rose J, Humiston C, Jewett G, Crummett W, Gehring P, Tirsell J and Brosier J. 1973. Toxicological and environmental factors involved in the selection of decabromodiphenyl oxide as a fire retardant chemical, Applied Polymer Symposia. No.22. Polymeric Materials for Unusual Service Conditions. 195-219.

Norris J, Ehrmantraut J, Gibbons C, Kociba R, Schwetz B, Rose J, Humiston C, Jewett G, Crummett W, Gehring P, Tirsell J and Brosier J. 1974. Toxicological and environmental factors involved in the selection of decabromodiphenyl oxide as a fire retardant chemical. JFF/Combustion Toxicology 1, 52-77.

Norris J, Kociba R, Schwetz B, Rose J, Humiston C, Jewett G, Gehring P and Mailhes J. 1975. Toxicology of octabromobiphenyl and decabromodiphenyl oxide. Environ Health Perspect 11, 153-161.

OSHA Standard 1990. http://www.osha-slc.gov/SLTC/carcinogens/index.htm1.

Ohta S, Ishizuka, D, Nishimura, H, Nakao T, Aozasa O, Shimidzu Y, Ochiai F, Kida T and Miyata H. 2000. Real Situation of Contamination by Polybrominated Diphenyl Ethers as Flame Retardant in Market Fish and Mother Milk of Japan. Organohalogen Compounds 47, 218-211.

Orn U and Klasson Wehler E. 1998. Metabolism of 2,2',4,4'-tetrabromodiphenyl ether in rat and mouse. Xenobiotica 28, 2, 199-211.

Patterson D. 2002. Personal communication. September 2002.

Päpke O, Bathe L, Bergman A, Fürst P, Guvenius D, Herrmann T, Norén K. 2001. Determination of PBDEs in Human Milk from the United States. Organohalogen Compounds.

Pinkerton M, Kociba R, Petrella R, McAllister D, Willis M, Fulfs J, Thoma H and Hutzinger O. 1989. A preliminary report on the investigation of the comparative toxicity of combustion products of high impact polystyrene with and without decabromodiphenyl oxide/antimony trioxide as a flame retardant using 2,3,7,8-tetrabromodibenzo-p-dioxin and 2,3,7,8-tetrabromodibenzofuran as positive controls. Chemosphere 1-6, 1243-1249.

Pomerantz I, Burke J, Firestone D, McKinney J, Roach J and Trotter W. 1978. Chemistry of PCBs and PBBs. Environmental Health Perspectives 24, 133-146.

Pons G, Rey E, and Matheson I. 1994. Excretion of psychoactive drugs into breast milk. Pharmacokinetic principles and recommendations. Clin Clin Pharmacokinet, Oct; 27(4):270-89.

Porch J and Krueger H. 2001. Decabromodiphenyl oxide (DBDPO): A toxicity test to determine the effects of the test substance on seedling emergence of six species of plants. Final Report. Project Number: 439-101. Wildlife International, LTD., Easton, MD.

Ranken P, Freiberg M, Mazac C, Bauer M, Varcoe F and Tondeur Y. 1994. Definitive study of the determination of polybrominated dibenzo-p-dioxins and polybrominated dibenzofurans in decabromodiphenyl oxide and tetrabromobisphenol A. Bull. Soc. Chim. Belg. EUROPEAN SECTION. Vol. 103/n 5-6, pp. 219-233.

W.A. Ritschel. Handbook of Basic Pharmacokinetics. 2nd Edition. 1982. Drug Intelligence Publications, Inc., Hamilton, IL.)

Ryan J and Patry B. 2001. Body Burdens and Food Exposure in Canada for Polybrominated Diphenyl Ethers (BDES). Organohalogen Compounds.

Schaefer E and Flaggs R. 2001a. Potential for biotransformation of radiolabelled decabromodiphenyl oxide (DBDPO) in anaerobic sediment. Final Report. Project No: 439E-104. Wildlife International, Ltd. Easton, MD.

Schaefer E and Flaggs R. 2001b. Potential for biotransformation of radiolabelled tetrabromodiphenyl oxide (TeBDPO) in anaerobic sediment. Final Report. Project No: 439E-105. Wildlife International, Ltd. Easton, MD.

Schaefer and Siddiqui. 2001. Decabromodiphenyl oxide: an activated sludge, respiration inhibition test. Final Report. Project No: 439E-106. Wildlife International, Ltd. Easton, MD.

Schaefer E, Ariano J and Rothenbacher K. 2001. Fate Of Decabromodiphenyl Ether In Anaerobic Freshwater Sediment. Organohalogen Compounds.

Schroeder R. 2000. Prenatal oral (gavage) developmental toxicity study of decabromodiphenyl oxide in rats. MPI Research. Mattawan, MI.

Sellstrom U, Soderstrom G, de Wit C and Tysklind M. 1998. Photolytic debromination of decabromodiphenyl ether (DeBDE). Organohalogen Compounds 35:447-450.

Sjodin A, Hagmar L, Klasson-Wehler E, Kronholm-Diab K, Jakobsson E and Bergman A. 1999. Flame retardant exposure: polybrominated diphenyl ethers in blood from Swedish workers. Environmental Health Perspectives 107, 643-648.

Sjodin A. 2000. Doctoral Dissertation: Occupational and Dietary Exposure to Organohalogen Substances, with Special Emphasis on Polybrominated Diphenyl Ethers. Department of Environmental Chemistry, Stockholm University.

Sjodin A, Patterson d, Bergman A. 2001. Brominated flame retardants in serum from U.S. blood donors. Environmental Science and Technology. 35(19):3803-3833.

Sjödin, A., H. Carlsson, K. Thuresson, S. Sjölin, Å. Bergman, and C. Östman. 2001a. Flame retardants in indoor air at an electronics recycling plant and at other work environments. Environmental Science and Technology 35:448–454.

Sjödin A, D.G. Patterson, Jr., and Å. Bergman. 2001b. Brominated flame retardants in serum from U.S. blood donors. Environmental Science and Technology 35(19):3830–3833.

Stanley J, Cramer P, Ayling R, Thornberg K, Remmers, Breen J and Schwemberger J. 1990. Determination of the prevalence of polychlorinated diphenyl ethers (PCDPES) in human adipose tissue samples. Chemosphere 20:981-985.

Strandberg B, Dodder N, Basu I and Hites R. 2001. Concentrations and spatial variations of polybrominated diphenyl ethers and other organohalogen compounds in Great Lakes air. Environmental Science and Technology 35:1078–1083.

Strandman T, Koistinen J and Vartiainen T. 2000. Polybrominated Diphenyl Ethers (PBDEs) in Placenta and Human Milk. Organohalogen Compounds 47, 61-64.

Stenzel J and Markley B. 1997. Decabromodiphenyl oxide (DBDPO): Determination of the water solubility. Wildlife International Ltd., Easton, MD.

Stenzel J and Nixon W. 1997. Decabromodiphenyl oxide (DBDPO): Determination of the vapor pressure using the spinning rotor gauge. Wildlife International Ltd., Easton, MD.

Stevens G and Mann A. 1999. Risks and Benefits in the Use of Flame Retardants in Consumer Products. URN 98/1026. Polymer Research Centre, School of Physical Sciences and School of Biological Sciences, University of Surrey, Guildford, Surrey GU25XH, UK, DTI.

Thuresson K, Jakobsson, K, Hagmar L, Englyst V and Bergman A. 2002a. Work related exposure to brominated flame retardants when recycling metals from printed circuit boards. Organohalogen Compounds, 58: 249-252.

Thuresson K, Jakobsson, Hagmar L, Sjodin A and Bergman A. 2002b. Decabromodiphenyl ether exposure to workers manufacturing rubber and in an industrial setting producing rubber coated electric wires. Organohalogen Compounds, 58: 165-168.

Tulve N, Suggs J, McCurdy, T, Hubal E, and Moya J. 2002. Frequency of mouthing behavior in young children. Journal of Exposure Analysis and Environmental Epidemiology. 12:259-264.

U.S. EPA. 2000a. 65(248) Fed. Reg.:81700–81718). U.S. Environmental Protection Agency, Washington, DC.

U.S. EPA. 2000b. Child-specific exposure factors handbook. External review draft. Prepared for U.S. Environmental Protection Agency, National Center for Environmental Assessment, Office of Research and Development, Washington, DC. NCEA-W-0853. Versar, Inc. June.

USFA. 2002. This Is Fire! A Fact sheet on the nature of fire. United States Fire Administration http://www.usfa.fema.gov/dhtml/public/safety.cfm.

Viberg H, Fredriksson A, Jakobsson E, Orn U and Eriksson P. 2001. Brominated Flame Retardant: Uptake, retention and developmental neurotoxic effects of decabromodiphenyl ether (PBDE 209) in the neonatal mouse. BFR 2001 Stockholm. The Second International Workshop on Brominated Flame Retardants. May 14-16, 2001. Stockholm University, Sweden. pp 279-282.

Walsh G, Yoder M, McLaughlin L, and Lores E. 1987. Responses of marine unicellular algae to brominated organic compounds in six growth media. Ecotoxoxicology and Environmental Safety 14,215 –222.

Watanabe I and Tatsukawa R. 1987. Formation of brominated dibenzofurans from the photolysis of flame retardant decabromobiphenyl ether in hexane solution by UV and sunlight. Bulletin of Environmental Contamination and Toxicology 39,953 –959.

Wagner V and Klug M. 1998. Bacterial Reverse Mutation Assay: decabromodiphenyl oxide. Study Number: G98AV87.503. MA BioServices, Inc., Rockville, MD.

Wania F and Dugani C. 2002. Assessing the long range transport potential of polybrominated diphenyl ethers: a comparison of four multimedia models. Final Report. University of Toronto at Scarborough, Scarborough, Ontario.

Wester R and Maibach H. 1983. Cutaneous pharmacokinetics: 10 steps to percutaneous absorption. Drug Metabolism Reviews 14(2), 169-205.

Wild S and Jones K. 1992. Organic chemicals entering agricultural soils in sewage sludges: screening for their potential to transfer to crop plants and livestock. Science of the Total Environment 119, 85-119.

Willet L and Durst H. 1978. Effects of PBBs on cattle. IV. Distribution and clearance of components of FireMaster BP-6. Environmental Health Perspectives Vol. 23, 67-74.

Wilson J. 1983. Determinants and consequences of drug excretion in breast milk. Drug Metabolism Reviews 14(4), 619-652.

WHO. 1994. Brominated Diphenyl Ethers. World Health Organization International Programme on Chemical Safety Environmental Health Criteria Document Number 162. Geneva.

Wong A, Lei Y, Alaee M and Wania R. 2001. Vapour pressures of the polybrominated diphenylethers. BFR 2001 Stockholm. The Second International Workshop on Brominated Flame Retardants. May 14-16, 2001. Stockholm University, Sweden. pp 195-197.

Zweidinger R, Cooper S, Erickson M, Michael L, and Pellizari E. 1977. Sampling and analysis for semivolatile brominated organics in ambient air. Monitoring Toxic Substances. ACS Symposium Series No 94. D. Schuetzle et al. (Ed.). American Chemical Society, Washington DC, pp. 217–231. Cited, not seen.

Zweidinger R, Cooper S, and Pellizzari E. 1978. Identification and quantitation of brominated fire retardants. Measurement of organic pollutants in water and wastewater. ASTM STP 686, C E Van Hall (Ed.). American Society for Testing and Materials, 234–250. Cited, not seen.

APPENDIX I

M. Hardy. 1997. The Importance of Flame Retardants in Today's Plastics.

THE IMPORTANCE OF FLAME RETARDANTS IN TODAY'S PLASTICS

M. L. Hardy
Albemarle Corporation 1997 Far East Seminars

I. INTRODUCTION: FIRE LOSSES DUE TO ELECTRONIC EQUIPMENT

The development and extensive use of synthetic polymers in varied applications has intensified the need and concern for flame retardancy. Synthetic polymers are not necessarily more flammable than natural polymers, but synthetic polymers are more easily used in forms such as electrical applications that can result in an increased fire safety problem. Further, polymers have fuel values comparable to common fuels such wood, oil, or alcohol and will contribute to the burning process in a typical fire. Early in their development and use, the small size of fabricated plastic articles and their relative scarcity made fire retardancy a secondary consideration. However, technology has led to more numerous and increasingly large-scale applications so that the potential contribution of polymers to fires cannot be overlooked. Plastics will help fuel a fire and merely isolating electrical components within the appliance will not solve the problem that plastic housings can contribute to fire.

Fire takes a tremendous toll on society. The total number of fire fatalities in 7 countries (Japan, United States, Canada, United Kingdom, Germany, France and Italy) exceeds 8,800 each year according to recent statistics from the World Health Organization. In Japan alone fire fatalities are approximately 1,200 per year *(Tsuda, Y. J. Investigation of Fire Fatalities: A Study of the Influence of Toxic Gases on Fire. Med. Soc. Toho, Japan, 1996, 43,3: 188-192)*. The United States (U.S.) has the highest human losses per capita in the industrial world. The total dollar value associated with fire in the U.S. is also very high. For the most recent year available, 1994, the total dollar value either lost to fire, spent to avoid or deal with fire, or donated to avoid or deal with fire in the U.S. was $115-154 billion (*Hall, J. R. The Total Cost of Fire in the United States Through 1994. National Fire Protection Agency, Quincy, MA, 1997)*. This can be compared to annual costs in the U.S. of $66 billion for acquired immune deficiency (AIDS) and $43 billion for coronary heart disease *(Hirschfeld, R. et al. JAMA, Jan 22/29, 1997, Vol 277, No. 4.)*. Thus, fire has a tremendous impact on the way the U.S. uses its resources.

Electronic equipment is a part of the fire problem worldwide. The U.S. arguably has some of the highest standards of fire safety for electronic equipment, including the voluntary compliance with Underwriters Laboratories (UL) test method V-0 for plastics used in these applications. Yet even in the U.S. more than a thousand structure fires a year are reported to U.S. fire departments as originating in electronic equipment rooms or areas. Civilian deaths have been rare, but have occurred in three of the last four years. Direct property damage has averaged roughly 30 million a year in recent years (*Hall, J.R. Special Analysis Package Computer Equipment and Computer Areas, National Fire Protection Agency, Quincy, MA, 1996)*.

In the U.S. during 1989-1993, televisions, radios, VCRs and phonographs accounted for the largest number of civilian fire deaths and placed third in number of civilian fire injures and dollar loss in all appliance or tool fires. There was an average of 35 civilian deaths, 166 civilian injuries and $34.8 million in direct property damage per year resulting from an estimated 2,400 home fires per year starting in this kind of equipment. Short circuits or ground faults were the leading cause of ignition, and electrical wire or cable insulation was the leading form of material first ignited. Appliance housing or casing was the second leading form of material first ignited but was associated with more civilian fire deaths and injuries than any other form of material. *(Slayton, D. M. The U.S. Home Product, 1989-1993 (Appliance and Equipment), National Fire Protection Agency, Quincy, MA, 1996.)*

Other U.S. data for the years 1990-94 shows there were an average of 1,179 structure fires originating per year in electronic equipment rooms or areas, with an annual average of 1 civilian death, 36 civilian injuries and $28.9 million in direct damages. Dwellings, duplexes, and manufactured homes collectively ranked first among properties with these fires. General business offices ranked second. Most of these fires began with electrical distribution system equipment or "other equipment", a large category that includes electronic equipment. Electronic equipment specifically was involved in ignition of 148 fires (annual average of 1990-94) at a direct property damage of $7.29 million. Office machines and televisions, radios, VCRs or phonographs were involved in the ignition of 21 and 15 fires, respectively, at a direct property cost of $0.27 or $0.12 million, respectively. *(Hall, J. R. The Total Cost of Fire in the United States Through 1994. National Fire Protection Agency, Quincy, MA, 1997, and Hall, J.R. Special Analysis Package Computer Equipment and Computer Areas, National Fire Protection Agency, Quincy, MA, 1996.)*

Examples of recent fires in the U.S. involving electronic equipment include:

- May 1995, Florida. Computer monitor from group with a problem history caused a fire. Fire damage was $125,000 to the building and $275,000 to the contents.

- October 1995, Texas. Fire from an undetermined problem in electronic audio equipment ignited the combustible housing of the unit in a home. Resulted in 5 deaths; 3 under the age 6.

- March 1994, Wisconsin. Fire in a computer uninterrupted power supply. Fire deterred by water sprinkler system. Total loss was $125,000.

- January 1992, Michigan. Fire in microfilming room, suspected due to a malfunctioning microfilm printer, caused $1.5 million in property loss.

- October 1990, Arkansas. Computer suffered electrical malfunction and ignited. Fire and smoke spread and caused property damage of $2 million.

- December 1989, Ohio. Electrical short circuit in a copier machine triggered over $2 million dollars damage.

- April 1987, Minnesota. Electrical malfunction of a computer component caused overheating and ignited the internal wiring. Damage to the building and its contents were in excess of $450,000.

- September 1981, California. An electrical short circuit in one CPU circuit board caused a fire that burned part of the board away and melted 16 other memory units. The fire self-extinguished. Damage was $42,000.

- July 1980, Alabama. Overheated components ignited plastic housing within a computer. Damage losses were $200,000.

- October 1967, Massachusetts. An electrical short circuit or malfunction of a cooling fan inside the computer ignited printed circuit boards made of pressed paper and PVC insulation on wires and cables.

These examples demonstrate that even with the high fire safety standards for electrical and electronic equipment in the U.S., fires continue to be a problem both in terms of the number of fires and the losses incurred due to fires. Further, the growth in the total cost of fire in the U.S. has been led not by fire losses but by other cost components. These "other" cost components totaled about $30 billion in 1991. The largest share of this $30 billion was associated with the manufacturing costs of equipment meeting UL or other standards in order to reduce the propensity of products to contribute to fire as a heat or fuel source; this is an especially important in electrical systems equipment and "smart" equipment with its greater use of computer components. This one cost component (manufacturing costs) accounted for $18 billion or 12-15% of the total cost of fires in the U.S. In contrast, costs associated with fire retardants and all product testing associated with design for fire safety were only about $2.5 billion or about 2% of the total fire costs. The growth in the "other" cost component of the total U.S. fire costs clearly indicates a need for product innovations that improve fire safety or lower cost. Therefore, the U.S. has a dual interest in reducing U.S. fire losses and in seeking ways to achieve equivalent fire safety at lower costs. *(Hall, J. R. The Total Cost of Fire in the United States Through 1994. National Fire Protection Agency, Quincy, MA, 1997.)*

II. VARYING FIRE SAFETY STANDARDS FOR ELECTRONIC EQUIPMENT: V-0 V.S. IEC 65

While most plastic used to enclose information technology and video display equipment such as televisions or computer monitors is flame retarded to a standard of V-0 in the U.S., exceptions to this standard occur in Europe and in some developing countries. The International Electrotechnical Commission (IEC) 65 is used as a global guide for designing safety into televisions and IEC 950 is used for information technology equipment. IEC 65, however, is not as stringent a fire safety standard as V-0. V-0 requires materials to be self-extinguishing, non-dripping, and have an after flame lasting less than 5 seconds. Materials that do not meet V-0 flammability standard can pass IEC 65. IEC 65's weaker flammability standards allows European manufacturers, in many cases, to use thick walled cabinets made of HB (non-flame retarded) resins to house their products. Most non-European OEMs still elect to use V-0 resin to

insure their products are as fire safe as possible. Maximum design flexibility in the form of thinner walled cabinets can also retained with V-O. IEC 950 calls for one of several grades of flame retarded resin depending on size and design of the enclosure and here most OEMs elect to use V-0-rated material.

A fire can start in an electrical appliance, in a fault condition, with a sustained power of as little as 15 volt-amps. If HB-resin is used to house this equipment, there is no ability for the plastic to self-extinguish and contain the fire. Models show that a fire with as little as 5 kg of HB-rated plastic can precipitate a flashover fire in an office or residence. In the early 1970s changes to safety requirements for television sets in the U.S., including cabinets of V-0-rated plastics, reduced the number of fire incidents involving televisions from a total of 20,000 including 800 life-threatening fires to just a handful today. In addition, HB resins provide no protection from fires that are external to the enclosure. A burning match can ignite HB plastic in 7-10 seconds. Further, an additional hazard is encountered when thicker walled cabinets made of HB-resin are used; that is, a larger fuel source is present in the electronic enclosure with the accompanying potential for greater heat release in the event of a fire.

III. RESPONSE OF PLASTICS IN A FIRE

The principal thermoplastics used in electronic enclosures can be divided into two classes based on their behavior when burning. The styrenic polymers melt, drip, and depolymerize to form volatile monomers, dimers and trimers when exposed to heat. These polymers require a gas phase flame retardant because of the way they burn. Styrenic polymers include polystyrene, acrylonitrile-butadiene-styrene (ABS), polyphenylene oxide/polystyrene blends (modified PPO) and polycarbonate/ABS blends. The second group of polymers used in electronic enclosures form a char when burned. These polymers decompose when exposed to heat but do not completely volatilize and instead form a carbonaceous char. These polymers can use a flame retardant that acts in the solid or condensed phase. Condensed phase flame retardants are often used with gas phase flame retardants to capitalize on both types of flame retardant mechanisms. Polymers that char when burned include polyphenylene oxide (PPO), polycarbonate (PC), polyethylene terephthalate (PET) and polybutylene terephthalate (PBT). *(Anderson, G. A. and Christy, M. R., "Standards, Bans and Flame Retardants", Presented at Structural Plastics '92, Structural Plastics Division of the Society of the Plastics Industry, Inc., Grand Kempinski Hotel, Dallas, TX, April 5-8, 1992 and Kirk-Othmer Encyclopedia of Chemical Technology, Volume 10, 1980, John Wiley & Sons, New York.)*

IV. CHEMICAL ELEMENTS WITH FLAME RETARDANT PROPERTIES

The chemical elements primarily responsible for flame retardance in engineering thermoplastics are phosphorus, bromine and chlorine. Phosphorus compounds affect char formation, that is, condensed phase reactions. Bromine and chlorine form gaseous species that react in the gas phase with high energy radicals to terminate the combustion reaction, that is, gas phase reactions. Bromine is unique in its efficacy as a flame retarding species and its compatibility with engineering thermoplastic formulations. No other chemical element provides equivalent flammability protection for materials requiring gas phase flame retardancy.

V. FLAME RETARDANTS' MECHANISM OF ACTION

Fire is an exothermic, gas-phase reaction. A plastic will burn as long as the heat supplied is enough to sustain thermal degradation. The combustion reaction is maintained by free radicals and radiant heat. The reaction proceeds at an increasing rate until flashover as long as the available free radicals and heat exceed the energy required for combustion. Conversely, the rate of combustion will decrease to extinction if the available energy is less than required to maintain equilibrium. Flame retardants take advantage of this fact and reduce the heat supplied to the polymer to below the critical level needed to maintain combustion. Flame retardants do this by scavenging the free radicals that propagate combustion, limiting the heat and mass transfer across the solid-gas phase boundary, or by creating a heat sink.

A. Scavenging Radicals In The Gas Phase

Gas phase flame retardants out-compete oxygen for the free radicals generated in the combustion process and thereby terminate the reaction. The flame retardant must form a gaseous component in order to do this. Further, the flame retardant must produce the gaseous component at the same temperature as where the polymer decomposes. Very few elements have the ability to form gaseous compounds. Halogens are some of the few chemical elements with this ability and there are very few halogens that are effective flame retardants. The order of reactivity of the halogens as radical scavengers is I > Br > Cl > F. Iodine is the most effective scavenger, but is very expensive and lacks the thermal and photolytic stability required for most thermoplastic applications. Bromine is the next most effective radical scavenger and is the element most widely used by gas phase flame retardants. Chlorine is considerably less effective than bromine because it only marginally competes with oxygen for hydrogen radicals and the aromatic C-Cl bond is too stable. Fluorine has virtually no effect as a flame retardant due to the stability of C-F and H-F bonds. Because of these limitations, there are very few gas phase flame retardants.

Antimony trioxide is typically added as a synergist to polymers using halogenated flame retardants. Antimony trioxide facilitates release of bromine into the gas phase at the proper time, temperature and concentration thereby enhancing bromine's ability to act as a flame retardant.

In summary, to function as a gas phase flame retardant, the compound must: (1) decompose to form a gaseous radical-scavenging species at the temperature the polymer begins to burn, and (2) successfully compete with oxygen for high energy free radicals to terminate the combustion reaction. The bottom line for plastics is that no other gas phase flame retarding species is as efficient or effective as bromine.

B. Limiting Transfer Across Solid-Gas Phase Boundary

Some polymers form a carbonaceous char when decomposed by heat. This char increases ignition resistance by reducing the amount of available fuel and by providing a heat barrier. Phosphorus is the principal condensed phase flame retardant. Phosphorus's mechanism of action in oxygen-containing polymers is through thermal decomposition to phosphoric acid.

Phosphoric acid extracts water from the burning substrate and thereby increases the amount of char.

Phosphorus compounds are effective condensed phase flame retardants for polymers with char-forming tendencies, such as polycarbonate and polypheneylene oxide. In general, polymers that do not inherently form char as they burn cannot utilize condensed phase chemistry for flame retardancy.

C. Physical Action As A Heat Sink

Physical action flame retardants act as heat sinks. These flame retardants are inorganic compounds that give off nonflammable gases such as water and carbon dioxide in endothermic reactions and cool the burning substrate. Aluminum hydroxide and magnesium hydroxide are two examples of physical action flame retardants.

Polymers begin to burn at temperatures between 150 and 400°C. In order to be effective, the flame retardant must decompose in the temperature range of the decomposing polymer. Aluminum hydroxide begins to decompose at about 230°C that is too low to function as flame retardant in engineering thermoplastics. It also requires very high loadings of between 40-80% by weight that are detrimental to the performance properties of the polymer. Aluminum hydroxide is primarily used in polyesters and latexes, and it is the largest volume flame retardant in the world. Magnesium hydroxide decomposes at about 300°C and it is used primarily in polypropylene and polyethylene terephthalate.

VI. BENEFITS AND DEFICIENCIES OF DIFFERENT FLAME RETARDANT SYSTEMS

The benefits derived from using a brominated aromatic flame retardant with antimony oxide in plastic include the ability to achieve a high standard of fire safety. Brominated flame retardants with antimony trioxide can achieve a V-0 UL-94 flammability rating at a low cost. The low cost can be achieved because of the superior efficiency of the brominated flame retardants. Because the brominated flame retardants are so efficient, they can be used at low concentrations in the polymers and achieve high fire safety standards. The ability to use the brominated flame retardants at low concentrations aids in maintaining the physical properties of the polymer and allows good performance characteristics. The low load level of brominated flame retardant in the polymer, compared to other flame retardants, is also good environmental stewardship - less total additive is needed which reduces the total impact on the environment from manufacture to transport to disposal. Brominated flame retardants provide the most attractive combinations of mechanical properties, flame retardant efficiency, and cost in the resins in which they are used.

Deficiencies of non-halogen flame retardants or brominated aromatic flame retardants with an alternate synergist to antimony trioxide include an inability to achieve a V-0 UL-94 flammability rating in some plastics (i.e., HIPS). The non-halogen flame retardants are higher in cost than brominated flame retardants. This has direct implications for equipment manufacturers and also for society. An increase in the cost of flame retardancy will increase the already high total fire

cost. The non-halogen flame retardants require higher load levels than the brominated flame retardants to achieve the same level of fire protection. In addition to directly impacting cost, these higher load levels adversely affect polymer performance and physical properties. Further, the higher load levels required to achieve a lesser standard of fire safety places an increased burden on the environment. More plastic additive must be manufactured, transported and ultimately disposed of than if brominated flame retardants had been used.

VII. SUMMARY

Bromine is unique in its efficacy as a flame retarding species and in its ability to flame retard engineering thermoplastic formulations. No other chemical element can match bromine's performance in these applications. Brominated flame retardants provide the most attractive combinations of mechanical properties, flame retardant efficiency, and cost in the resins in which they are used. Brominated flame retardants allow the use of plastic materials in applications that would otherwise present a fire risk. Brominated flame retardants save human lives and property.

In many end use applications, antimony trioxide is used as a synergist along with the brominated flame retardant. The antimony trioxide enhances the flame retardant action of the brominated flame retardant. This allows less flame retardant to be used in the resin, which helps to maintain the resin's physical properties, reduces the amount of additive in the resin and keeps down cost. Without antimony trioxide, approximately three times the amount of brominated aromatic flame retardant was required to achieve a V-0 UL-94 flammability rating in HIPS. The higher loading adversely affected the physical properties of the polymer. No alternate synergist has been identified in our extensive research program *(Nalepa, C. "Studies of Alternative Synergists for Bromoaromatics in FR-HIPS Systems", Sixth Annual BCC Conference on Flame Retardancy, Stamford, CN, May 23-25, 1995).*

If a brominated flame retardant must be substituted in a particular resin, another bromine chemical is the most feasible, and sometimes only, substitute. This is because the way a plastic burns dictates the kind of flame retardant used in that plastic. An example of this kind of substitution is using TBBPA instead of octabromodiphenyl oxide in ABS. The next most feasible substitution for a brominated flame retardant is to change to totally different polymers and flame retardants. An example is substituting phosphorus-containing ABS/PS alloys for brominated diphenyl oxide-containing high impact polystyrene or ABS. However, these substitutions are likely to be costly and time consuming. Replacing a brominated flame retardant in the same polymer type by chlorine, phosphorus or inorganic flame retardants is not appropriate. This is because differences in physical properties, processability, and cost are too great. In some instances, this kind of substitution is impossible. Antimony trioxide is essential to brominated flame retardants and brominated flame retardants are essential in many applications to provide the needed fire safety.

APPENDIX II

Hardy M, Schroeder R, Biesemeier J, and Manor O. 2002. Prenatal Oral (Gavage) Developmental Toxicity Study of Decabromodiphenyl Oxide in Rats. International Journal of Toxicology, 21:83-91.

NOT AVAILABLE IN ELECTRONIC FORMAT.

APPENDIX III

Abstract. Final Report: Toxicology and carcinogenesis studies of decabromodiphenyl oxide in F344/N rats and B6C3F1 mice (Feed Studies). National Toxicology Program. Technical Report Series. No. 309. 1986. U.S. Department of Health and Human Services.

DECABROMODIPHENYL OXIDE

CAS No. 1163-19-5

$C_{l2}Br_{10}O$ Molecular weight 960

Synonyms: Decabromodiphenyl ether; Bis(pentabromophenyl)ether; DBDPO

ABSTRACT

Toxicology and carcinogenesis studies of decabromodiphenyl oxide, a flame retardant for plastics and other materials, were conducted by exposing groups of 50 male and 50 female F344/N rats and B6C3Fl mice at 0, 25,000, and 50,000 ppm in the diet for 103 weeks. These concentrations were selected because no toxicity was observed at any dose in the 14-day or 13-week studies and 50,000 ppm chemical in the diet is considered to be the highest dose to which rats and mice can be exposed for extended periods of time without reducing the nutritional value of the diet. No compound-related gross or microscopic pathologic effects were observed in the 14-day or 13-week studies.

Body weights of dosed male and female rats and mice in the 2-year studies were comparable to those of the controls. Decreased survival of low dose male rats was not believed to be compound related. No other effects on survival were observed in the 2-year studies. Loss of control male mice (presumably due to fighting) was significant during the first part of the study.

In the 2-year studies, nonneoplastic lesions were observed at increased incidences in rats and mice of each sex. Thrombosis and degeneration of the liver, fibrosis of the spleen, and lymphoid hyperplasia were observed in high dose male rats. Degeneration of the eye was observed in low dose female rats. Nonneoplastic lesions observed in dosed mice were granulomas in the liver of low dose males and hypertrophy in the liver of low dose and high dose males. Follicular cell hyperplasia was observed in thyroid glands of dosed male mice (control, 2/50; low dose, 10/50; high dose, 19/50).

The incidences of neoplastic nodules in the liver of low and high dose male rats (1/50; 7/50; 15/49) and high dose female rats (1/50; 3/49; 9/50) were significantly greater than those in the controls. Mono- nuclear cell leukemia occurred in dosed male rats with a positive trend (30/50; 33/50; 35/50); this marginal increase was not considered biologically significant. Acinar cell adenomas were observed in the pancreas of four high dose male rats, and a sarcoma was observed in the spleen of one low dose and one high dose male rat. Hepatocellular adenomas or carcinomas (combined) occurred at marginally increased incidences in dosed male mice (8/50; 22/50; 18/50). The incidences of thyroid gland follicular cell adenomas or carcinomas (combined) were increased in dosed male mice (0/50; 4/50; 3/50).

A study of decabromodiphenyl oxide absorption from the gastrointestinal tract indicated that absorption was minimal, possibly less than 1 %, at the doses administered in the 2-

year studies. Additional chemical analysis indicated that the decabromodiphenyl oxide used in these studies contained several less brominated diphenyl oxides. Therefore, since absorption and toxicity of minor impurities are unknown, effects observed in these studies must be attributed to the approximately 95% pure preparation used rather than to pure decabromodiphenyl oxide.

Decabromodiphenyl oxide was not mutagenic in strains TA1535, TA1537, TA9S, or TAI00 of *Salmonella typhimurium* in the presence or absence of Aroclor 1254-induced Sprague-Dawley male rat or Syrian male hamster liver S9 when tested according to the preincubation protocol. Decabromodiphenyl oxide was not mutagenic in the mouse lymphoma L5178Y/TK $^{+/-}$ assay in the presence or absence of Aroclor 1254-induced F344 male rat liver S9. Decabromodiphenyl oxide did not induce sister-chromatid exchanges or chromosomal aberrations in Chinese hamster ovary cells in vitro in the presence or absence of S9 prepared from livers of Aroclor 1254-induced male Sprague-Dawley rats.

An audit of experimental data was conducted for these 2-year studies on decabromodiphenyl oxide.

No data discrepancies were found that influenced the final interpretations.

Under the conditions of these 2-year feed studies of decabromodiphenyl oxide, there was *some evidence of carcinogenicity* for male and female F344/N rats as shown by increased incidences of neoplastic nodules of the liver in low dose (25,000 ppm) males and high dose (50,000 ppm) groups of each sex. There was *equivocal evidence of carcinogenicity* for male B6C3F1 mice as shown by increased incidences of hepatocellular adenomas or carcinomas (combined) in the low dose group and of thyroid gland follicular cell adenomas or carcinomas (combined) in both dosed groups. There was *no evidence of carcinogenicity* for female B6C3F1 mice receiving 25,000 or 50,000 ppm in the diet. Several non- neoplastic lesions were observed at increased incidences, the most notable being thyroid gland follicular cell hyperplasia in male mice.

APPENDIX IV

Viberg H, Fredriksson A, Jakobsson E, Orn U and Eriksson P. Brominated Flame Retardant: Uptake, retention and developmental neurotoxic effects of decabromodiphenyl ether (PBDE 209) in the neonatal mouse. 2001. Proceedings: The Second International Workshop on Brominated Flame Retardants. BFR2001. Stockholm, SE. pp 279-282.

Brominated Flame Retardant: Uptake, retention and developmental neurotoxic effects of decabromodiphenyl ether (PBDE 209) in the neonatal mouse

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Summary

This study shows that PBDE 209 can be taken up and retained in the neonatal mouse brain. In addition, neonatal exposure to PBDE 209 induces behavioural disturbances in adult mice, disturbances that worsen, with age.

Introduction

Polybrominated diphenyl ethers (PBDEs) are group of chemical substances used as additive flame-retardants. Product~ that contain flame-retardants are electrical appliances such as computers and television sets, textiles, building materials and other objects. PBDEs are not fixed in the polymer products and can thus leak into the environment (1, 2). Studies have shown that PBDEs are present in the global environment (3) and that levels of PBDEs are increasing in the Swedish environment (4, 5, 6). A recent report has shown the presence of PBDEs in Swedish mother's milk and also that the PBDEs have increased exponentially since 1972 (7). Samples of human blood have also been shown to contain PBDEs (8), including workers in the electrical dismantling industry who show levels of PBDEs in their blood, including the deca-brominated congener PBDE 209 (9). This indicates that humans are exposed to PBDEs both as infants and as adults. In recent studies we have shown that neonatal exposure to flame-retardants, during the period of rapid brain growth, known as the "brain growth spurt" ("BGS"), can induce persistent dysfunction in adult mice, manifested as deranged spontaneous behaviour, an effect that also worsens with age (10) and that these effects are inducible during a restricted period of neonatal life (11). In view of the increasing amounts of PBDEs in the environment and in mother's milk and the fact that other PBDE congeners have been shown to induce persistent dysfunctions in mice, this study was undertaken to study uptake and retention of PBDE 209 in neonatal mice, as well as possible behavioural effects of PBDE 209 when given during the rapid development of the brain.

Materials and methods

Both [^{14}C]PBDE 209 and PBDE 209 were synthesized at the Wallenberg Laboratory, University of Stockholm, Sweden, and were administered as one single oral dose to neonatal NMRI mice on postnatal day 3, 10 or 19.

In order to study uptake and retention two litters in each age categories were given 1.5 [^{14}C]PBDE MEq/kg body weight (40.5 μCi/kg body weight). Each of the two litters from the three different age categories was sacrificed, by decapitation, 24 h or 7 days, respectively, after administration. The skull was opened and the brain sectioned just behind the cerebellum. The brains were solubilized and the radioactivity was counted in a scintillation analyser .

In the behavioural study 3-days-old and 19 days-old mice were given 2.22 or 20.1 mg PBDE 209/kg b.wt. (2.3 or 21 μmol/kg b.wt.) and 10 days-old mice were given 1.34, 13.4 or 20.1 mg PBDE 209/kg b.wt. (1.4, 14, or 21 μmol/kg b.wt.). Mice serving as controls received 10 ml/kg b. wt. of the 20% fat emulsion vehicle in the same manner. Each group contained 3-5 litters. The spontaneous behaviour test was conducted at 2, 4 and 6 months of age. The test measures *locomotion:* horizontal movements, *rearing:* vertical movements, and *total activity:* all types of vibrations within the test cage.

Results and discussion

The data from the uptake and retention study showed that [^{14}C]PBDE can be taken up and be distributed in the neonatal mouse, but that there is a difference in the amount of radioactivity found in the mouse brain in the different age categories. Mice exposed to [^{14}C]PBDE on postnatal day 3 or 10 displayed around 4% of the administered amount of radioactivity in the brain 24 h after administration, whereas mice exposed to [^{14}C]PBDE on postnatal day 19 had only about 0.6% of the administered amount of radioactivity in the brain 24 h after administration. 7 days after administration the amount of radioactivity in the brain had increased almost two-fold in mice exposed to [^{14}C]PBDE on postnatal day 3 or 10, whereas mice exposed on postnatal day 19 showed the same amount of radioactivity as they did 24 h after administration. This shows that PBDE 209 can be taken up and find its way to the brain during the critical "BGS", but the retention pattern differs from other similar compounds during this period, for example PBDE 99 (11) and some PCBs (12).

The spontaneous motor behaviour data states a disruption of habituation in adult mice exposed to PBDE 209 on postnatal day 3, but this disruption in habituation can not be seen in mice exposed to PBDE 209i on postnatal day 10 or 19. Habituation, defined here as a decrease in *locomotion, rearing* and *total activity* variables in response to the diminishing novelty of the test chamber over the 60 min test period, was demonstrated in the control groups of the three age categories as well as in the animals exposed to PBDE 209 on postnatal day 10 or 19. The animals exposed to the highest dose of PBDE 209, on postnatal day 3, showed a non-habituating behaviour profile at 2, 4 and 6 months of age. At 6 months of age mice exposed to the lower dose of PBDE 209, on postnatal day 3, showed this non-habituating behavioural profile. Certain PCBs (12) as well as PBDE 99 (11, 12) have been shown to induce this type of behavioural profile when administered on postnatal day 3, but this response is always accompanied by a response in animals exposed to the toxic compound on postnatal day 10. In this study the explanation can be that the amount of substance reaching the brain is not enough to induce disturbances but as time goes by the amount is increasing, which is seen in the retention study. Another possible explanation is that PBDE 209 is metabolised to a metabolite that reaches the brain just in time for the critical window of "BGS" and induces the persistent effect. In addition, the neurotoxic effects of PBDE 209 were more pronounced in the older the animals, which indicate the advance of a brain dysfunction process induced at the time of rapid brain development in the neonatal mouse.

The present investigation shows that [^{14}C]PBDE can be taken up in the neonatal mouse and that the uptake is more efficient in younger animals. Radioactivity is found in the brain and increases during the first week after administration. This study also shows that neonatal exposure to PBDE 209 can induce neurotoxic effects, manifested as aberrations in spontaneous motor behaviour in the adult animal, effects that also worsens with age.

Acknowledgements

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References

[1]Hutzinger, 0., Sundström, G. and Safe, S., Chemosphere, 1976,1, 3-10.
[2]Hutzinger, 0. and Thoma, H., Chemosphere, 1987, 16, 1877-1880.

[3]de Boer, J., Wester, P. G., Klamer, H. J., Lewis, W. E. and Boon, J. P., Nature, 1998, 394, 28-9.
[4]Andersson, 0. and Blomkvist, G., Chemosphere, 1981, 10, 1051-1060.
[5]Nylund, K., Asplund, L., Jansson, B. and Jonsson, P., Chemosphere, 1992, 24, 1721-1730.
[6]Sellström, U., Jansson, B., Kierkegaard, A., de Wit, C., Odsjö, T. and 01sson, M., Chemosphere 1993, 26, 1703-1718.
[7]Meironyté, D. and Norén, K., Journal of Toxicology and Environmental Health Part A, 1999, 58, 329-341.
[8]Klasson-Wehler, E., Hovander, L. and Bergman, Å., Organohalogen Compounds, 1997, 33: 420-421.
[9]Sjödin, A., Hagmar, L., Klasson-Wehler, E., Kronholm-Diab, K., Jakobsson, E. and Bergman, Å., Environmental Health Perspectives, 1999, 107, 643-648.
[10]Eriksson, P, Jakobsson, E. and Fredriksson, A. (2001a). "Neonatal exposure to polybrominated diphenyl ethers causes behavioural derangements in mouse that deteriorate with age." Submitted.
[11]Eriksson, P., Viberg, H., Jakobsson, E., Örn, U. and Fredriksson, A. (2001b). "A brominated flame retardant, 2,2',4,4',5-pentabromodipheny1 ether: Uptake, retention and induction of neurobehavioura1 derangement in mice, during a critical phase of neonatal brain development." Submitted.
[12]Eriksson, P., Swedish Environmental Protection Agency, 1998, Report 4897, 56pp.

APPENDIX V

NAS. 2000. Toxicological Risks of Selected Flame-Retardant Chemicals. Excerpt from Chapter 5: Decabromodiphenyl Oxide. National Academy Press. Washington, D.C. http://www.nap.edu. pp 88-93.

The terms RfC and RfD used in the NAS report refer to reference concentration and reference dose, respectively. The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark concentration, with uncertainty factors generally applied to reflect limitations of the data used, and is generally used in EPA's noncancer health assessments. Similarly, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used, and is generally used in EPA's noncancer health assessments.

BEGINNING OF NAS QUOTATION, page 84 of that document.

"**QUANTITATIVE TOXICITY ASSESSMENT**

Noncancer

Dermal Assessment

Available data suggest that DBDPO is not irritating to the skin and is not a dermal sensitizer. Systemic effects of short- or long-term dermal exposures to DBDPO have not been adequately studied. There were no treatment-related changes in body weight gain or survival in rabbits following a single application of ~2,000 mg/kg (IRDC 1974; Great Lakes 1977). There were no treatment-related changes in body weight or gross pathological effects in rabbits treated with 40 mg/kg-d DBDPO (rubbed into external ear canal skin) for 4 wk in an acnegenesis assay (Pharmakon 1981). There is insufficient information on toxicity of DBDPO from sub chronic or chronic dermal exposures to estimate the dermal RfD.

Inhalation RfC

The subcommittee identified no inhalation studies of sufficient duration (i.e., subchronic or chronic) for deriving an RfC, since the available data are limited to an acute inhalation study (IRDC 1974; Great Lakes 1984) and an acute intra- tracheal study (Dow 1976). Therefore, the subcommittee did not derive and inhalation RfC for DBDPO.

Oral RfD

EPA's reference dose (RfD) of 1 x 10-2 mg/kg-d for DBDPO is based on the 1 mg/kg-d NOAEL for histopathology and other toxicity endpoints in rats exposed via diet for 2 yr (Kociba et al. 1975). Doses higher than 1 mg/kg-d were not tested in this study, precluding identification of a LOAEL.

The reason the NTP (1986) 2-yr toxicology/carcinogenesis bioassay for DBDPO was not considered in the current Risk summary in IRIS (EPA 1999) is because it was not available at the time of the Risk derivation (1984-1985). The subcommittee believes that it was appropriate to reevaluate the RfD considering the NTP data, because of the higher compound purity (~ 99% versus 77.4 %), reflecting the actual chemical composition applied as a flame retardant (Marcia Hardy, Albermarle Corporation, Pers. Commun., February 9, 1999); the larger number of animals (50 versus 25 rats/sex/dose); the higher dose levels; and the use of a second species in the NTP (1986) bioassay in comparison to the Kociba et al. *(1975)* study.

The subcommittee derived an oral RfD for DBDPO by using the chronic NOAEL of 1,120 mg/kg-d, based on liver thrombosis and degeneration observed in rats at the next higher dose (NTP 1986), and a composite uncertainty factor of 300, resulting in an RfD of 4 mg/kg-d (RfD = NOEL ÷ 300) (see Table 5-3). The composite uncertainty factor is composed of 3 uncertainty factors: 10 for interspecies extrapolation, 10 for intraspecies variability, and 3 for database uncertainties (10A x 10H x 3D = 300). The RfD is based on a well-designed chronic toxicity study of DBDPO in two species. Data on chronic, developmental, and reproductive toxicity are available from other studies in rats. However, limitations in these studies (particularly compound purity (77.4%), lack of a second species, and use of low dose levels in that chronic study; lack of longer than one-generation testing in the reproductive study) indicate that there is some uncertainty in the DBDPO database. Based on these considerations, an uncertainty factor of 3, instead of 10, for database insufficiency was used.

TABLE 5-3 Oral Reference Dose for DBDPO

Critical effect	Species	Effect level (mg/kg-d)	Uncertainty factors	RfD (mg/kg-d)	Reference
Liver thrombosis and degeneration observed at the LOAEL of 2,240 mg/kg-d	Male and female rats	NOAEL: 1,120	UF_A:10 UF_B:10 UF_D: 3 Total: 300	4.0	NTP (1986)

NOAEL, no-observed-adverse-effect level; RfDl, reference dose; UF_A extrapolation from animals to humans; UF_B, extrapolation for intraspecies variation; UF_D, inadequate or deficient toxicity database.

Confidence in the key study (NTP 1986) is high because it was well conducted and because it used two species, a sufficient number of animals, a dose range adequate to identify a NOAEL and LOAEL for a known sensitive effect, and a high-purity test formulation. However, confidence in the database is low because of limitations in the available developmental, reproductive, and supporting chronic toxicity studies of DBDPO, as well as use of low-purity compound, lack of testing in species other than the rat, lack

of multigenerationnal reproductive tests, and a low range of chronic dose levels. Therefore, confidence in the provisional RfD is medium to low.

Cancer

Oral

There are no epidemiological data available on the carcinogenicity of DBDPO. However, the carcinogenicity of DBDPO has been assessed in two chronic bioassays (Kociba et al. 1975; NTP 1986). No evidence for carcinogenicity was observed in male or female Sprague-Dawley rats fed dose levels of 0, 0.01,0.1, or 1 mg/kg-d DPDPO in their diet for 2 yr (Kociba et al. 1975). However, this study has several limitations including use of an inadequate number of animals (25/sex/dose), dosing with impure DBDPO (77.4%DBDPO, 1.8% NBDPO, 0.8% OBDPO), and utilization of dose levels that were probably below the maximum tolerated dose (Mill) (see NTP 1986). "Some evidence of carcinogenicity" was reported by NTP (1986) for male and female rats fed DBDPO in their diet at dose levels of up to 50,000 ppm for 2 yr. NTP (1986) reports that there was "equivocal evidence of compound-related carcinogenicity" for mice exposed to DBDPO in their diet for 2 yr at dose levels of 5,000 or 50,000 ppm. The EPA weight-of -evidence cancer classification for DBDPO in accordance with the currently used 1986 Guidelines for Carcinogenic Risk Assessment EPA 1986) is Group C, possible human carcinogen (EPA 1999). This is based n no human data and limited evidence of carcinogenicity in animals (NTP 1986), specifically, statistically significant increases in the incidences of "neoplastic nodules" of the liver in male and female rats and hepatocellular adenomas and carcinomas combined in male mice. Under the newer Proposed Guidelines for Carcinogenic Risk Assessment (EPA 1996), which take into account genotoxicity data, the weight-of-evidence for the carcinogenic potential of DBDPO in humans would be termed "suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential." The subcommittee has concluded that the weight-of-evidence, based on that currently available, suggests that DBDPO is a possible carcinogen in rats.

Because there is uncertainty concerning the carcinogenicity of this compound, the subcommittee concluded that derivation of a cancer risk estimate was warranted and should be used for assessing the potential carcinogenic risk associated with this compound when used as a flame retardant in residential furniture. The subcommittee believes that derivation of a cancer potency factor ($0.1/LED_{10}$) as opposed to a hazard index is justified in this case because a NOAEL was not detected for liver nodules in rats (NTP 1986).

The subcommittee has not concluded that DBDPO is a carcinogen in humans but believes that a conservative approach is justified at this time in order to be protective of human health. The subcommittee acknowledges that the increased incidence of "neoplastic nodules" of the liver in male and female rats and male mice does not constitute sufficient evidence for the carcinogenicity of DBDPO and is aware that there is controversy over the

significance of these lesions in determining cancer risk (Maronpot et al. 1986). However, the finding of margina1 increases in follicular cell tumor incidence in conjunction with an increased frequency of hyperplasia in dosed animals as compared with controls is suggestive of a carcinogenic response and adds weight for the use of a conservative approach for evaluating the carcinogenicity of DBDPO. This approach is further justified by other scientists who have concluded that hyperplasia is a stage in the thyroid follicular cell carcinogenic process (Hill et al. 1989, 1998; EPA 1998; Hard 1998).

The cancer potency factor ($0.1/LED_{10}$) was derived for DBDPO using both the censored (for early deaths) and uncensored neoplastic nodule incidence data for male rats from NTP (1986). Use of the censored data produced a $0.1/LED_{10}$ of 9×10^{-4} /mg/kg-d as compared with 7×10^{-4} /mg/kg-d when the uncensored data were used in the derivation (see Table 5-4). Use of censored data represents a crude attempt to adjust for differential mortality among male rats (low- and high-dose groups). Survival of the low-dose male rats was reduced as compared to controls and to males in the high-dose group; these differences were statistically significant by the end of the study. The number of neoplastic nodules produced might have been greater in the animals of this dose group if a greater number had survived until the 2-yr termination point.

Data for female rats were not used to calculate a cancer slope factor because LED_{10} values derived from female liver neoplastic nodule incidence data were roughly two- fold greater (less protective) than those for male rats. LED_{10}s were also derived using liver adenoma and carcinoma incidence (combined) in male mice, but model fit was "poor" when either censored or uncensored data for these tumor types were used. This is primarily because of the higher incidence of tumors in the low- versus high-dose groups. These values were higher, and thus less protective, than LED_{10} values derived from female rat liver nodule data.

TABLE 5-4 Cancer Assessment for DBDPO Based on Hepatic Neoplastic Nodules Reported for Male Rats in NTP (1986) Study (oral exposure)

	Daily dose level (mg/kg-d)			$0.1/LED_{10}$ derivation		
Nodules	0	1,120	2,240	LED_{10}b (mg/kg-d)	LED_{10} (mg/kg-d), adjusted[c]	$0.1/LED_{10}$d (mg/kg-d)
Uncensored	1/50	7/50	15/49	516	137	7 x 10-4
Censored[a]	1/45	7/38	15/45	435	115	9 x 10-4

LED, lowest effective dose

[a]Animals in all groups that died prior to the occurrence of the first hepatic neoplastic nodule in either treated group (wk 87) were removed from the denominator on the assumption that these animals had insufficient opportunity to develop the tumor .

[b]Calculated using a multistage model fit to the dose-response data and based on extra risk.

[c]Dose adjusted for human equivalency by taking the ratio of human body weight to rat body weight over human to rat body weight to the 0.75 power (EP A 1992). Defaults used: human body weight, 70 kg; rat body eight, 0.35 kg calculated as 0.1/LEDlo, as per the EPA (1996, 1999) proposed cancer guidelines.

The subcommittee has low-to-moderate confidence that the NTP (1986) bioassay results accurately characterize the carcinogenic potential of DBDPO. While the assay was not conducted at the MTD, the dose levels administered were the highest recommended for use in NTP studies. Mortality was significantly elevated among male rats in the low-dose group and in male control mice and is an issue when judging the quality of the study.

The subcommittee places moderate confidence in the derived LED_{10}, and subsequently the $0.1/LED_{10}$. The NOAEL for liver neoplastic nodules in the NTP (1986) bioassay was not determined, which raises the concern that these effects could occur at lower dose levels than the LED_{10}.

EXPOSURE ASSESSMENT AND RISK CHARACTERIZATION

Noncancer

Dermal Exposure

The assessment of noncancer risk for the dermal route of exposure is based on the dermal exposure scenario described in Chapter 3. This exposure scenario assumes that an adult spends 1/4th of his or her time sitting on furniture upholstery back coated with DBDPO and also assumes that 1/4th of the upper torso is in contact with the upholstery and clothing presents no barrier. Exposure to other chemicals present in the back coating were not included in this assessment.

First Iteration

As a first estimate of exposure, it was assumed that the skin and clothing of the person sitting on the couch, and the fabric of the couch, would present no barrier to movement of DBDPO. In addition, it was assumed that there would be sufficient water present (e.g., from sweat) to allow dissolution of the DBDPO in the water, and transfer to the skin and into the body of the sitting individual. The only limiting factor on the transfer rate using these assumptions results from the limited dissolution rate from the fabric-all the DBDPO that dissolves is assumed to be absorbed immediately by the sitting individual.

Dermal exposure was estimated using Equation 1 in Chapter 3. For this calculation, the subcommittee estimated an upholstery application rate (S_a) for DBDPO of 5 mg/m2. The extraction rate (μ_w) by water for DBDPO was estimated to be 0.025/d based on extraction data for hexabromocyclododecane in polyester fiber (McIntyre et al. 1995). This release rate was calculated as 0.04/d at 28°C from the fiber, with a correction from fiber to film of a factor of 0.63 ($2^{nd}/2\pi R$ for film thickness *d,* fiber radius *R).*

Using these values specific to DBDPO, the estimated dermal absorbed dose rate was determined to be 0.98 mg/kg-d. Although lack of sufficient data precludes deriving a dermal RfD, the oral RfD (4 mg/kg-d)

was used to calculate a hazard index. The hazard index of 0.25, derived by dividing the dermal absorbed dose rate of 0.98 mg/kg-d by the oral RfD of 4 mg/kg-d, indicates that DBDPO does not pose a noncancer risk by the dermal absorption route when used as an upholstery fabric flame retardant. Nevertheless, an alternative iteration of the exposure assessment was performed because of concerns about potential cancer risk (see below).

Alternative Iteration

For the alternative iteration of the dermal assessment, the same exposure assumptions were made as in the first iteration, except that the assumption of immediate absorption of all the DBDPO that dissolves was modified. Instead, an estimate of the rate at which DBDPO can penetrate the skin was determined, assuming that DBDPO dissolves up to its solubility limit in water. This rate of penetration was then factored into the exposure assessment.

The rate of penetration of a chemical through skin may be estimated using the skin permeability coefficient *(Kp,* with dimensions of velocity)-the total mass penetration rate is the product of water concentration, permeability coefficient, and skin area. This coefficient has not been measured for DBDPO. However, it was estimated from the octanol-water partition coefficient (Kow, dimensionless) and molecular weight (m, mass/unit amount of substance) using a correlation (Potts and Guy 1992) based on Equation 2 in Chapter 3. The value estimated from this correlation is 3.21×10^{-4} cm/d for DBDPO.

Using Equation 5 in Chapter 3 in conjunction with the permeability coefficient (3.21×10^{-4} cm/d) and the water solubility specific to DBDPO (0.1 ug/L), the dose rate, using this alternative iteration, was estimated to be 1.33×10^{-9} mg/kg-d. The hazard index was then recalculated by dividing the dermal absorbed dose rate (1.33×10^{-9} mg/kg-d) by the oral RfD (4 mg/kg-d), as the best estimate for internal dose from dermal exposure. The hazard index of 3.34×10^{-10}, again demonstrates that DBDPO, used as an upholstery fabric flame retardant, is not likely to pose a noncancer risk from dermal exposure.

Inhalation Exposure

Particles

Inhalation exposure estimates for DBDPO were calculated using the exposure scenario described in Chapter 3. This scenario assumes that a person spending a quarter of his or her life in a room with low air-change rates (0.25/hr) and with a relatively large amount of fabric upholstery (30 m^2 in a 30 m^3 room), with the DBDPO treatment gradually being worn away over 25% of its surface to 50% of its initial quantity over the 15 yr lifetime of the fabric. A small fraction, 1 %, of the worn-off DBDPO is released into the indoor air as small particles that may be inhaled.

Particle exposure was estimated using Equations 4 through 6 in Chapter 3. The release rate (μ_w) for DBDPO from upholstery, 2.3×10^{-7} /d (Equation 5), was used in conjunction with the upholstery application rate (S_a

for DBDPO of 5 mg/cm^2 to calculate a room airborne particulate concentration of 1.9 mg/m^3 (Equation 4). Factoring in the fraction of a day a person spends in the room containing upholstery (0.25), the time-average exposure concentration was determined to be 0.48 mg/m^3 (Equation 6).

For the purpose of estimating a hazard index for the inhalation of DBDPO and in the absence of relevant inhalation exposure data, the subcommittee chose to estimate the inhalation RfC from the oral RfD. The subcommittee, however, recognizes that this is not an ideal approach and also recognizes that the estimated RfC might be considerably different than the actual reference concentration (if inhalation data were available). Extrapolating from one route of exposure (oral) to another (inhalation) requires specific knowledge about the uptake kinetics into the body by each exposure route, including potential binding to cellular sites. The subcommittee believes that its extrapolation of the oral RfD to the inhalation RfC is highly conservative; it assumes that all of the inhaled compound is deposited in the respiratory tract and is completely absorbed into the blood. The NRC Committee on Toxicology (NRC 1985) has used this approach when inhalation exposure data were insufficient to derive inhalation exposure levels. The subcommittee believes that such an approach is justified for conservatively estimating the toxicological risk from exposure to DBDPO. The provisional RfC should be used as an interim or provisional level until relevant data become available for the derivation of an inhalation RfC for calculating the hazard index.

Based on this, a provisional RfC of 14 mg/m^3 was derived from the oral RfD of 4.0 mg/kg-d and Equation 7 in Chapter 3. A hazard index of 3.4 x 10^{-5} was estimated by dividing the exposure concentration (0.48 ug/m^3) by the provisional inhalation RfC (14 mg/m^3). This indicates that under the worst case exposure assumptions, DBDPO, used as an upholstered flame retardant, does not pose any noncancer risk via inhalation of DBDPO in the particulate phase.

Vapors

In addition to the possibility of release of DBDPO in particles worn from upholstery fabric, the subcommittee considered the possibility of its release by evaporation. The approach is described in Chapter 3 and uses a scenario similar to that previously described for exposure to DBDPO in the particulate phase.

Using Equations 8 through 10 in conjunction with the saturation vapor concentration (Cv) (1.8 x 10^{-3} mg/m^3) and the application density (S_a (5 mg/cm^2) for DBDPO, the equilibrium room-air concentration of DBDPO was estimated to be 1.52 x 10^{-3} mg/m3. From Equation 11, it was determined that this vapor concentration could be maintained for approximately 390 yr. Factoring in the fraction of a day a person spends in the room containing upholstered fabric (0.25), the time-average exposure concentration was determined to be 3.8 x 10^{-4} mg/m3.

Division of this exposure concentration (3.8 x 10^{-4} mg/m3) by the provisiona1 inhalation RfC (14 mg/m3) results in a hazard index of 2.71 x 10^{-}

^{5}, indicating that under the worst case scenario, exposure to DBDPO, used as an upholstery flame retardant, is not likely to pose a noncancer risk via the inhalation route, when exposure occurs in the vapor phase.

Oral Exposure

The assessment of the noncancer risk for the oral exposure route is based on the scenario described in Chapter 3. This scenario assumes a child is exposed to DBDPO through sucking on 50 cm2 of fabric back coated with DBDPO daily for 2 yr, 1 hr/d. The dose rate to the child was calculated using Equation 15 in Chapter 3. DBDPO specific parameters used in this calculation included an upholstery application rate (Sa) of 5 mg/m2 and an extraction rate (μ_w) by saliva of 0.025/d. This extraction rate was based on data for hexabromocyclododecane in polyester fiber (McIntyre et al. 1995) and was calculated as 0.04/d at 28°C from the fiber, with a correction from fiber to film of 0.63 *(2d/2πR* for film thickness *d,* fiber radius *R).*

Using these values, the average oral dose rate was estimated to be 2.6 x 10^{-2} mg/kg-d, compared with an oral RfD of 4 mg/kg-d, giving a hazard index of 6.5 x 10^{-3}. It was concluded that DBDPO used as an upholstery fabric flame retardant does not pose any noncancer risk via the oral route.

Cancer

Dermal Exposure

Human cancer risk for dermal exposure to DBDPO was calculated by multiplying the lifetime oral cancer potency factor for DBDPO by the lifetime average dermal dose rate. Using the lifetime average dermal dose rate of 1.33 x 10^{-9} mg/kg-d, obtained in the alternative dermal exposure iteration (see the Noncancer Dermal Exposure section), and multiplying this by the cancer potency estimate of 9 x 10^{-4} kg-d/mg, a lifetime risk estimate of 1.20 x 10^{-12} is obtained. This estimate is small enough that the cancer risk through dermal contact with DBDPO used as an upholstery-fabric flame retardant, can be considered negligible.

Inhalation Exposure

For DBDPO, no inhalation cancer unit risk is available. However, an inhalation cancer unit risk of 2.57 x 10^{-7} per ug/m3 was estimated from the oral carcinogenic potency using Equation 14 in Chapter 3.

Particles

The average room-air concentration and average exposure concentration to DBDPO were obtained as described in the Noncancer section. Using the estimated unit risk (2.57 x 10^{-7} per ug/m3), the lifetime risk estimate from exposure to DBDPO as particles is 1.2 x 10^{-7}. From this estimate, DBDPO, used as an upholstered flame retardant, poses a negligible cancer risk via inhalation in the particulate phase.

Vapors

The equilibrium concentration of vapor-phase DBDPO in room. air was estimated as described in the Noncancer Inhalation Exposure Section. The long term time-average vapor exposure concentration was estimated from the equilibrium vapor concentration in room air using Equation 13 in Chapter 3.

Using the estimated unit risk of 2.57 x 10-3 per ~g/m3, the lifetime risk estimate for exposure to DBDPO in the vapor phase is 9.74 x 10-8. This estimate indicates that DBDPO, used as a flame retardant, poses a negligible cancer risk via inhalation in the vapor phase.

Oral Exposure

For DBDPO, the lifetime average dose rate estimate by the oral route was 7.4×10^{-4} mg/kg-d. This dose rate estimate is multiplied by the cancer unit risk of 9.0×10^{-4} mg/kg-d, giving a lifetime cancer risk estimate of 6.7×10^{-7}. This estimate is small enough that the cancer risk via the oral route can be dismissed as, negligible when DBDPO is used as an upholstery fabric flame retardant."

END OF NAS QUOTATION, page 93 of that document.

ACC BFRIP
Brominated Flame Retardants Workshop
November 13, 2001, Washington D.C.

Assessment Of Reported Decabromodiphenyl Oxide Blood And Air Levels In Swedish Workers And Their Workplace

Marcia L. Hardy[1]

ABSTRACT Decabromodiphenyl oxide (DBDPO), the second largest volume brominated flame retardant, is a highly effective flame retardant used primarily in electrical and electronic equipment. Two recent papers report the detection of DBPDO, and other polybrominated diphenyl oxide isomers, in Swedish workers dismantling electronic equipment and in computer technicians. The aim of this communication is to evaluate the significance of the reported DBDPO blood levels in relation to DBDPO's toxicology, in terms of what could be expected based on DBPDO pharmacokinetics, and in comparison to the American Industrial Hygiene Association's (AIHA) DBDPO Workplace Environmental Exposure Level (WEEL) of 5 mg/m^3. The DBDPO blood levels reported in electronics dismantlers (5 pmol/g lipid) and computer technicians (1.6 pmol/g lipid) were extremely small. The electronics dismantlers' internal dose (1.2 ng/kg body weight), based on their measured DBDPO blood concentrations, was comparable to the concentration expected (0.57 ng/kg body weight) calculated from the measured air concentration. The DBDPO measured air concentration (0.2 ug/m^3) in the electronics recycling plant was 0.004% of the AIHA WEEL (5 mg/m^3). Based on DBDPO's toxicology and its measured air and blood concentrations, no impact on human health is expected in either the electronics dismantlers or computer technicians.

INTRODUCTION Brominated flame retardants (BFRs) comprise about 25% of the world volume of flame retardants (FR) and are used in applications requiring high FR performance or in resins needing a FR active in the gas phase.[1] The bromine portion of the compound is responsible for the molecule's flame retardant activity and is unique in its ability to provide flame retardancy in the gas phase. BFRs as a class are structurally diverse and include aromatic diphenyl oxides (a.k.a. ethers), cyclic aliphatics, phenolic derivatives, aliphatics, phthalic anhydride derivatives and others.[2] Within the BFR group known as polybrominated diphenyl oxides (PBDPO) or polybrominated diphenyl ethers (PBDE) are three commercial products: decabromodiphenyl oxide (DBDPO), octabromodiphenyl oxide (OBDPO) and pentabromodiphenyl oxide (PeBDPO). The composition, production volumes, uses and toxicology of the three commercial PBDPO flame retardants DBDPO, OBDPO and PeBDPO are distinctly different.[2]

Decabromodiphenyl oxide (DBDPO, CAS# 1163-19-5), also known as decabromodiphenyl ether, is a highly effective flame retardant used primarily in electrical and electronic equipment and secondarily in upholstery textiles.[1] Global market demand in 1999 for DBDPO was estimated at 54,800 metric tons, which makes it the second largest volume BFR in use today after tetrabromobisphenol A (TBBPA).[3] Together, DBDPO and TBBPA account for approximately 50% of all BFR usage globally. Two papers report the detection of DBPDO, and other polybrominated diphenyl oxide (a.k.a. ether) isomers, in Swedish workers dismantling electronic equipment[4] and in computer technicians[5]. The aim of this communication is to evaluate the reported DBDPO blood

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levels in relation to DBDPO's toxicology and pharmacokinetics, and to compare the reported air levels to the American Industrial Hygiene Association's (AIHA) Workplace Environmental Exposure Level (WEEL) for DBDPO of 5 mg/m^3.

DBDPO TOXICOLOGY AND PHARMACOKINETICS DBDPO is a high molecular weight substance (959.21) with negligible solubility in water (<0.1 ug/L) and organic solvents (acetone 0.05%, benzene 0.48%).[2;6] DBDPO has undergone a wide range of toxicology tests in mammalian species. DBDPO was not: acutely toxic, irritating, a skin sensitizer, genotoxic or an inducer of hepatic enzymes.[6-11] The no-adverse-effect-level in 14 and 90 day studies in rats and mice was > 1,000 mg/kg/body weight.[10] The no-effect-level for developmental effects in rats was $\geq$ 1,000 mg/kg/day administered on days 0-19 of gestation.[12] Doses as high as 2,550 or 7,780 mg/kg/d administered to rats and mice, respectively, for 2 years were well tolerated with no effect on body weight or mortality, only minimal evidence of organ effects, and no, equivocal or some evidence of carcinogenicity (dependent on genus and sex).[10] No evidence of carcinogenicity was found in female mice at doses of ~3,760 or 7,780 mg/kg/d.[10] Equivocal evidence was found in male mice due to the combined incidence of hepatic adenomas or carcinomas at ~3,200 or 6,650 mg/kg/d; however, a large number of early deaths in the control may have influenced the statistical significance detected and the incidence was within the historical range.[10] Some evidence was found in male and female rats at doses of ~1,120, 2,240 or 2,550 mg/kg/d due to hepatic "neoplastic nodules"[10]; terminology which is no longer used to describe hepatoproliferative lesions in rats. DBDPO is not listed as a carcinogen by the U.S. National Toxicology Program (NTP),[13] the International Agency for Research on Cancer (IARC)[14] or the U.S. Occupational Safety and Health Administration (OSHA)[15].

DBDPO's low toxicity is likely related to its poor absorption and rapid elimination.[10;16;17] Pharmacokinetic studies have shown that DBPDO is poorly absorbed (<0.3 – 2% oral dose), has a short half life (<24 hr) compared to PCB 153 (only 2% of an oral dose was eliminated by rats in 21 days), can be metabolized, and is rapidly eliminated in the feces (>99% in 72 hr).[7-10;16;17]

MEASURED VERSUS THEORETICAL DBDPO BLOOD LEVELS DBPDO oral absorption is minimal (<0.3 to 2% of an oral dose), but no data on its pulmonary absorption is available. Although the absorptive processes in the lung and gastrointestinal (GI) tact are similar, DBDPO absorption from the respiratory tract is expected to be less than from the GI tract. The respiratory membrane has a surface area of 160 m^2 versus 250 m^2 for the intestinal mucosal villi,[18] and the lung's absorptive surface is therefore ~64% of that of the small intestine. Further, Sjodin reported that μ 99% of the DBDPO detected in air at the electronics dismantling plant was associated with particulate matter.[19] DBDPO has negligible solubility, and thus inhaled particle-bound DBPDO can be expected to behave similar to other inert insoluble particles deposited in the respiratory tract. Insoluble particles deposited within the ciliated airways of the respiratory tract (e.g., the nasal passages and tracheobronchial tree) undergo passive transport via the mucuciliary escalator to the pharynx and are subsequently swallowed.[20] Insoluble particles reaching the alveoli are predominantly cleared by alveolar macrophages that phagocytize the particles and transport them proximally on the bronchial tree to be swallowed. Absorption of insoluble particles from the alveoli directly into the bloodstream is low and exceedingly slow. Thus, it appears unlikely that absorption of DBPDO from the respiratory tract is greater than that of the gastrointestinal tract, and that a theoretical DBDPO blood concentration can be calculated using the percent oral absorption as an indicator of respiratory uptake. Using a maximum absorption of 2% of the dose and at maximum measured DBDPO air concentration of 0.2 ug/m^3 in the electronics dismantling plant[4], the absorbed dose would be 0.04 ug DBDPO/70 kg man or 0.57 ng DBDPO/kg body weight.

The amount of a substance absorbed (A_{dose}) through the respiratory tract over a given period of exposure can be calculated using the concentration in air in mg/m^3 (A_c), the duration of exposure in hours (T), the ventilation rate in m^3/hour (V), and the absorption rate (A_{bs}): $A_{dose} = A_cTVA_{bs}$.[21] Using a maximum absorption of 2% of the dose, a ventilation rate of 10 m^3/8 hr work shift and at maximum measured DBDPO air concentration of 0.2 ug/m^3 in the electronics dismantling plant [4], the absorbed dose would be 0.04 ug DBDPO/70 kg man or 0.57 ng DBDPO/kg body weight. At a measured DBDPO serum lipid level of 4.8 ng/g lipid in the electronics dismantling workers,[4] the DBDPO plasma level would be 0.0288 ng/ml plasma. Assuming 3,000 ml plasma in a 70 kg man and a normal plasma lipid concentration of 0.6%,[18] the 0.0288 ng DBDPO/ml plasma represents a total blood volume content of 86.4 ng DBDPO/70 kg man or 1.2 ng/kg body weight. Thus, the theoretical DBDPO internal dose (0.57 ng/kg body weight) due to a measured air concentration of 0.2 ug/m^3 compares favorably with the actual dose of 1.2 ng/kg body weight in the electronics dismantling workers calculated from their measured blood values. The theoretical and measured values are well within the variation expected due to the assumptions used in calculating the expected values and the collection and analytical methods.

MEASURED AIR LEVEL VERSUS ACCEPTABLE DBDPO WORKPLACE EXPOSURE LEVEL The measured DBDPO air level at the electronic recycling plant was 0.0002 mg/m.[4] The American Industrial Hygiene Association (AIHA) evaluated DBDPO's toxicology and set a Workplace Environmental Exposure Level (WEEL) of 5 mg/m^3, e.g. that of a nuisance dust.[22] Thus, the measured DBDPO air level at the electronics dismantling plant was 25,000 times below the AIHA level to which workers could be exposed every day with the expectation of no adverse effects. Further, using the equation $A_{dose} = A_cTVA_{bs}$ and a maximum absorption of 2%, the estimated internal DBDPO dose from an 8-hr exposure at the AIHA WEEL of 5 mg/m^3 would be 0.11 mg/kg body weight. The internal dose of the electronic recycling workers was 1.2 ng/kg or 0.001% of the internal dose that could be received at a DBDPO exposure equal to the AIHA WEEL. Finally, in the event that DBDPO absorption from the respiratory tract was greater than 2%, the internal dose of the electronic recycling workers at a measured DBDPO air level of 0.0002 mg/m^3 would remain substantially below that achievable at the AIHA WEEL. For example, if DBDPO absorption equaled 100%, the internal dose due to a workplace air level of 0.0002 mg/m^3 would be 0.004% of that dose which could be received at a DBDPO exposure equal to the AIHA WEEL.

DISCUSSION The DBDPO blood levels reported in Swedish electronics dismantling workers (5 pmol/g lipid) and computer technicians (1.6 pmol/g lipid) were extremely small and are representative of our increasing ability to detect minute amounts of chemicals in various media. The DBDPO blood levels were far below those of PCB 153 (dismantlers, 760 pmol/g lipid; technicians, 290 pmol/g lipid) measured in the same workers. Further, the electronics dismantling workers' internal DBDPO dose (1.2 ng/kg body weight) based on their measured blood level was comparable to the level expected (0.57 ng/kg body weight) calculated from the measured air levels. A similar comparison was not possible for the computer technicians because air values were not reported for that workplace. In addition, the DBDPO measured air level (0.2 ug/m^3) in the electronics recycling plant was approximately 25,000 times below the acceptable DBDPO workplace exposure level of 5 mg/m^3. This acceptable workplace exposure level, set by the AIHA, was based on an evaluation of DBDPO toxicology data. Thus, no impact on human health from DBDPO is expected in either the electronics dismantlers or computer technicians.

The tendency of some investigators has been to assume that any detectable level of an exogenous substance is equivalent to an adverse effect. This approach is both unscientific and misguided, however, because it fails to consider dose-response relationships, detoxification mechanisms, and

other important factors that must be taken into consideration when evaluating whether specific chemical exposures might be related to health effects.[23] This approach also fails to consider that virtually all substances to which a human is exposed are absorbed into the blood in some amount.[23] As a result of these exposures, human tissues and body fluids contain trace levels of virtually any and all the foreign substances to which we are exposed on a daily basis. Knowledge that this occurs is not new.[24] Further, occupational exposure limits (OEL) are based on the premise that, although all chemical substances are toxic at some concentration when experienced for a specific period of time, a concentration (e.g., dose) does exist for all substances at which no injurious effect should result no matter how often the exposure is repeated.[21] OEL are set based on the best available information from industrial experience, experimental human and animal studies, and from a combination of the three when possible. In addition, a safety factor is usually applied to animal data to allow for the possibility that humans may be more sensitive to the substance than the species tested. The principles used in setting OEL are similar to those used to identify safe doses of food additives and pharmaceuticals. Thus, the establishment of exposure limits, by their very nature, implies that at some concentration or dose, exposure to a chemical substance can be expected to pose no significant risk of harm to exposed persons.[21] Given that human tissues and body fluids contain trace levels of virtually any foreign substance to which we are exposed, it then follows that exposure to a substance at or below its OEL is likely to result in some measurable quantity in human body fluids. The mere detection of such levels should not be construed as an adverse effect or a cause for alarm; to do so would mean abandoning the concept of OEL as a means to prevent or minimize occupational disease.

References

[1] Hardy M, *Organohalogen Compounds,* **2000**, *47*, 41-44.
[2] Hardy M, *Chemosphere*, 2002, *In press*.
[3] Bromine Science and Environmental Forum (BSEF) Web Site. http://www.bsef.com. 2001.
[4] Sjodin A, *Environ Health Perspect,* **1999**, *107*, 643-648.
[5] Hagmar L, *Organohalogen Compounds,* **2000**, *47*, 202-205.
[6] World Health Organization. IPCS Environmental Health Criteria Document. Report No. 162. World Health Organization, Geneva, 1994.
[7] Norris J et al., *Environ Health Perspect,* **1975**, *11*, 153-161.
[8] Norris J et al., *JFF/Combustion Toxicology*, **1974**, *1*, 52-77.
[9] Norris J et al., *Applied Polymer Symposia, No.22. Polymeric Materials for Unusual Service Conditions,* **1973**, 195-219.
[10] National Toxicology Program (NTP). *Report No. NTP Technical Report Series No. 398.* U.S. Department of Health and Human Services. Public Health Service. National Institutes of Health., Research Triangle Park, NC, 1986.
[11] Carlson G, *Toxicol Lett,* **1989,** *5*, 19-25.
[12] Schroeder R, Report # 474-003, MPI Research, Mattawan, MI, 2000.
[13] NTP 9th Report on Carcinogens 2000. Available: http://ehis.niehs.nih.gov/roc/toc9.html
[14] IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Some Flame Retardants and Textile Chemicals, and Exposures in the Textile Manufacturing Industry. Volume 48: 73-84. 1990.
[15] OSHA Standard 1990. Available: http://www.osha-slc.gov/SLTC/carcinogens/index.html
[16] El Dareer S et al. *J Toxicol Environ Health,* **1987**, *22*, 405-415.
[17] Morck A and Klasson Wehler E. The Second International Workshop on Brominated Flame Retardants. May 14-16, 2001. Stockholm University, Sweden.
[18] Guyton A. Textbook of Medical Physiology (W.B. Saunders Company, Philadelphia, ed. Seventh Edition, 1986).
[19] Sjodin A. Thesis, Stockholm University (2000).

[20] Lippmann M et al., *British Journal of Industrial Medicine,* **1980**, *37*, 337-362.
[21] Patty's Industrial Hygiene and Toxicology. (John Wiley and Sons, New York, 1994).
[22]Workplace Environmental Exposure Level: Decabromodiphenyl Oxide. AIHA. American Industrial Hygiene Association, 2700 Prosperity Avenue, Suite 250, Fairfax, VA 22031, 1996.
[23] American Council on Science and Health. Traces of Environmental Chemicals in the Human Body: Are They a Risk to Health? Available: http://www.acsh.org.
[24] Kroger M. General environmental contaminants occurring in milk. In: Lactation, Nutrition and Biochemistry of Milk/Maintenance, Vol III (Larson BL and Smith VR, eds). New York: Academic Press, Inc.; 1974:135-158.

City of Seattle
Gregory J. Nickels, Mayor

Office of Sustainability and Environment
Steven Nicholas, Director

Department of Ecology
Attn: Cheri Peele
PO Box 47600
Olympia, WA 98504

Re: Comments on Draft PBDE Chemical Action Plan

Dear Ms. Peele;

Thank you for the opportunity to comment on the Department of Ecology's and Health's Draft PBDE Chemical Action Plan. I serve as director of the City's Office of Sustainability and Environment, and am writing to express the City's support for the recommendations provided in the plan to reduce threats posed by PBDEs.

Given the persistent and accumulative nature of these products we believe we need to move now to reduce exposures to humans and the environment and to find viable alternatives to these products. As you may know, the City has been very active in identifying areas where we can reduce or eliminate our use of persistent, bioaccumulative toxic chemicals such as PBDEs. For example, we have stopped purchasing pentachlorophenol treated utility poles and creosote-treated wood, we use ultra-low sulfur diesel and biodiesel to reduce PAHs in our diesel fleet, and we are removing mercury containing trunk switches when vehicles are retired.

We agree that additional study is needed prior to developing final recommendations on how to handle products that contain PBDEs in the waste stream. This is a complex issue that needs careful consideration. We support prohibiting the manufacture, distribution, and sale of new products containing Penta-BDE and Octa-BDE. We think the industry phase out of Penta-BDE and Octa-BDE and the EPA rule prohibiting importation of these substances will minimize the potential impact of this provision. While there are some questions regarding the safety of Deca-BDE, we agree that the best solution is to take precautionary measures to reduce the potential for its increased use. It is encouraging to see manufactures of products containing Deca-BDEs, especially electronic manufactures, phasing out their use over the next several years.

Thank you again for the opportunity to comment and we look forward to working with you to reduce exposure to PBDEs as quickly as possible.

Sincerely,

Steve Nicholas, Director
Office of Sustainability and Environment

Tacoma City of Tacoma
Public Works Department

November 8, 2004

Department of Ecology
Attention: *Cheri Peele*
P. O. Box 47600
Olympia, Washington 98504-

Dear Ms Peele:

Thank you for the opportunity to comment on Ecology's draft PBDE Chemical Action Plan. The City of Tacoma wastewater utility operates two secondary wastewater plants. We treat an average of 45 million gallons of wastewater everyday and produce approximately 3500 dry tons of Biosolids each year. Tacoma is committed to the protection of public health and the preservation and enhancement of the aquatic environment in the greater Tacoma area. As an organization we base our management on science. We believe that using the best science available produces the greatest protection of human health and the environment because it focuses resources on the most significant risks.

The potential toxic effects of PBDEs are just now beginning to be understood. As you have pointed out the data gaps are legion. We commend Ecology for taking on the task of examining the PBDE problem and making recommendations. We believe that the greatest value of your work has been to open up a dialogue on what we do and mostly do not know about PBDEs. Your efforts have pointed out the vast amount of work that must be done to adequately characterize the environmental and human health risk posed by PBDEs.

The comments provided by the external scientific review were excellent. We strongly recommend that you include them as an appendix to the chemical action plan.

We agree with the general approach Ecology has taken with the recommendations contained in the draft chemical action plan. Leveraging resources with other states and the federal government in conducting the debromination and toxicological studies will be absolutely necessary.

We do have some concerns with the Chemical Action Plans recommended approach to monitoring. The background information provided in the draft plan and the comments provided by external experts points out how little we know about fate and transport of PBDEs. We know even less about exposure pathways. We believe that in order to be efficient and effective in protecting public health and the Environment we need to know the pathways of exposure. Only after the exposure pathways have been determined and the fates of the various congeners of PBDE have been determined can we begin to know what and where to monitor.

It is important to note here that the two routes of exposure cited in the Chemical Action Plan and in the comments by outside experts are dietary (primarily fish) and indoor airborne particulate. Given these primary routes of exposure we wonder why it would be important to monitor PBDE content of Biosolids, which are generally not found indoors and are barred from use in aquatic environments. We think there are some very valid reasons to explore other facets of the PBDE issue before adding another obligation to an already heavily regulated sector of the environmental services community.

Tacoma believes that the recommendation that Ecology monitor Biosolids for PBDE content be delayed or dropped until it is determined if biosolids is an important pathway for exposure. We believe Ecology should prioritize its actions to focus first on exposure routes, fate, and toxicity and use this information to design a more cost effective monitoring program. Analytical methods for conducting any survey work conducted by Ecology need to be recognized by appropriate regulatory authorities.

The chemical action plan has recommended a ban on penta, octa, and deca BDE. It appears to us that the evidence presented in justifying this action, particularly in the case of deca BDE, is less than compelling. There may be reasons for a ban that are not related to risk (for example demonstrated safe alternatives are available and easily substitutable). If this is the case we urge Ecology to address this overtly. Separating risk related concerns from precautionary measures strengthens the recommendations by differentiating policy from science.

Thank you for this opportunity to comment on Ecology's chemical action plan for PBDE. If you have any questions on our comments or would like further clarification on our positions please contact me at (253) 502-2191.

Sincerely

Dan C. Thompson Ph. D
Environmental Services Wastewater Operations Division Manager
City of Tacoma Public Works

DCT: js (W19531)

File: Wastewater Management Source Control

INDEPENDENT BUSINESS ASSOCIATION

November 9, 2004

Cheri Peele
Department of Ecology
PO Box 47600
Olympia, WA 98504-7600

Dear Ms. Peele:

Thank you for considering our comments regarding the draft PBDE Chemical Action Plan. We have the following comments:

- We have received input from the vehicle recycling industry with the following comments:
  - They feel strongly that "auto fluff" should continue to be used as a cap for landfills. They strongly object to the policy option statement, "*Restrict the disposal of products containing PBDE's to landfills that do not release leachate into the environment or to waste water treatment plants.*"
  - If there are special handling or disposal requirements imposed on PBDE containing materials, the cost should be borne by the manufacturer or society at-large and not imposed on those responsible for disposal. In the case of the vehicle recycling industry, if new handling and disposal costs are imposed on vehicle recyclers, it may destroy the industry.

- Small businesses have very serious concerns about identifying PBDE as a "special waste." It is unclear what "special waste" means. We know of no statutory basis for classifying PBDE as a "special waste." We recommend it be removed from the CAP.

- Small businesses are oppose the recommendation: "*The Legislature should pass a ban on the manufacture, distribution, and sale of new electronics and electrical equipment containing Deca-BDE, in Washington State effective July 2008.*" We find this recommendation very premature with inadequate analysis of the effects of such a ban and inadequate analysis of the alternative fire retardants that would be used in its place. At the most, the CAP should call for a complete review and analysis of the effects of such a ban if it were to be but in place so that the Legislature and the citizens at large have a much better knowledge of the full effects of such a policy before it is proposed or enacted.

- Small businesses are concerned that the efforts to eliminate the use of Deca-BDE will result in either reduced fire safety or forcing manufacturers into using other types of fire retardants that have not been fully assessed for health and environmental risks only to find out later they have such risks. Neither outcome is acceptable.

Thank you for considering our comments.

Sincerely,

Gary Smith
Executive Director

16541 Redmond Way #336C Redmond, Washington 98052 (425) 453-8621 Email iba@isomedia.com

INSTITUTE for
CHILDREN'S
ENVIRONMENTAL
HEALTH

PBDE Public Hearing
October 19, 2004
Seattle, WA

RECEIVED
OCT. 27 2004

Testimony

First, I want to commend the Department of Ecology for all your work to date on polybrominated diphenyl ethers (PBDEs). It is clear from your draft "PBDE Chemical Action Plan" that you know what they are and what they can do to human health. You know PBDEs are a group of flame retardants widely used in many products. You know that PBDEs persist in the environment and build up in animals and people. You also know that PBDEs impair memory, learning and behavior in laboratory animals and they can affect thyroid hormones—which are essential for healthy brain development. And you know that exposures put developing fetuses, infants, and young children most at risk because their biological systems, including their nervous systems and brains, are still developing.

What you may not know, though, is that as executive director of the Institute for Children's Environmental Health and coordinator of the Learning and Developmental Disabilities Initiative, I work nationally with groups who are concerned about the increasing numbers of children with learning and developmental disabilities. These groups include the Learning Disabilities Association, the American Association on Mental Retardation, the Autism Society, the American Association for the Dually Diagnosed and many others. These colleagues, in addition to witnessing the increasing numbers, observe the increasing emotional and social costs of learning and developmental disabilities to these children, their families and communities. They also see the increasing economics costs of lost income, special education, lower intellectual capacity, school drop outs and incarceration.

What these groups also know is that though many factors can contribute to learning and developmental disabilities—nutrition, genetics, social factors—the one factor that is often the least acknowledged and the easiest to prevent are environmental factors. We've seen this with lead and tobacco and with PCBs, which are molecularly so similar to PBDEs.

Now these learning and developmental disabilities groups, along with physicians, nurses, public health officials, child advocates and environmental health organizations are taking a stand—we're saying it's not okay to expose our children to known and suspected neurotoxicants, such as PBDEs.

We're saying if the cornerstone of public health is prevention, then why are we waiting? The evidence is clear that PBDEs can harm developing brains and alternatives exist. Not only could we save the emotional and social costs of more kids with learning and developmental disabilities, we could save the economic costs. In fact, in a study undertaken by doctors and researchers at Mt. Sinai School of Medicine, estimated that the environmentally attributable fraction of health care costs for neurobehavioral disorders in children nationally are conservatively: $4.6 billion annually. This means that if those environmental pollutants are removed then we could collectively, on a societal level, save $4.6 billion annually. This means that by phasing out PBDEs, we could take a step towards reducing these health care costs.

When there's the political will to act, we have shown we can create a healthier environment for our children as we did by reducing lead and tobacco exposures. We have another opportunity to do the right thing now. Let's have Washington State lead the nation by phasing out all forms of PBDEs, by using alternatives and by saying 'yes' to our children reaching their fullest potential.

Thank you.

Elise Miller, M.Ed.
Executive Director
Institute for Children's Environmental Health
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Emiller@iceh.org

Local Hazardous Waste Management Program in King County

November 9, 2004

Dear Cheri,

Thank you for this opportunity to comment on the State's draft PBDE Chemical Action Plan.

The following general comments and suggestions are offered for your consideration.

- The Local Hazardous Waste Management Program is strongly supportive of the recommendations to prohibit the manufacture, distribution and sale of new products containing Penta-, Octa- and Deca-BDE's as proposed in the draft plan.
- The Program encourages the Department of Ecology to develop, together with interested local jurisdictions, a state-wide strategy for the disposal and recycling of products containing PBDEs. (Note: The recommendation in the draft plan calls for Ecology to examine and revise disposal practices - it is not clear to me what that really means.) The Program further encourages Ecology to set a target completion date for the development of a state-wide disposal and recycling strategy before the end of 2005.
- The Program is very supportive of requiring manufacturers to fund PBDE monitoring and research activities. The State should also consider the possibility of requiring manufacturers of PBDE-containing products to fund, at least in part, any state-developed plan for the disposal/recycling of PBDE-containing products. Involving manufacturers financially not only allocates end-of-life costs more appropriately - but will likely result in a more sustainable system. Although getting manufacturer involvement may be a challenge perhaps the best way to bring their research and development resources to bear is by having them share in the end-of-life costs.
- Expanding on the (draft) Plan's recommendation to research alternatives to brominated fire retardants - the Program recommends that upon completion of such research the State list those alternatives as the only acceptable fire retardants for use in products manufactured, sold, or distributed in Washington State.

I applaud the State's efforts to develop a comprehensive and forward-looking plan to deal with this ubiquitous and problematic chemical.

Attached are some additional, more detailed, comments relating to the draft plan. Questions regarding the details contained in the attached comments should be referred to Alice Chapman, who can be reached at 206-263-3058.

Please feel free to call me if you have any other questions.

Sincerely,

Kenneth W. Armstrong
Administrator
The Local Hazardous Waste Management Program in King County
150 Nickerson Street, Suite 100
Seattle, WA 98109
206-352-8163

Comment 1

Conduct a Root Cause Analysis and Develop Preventive Recommendations (Page 24, 35 and 57)

As presented on page 35, wastes containing PBDE flame retardants likely designate under Washington's persistent halogenated organic compound (HOC) regulations. The criteria for HOC persistence was established around thirty years ago. Historically, these regulations appear to have been poorly understood by generators and a relatively low rate of proper designation and compliance resulted. Further, it's difficult for generators to know what wastes to evaluate for HOCs such as PBDE flame retardants. The possibility that furniture, mattresses and other common items used in intimate contact with humans both in businesses and in homes should be evaluated for "dangerous waste" criteria just didn't occur to most of us.

Purchasers didn't know they were buying a product that at end of life required costly hazardous waste management according to State regulations. Regulators with hazardous waste expertise didn't know HOC-laden products were being sold in such volumes. Had the proper knowledge been readily accessible when PBDEs were first being used, the true cost of the purchase could have been known and perhaps market forces would have reduced PBDE use.

The current recommendation (see page 61) to require disclosure of PBDEs for bidders on State contracts is an improvement, but it doesn't go far enough to help the many small businesses and residents in Washington.

Future research will tell us whether the greater danger to human health and the environment is in the daily use of PBDE-laden products or in end-of-life management. A sound rationale underlies the persistent HOC regulations and the discovery of environmental problems with PBDEs bears this out. Somehow, these regulations have failed to protect us. As a State, we find ourselves needing to research (see page 57, recommendation 1 and page 58 Rationale) the types of environmental or human health problems of municipal waste management of PBDEs that our State's regulations already classified as dangerous waste.

The situation with PBDEs teaches us again the lesson we learned in the 1970's with PCBs: connect product management with waste management. The PBDE situation is a symptom of a problem, and the proposed recommendations of the Chemical Action Plan take steps to reduce the damage of the symptoms and to assess the extent of PBDE damage. The Local Hazardous Waste Management Program would like to see additional discussions and recommendations that address the root cause.

- How did such a vast waste stream avoid proper management and disposal under existing State dangerous waste regulations? As discussed on page 24, waste with PBDEs that is likely dangerous waste now makes up 6.5 percent of municipal solid wastes.
- How did a persistent bioaccumulative toxin at high concentrations so thoroughly saturate our homes, workplaces, environment and our bodies without notice for years?
- What other ubiquitous chemicals, including other halogenated flame retardants, pose similar and yet undiscovered risks? How can we find them?
- Average consumers shouldn't bear the full burden for keeping themselves safe from hidden chemical risks in everyday products.

Comment 2
What if the PBDE prohibition is not passed by the legislature and signed into law?

If the PBDE bans don't go through, manufacturers of products that contain PBDEs need to identify PBDEs in a way that is easily accessible to the purchaser. Businesses or households must be able to readily identify sources of PBDEs in order to reduce exposure to PBDEs and make informed choices about purchasing products that may contain them. Ask manufacturers who used PBDEs in the past to identify where PBDEs were used. Adequate waste management or recycling systems (still to be developed) will be difficult to implement without an easy way to identify items containing PBDEs. Identification thru State contract bids alone (page 61, State purchasing recommendation) is a start but doesn't address the needs of most Washington businesses or residents.

As mentioned in the last paragraph on page 7 of the Chemical Action Plan, "It appeared that companies fabricating items from plastic or foam feedstock did not know which, if any, flame retardants were added to their materials." This statement confirms the experience that the Local Hazardous Waste Management Program has had in attempting to identify sources of halogens detected in waste samples. Currently, PBDEs are often not identified on product material safety data sheets. Product compositions may be even more difficult to identify when manufactured outside the United States.

Comment 3
End-of-Life Management (Page 57 and 61)

The end-of-life management section acknowledges that many wastes with PBDEs designate as persistent dangerous waste now. Until new recommendations are developed in July 2007, waste management guidance for proper disposal during this interim period is needed. Otherwise, generators may be at risk of knowingly disposing of dangerous wastes improperly.

Consider shortening the timeframe for completing end-of-life management recommendations. Page 61 has a recommendation for Department of Health to educate the general public about reducing PBDE exposures and the general public will want to know about disposal options. If disposal to municipal solid waste is the only option presented during these first two years, an opportunity is missed.

Comment 4
End-of-Life Management (Page 57)

We encourage PBDE staff to work closely with Ecology staff (Alex Stone and Leatta Dahlhoff) updating the persistent HOC designation guidance in Ecology's publication 97-407, *Chemical Testing Methods for Designating Dangerous Waste*. Testing data and technical resources they are gathering should prove valuable in determining the next steps for PBDE end-of-life management. Generators of HOC wastes need the PBDE Chemical Action Plan to be consistent with dangerous waste management regulations and guidance.

Comment 5
Coordinate PBDE and PBT initiatives for dioxin reduction (Page 26 and 27)

While this PBDE Chemical Action Plan is being developed, a broader program for Persistent Bioaccumulative Toxins is also being reviewed. These two plans clearly overlap and we encourage Department of Ecology and other state departments to ensure the activities of these two groups are coordinated. Two specific examples are provided below:

(Page 26) "Biosolids & Sewage" indicates that regulations for sewage sludge incineration lack minimum temperature ranges. For dioxin reduction (a goal of both PBT and PBDE plans) perhaps minimum temperatures should be considered.

(Page 27) ash reuse may also have potential actions for dioxin reduction which could benefit both PBT and PBDE plans.

Comment 6
(Page 57, Recommendation 1)

Add vent and duct cleaners to the list of service industries to assess.

Comment 7
(Page 57, Recommendation 3, "Special Waste")

A "Special Waste" management option exists in current Dangerous Waste regulations and it already poses some difficulties. It is hard to explain to small businesses that their hazardous waste is "special" and what that really means. To our knowledge, few counties statewide have access to facilities that are allowed to manage "special waste" so it often ends up being managed as hazardous waste anyway. Where it is available, generators may have to haul the "special waste" directly to the landfill themselves, because of transfer station limitations.

Consider managing PBDE waste, and perhaps other wastes with halogenated flame retardants, as state-only "Universal Wastes." PBDE waste is now universally generated and could have similar management needs, such as many drop-off locations, as the other universal wastes (mercury lamps, thermostats, batteries, etc.)

Comment 8
(Page 58/59 ban on penta-BDE and octa-BDE)

The Local Hazardous Waste Management Program supports the recommended ban on new products with penta- or octa-BDE by July 2006.

Comment 9
(Page 34, 42 and 59, ban on deca-BDE)

We agree with the statement on page 34 regarding deca-PBDE, that sufficient evidence exists to warrant concern and action. The experience of Germans (see page 42) with the voluntary agreement to discontinue production and use of PBDEs since 1989 seems to indicate that alternatives are available. The Local Hazardous Waste Management Program supports a ban on electronics, electrical equipment and new upholstered fabrics by July 2008.

Comment 10
(Page 63, human health monitoring and environmental monitoring)

Ensure biomonitoring proposals include routine breast milk testing. Breast milk should be monitored for many potential contaminants, not simply PBDEs. Likewise, ensure research needs address potential risks other than PBDEs. A better early warning system is needed in Washington to detect human health and environmental risks.

Comment 11
General comment (Page 2, fourth paragraph)

Thank you for obtaining peer review by qualified and internationally recognized technical experts; this enhances the document's credibility.

Comment 12
Minor editing or typographical comment (Page 5, Table 3)

Please line up the numbers in columns by decimal point location. This will enhance the readability of the table.

Comment 13
Minor editing or typographical comment (Page 24, Figure 7)

The first box under "PBDE Content" reads "Polyurethane Poam" and should be changed to "Polyurethane Foam".

Comment 14
Minor editing or typographical comment (Page 39, paragraph 5 of European Union summary)

Change sentence as follows: "First, it ~~first~~ concluded that there is a need..."

Comment 15
Minor editing or typographical comment (Page 59, paragraph 2 of Deca-BDE Key Findings.)

Change paragraph end as follows: "... redesign or retooling to replace Deca-DBeE later on.~~.~~"

From: SwansonD@MKAmerica.com
Sent: Thursday, November 11, 2004 2:00 PM
To: Peele, Cheri
Subject: Re: Draft PBDE CAP

Good afternoon Cheri. Sorry I am late with comments, however....

Overall, DOE's PBDE plan is well thought out. I totally agree with the Octa and Penta ban. As a company, Matsushita (as well as other manufacturers have followed the EU and the Rohs). Matsushita has a ban on all PBDE material used in their manufacturing products taking effect 3/31/05 (as well as a ban on lead, lead solder, mercury, cadmium, and hexavalent chromium). Consumer electronics manufacturers use flame retardant material as one part of the UL (Underwriters Laboratories) approval. The flame retardant material is not primarily for electronics-derived fires as they are few. The flame retardant material in electronics bodies is to restrict (or not increase) the spread of the fire that started elsewhere.

Being on the PBDE Advisory Board educated me to a lot of the risks associated with the penta and octa. The hard evidence from the deca is still not convincing to me.

For a manufacturer such as ours to make a change from a PBDE to a non-PBDE adds about 15% to the material cost. The new material being changed to, though not a PBDE is still new. This can create havoc on companies' bottom line and the reason that they are in business.

Some interesting facts are coming out of Europe, which has either banned or restricted FR material. There has been an increase of fatal structure fires. I am worried that companies not originally obtaining the UL certification, will now advertise their products as PBDE free.

Dale Swanson
Environmental Engineering/ISO 14001
Matsushita Kotobuki Electronics Industries
Swansond@MKamerica.com
(360)-567-0341Direct
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"Peele, Cheri" <CHEP461@ECY.WA.GOV>

10/12/2004 03:57 PM

To: "'lgcosta@u.washington.edu'" <lgcosta@u.washington.edu>, "'gdana@autoalliance.org'" <gdana@autoalliance.org>, "'mfg@environmentalhomecenter.com'" <mfg@environmentalhomecenter.com>, "'sego.jackson@co.snohomish.wa.us'" <sego.jackson@co.snohomish.wa.us>, "'clorch@totalreclaim.com'" <clorch@totalreclaim.com>, "'mo@washpirg.org'" <mo@washpirg.org>, "'tmcdonal@oehha.ca.gov'" <tmcdonal@oehha.ca.gov>, "'maryanno@u.washington.edu'" <maryanno@u.washington.edu>, "'mel.oleson@boeing.com'" <mel.oleson@boeing.com>, "'ivy@pugetsound.org'" <ivy@pugetsound.org>, "'dsanders@glcc.com'" <dsanders@glcc.com>, "'iba@isomedia.com'" <iba@isomedia.com>, "'swansond@Mkamerica.com'" <swansond@Mkamerica.com>, "'lvaleriano@watoxics.org'" <lvaleriano@watoxics.org>, "'awolk@ci.camas.wa.us'" <awolk@ci.camas.wa.us>, 'Jason Lewis' <jason.lewis@retailassociation.org>
cc: "'ron.bowen@wsp.wa.gov'" <ron.bowen@wsp.wa.gov>, "Bowhay, Dennis L." <dbow461@ECY.WA.GOV>, "'dancoyne@coynejesernig.com'" <dancoyne@coynejesernig.com>, "Duff, Robert" <Robert.Duff@DOH.WA.GOV>, "'steve@duncanlabs.com'" <steve@duncanlabs.com>, 'Pete Erickson' <peter@cascadiaconsulting.com>, "Gallagher, Mike (ECY)" <MGAL461@ECY.WA.GOV>, "'Green9600@comcast.net'" <Green9600@comcast.net>, "Helbrecht, Lynn" <Lynn.Helbrecht@OFM.WA.GOV>, "Hutchison, Sheryl" <shut461@ECY.WA.GOV>, "LaFlamme, Denise" <Denise.LaFlamme@DOH.WA.GOV>, "'Susan_Landry@albemarle.com'" <Susan_Landry@albemarle.com>, "Manugian, Richard" <RICM461@ECY.WA.GOV>, "'grantn@awb.org'" <grantn@awb.org>, "'peter_o'toole@was.bm.com'" <peter_o'toole@was.bm.com>, "'Marley B. Shoaf'" <marley@cascadiaconsulting.com>, "Shultz, Ron" <Ron.Shultz@OFM.WA.GOV>, "'gsmall@watoxics.org'" <gsmall@watoxics.org>, "Sturdevant, Ted (ECY)" <TSTU461@ECY.WA.GOV>, "Whittaker, Stephen G (LNI)" <WHIW235@LNI.WA.GOV>, "'wildermu@nwrain.com'" <wildermu@nwrain.com>, "Zehm, Polly" <pzeh461@ECY.WA.GOV>, 'Marc

Daudon' <marc@cascadiaconsulting.com>, "'cirone.patricia@epamail.epa.gov'" <cirone.patricia@epamail.epa.gov>, "'Layton@carneylaw.com'" <Layton@carneylaw.com>, "'Springer.Pat@epamail.epa.gov'" <Springer.Pat@epamail.epa.gov>, "'Dthompso@ci.tacoma.wa.us'" <Dthompso@ci.tacoma.wa.us>, "'watson.michael@epamail.epa.gov'" <watson.michael@epamail.epa.gov>, 'Robert Campbell' <RCAMPBEL@glcc.com>, "Brace, Sarah" <SBrace@PSAT.WA.GOV>, "Peele, Cheri" <CHEP461@ECY.WA.GOV>, "Sorlie, Greg" <gsor461@ECY.WA.GOV>, "Upthegrove, Dave" <upthegro_da@leg.wa.gov>, "Backous, Bill" <BBAC461@ECY.WA.GOV>, "Prado, Joanne" <Joanne.Prado@DOH.WA.GOV>, 'Terwille_ka@leg.wa.gov'

Subject: Draft PBDE CAP

The draft PBDE CAP has been posted for the 30 day public comment period this morning at Ecology's website. You can find it at www.flameretardants.org. The recommendations reflect much of what we have heard from the committee and what was presented at the August 25 Advisory Committee meeting, but certainly not everything.

I want to thank you for your hard work and your input so far, but as you know, we are not yet done. I expect many of you may have specific comments when you see the draft plan. Comments can be sent to me by e-mail, fax, or hard copy through November 9. I would prefer to receive comments electronically to facilitate their distribution and consideration. The last Advisory Committee meeting will be held December 1 to discuss how the Departments of Ecology and Health might revise the plan based on the comments we receive.

Finally I want to remind you of the two public meetings we have scheduled; October 19 in Seattle and October 26 in Spokane. The time and location of the meetings can be found at www.flameretardants.org.

If you have any questions, please don't hesitate to call me.

Cheri

Cheri Peele
Policy Analyst
Washington State Department of Ecology
PO Box 47600
Olympia, WA 98504-7600
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MBA Polymers, Inc.

The Plastics Recycling Professionals

November 5, 2004

Department of Ecology
Attn: Cheri Peele
PO Box 47600
Olympia, WA 98504

Re: Draft PBDE Chemical Action Plan

Dear Department of Ecology Members:

We support your efforts to protect human health and the environment. We are writing to voice our concern on your recommendation to completely prohibit the manufacture, distribution, and sale of new products containing PBDEs. We are especially concerned of the impact that may have on the manufacture of durable goods (consumer and commercial products) using recycled plastics.

MBA Polymers recycles plastics from a range of consumer electronics that contain plastics with Octa-BDE and Deca-BDE. We convert the raw material to high value engineering plastics for reuse in similar applications. This is a relatively new industry, and MBA Polymers is at the cutting-edge of developing postconsumer engineering-grade plastics that have been successfully reused in the manufacture of new consumer electronics. Our impact on the plastics industry has been featured in nearly 100 articles and TV stories.

We want to alert the Department of Ecology and the Department of Health to the unintended consequences of prohibiting the use of products containing PBDEs. The ability to recycle plastics for the postconsumer market is a complex process. In evaluating the Draft PBDE Chemical Action Plan, we are particularly concerned about the following real-world scenario as a plastics recycler:

- PBDEs are melt-blended into plastics reacted into the polymer backbone, or dispersed into plastics.
- Octa-BDE and Deca-BDE -- which is used in TV cabinets, computer housings, electronic circuit boards and business machines -- is present in many plastics used in consumer electronic products. It is very difficult – if not impossible - to completely segregate them (plastics with Octa-BDE and Deca-BDE) from the general plastic waste stream from electronics, appliances, etc.
- If the use of PBDEs were completely prohibited today, without allowing for small allowances stemming from recycled plastics, it would be virtually impossible to implement cost-effective consumer electronic recycling programs and the legislation

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TEL (510) 231-9031 * FAX (510) 231-0302

would substantially undermine the market for recycled material in durable goods applications.

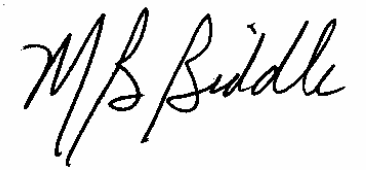
- The legislation would also make it economically impossible for manufacturers to use recycled plastics recovered from consumer electronics in new products because PBDEs are present in essentially all of these streams. Many US manufacturers are increasing their use of recycled plastics and depend on these feedstocks to continue to grow these programs. These programs, in turn, help sustainable manufacturing, lower greenhouse gases, reduce energy consumption (our plastics manufacturing requires less than 10% of that required to make the same amount of virgin plastics), help the manufacturers remain competitive in the marketplace, and reduce material being sent to landfills or incinerated. And finally,
- If we can't recycle these plastics, new plastics must be made to meet the needs of manufacturers, which will result in use of much more precious energy, increased greenhouse gas generation and higher costs to manufacturers. We don't think that this is the intent of the legislation.

At the end of its life cycle, most plastics from consumer electronics are disposed of in landfills, incinerated with other waste streams, recycled, or exported to other countries. Clearly, the most environmentally sound option is to recycle the plastic from these products. From a practical matter, banning the reuse of plastics containing Octa-BDE and Deca-BDE domestically does not solve the problem because it facilitates the export of these plastics to countries without restrictions that, in today's global marketplace, are often the ones that make new consumer electronics and other plastic products that are then sent back to the US.

Until consequences such as this can be prevented, we cannot support your recommendation to completely prohibit the manufacture, distribution, or sale of new products containing PBDEs. It is important to recognize that products that are in use today – televisions, computers and home appliances – will be a part of the consumer electronics waste stream for the next 10-20 years. We hope that you understand our concerns from the perspective of a durable goods recycler and further consider the disposition of recycled plastics containing PBDEs. We believe that reuse of this plastic is an environmentally sound solution considering today's alternatives.

Please contact us if we can provide you with additional information.

Sincerely,



Darren F. Arola

Dr. Mike Biddle
Chief Executive Officer

Dr. Darren F. Arola
Director - Product Development and Sales

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NORTHWEST BIOSOLIDS MANAGEMENT ASSOCIATION

201 S. Jackson St., KSC-NR-0512; Seattle, WA, 98104-3855 USA; Phone: (206) 684-1145; Fax: (206) 263-3485

Department of Ecology
Attn: *Cheri Peele*
PO Box 47600
Olympia, WA 98504

RECEIVED
NOV - 9 2004

Dear Ms Peele:

Thank you for the opportunity to comment on Ecology's draft PBDE Chemical Action Plan. The Northwest Biosolids Management Association (NBMA) represents over 200 public municipalities, sewer districts, and private companies most of which are located or have worked with Biosolids in Washington State. The NBMA is committed to the advancement of environmentally sound biosolids management. As an organization and an industry, we base our management recommendations on science. We believe that using the best science available produces the greatest protection of human health and the environment because it focuses resources on the most significant risks.

The potential toxic effects of PBDEs are just now beginning to be understood. As you have pointed out, the data gaps are legion. We commend Ecology for taking on the task of examining the PBDE problem and making recommendations. We believe that the greatest value of your work has been to open up a dialogue on what we do and mostly do not know about PBDEs. Your efforts have pointed out the vast amount of work that must be done to adequately characterize the environmental and human health risk posed by PBDEs.

The comments provided by the external scientific review were excellent. We strongly recommend that you include them as an appendix to the chemical action plan.

We agree with the general approach Ecology has taken with the recommendations contained in the draft chemical action plan. Leveraging resources with other states and the federal government in conducting the debromination and toxicological studies will be absolutely necessary.

We do have some concerns with the Chemical Action Plans recommended approach to monitoring. The background information provided in the draft plan and the comments provided by external experts points out how little we know about fate and transport of PBDEs. We know even less about exposure pathways. We believe that in order to be efficient and effective in protecting public health and the environment, we need to know the pathways of exposure. Only after the exposure pathways have been determined and the fates of the various congeners of PBDE have been determined can we begin to know what and where to monitor.

It is important to note here that the two routes of exposure cited in the Chemical Action Plan and in the comments by outside experts are dietary (primarily fish) and indoor airborne particulate. Given these primary routes of exposure we wonder why it would be important to monitor PBDE content of biosolids, which are generally not found indoors and are barred from use in aquatic environments. We think there are some very valid reasons to explore other facets of the PBDE issue before adding another obligation to an already heavily regulated sector of the environmental services community. We are also disappointed that nobody associated with the Ecology PBDE effort took the time to meet with us to explore options or implications before presenting them in the draft action plan.

The NBMA believes that the recommendation that Ecology monitor biosolids for PBDE content be delayed or dropped until it is determined if biosolids is an important pathway for exposure. We believe Ecology should prioritize its actions to focus first on exposure routes, fate, and toxicity and use this information to design a more cost effective monitoring program. Analytical methods for conducting any survey work conducted by Ecology need to be recognized by appropriate regulatory authorities.

The chemical action plan has recommended a ban on penta, octa, and deca BDE. It appears to us that the evidence presented in justifying this action, particularly in the case of deca BDE, is less than compelling. There may be reasons for a ban that are not related to risk (for example demonstrated safe alternatives are available and easily substitutable). If this is the case we urge Ecology to address this overtly. Separating risk related concerns from precautionary measures strengthens the recommendations by differentiating policy from science.

Thank you for this opportunity to comment on Ecology's chemical action plan for PBDE. If you have any questions on our comments or would like further clarification on our positions, please contact our Regulations Committee Co-chair, Dan Thompson, at (253) 502-2191.

Sincerely

Jim Fleming
President
Northwest Biosolids Management Association

Comments on the Washington State PBDE Chemical Action Plan,
Department of Ecology Publication No. 04-03-045,
Department of Health Publication No. 333-060,
Draft, October 11, 2004

Submitted by John Abbotts and Clark Williams-Derry
Northwest Environment Watch
November 9, 2004

We appreciate the preparation by state agencies of the Draft PBDE (polybrominated diphenyl ethers) Chemical Action Plan, and the opportunity for public comment. Northwest Environment Watch (NEW) is a Seattle-based non-profit research and communication center that covers the Pacific Northwest, including Washington, Oregon, Idaho, and British Columbia. Among other activities, NEW monitors indicators of sustainability through its Cascadia Scorecard. In connection with the Scorecard's pollution indicator, this September NEW reported on the detection of high levels of PBDE flame retardants in the bodies of each of 40 women tested in the Pacific Northwest (report and supplementary data available at *www.northwestwatch.org/toxics*). That report made several recommendations for Northwest jurisdictions, and our comments evaluate the Draft Action Plan in relation to those recommendations.

In summary, our comments address the following points:

1. We commend the Washington state government for its leadership on persistent toxic chemicals.
2. We recommend that the action plan incorporate a full phase out for the Deca-BDE formulation.
3. We commend state agencies for their plans to evaluate the issue of PBDE sources in homes and offices, and develop recommendations for consumers and businesses.
4. We are gratified that the Action Plan considers a biomonitoring option. We recommend more discussion of the rationale for the particular option selected.
5. The PBDE story nationally demonstrates the systemic weaknesses of federal requirements for testing toxic chemicals. We recommend that state agencies address what actions they might take to prepare for future chemical surprises that seem inevitable given the current system.

Each of these points is developed in more detail in the following sections:

1. <u>The state program</u>.

We commend the Washington state government for its leadership on persistent toxic chemicals, including the development of a state program on such chemicals, the Governor's executive order in early 2004 directing that PBDE flame retardants be incorporated into that state program, and for the development of the Draft Chemical Action Plan issued in October. The Draft contains a comprehensive compilation of

information on PBDEs; and in that regard stands as a very useful reference document in its own right.

2. Recommendation on the Deca-BDE formulation.
At the public meeting in Seattle on October 19, we formally submitted for the record the detailed analytical data that supplement the NEW September report (also accessible via *www.northwestwatch.org/toxics*). Those data include the clear detection of deca in 24 of the 40 women tested. Ten of the women carried deca levels above 1 part per billion, with 4.3 parts per billion representing the highest level detected. This latter level of the deca congener alone exceeds the levels of *total* PBDEs typically found in European and Japanese populations. Levels of deca in the most exposed residents of the Pacific Northwest are comparable to those of Swedish electronics dismantlers, who are occupationally exposed to Deca-BDE.

The Draft Action Plan also cites the earlier work of Dr. Arnold Schecter at the University of Texas as another study that detected the deca congener [Draft Action Plan, p. 11]; the maximum level of deca among Dr. Schecter's study population was 8.2 parts per billion. We thus believe the Draft Action Plan reflects the current scientific consensus when it finds that deca has been detected in members in the general population [p. 11], and when it further finds that, "There is a weight of evidence suggesting that highly brominated PBDEs are precursors of the more bioaccumulative and persistent lower brominated PBDEs, as well as PBDFs [polybrominated dibenzofurans]." [p. 34] With this weight of evidence, the state action plan is justified in expressing the policy goal that, "Deca-BDE use should be decreased, not allowed to increase." [p. 58]

The Draft Action Plan recommends that manufacture, distribution, or sale of the new products containing the Penta-BDE and Octa-BDE formulations be prohibited in the near future. The approach to the Deca-BDE formulation is slightly different, recommending that manufacture, distribution, or sale of Deca-BDE be prohibited in designated products, specifically consumer electronics and textiles. [Draft Action Plan, p. v]

We express our concern over the possibility of "deca creep," that is, the potential for Deca-BDE use to increase if it were to replace current uses of Penta- and Octa-BDE as those commercial formulations are phased out. The Draft explicitly recognizes that potential for textiles by designating those products in the Deca-BDE recommendation. It seems preferable to close all such potential loopholes. The September NEW report recommended that Northwestern jurisdictions should ban PBDEs from commerce, including a phase out of the Deca-BDE formulation. We reiterate that suggestion here, and recommend that as a means of achieving the state's policy goals, the Action Plan should incorporate a full phase out of all uses of Deca-BDE, rather than limiting the phase out to specified products.

3. Existing PBDE sources.
The September NEW report also recommended that Northwest jurisdictions should develop strategies and advice to help people remove PBDEs from their homes and workplaces. We recognize that the wholesale replacement of items such as furniture and

electronic devices would be prohibitively expensive for many Washington residents, and could be counter-productive in addition if such activities increase exposures by suspending PBDE-laden dusts.

Nonetheless, as the Draft Action Plan recognizes, "Even if no new PBDE products were produced or sold, merely dealing with existing products will require programs to limit human exposure and prevent the continued release of PBDEs into the environment for decades to come." [p. iv] However, with the current lack of knowledge on pathways for human exposure to PBDEs, the agencies' plans to evaluate this issue and develop recommendations for consumers and businesses seem judicious. We commend the agencies for their approach to this problem.

4. Biomonitoring.
The NEW September report also recommended biomonitoring of blood and breastmilk for PBDEs and other toxic substances. Such programs would serve as early warning systems to catch emerging toxic exposures; they would also provide indicators of success in reducing sources of exposure.

We are gratified that the Draft Action Plan considers biomonitoring of the blood of workers who may be most highly exposed to PBDEs. We also recognize that biomonitoring represents a new responsibility for state agencies, and funding limits may represent obstacles to a wider program. If funding does represent a limit, then efforts to coordinate with federal agencies such as the Centers for Disease Control (CDC), which already conducts biomonitoring, seem logical. Our understanding is that CDC does intend to test for PBDEs sometime in the future, although the Centers do not currently list these chemicals for their next national biomonitoring report (at *www.cdc.gov/exposurereport/pdf/third_report_chemicals.pdf*). In addition, it seems that a current drawback in relying on CDC data is that the Centers do not report results by geographic region. Such a distinction would be necessary before Washington state agencies could use CDC data to gauge the effectiveness of their own regulatory actions.

Unlike other recommendations in the Draft Action Plan, the Monitoring and Research category does not contain a "Rationale" section. We recommend that the Plan include a discussion of the practical obstacles to a wider biomonitoring plan at the state level, along with a discussion of changes to the CDC program necessary to make it more useful for the purposes Washington state.

5. Federal regulatory framework for toxic chemicals.
The chronology of PBDEs illustrates the systemic weaknesses of the current federal regulatory framework for toxic chemicals: federal regulations do not require sensible precautionary measures, including adequate health and safety testing, for industrial chemicals to be used in the marketplace. Although PBDEs are close chemical cousins of PCBs (polychlorinated biphenyls), they remained in commerce after manufacture of PCBs was prohibited in North America in the 1970s. Information on the toxicity of PBDEs and their accumulation in human bodies was provided not by the chemical industry, but by independent scientists in Europe, Japan, and North America. At best, the

chemical industry was "missing in action" with regard to the public release of toxicity information on these chemicals. And even though the U.S. EPA had the regulatory authority to require toxicity testing, the agency did not request such testing on these chemicals. Moreover, regulatory actions on PBDEs were taken first in Europe, before EPA reached a negotiated settlement with the only U.S. manufacturer to phase out production of the Penta- and Octa-BDE formulations.

We reiterate the commendation of state agencies for developing the PBDE Action Plan, but the plan does burden state government with new responsibilities. As long as the manifest deficiencies of the federal system remain in place, the question seems when, not whether, the PBDE story will be replicated in the future with other chemicals. With this reality, it seems useful for the Action Plan to include a "lessons learned" section with regard to the regulatory framework, and address what measures Washington state agencies could establish as an early warning system to prepare for future situations where other toxic chemicals might break into public attention, unanticipated by federal agencies.

Statement of Northwest Environment Watch
Before the Washington State Public Meeting on PBDEs (polybrominated diphenyl ethers)
Seattle, October 19, 2004

Members of the meeting panel, thank you for the opportunity to present a statement. My name is John Abbotts; I am research consultant to Northwest Environment Watch (NEW), a Seattle-based non-profit research and communication center that covers the Pacific Northwest region. At the end of September NEW reported on the detection of high levels of PBDE flame retardants in the bodies of each of 40 women tested in the Pacific Northwest (report available at www.northwestwatch.org/toxics).

We wish to commend the Washington state government for its leadership on persistent toxic chemicals, including the development of a state program on such chemicals, the Governor's executive order directing that PBDE flame retardants be incorporated into that state program, and for the development of the draft chemical action plan presented this evening. We are also gratified that state agencies cited the September NEW report in the draft action plan, including the detection of the deca-PBDE congener in human bodies.

On that topic, we wish to submit for the record the detailed analytical data that supplement the NEW report, including the clear detection of deca in 24 of the 40 women tested. Ten of the women carried deca levels above 1 part per billion, with 4.3 parts per billion representing the highest level detected. This latter level of the deca congener alone exceeds the levels of *total* PBDEs typically found in European and Japanese populations. We believe the draft state action plan reflects the current scientific consensus when it finds that deca has been detected in members in the general population [p. 11], and when it further finds that, "There is a weight of evidence suggesting that highly brominated PBDEs are precursors of the more bioaccumulative and persistent lower brominated PBDEs, as well as PBDFs [polybrominated dibenzofurans]." [p. 34]

With this weight of evidence, the state action plan is justified in expressing the policy goal that, "Deca-BDE use should be decreased, not allowed to increase." [p. 58] On that particular point, we express our concern for the possibility of "Deca creep," that is, the potential for Deca use to increase if it were to replace current uses of Penta and Octa-BDE as those commercial formulations are phased out. As a means of achieving the state's policy goal, we therefore wish to recommend that the action plan incorporate a full phase out of all uses of Deca, rather than limiting the phase out to specified products.

Thank you again for holding this public meeting, and for allowing spoken comments.

Flame Retardants in the Bodies of Pacific Northwest Residents

By Northwest Environment Watch, http://www.northwestwatch.org/toxics

Data Supplement: PBDE levels in the Pacific Northwest

Levels were reported as parts per trillion in milk fat; divide by 1000 to convert to parts per billion.
Levels were determined on a weight wet basis, then converted to a fat basis based on fat% of each sample, and generally reported to three significant digits; consequently there may be small inconsistencies in totals and subtotals.
Labels to the right of reported values: *=less than method detection limit; B=below method quantitation limit; these designations are as provided by the performing laboratory.

	PBDE congeners:																				
Sample	PBDE-32		PBDE28/33	Total Tri-PBDE	PBDE-71		PBDE-47	PBDE-66	Total Tetra-PBDE	PBDE-100	PBDE-99	PBDE-85	Total Penta-PBDE	PBDE-154	PBDE-153	Total Hexa-PBDE	PBDE-183	Total Hepta-PBDE	Deca-PBDE, PBDE-209		Total PBDEs
British Columbia																					
1	7	*	576	583	25		4,920	22	4,960	738	789	5	1,530	50	842	892	91	91	285	*	8,350
2	36		1,360	1,400	123		15,600	108	15,800	1,590	3,120	231	4,940	1,440	1,390	2,840	358	358	1,160		25,300
3	239		2,510	2,750	1,060		78,300	1,030	80,300	13,200	37,700	3,350	54,200	1,270	4,600	5,870	177	177	721		144,000
4	124		1,660	1,790	267		13,400	134	13,800	3,800	8,950	574	13,300	417	1,720	2,140	314	314	402		31,800
5	11		1,320	1,330	34		14,600	30	14,700	1,940	2,890	275	5,110	152	841	993	203	203	441	B	22,800
6	9		257	266	40		2,630	42	2,720	549	810	4	1,360	25	1,070	1,100	895	895	1,460		6,340
7	910		20,000	20,900	771		201,000	848	203,000	37,200	27,600	4,020	68,800	1,790	12,500	14,300	395	395	553		308,000
8	118		1,270	1,390	182		19,500	140	19,800	5,620	4,020	557	10,200	285	4,840	5,130	106	106	484		37,100
9	9	*	1,570	1,570	34		18,100	29	18,100	2,060	4,110	261	6,430	183	850	1,030	248	248	4,240		31,600
10	11	*	364	375	230		8,630	29	* 8,890	3,640	2,470	9	* 6,120	174	33,500	33,700	268	268	639		50,000
Montana																					
11	5	*	931	937	22	*	5,710	16	* 5,750	1,100	1,700	125	2,920	97	1,510	1,610	91	91	262		11,600
12	159		4,560	4,720	380		20,900	19	* 21,300	1,930	2,220	5	* 4,160	157	1,680	1,840	283	283	2,700		34,900
13	645		7,370	8,020	867		98,500	707	100,000	35,500	19,500	2,600	57,500	1,290	24,600	25,900	390	390	273	*	192,000
14	3	*	716	719	134		4,020	14	* 4,170	495	1,030	53	1,580	50	1,340	1,390	114	114	774		8,740
15	206		1,220	1,430	352		18,600	232	19,200	8,970	4,620	408	14,000	360	28,400	28,800	229	229	1,300	B	64,900
16	7	*	1,660	1,670	35	*	13,400	26	* 13,400	3,770	3,270	286	7,320	160	4,630	4,790	147	147	1,570		28,900
17	553		5,710	6,270	19	*	142,000	14	* 142,000	76,500	13,400	4,440	94,300	3,940	72,800	76,700	373	373	1,250		321,000
18	904		17,400	18,300	3,430		164,000	2,880	170,000	21,600	49,200	4,730	75,600	2,010	8,180	10,200	160	160	878		275,000
19	142		2,570	2,710	1,010		66,800	756	68,600	16,100	22,800	1,450	40,400	1,280	34,700	36,000	204	204	1,360		149,000
20	150		2,340	2,490	13	*	26,000	12	* 26,100	6,850	3,520	406	10,800	392	4,740	5,130	167	167	1,620		46,200
Oregon																					
21	5	*	1,240	1,250	235		19,300	135	19,700	5,180	5,220	478	10,900	286	5,250	5,540	278	278	665		37,700
22	1,300		16,800	18,100	856		201,000	936	203,000	27,500	19,800	3,420	50,600	1,540	11,400	12,900	117	117	508	B	285,000
23	1,090		5,380	6,470	2,800		144,000	1,980	149,000	19,300	40,900	3,640	63,900	1,950	19,500	21,400	168	168	412	B	241,000
24	22	*	2,000	2,270	494		27,000	427	27,900	5,080	7,130	625	12,900	271	5,830	2,710	57	57	60	*	47,200
25	34	*	2,489	2,510	105	*	29,600	99	* 29,600	15,400	4,110	22	* 19,500	709	89,300	90,000	120	120	131	B	142,000
26	141		6,970	7,110	496		47,600	263	48,300	16,800	6,310	1,400	24,500	967	20,900	21,900	440	440	270		102,000
27	103		1,780	1,880	354		30,600	298	31,200	5,310	6,570	697	12,600	340	3,590	3,930	290	290	4,260		54,200
28	454		5,520	5,980	30	*	103,000	26	* 103,000	21,000	13,000	2,010	36,000	1,300	16,800	18,100	257	257	662	B	164,000
29	411		4,500	4,910	630		58,600	468	59,700	12,100	6,660	1,030	19,800	687	10,600	11,300	221	221	101	*	96,000
30	78		536	614	151		7,170	12	* 7,340	1,450	1,280	100	2,830	89	3,260	3,350	152	152	193	B	14,500
Washington																					
31	459		6,630	7,090	652		87,800	34	* 88,500	23,200	15,700	2,460	41,400	1,100	18,000	19,100	117	117	76	*	156,000
32	8	*	3,780	3,790	783		73,300	693	74,800	10,300	18,800	1,700	30,800	2,100	5,580	7,680	170	170	286		117,000
33	7	*	884	890	39	*	7,950	38	* 8,030	884	1,840	92	2,810	64	992	1,060	6	* 6	48	*	12,800
34	438		5,460	5,900	1,020		74,500	644	76,200	14,600	14,900	1,030	30,500	713	12,300	13,000	157	157	230		126,000
35	241		1,520	1,760	499		33,600	414	34,500	4,640	12,100	934	17,700	569	1,750	2,320	141	141	161		56,200
36	10	*	641	651	45	*	7,010	44	* 7,100	1,070	1,400	105	2,570	194	* 2,080	2,280	133	133	374		12,700
37	22	*	5,780	5,800	75	*	76,200	71	* 76,400	38,200	14,300	2,190	54,700	2,980	169,000	172,000	251	251	207	B	309,000
38	10	*	745	754	158	B	7,940	29	* 8,130	921	1,720	128	2,770	140	1,120	1,260	511	511	137	*	13,400
39	47	*	1,620	1,670	162	*	33,100	142	* 33,400	3,860	5,490	712	10,100	725	* 2,370	3,100	1,550	1,550	399	B	49,800
40	24	*	749	772	81	*	12,800	71	* 12,900	2,860	4,020	288	7,170	209	* 4,690	4,900	358	358	334		26,100

PEOPLE FOR PUGET SOUND

pugetsound.org

(VIA EMAIL AND FAX)

November 11, 2004

Cheri Peele
Department of Ecology
PO Box 47600
Olympia, WA 98504

Dear Cheri,

On behalf of People For Puget Sound, I am submitting comments on the *Draft Washington State PBDE Chemical Action Plan.* People For Puget Sound is a citizens' organization that works to protect and restore Puget Sound and the Northwest Straits. We focus on protecting water quality, preserving and restoring critical marine habitat, and safeguarding the quality of life of the people and wildlife that make the area their home.

PBDEs are a threat to human health and the environment. They are toxic at very low levels and move up the food chain, ultimately winding up in our bodies. Studies have shown that PBDEs can cause memory and learning impairments, delay sexual development, and affect thyroid hormone levels.

We are especially concerned about the levels of PBDEs found in the Puget Sound region. Recent studies have found PBDEs in Puget Sound orcas, salmon, ospreys, Columbia River fish, and Northwest women's breast milk. The levels found in orca whales are 2-10 times higher than those found in other whales around the world and the levels found in breast milk were 20 to 40 times higher than levels found in Europe and Japan. This evidence that PBDEs are contaminating Washington state's wildlife and citizens should serve as ample warning that we need to take swift and decisive action to eliminate these chemicals from our environment.

The need for swift action on PBDEs is underscored by the fact that we are already dealing with a toxic legacy in Puget Sound created by PCBs, the chemical cousins of PBDEs. Even though PCBs have been banned for 30 years, Puget Sound continues to suffer from their effects. PCB-contaminated sites can be found throughout Puget Sound. PCBs have contaminated the Puget Sound food chain, including salmon, rockfish, and orca whales. Declines in populations of these species are thought to be due in part to contamination from chemicals like PCBs. In fact, PCBs have helped to make Puget Sound's orca whales one of the most contaminated marine mammals in the world. Taxpayers, businesses, and local and state governments have spent millions of dollars cleaning up PCB contamination and millions of dollars more are still needed to complete the cleanup.

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We should heed the lessons learned from PCBs in the Sound and take decisive action now to eliminate PBDEs. Such precautionary action will help ensure that our children will not have to contend with another damaging toxic legacy in Puget Sound 30 years from now.

General Comments

We are extremely pleased that the Departments of Ecology and Health have put forward this visionary plan to phase out PBDEs in Washington. Overall, the PBDE CAP is well-researched, well-documented, and well-written. We appreciate the time and effort that went into crafting the document, especially given the limited timeframe and resources available to Ecology and Health.

The plan contains many recommendations that when implemented will help to protect Puget Sound and the people living in the region, including the recommendations on human health monitoring, occupational exposure, environmental sampling, and state purchasing of products containing penta-BDE (penta) and octa-BDE (octa). However, there are several recommendations that should be strengthened, especially the recommendations on deca-BDE (deca).

Specific Comments on Recommendations

- **Ban The Manufacture, Sale and Use of Products Containing Penta-BDE and Octa-BDE By January 1, 2006.** We support the recommendation to ban the manufacture, sale, and use of products containing penta and octa. However, the timeline for the phaseout should be moved to January 1, 2006. A longer timeline is unnecessary because industry has voluntarily agreed to stop using penta and octa by 2005. An effective date of January 1, 2006, would help ensure industry will comply with the voluntary agreement and also synchronize Washington's ban with the bans recently passed in Maine and Hawaii, both of which are effective January 1, 2006.

- **Ban The Manufacturer, Sale, and Use of All Products Containing Deca-BDE by January 1, 2006**
  The CAP should contain a ban on all products containing Deca-BDE effective January 1, 2006, for the following reasons:

  1. *Deca breaks down into lower brominated forms, including penta and octa.* One of the biggest concerns around deca is its ability to breakdown into lower brominated forms, including the highly toxic forms of penta and octa, when exposed to sunlight or metabolized by fish. The plan calls for phasing out these lower congeners on a more aggressive timeline and it only makes sense that deca be phased out on the same timeline. We would also suggest referring to the breakdown of deca in the discussion on the toxicity of deca on p. 19 of the CAP.

2. *Serious concerns exist that deca is persistent, toxic and bioaccumulates in people and wildlife.* While there is not as much information on the toxicity of deca as other congeners of PBDEs, serious questions remain about the safety of deca itself. For example:
    - Deca has been found in the blood and breast milk of humans, livers of gulls in Polar Regions, and in polar bears.
    - Studies in mice show that exposure over a short amount of time to just small amounts of deca affect brain growth and result in neurological damage.
    - Because the body absorbs smaller amounts of deca than other forms of PBDEs, exposure to deca overtime may result in higher exposure rates and long-term health and environmental threats.
    - The relative toxicity of deca must also be considered in light of the cumulative impacts of deca with other PBTs humans and wildlife are exposed to everyday, including PCBs, mercury, and dioxin.

3. *Deca use in North America is growing.* Deca is the largest source of PBDEs in the United States, making up 85-90% of PBDEs used in North America. It is not just found in electronic products and upholstered fabric, but also drapes, carpets, cables, and throughout the transportation sector. With the phase out of other forms of PBDEs and possible action by the CPSC and EPA regarding newer flammability standards, the use of Deca is expected to rise. By banning deca on a shorter timeframe and for all products, Washington state will can curb this trend and end all uses of this toxic chemical.

4. *A ban would provide needed incentives for business to find suitable alternatives.* In response to the EU ban on deca, electronic manufacturers are switching out of deca. Without a ban in place, manufacturers do not have an incentive to find suitable alternatives.

As we continue to learn more about deca, it is clear that it poses a serious threat to the environment and public health. We should not wait to take action. Precautionary action is needed to remove deca from the environment and our bodies.

- **Strengthen the Recommendation on PBDEs at End-of Life**
  Overall, we support the recommendation to examine current disposal and recycling practices and to determine how PBDE-containing products should be handled at end-of-life. This recommendation should be strengthened, however, by incorporating the following:

1. **Move up the timeline for completing the investigation of what products contain PBDEs and ensuring proper disposal of these products.** The millions of products currently in the stream of commerce that contain PBDEs represent a significant source of PBDEs. Steps must be taken to ensure these products do not contaminate the environment or our bodies. Ecology should complete this work in 2005, instead of 2007.

2. **Require manufacturers to sort products at end-of-life.** To ensure that PBDEs are not recycled back into products, manufacturers must be required to separate PBDE-containing products from products that do not contain PBDEs.
3. **Require manufacturers to report whether their products contain PBDEs.** Manufacturers should be required to report to Ecology whether their products contain PBDEs instead of Ecology and Health researching the issue. One of the largest impediments to ensuring proper disposal of these toxic products is that consumers, recyclers, and government agencies do not know what products contain PBDEs. This makes it impossible for consumers, businesses, and recyclers to properly handle the products at end-of-life. It does not make sense for Ecology or Health to spend scarce resources searching for this information when manufacturers have this information and could easily provide it.
4. **Ban the incineration of products containing PBDEs.** The third bulleted item under 3. of the "End-of-Life" section infers that disposing of PBDEs in waste disposal facilities is a safe option. Incineration is not a safe option. The incineration of PBDEs results in the formation of dioxins and furans, chemicals that are known to cause health problems in humans and wildlife. Solid waste incinerators cannot burn PCBs, the chemical cousins of PCBs. PBDE-containing products may qualify as hazardous waste and hazardous waste cannot be incinerated. Thus, incineration should not be an option for disposal.

- **Require Washington state to give preference in state contracts to products that do not contain deca.** The recommendation on state purchasing for deca should be strengthened to require the Department of General Administration to specify a preference for products that do not contain deca. This is consistent with Section 5 of Governor Locke's Executive Order 04-01, which requires the Department of General Administration to give preference to products that do not contain PBTs when purchasing equipment, supplies, and other products on state contracts.

- **Establish an institute in the Washington State university system to research alternatives to PBDEs and other persistent toxic chemicals.** Because of the failure of US chemicals policy, little is known about the safety of some of the alternatives to PBDEs. While the lack of information around some of the alternatives should not be used as an excuse not to eliminate flame retardants we know are harmful, it is important that Washington investigate potential alternatives and encourage the use of non-toxic substitutes. Establishing an institute in the Washington State university system would ensure substitutes for toxic flame retardants are safe, help encourage business innovation, establish Washington state as a leader in "clean product design and production", and assist businesses in finding alternatives to PBDEs and other persistent toxic chemicals.

- **Develop a more extensive fish monitoring program.** While we support using existing systems for environmental monitoring of PBDEs, it is urgent that Washington state expand its fish tissue monitoring program not only for PBDEs but all persistent toxic chemicals. Expanding fish monitoring will result in better information of the extent of chemical contamination in the environment and help identify problems of toxic contamination early on.

- **Ensure environmental monitoring includes testing of sediments, marine mammals, and other wildlife, and incinerators.**

- **Require labeling of PBDE-containing products.** Especially if the phaseout of deca is on a longer timeline than January 1, 2006, manufacturers should be required to label a product that contains deca. This will allow consumers, retailers, and others to make choices about what products they want to purchase and to know how to properly dispose of the product.

Thank you for the opportunity to submit comments on the plan and for your excellent work. We look forward to working with Ecology and Health as you finalize the plan. Please feel free to contact me at (206) 382-7007 or ivy@pugetsound.org if you have any questions or concerns.

Sincerely,

Ivy Sager-Rosenthal
Policy Associate

Pierce County
Public Works and Utilities

Brian J. Ziegler, P.E.
Director

Environmental Services
9850 64th Street West
University Place, Washington 98467-1078
www.piercecountywa.org/environmental

November 9, 2004

Department of Ecology
Attn: Cheri Peele
PO Box 47600
Olympia, WA 98504

e-mail: chep461@ecy.wa.gov

Dear Ms. Peele:

The Pierce County Department of Public Works and Utilities is pleased to submit these comments on the Draft PBDE Chemical Action Plan (Draft CAP). Some of the recommendations within the Draft CAP will impact the Department in three areas:

1. In the solid waste arena, Pierce County educates the public on proper solid waste recycling and disposal methods, contracts with a private company for municipal solid waste disposal services, and provides household hazardous waste disposal services through multiple contracts.
2. Our wastewater utility operates an award-winning treatment plant and is finalizing plans to produce a beneficial soil amendment from treated biosolids. That facility is designed and under construction.
3. Pierce County Responds is a nationally-recognized program that has resulted in the removal of thousands of nuisance vehicles from public rights-of-way and private properties.

Combined, Pierce County's programs for public education, solid waste disposal, wastewater treatment, biosolids management, and nuisance abatement target known and measurable pathways through which pollution enters the environment and impacts human health.

Research Is the Highest Priority

The Draft CAP appropriately acknowledges the state of the science and research concerning PBDEs. Clearly, we need more research on the human health impacts of PBDEs as well as a better understanding on the most prevalent and controllable pathways by which these chemicals impact human health and the environment. Pierce County strongly encourages the Department of Ecology and the Department of Health to make this research its highest priority in its approach to PBDEs.

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Solid Waste
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Water Programs
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End of Life Controls

A serious shortcoming of the current Draft CAP is that end-of-life controls, rather than research or source control, appear to be seen as the State agencies' highest priority. We come to this conclusion by examining the manner in which the end-of-life control recommendations are placed in the document, the level of detail provided for end-of-life controls, and by the setting of specific deadlines for achieving end-of-life controls. With two exceptions (each being bans on certain types of PBDEs), the end-of-life recommendations are the only recommendations in this report with "due dates." The scientific evidence presented in the Draft CAP supports neither the prioritization, nor such attention on deadlines given to end-of-life control measures. In fact, recommendations on pages 62 (General Population and Occupational Exposure), 63 (Human Health Monitoring), and 64 (Environmental Monitoring and Research) would seem to us to be necessary precursors to setting end-of-life controls.

Focusing specifically on the end-of-life control recommendations (page 57), Pierce County Public Works and Utilities is most concerned about recommendation 3 and the bullet points which follow.

1) The recommendation focuses too narrowly on Chapter 173-303 WAC. Research may determine that Municipal Solid Waste Landfills permitted under Chapter 173-351 WAC, and recycling and transfer facilities permitted under Chapter 173-350 WAC may be suitable to contain PBDEs. If attention remains focused on Chapter 173-303, the end-of-life controls discussed in the bullet list will require the development of parallel waste collection, handling, and disposal programs for consumer products which may contain PBDEs. Chapter 173-303 WAC is not the appropriate mechanism to regulate solid wastes that have traditionally been in the sphere of municipal solid waste.

2) The first bullet lists a goal "to isolate PBDEs and remove them from the waste stream." This is impractical. PBDEs are going to be waste somewhere. If the bullet meant to refer to the "municipal solid waste stream", we believe that much additional research is necessary to determine whether the added costs are worth the benefits when so little is still known about pathways of exposure.

3) The fourth and fifth bullets should reflect the prevailing current wisdom that PBDEs are safely contained in mixed municipal solid waste landfills permitted under Chapter 173-351 WAC. Nothing in the Draft CAP demonstrates otherwise.

We recognize that future scientific research may show that current practices are not working as anticipated. That new research, combined with advanced financial modeling, should be presented to allow the Legislature and other stakeholders to assess potential costs (which would have to be passed to local governments, the public we serve, and industry) versus the environmental and human health benefits.

The bias toward end-of-life controls appears not just in the recommendations, but throughout the document. We compared Page 12 (the results of tests tracing PBDE exposure by infants) and Page 25 (landfill disposal of "auto fluff"). The Draft CAP documents that breast-feeding is a

known pathway by which PBDEs are passed from mother to child. And, the many health benefits of breast-feeding are considered to outweigh a risk that has some scientific credence.[1] On the other hand, even though the Draft CAP is unable to explain the potential pathway connecting landfill disposal to human exposure, the recommended action is much more conditional.[2] This significant bias should be modified in the Final CAP.

Biosolids Issues

Pierce County is equally concerned about the impact the Draft CAP portends for biosolids management. The Pierce County Wastewater Utility is a member of the Northwest Biosolids Management Association and is in complete support of President Jim Flemming's letter to Ecology on their proposal to test sewage sludge (biosolids) for the presence of PBDEs. We echo Mr. Flemming's comment regarding the background information and comments by external experts about how little we know about the fate and transport of PBDEs as well as exposure pathways.

In the Executive Summary of the CAP, it is pointed out that "PBDEs build up in the body at steadily increasing levels because they reside in fatty tissue and are not processed out of the system". If there is no supporting documentation that proves that PBDEs are processed by the body and enter the waste stream, we question the need to test for PBDEs in biosolids. In Ecology's reference to studies in Canada and the United Kingdom regarding the intake of PBDEs through diet, are there any studies that suggest that PBDEs are transmitted to biosolids to verify the need to test biosolids?

We strongly urge Ecology to reconsider the proposed recommendation to test sewage sludge (biosolids) until more compelling evidence is found to suggest that sewage sludge is indeed a transporter of PBDEs.

Pierce County Responds

Pierce County established *Pierce County Responds* in 2001 to combat blight and public nuisances in our community. One of the main programs coordinated by Pierce County Responds is the removal of nuisance vehicles from public and private properties throughout the County. Nuisance vehicles attract other forms of blight, lower property values, harm water quality, provide an attractive nuisance where children can be hurt, foster the growth and spread of rodents and disease-carrying insects and are a known haven for illegal activities. By tackling the problems created by an estimated 25,000 nuisance vehicles in Pierce County, the County is committed to disconnecting a pollution pathway that causes health, environmental, and social harm.

Regulatory changes that impact the disposal of vehicle components will have a major financial impact on the County. A decision which restricts the use of "auto fluff" as daily cover would translate into higher vehicle disposal facility costs and lower scrap prices. Pierce County Public

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[1] While the levels of PBDEs in break milk are a concern and will likely be monitored further by researchers, health agencies including DOH continue to recommend breastfeeding as the best choice for feeding infants. (Page 12)

[2] The environmental impact of this practice is unknown; it is possible that using auto fluff as daily cover is the best waste management practice with regard to PBDEs. (Page 25)

Works and Utilities conservatively estimates that lower scrap prices would result in $70,000 to $100,000 in additional annual costs to Pierce County government. Further, members of the public who have agreed to remove vehicles voluntarily at their own expense, may seek financial assistance from the County if scrap prices don't offset the expense of getting a vehicle to the scrap yard.

Before making recommendations that could harm Pierce County's very successful abatement program, we ask for a full evaluation of PBDE content in "auto fluff" and a full examination of the potential pathways, if any, by which PBDE might reach the greater environment. We encourage the agencies to also explore the very real possibility that changes in automobile disposal practices might result in more nuisance vehicles being legally and illegally stockpiled in our communities.

Next Steps

It is right for all of us to be concerned and act to reduce the prevalence of PBDE and other persistent, bioaccumulative, toxic chemicals. *It is not right, however, to place the burden of change in the wrong location.* End-of-life controls treat the symptoms and not the root problem. End-of-life controls also have a strong potential to direct the economic costs to the wrong audience, i.e., a consumer base that has not been educated in the potential effects of PBDE nor provided marketplace options to choose alternative products.

At the same time, we realize that source control is not the only solution. Even if PBDEs were banned tomorrow, PBDEs could impact human health and the environment for decades to come. Pierce County has well-established environmental programs that educate the public on making environmentally beneficial choices. As the scientific data becomes more solid, we will be happy to work with you to spread the message and educate the public on the economic and environmental impact of their many purchasing choices.

If you have any questions about this letter, please feel free to contact either Steve Wamback or myself at (253) 798-4050.

Sincerely,

Brian J. Ziegler, P.E.
Director

cc: Steve Wamback, Solid Waste Administrator, Public Works & Utilities
Tim Ramsaur, Wastewater Utility Manager, Public Works & Utilities
Robin Ordonez, Supervisor of Engineering, Wastewater Utility
John Sherman, Tacoma-Pierce County Health Department
Cullen Stephenson, Solid Waste & Financial Assurance Program, Department of Ecology

Pierce County Recycling, Composting and Disposal, LLC dba LRI
17925 MERIDIAN STREET EAST • PUYALLUP, WASHINGTON 98375
OFFICE: (253) 847-7555 • FAX: (253) 847-7713 Web-site at www.lrilandfill.com

November 8, 2004

Ms. Cheri Peele
Environmental Assessment Program
Department of Ecology
P O Box 47600
Olympia, WA 98504-7600

Delivered via email to chep461@ecy.wa.gov

RE: Washington State PBDE Chemical Action Plan

Dear Ms. Peele:

Thank you for the opportunity to review and provide comment on the Washington State Draft PBDE Chemical Action Plan. Although, I applaud the Departments of Ecology and Health for identifying actions the state may take to reduce threats posed by PBDEs. I have concerns over several fundamental statements and approaches.

In the Executive Summary, the statement is made that the departments of Ecology and Health recommend a strategy that guides the handling and disposal of existing PBDE products and reduces the manufacture and sale of new PBDE products. As I review the list of participants I find a list of manufactures, retailers, environmental groups, local, state and federal governments representation, however, although many of the strategies focus on " handing and disposal" I do not see a representative from the solid waste industry that is actually responsible for the handling and disposal of these materials. Such a representative could provide valuable insight on this matter.

In Section III under the heading of Products Containing PBDE's at End-of-Life, the statement is made "while pathways for PBDEs from products to the environment is unknown, it is thought that much of the substance is likely released at the time of disposal". In the executive summary it is stated "PBDEs have been detected in everything from food to house dust to indoor air, exactly how people are exposed to PBDEs is an area of ongoing study". It seams to me that we are leading the reader to the conclusion that the pathway to the environment is at the time of disposal even though it was previously stated it is found in house dust and indoor air etc. Is it not as likely that the environment can be receiving PBDEs from this route also?

Under the section entitled Landfills the statement is made "most PBDEs are probably landfilled in Washington. The fate of PBDEs in the landfill environment is unknown." I find it unfortunate

that the LRI landfill is singled out due to the receipt of Auto fluff even though PBDEs are found in so many waste streams (as indicated in the document) and accepted by every landfill.

Section IV PBDEs and the Regulatory Environment states, "Under this criteria (WAC 173-303) many products containing PBDEs would probably be considered dangerous waste at end-of-life". Is this where we really want to end up designating carpet, chairs, foam, and interiors of cars as dangerous waste? Creating a "special waste" designation will be costly for the consumer. Do we want to start manifesting large quantities of consumer household products at the end-of-life? Would this possibly lead to and even bigger problem, the increase of illegal dumping and wide spread contamination rather than controlling the destination in an environment built specifically to control such releases.

Washington State has always been a leader in solid waste handling with high aspirations of reducing waste, reusing and recycling materials. This draft PBDE plan has the potential of sending the State in the opposite direction if we are to work with charities and businesses to "minimize the resale of upholstered furniture" and "remove materials from the recycling stream…" We need to focus on the beginning of life.

Once again thank you for the opportunity to respond to the Draft PBDE Chemical Action Plan. If you have any questions regarding this correspondence I can be reached at 253-927-6710.

Sincerely,

Jody L Snyder
Director of Regulatory Services

Cc: Eddie Westmoreland, Vice President, LRI
Norman LeMay, Treasurer, LRI
Cullen Stevenson, Department of Ecology
John Sherman, Tacoma Pierce County Health Department
Steve Wamback, Pierce County Solid Waste

STATE OF WASHINGTON
PUGET SOUND ACTION TEAM
OFFICE OF THE GOVERNOR
P.O. Box 40900 • Olympia, Washington 98504-0900
(360) 725-5444 • (360) 725-5456

November 9, 2004

Dear Cheri,

Thank you for your hard work in compiling the draft *Washington State PBDE Chemical Action Plan*. The information included provides powerful evidence that flame retardants must be treated as a serious threat to human health, wildlife and the environment in Washington. This plan is an important tool to spur management of PBDEs in the most informed and responsible way.

The Puget Sound Action Team is charged by the legislature with responsibility for defining, coordinating and helping to implement Washington's environmental agenda for Puget Sound. The Action Team works through a partnership structure, including a chair appointed by the governor, directors from 10 state agencies and representatives from tribal, federal and local governments with direct responsibilities and authorities for conservation and restoration of the Puget Sound. This letter and comments are provided in my role as Director of the Puget Sound Action Team staff rather than as chair of the partnership

We have several comments on the draft which are provided below:

- Persistent bioaccumulative toxics are a significant concern in the Puget Sound ecosystem. The reduction in use of these compounds - with the aim of reducing the harm from such toxics - are a stated goal of the Puget Sound Management Plan.

- Accordingly, the Action Team staff supports a ban of Penta-BDE and Octa-BDE in the manufacture, distribution or sale of new products, beginning in July 2006.

- The Action Team staff urges a ban of Deca-BDE, and within in the same timeframe as the ban of Penta-BDE and Octa-BDE. As suggested in the draft Chemical Action Plan (CAP), Deca-BDEs degrade into more bioaccumulative and potentially toxic compounds than in their original state, thus remaining a persistent threat to human and environmental health. According to the research conducted in the Baltic Sea, the largest concentrations of PBDEs measured were the decabrominated congeners. And, by phasing out all three compounds, there may be less confusion to the public as to why some PBDEs are presumed more 'safe' than others.

- The report includes great detail on the major PBDE congeners found in dust, food, biosolids, etc. Would it be possible to include a table (or expand Table 1) to indicate with which PBDE-containing household products specific congeners are associated? It would be helpful when reading the chapters on PBDEs and human health to relate the high

reported levels of specific congeners with likely sources (i.e. electronics, upholstered furniture, carpets, etc.).

- Considering the widespread distribution of PBDEs in the environment – much of it likely through airborne transport - we suggest the State re-examine the practice of permitting "auto fluff" (shredded waste material from scrap cars) in landfills as a "best waste management practice with regards to PBDEs" (pg. 25). Shredding plastics, foams and other non-ferrous materials from cars and then spreading this material over landfilled waste may release a significant amount of PBDEs into the air as well as the landfill leachate.

- Recommendations for environmental monitoring should be expanded to include wastewater, sediment, biosolids and air in addition to tissue sampling from resident and migratory fish (including bottom dwelling, forage fish and salmon), predatory birds and marine mammals.

If you have any questions about the comments provided, please feel free to contact the Sarah Brace (360-725-5464, sbrace@psat.wa.gov) or me (360-725-5437, back@psat.wa.gov). I look forward to viewing the final draft of the report.

Thanks,

Brad Ack
Director

From: Steve Richmond [s_t_e_v_e_r@hotmail.com]
Sent: Friday, November 05, 2004 10:29 AM
To: Peele, Cheri
Subject: Toxic flame retardants (PBDEs)

Toxic flame retardants (PBDEs) are unnacceptable to the people affected,
even if tracing cause and effect is blurred. If the science is solid, act
now, not in a delayed schedule.

If a compromise must be made, consider taxing the source of the problem
(consumer demand), and use that money to pay for best estimates of the
health costs, and for retraining of workers possibly affected, and for bonds
to retool industry toward cleaner alternatives.

Sincerely,

Steven K. Richmond
Co-chair, Seattle Chapter Fellowship of Reconciliation
6502 18th Ave S.W.
Seattle, WA 98106
(206) 650-9807
s_t_e_v_e_r@hotmail.com

November 11, 2004

Ms. Cheri Peele
Environmental Assessment Program
Department of Ecology
PO Box 47600
Olympia, WA 98504

Re: Draft PBDE Chemical Action Plan

Dear Ms. Peele:

Thank you for the opportunity to provide comment on the Draft PBDE Action Plan. This draft, along with related initiatives to address persistent bioaccumulative toxins (PBTs), is an important element in our effort to protect public health and the environment. In general, the Tacoma-Pierce County Health Department (TPCHD) encourages efforts to control the production and distribution of products containing PBDEs. This approach may be difficult, particularly since PBDEs are widely produced and distributed, but efforts in Washington will be assisted by significant national and international momentum.

The TPCHD recommends that the final plan emphasize data collection to further support data-driven policies, supports efforts to require disclosure of PBDEs in products, and applauds efforts to reduce or eliminate the production and use of PBDEs (and other PBTs).

The TPCHD does not endorse the reliance upon end-of-life controls as a means to address PBDEs. In fact, PBDEs appear more prevalent than the so-called 'universal wastes' defined in both federal and state hazardous/dangerous waste rule. End-of-life controls would place a significant burden upon the solid waste and hazardous waste management systems. While end-of-life controls would arguably be the simplest to implement at the state level, the TPCHD urges Ecology to avoid this 'easy' answer.

Finally, the publication of this draft document serves to bring attention to the need for the Department of Ecology to review the utility and application of the state-only dangerous waste designation (Chapter 173-303 WAC). Available data indicate that due to PBDEs many waste streams previously considered 'solid waste' are, in fact, state-only dangerous wastes. This is not a tenable situation for either the regulated community or the regulatory community. More importantly, there seems to be little or no data to suggest that proper disposal into a permitted and properly managed municipal solid waste landfill presents a public health or environmental hazard. Timely review and rule-revision will be necessary well before the 2007 target mentioned in the document.

These points and others are discussed in more detail on the enclosed page. Thank you again for the opportunity to provide comment. Please feel free to contact me at (253) 798-6528 if you have any questions.

Sincerely,

John Sherman
Environmental Health Liaison
Environmental Health Program

Enclosure: TPCHD Comments

X:\PBDE\public comments\Tacoma-Pierce County Health Department #1.doc

1. The Executive Summary for the document states that "Studies in Canada and the United Kingdom suggest that more than 90% of a person's total intake of PBDEs is through diet. PBDEs are believed to migrate from products into the air and dust that is then consumed by insects and moves up the food chain from there" Page 22 of the document states that "While pathways for PBDEs from products to the environment is [sic] unknown, it is thought that much of the substance is likely released at the time of disposal." The document further notes (page 27) that at least one study suggested that indoor air may serve as a significant source of PBDE to outdoor air (and hence to the environment).

These statements can, at some level, be construed as contradictory. Please clarify how these statements relate, and to what degree empirical evidence indicates that PBDE release to the environment occurs at the time or place of disposal.

2. Page 24, Table 7 Please clarify whether or not the values listed include auto-shredder residue (fluff). If not, what would be the impact of including that wastestream? Also, what is the source for this table?

3. Page 25; Landfills Is the LRI Landfill in Pierce County the only municipal solid waste landfill that accepts auto fluff?

4. Page 35, PBDEs and the Regulatory Environment. While the draft document does call out the fact the Chapter 173-303-100 pertains to PBDEs, the level of detail is inadequate. This is not an insignificant issue. Please address this issue in more detail and elaborate upon the implications of this designation upon generators and permitted solid waste recyclers and disposal facilities.

Also, please clarify whether or not any portions of either RCW 70.105D or Chapter 173-340 WAC are applicable.

5. Section VI Policy Recommendations:
a. Page 57, Recommention 3. 'Creating a "special waste" designation...' Please clarify whether or not the existing state-only designation pathway (for halogenated organic compounds; Chapter 173-303 WAC) applies to PBDEs, and, if so, whether or not the common products listed in this document would in fact designate as state-only dangerous waste.

'Special waste', as defined in the current Dangerous Waste Regulation (solid-phase, state-only), would very likely be an appropriate regulatory label for many common wastes containing PBDEs. The TPCHD recognizes that special wastes can be accepted, with certain conditions, at Chapter 173-351 WAC municipal solid waste landfills. However, the waste is still legally a dangerous waste, with all of the attendant management and tracking issues. In addition, many permitted solid waste landfills will be either unable or unwilling to accept 'special waste' due to specific land-use or solid waste permitting restrictions, or due to political factors. The existence of the current 'special waste' clause in Chaper 173-303 WAC would not meaningfully reduce the impact of state-only designation for PBDEs.

b. Do products containing PBDEs (at sufficient concentrations) meet the definition of 'hazardous substance' as defined for the purposes of the Model Toxics Control Act? If so, is assessment of the 'hazardous substances tax' applicable? If so, what are the implications?

c. Please describe, if possible, how the presence of PBDEs in (at least some) electronic wastes impacts the Hazardous Waste and Toxics Reduction Program's policy regarding e-waste management, and e-waste recycling, and disposal facilities.

BREAST CANCER FUND

PREVENTION STARTS HERE.

November 11, 2004

Cheri Peele
Environmental Assessment Program
Washington State Department of Ecology
P.O. Box 47600
Olympia WA 98504-7600
By e-mail: chep461@ecy.wa.gov

Dear Ms. Peele

This letter summarizes the Breast Cancer Fund's testimony delivered by Nancy Evans, Health Science Consultant and 14-year breast cancer survivor, at the Public Hearing on PBDEs on October 19, 2004. The Breast Cancer Fund (BCF) is the only national non-profit solely committed to preventing breast cancer. We work to fulfill that mission by helping to identify and advocate for the elimination of environmental causes of breast cancer. BCF supports the phaseout of *all* PBDEs in Washington state and throughout the nation.

Scientists don't know whether exposure to PBDEs causes breast cancer but they do know that women's bodies, especially their breasts, are increasingly contaminated by these chemicals and are finding their way into breast milk. Levels of PBDEs in American women are 10 to 40 times higher than in European women. Scientists also know that PBDEs impair thyroid and liver function and behave much like PCBs, chemicals that were banned in the 1970s and *are* associated with increased risk of breast cancer.

Other countries are way ahead of the U.S. in protecting public health from PBDE exposure. PBDEs have been banned in Sweden, and some forms of PBDE are scheduled for phaseout in the EU. The U.S. EPA announced an agreement to ban the two most troubling forms of PBDE from the U.S. market. However, the most widely used PBDE in computers and consumer electronics will remain in use unless there is public pressure to ban ALL PBDEs.

The Breast Cancer Fund urges the Washington Department of Ecology to resist industry pressure and adopt a strong phaseout plan for all forms and all uses of PBDEs. We don't have proof that these chemicals cause cancer but we have evidence. We *are* the bodies of evidence.

Sincerely,

Janet Nudelman
Director of Programs and Policy

1388 Sutter Street, Suite 400, San Francisco, CA 94109-5400
phone 415.346.8223 **fax** 415.346.2975 www.breastcancerfund.org

THURSTON COUNTY
WASHINGTON
SINCE 1852

COUNTY COMMISSIONERS
Cathy Wolfe
District One
Diane Oberquell
District Two
Robert N. Macleod
District Three

PUBLIC HEALTH AND
SOCIAL SERVICES DEPARTMENT

Sherri McDonald, RN, MPA
Director
Diana T. Yu, MD, MSPH
Health Officer

November 3, 2004

Department of Ecology
Attn: Cheri Peele
PO Box 47600
Olympia, WA 98504

Dear Ms. Peele,

I am writing today in support of Ecology's phase-out plan for all forms of PBDEs. This phase-out is a forward-thinking action and one that makes us proud to be in the state of Washington.

Numerous studies show that levels of PBDEs are rising rapidly in people and the environment. There is no way to tell people to avoid them. This is a clear case where the chemicals need to be removed at the industry level, and replaced by safer fire retardant materials.

This is of concern to us at the county level because PBDEs are a threat to public health. Like their chemical cousins PCBs, PBDEs persist and build up in the environment and in our bodies. They also are toxic at low levels. Studies on laboratory animals have shown that PBDEs can impair memory and learning, alter behavior, delay sexual development, and disturb thyroid hormone levels, among other toxic effects.

The dust from computers and other appliances has high amounts of Deca-BDE, which I would stress should be included in the phase-out. Deca has been shown to degrade rapidly in the environment to more harmful forms (penta and octa).

There is enough evidence that we need to act now to protect ourselves from a lasting legacy of these chemicals. We support Washington State in taking swift action, along with other states and governments around the world, to address the serious problems caused by PBDEs.

Sincerely,

Art Starry, Director
Environmental Health Division
Thurston County Public Health and Social Services Department

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www.co.thurston.wa.us/health

TOTAL RECLAIM INC
ENVIRONMENTAL SERVICES

November 15, 2004

Ms. Cheri Peele
P.O. Box 47600
Olympia, WA 98504-7600

Re: Comments on Washington State PBDE Chemical Action Plan

Dear Ms. Peele:

I am writing to provide comment on the draft Washington State PBDE Chemical Action Plan on behalf of Total Reclaim, Inc.

I want to commend you for the considerable effort you and your staff put into researching the issues and developing these recommendations. I believe that taken as a whole, the recommendations are well crafted and will help to protect human health and the environment in Washington State.

I have the following comments on the recommendations:

- As a note of clarification, on Page vi, I am incorrectly listed as representing EcoLights under the heading Electronics Recycling. That should be changed to Total Reclaim Inc.

- The recommendations include numerous references to having DOH and L&I measure and monitor occupational exposures to PBDE fire retardants. I agree that there is a need to identify, monitor, and possibly regulate the exposure to these materials. Unfortunately, to my knowledge, and from the pages of this report, there is very little research on the actual health impacts or toxicology of exposure to PBDEs.

  With no data on the risk and possible harm from exposure, how can DOH or L&I develop standards for exposure that have any basis in reality. To say it another way, just because we can measure it, does that mean it represents overexposure and needs to be regulated?

  I believe that prior to developing a monitoring and testing regime, DOH, L&I, or other agencies should provide information on the risk these materials pose to humans and regulatory exposure levels.

P.O. BOX 24996 | T (206) 343-7443
SEATTLE, WA 98124 | F (206) 343-7445 | WWW.TOTALRECLAIM.COM

- During the Task Force meetings it was commented that blood testing for PBDEs is expensive. Where the toxicology of materials is understood, as with lead or mercury, expensive testing may be justified, as it will protect employees from known dangers. In this case, there is no quantitative basis for evaluating occupational risk, and this requirement would likely place a significant burden on companies without a recognizable benefit.

- I support the proposed regulations to remove any and all types of PBDEs from products. At the same time, it should be recognized that if Washington bans the sale of new products containing Penta-BDE, Octa-BDE, or Deca-BDE, this represents a defacto ban on the recycling of products such as carpets, mattresses, computers, TVs, and other electronic equipment that contain these flame retardants. The only recourse will be to dispose of these materials in landfills or incinerators. This is in conflict with the current state policy to encourage recycling of these materials. If it is the intent of Ecology to require disposal of these materials, rather than recycling, this should be explicitly stated.

As I indicated in the meetings, I am very concerned about the widespread environmental contamination by PBDEs and the potential impact of these materials on workers in the electronics recycling industry. I applaud you and others at the Department of Ecology for elevating the understanding of the risks of these materials and beginning the process to control their spread in products and the environment.

If you have further questions or require additional information on the electronics recycling industry or Total Reclaim's services, please contact me at (206) 343-7443.

Thank you for you consideration.

Sincerely,

Craig Lorch
Total Reclaim, Inc

Dear Director Linda Hoffman and Director Mary Selecky:

We are writing today as members of the Toxic-Free Legacy Coalition, which represents tens of thousands of citizens in Washington State who are committed to eliminating persistent toxic chemicals like PBDEs.

Together, our coalition would like to thank the Department of Ecology and Department of Health for your excellent work on the draft Chemical Action Plan for PBDEs and to ask that the plan be strengthened to protect our children, wildlife, and food supply from the harmful effects of these chemicals.

We especially applaud your review of the mounting evidence, indicating that deca-BDE (deca) is a problem for public health and the environment. In particular, we support the following concerns, described in your draft plan:

- the plan points to new studies, which demonstrate that deca does build up in humans and suggest that infants may be exposed to higher levels of deca through dust.
- the plan presents strong scientific evidence that deca does break down into furans and more harmful forms of PBDEs.
- the plan recognizes that deca is used in large quantities and will be the only PBDE still in commercial production by December 2004.

This evidence raises serious concerns regarding deca-BDE, and we believe Washington State needs a stronger plan for deca. In order for us to act responsibly and take swift action to phase out all forms of PBDEs, we urge you to make the following changes to the draft PBDE Chemical Action Plan:

1) *Currently, a ban on deca is recommended only for consumer-based electronics and upholstery.* While this action would address a significant portion of the deca in use, a complete ban is needed to solve the problem of PBDEs in our breast milk, wildlife, and food supply. In addition, a ban on all uses of deca, including uses applicable to transportation, wiring and cables, and all textiles, would provide a necessary driver for innovation that replaces deca with safer alternatives. It would also ensure that the use of deca will not expand into other applications.

   The Departments of Ecology and Health should recommend a ban on deca-BDE for all new products.

2) *Currently, the deca ban is recommended for 2008.* Europe is set to ban deca in electronics by 2006, and many companies are already in compliance. There is no reason for us to wait. Added time only means that more deca will pollute our homes, wildlife, and our bodies. In addition, the solution will take longer and be more expensive.

The Departments of Ecology and Health should recommend a consistent timeline that phases out all PBDE products, including those with deca, by 2006.

3) *Currently, the state purchasing recommendation falls short, because it only addresses purchasing products free of penta and octa-BDE, which will already be phased out of products.* Since the plan states there is "sufficient evidence to warrant concern" about deca, the state has an obligation to end its purchase of all products that contribute to PBDE contamination of office buildings, schools, breast milk, and the environment, including products containing deca.

   The Departments of Ecology and Health should recommend that goods purchased on state contracts not contain penta, octa or deca-BDE.

4) *Currently, there are no labeling requirements for products containing deca-BDE.* Labeling is needed to ensure that consumers can make an informed choice about whether to buy a product containing deca-BDE until the ban is in place. In addition, labeling will allow retailers to know whether they are in compliance with the ban and help facilitate the proper disposal of the product. It will also enable recyclers to ensure that the product is properly managed at the end-of life stage.

   The Departments of Ecology and Health should recommend that deca products be labeled as containing deca-BDE.

Taking these steps towards deca phase-out are necessary to protect public health and the environment and to shift markets toward safer substitutes. This is a first priority for our coalition. We look forward to working with the Departments of Ecology and Health on a strong PBDE phaseout plan in the coming year.

Sincerely,

Pam Tazioli
The Breast Cancer Fund

Matthew Cacho
Healthy Building Network

Ivy Sager-Rosenthal
People for Puget Sound

Nancy Dickeman
Washington Physicians for Social Responsibility

Mo McBroom
Washington Public Interest Research Group (WashPIRG)

Laurie Valeriano
Washington Toxics Coalition

Aileen Gagney
American Lung Association of Washington

Dave Batker
Asian Pacific Environmental Exchange

Jim Puckett
Basel Action Network

Leslie Ann Rose
Citizens for a Healthy Bay

BJ Cummings
Duwamish River Clean-Up Coalition

Will Anderson
Earth Island Institute Orca Recovery Program

Steve Gilbert
Institute for Neurotoxicology and Neurological Disorders

Tim Coleman
Kettle Range Conservation Group

Mike Petersen
The Lands Council

Janet Primomo, PhD, RN
Nursing Program, University of Washington, Tacoma

Jane Harris
Oregon Center for Environmental Health

Wendy Steffensen
RE Sources for Sustainable Communities

Alice Woldt
Seattle Alliance for Good Jobs and Housing for Everyone (SAGE)

Suellen Mele
Washington Citizens for Resource Conservation

Ginny English
Healthy Mothers, Healthy Babies Coalition of Washington

Kim Radtke
Breast Feeding Coalition of Washington

John Boonstra
Washington Association of Churches

Elise Miller
Institute for Children's Environmental Health

Deb Abramson
S.H.A.W.L. Society (Sovereignty, Health, Air, Water, Land)

Michael Lang
Friends of the Columbia Gorge

Clark Williams-Derry
Northwest Environment Watch

Reverend Paul Benz
Lutheran Public Policy Office

Bruce Herbert
Newground Social Investment

Karen Ahern
Coalition for Environmentally Safe Schools

Karen Holt Luetjen
Seattle Tilth

Tanya Marcovna Barnett
Earth Ministry

Phoebe Yin, ND

Lilamrta Logue

WASHINGTON ACADEMY OF FAMILY PHYSICIANS

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RESOLUTION # 18: Phasing Out Flame Retardant

Adopted.

RESOLVED, that the Washington Academy of Family Physicians (WAFP) supports the phase out and elimination of the use of PBDEs (polybrominated diphenyl ethers) in Washington State while maintaining existing fire safety standards

18530 156th Avenue NE, #100, Woodinville WA 98072
Telephone: (425) 486-3530 (800) 621-8424 within Washington
Fax: (425) 486-0169 **Email:** admin@wafp.net

Washington Academy of Family Physicians
Resolution
Submittal: **May 2004 Annual Meeting**

Title: **Phasing Out Polybrominated Diphenyl Ethers (PBDEs) – Toxic Flame Retardants**

Whereas: Polybrominated diphenyl ethers, PBDEs, are linked to serious health effects including memory impairment, and learning and behavioral problems in laboratory animals at very low levels, and they have also been associated with disruption of thyroid hormone balance, non-Hodgkin's lymphoma in humans, and a variety of cancers in rodents; [i] and

Whereas: PBDEs are similar in chemical structure to polychlorinated biphenyls (PCBs), a highly toxic chemical banned in the United States for production in 1977, and for distribution in 1978; [ii] and

Whereas: PBDEs accumulate in the blood and breast milk of nursing mothers, and in the blood of infants;[iii] and

Whereas: Recent scientific studies have documented rapidly rising levels of PBDEs in human breast milk, with levels in Puget Sound women documented at levels from 20 to 40 times higher than their European and Japanese counterparts;[iv] and

Whereas: PBDE contamination is rapidly rising in fish, building up in sediment and other aquatic organisms, and fish consumption constitutes a major exposure pathway for humans[v]; and

Whereas: PDBEs are widely used in upholstered furniture, electronics, automotive interiors, and plastics to slow the spread of fire[vi]; and

Whereas: PBDEs can enter the environment during the production and disposal of materials containing PBDE flame-retardants, as well as during the lifetime of PBDE-containing products[vii]; and

Whereas: PBDEs are not chemically bound to plastics, and can evaporate into indoor air or outdoor environments[viii]; and

Whereas: Alternative measures including use of less hazardous flame retardant chemicals, and use of less flammable materials in manufacturing are available, and have been adopted for use by companies including IKEA, Intel, and others[ix]; and

Whereas: Maine banned the use of all forms of PBDEs in 2004, and California and Europe banned the use of PDBEs in 2003, in these instances citing the safe, cost-effective alternatives to PDBEs for use as flame retardants[x]; and

Whereas: Governor Locke signed an Executive Order in 2004 directing the Washington State Department of Ecology to develop a phase out plan for PBDEs, as part of its larger strategy to phase out persistent bioaccumulative toxic chemicals[xi]; and

Whereas: Phasing out use of potentially toxic chemicals is an essential step in reducing these exposures to infants through lactation, and in reducing these exposures to general populations through environmental, including fish, contamination[xii];

Therefore, Be It

Resolved, that: The Washington Academy of Family Physicians (WAFP) supports the phase out and elimination of the use of PBDEs in Washington State while maintaining existing fire safety standards.

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[i] Darnerud, P.O., *et al.* Polybrominated Diphenyl Ethers: Occurrence, Dietary Exposure, and Toxicology, Environmental Health Perspectives Journal 2001; 109(Supplement 1): p. 49-68 http://ehp.niehs.nih.gov/docs/2001/suppl-1/49-68darnerud/abstract.html

[ii] Hooper, K. and T.A. McDonald. The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs, Environmental Health Perspectives Journal 2000; 108(5): p. 387-392. http://ehp.niehs.nih.gov/docs/2000/108p387-392hooper/abstract.html

[iii] Hooper, K. and T.A. McDonald. The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs, Environmental Health Perspectives Journal 2000; 108(5): p. 387-392. http://ehp.niehs.nih.gov/docs/2000/108p387-392hooper/abstract.html

[iv] Northwest Environmental Watch: Flame Retardants in Puget Sound Residents, First Round of Results from a Study on Toxic Body Burdens; February 2004; p. 2 http://www.northwestwatch.org/pollution/

[v] She, Jianwen, et al. PBDEs in the San Francisco Bay Area: measurements in harbor seal blubber and human breast adipose tissue, Chemosphere 46 (2002) p. 697-707

[vi] Schecter, Arnold, MD, Birnbaum, Linda, et.al. Polybrominated Ethers (PBDEs) in US Mothers' Milk Environmental Health Perspectives Volume 111 Number 14 November 2003, p: 1723-1729 http://ehp.niehs.nih.gov/members/2003/6466/6466.html

[vii] Hooper, K. and T.A. McDonald. The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs, Environmental Health Perspectives Journal 2000; 108(5): p. 387-392. http://ehp.niehs.nih.gov/docs/2000/108p387-392hooper/abstract.html

[viii] Hooper, K. and T.A. McDonald. The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs, Environmental Health Perspectives Journal 2000; 108(5): p. 387-392. http://ehp.niehs.nih.gov/docs/2000/108p387-392hooper/abstract.html

[ix] Belliveau, Mike, Press Release, " Maine Leads Nation with Ban on Toxic Fire Retardants", April 8, 2004

[x] Belliveau, Mike, Press Release, " Maine Leads Nation with Ban on Toxic Fire Retardants", April 8, 2004

[xi] Washington State Department of Ecology, News Release, "Governor Locke Signs Executive Order to Protect the Public from Toxic Chemicals", January 28, 2004 http://www.ecy.wa.gov/../news/2004news/2004-026.html

[xii] Madsen, Travis and et al. Growing Threats: Toxic Flame Retardants and Children's Health, 2003 http://www.environmentcalifornia.org/reports/GrowingThreats03.pdf ; and Schecter, Arnold, MD, Birnbaum, Linda, et.al. Polybrominated Ethers (PBDEs) in US Mothers' Milk Environmental Health Perspectives Volume 111 Number 14 November 2003, p: 1723-1729 http://ehp.niehs.nih.gov/members/2003/6466/6466.html

WASHINGTON ACADEMY OF FAMILY PHYSICIANS

February 19, 2004

Dear Washington State Senators and Representatives:

The Washington Academy of Family Physicians supports full funding for the Washington State Department of Ecology's Persistent Bioaccumulative Toxic (PBT) program in the 2004 Supplemental Operating Budget. As family health practitioners concerned with the health of our citizens statewide, we urge you to restore funding for this critical program.

The Department of Ecology's PBT program takes a crucial step towards improving the health of communities throughout our state. The human and environmental health hazards posed by PBTs -- including mercury, dioxin and lead -- are substantial. These chemicals have been linked to serious health effects including birth defects, learning and behavioral disorders in young children, cancer, reproductive failure, and other serious health problems.

This program, at a cost of $436,000, is also a sound financial investment. These funds can make a positive impact against the healthcare and loss of productivity costs incurred as a result of the harmful effects of persistent pollutants. For example, our nation spends billions of dollars on expenses from environmental contributors to health problems, including lead poisoning, cancer and neurobehavioral disorders, as examined in "Environmental Pollutants and Disease in American Children", in Environmental Health Perspectives.[i] Applying this study's calculations to Washington State results in $925 million spent on the health consequences of neurobehavioral disorders including the losses due to childhood lead exposure. When looking at cancer occurrences alone, our state spent $3.4 billion during 2002[ii]. These costs include direct medical costs, the cost of lost productivity due to illness, and the cost of lost productivity due to premature death. The Washington State Department of Health estimates that some form of cancer will strike one in three Washingtonians in their lifetime. Funding the PBT program is an essential move forward in bringing these costs down, and in protecting public health from toxic pollution.

The PBT program is a small investment that can reap an enormous economic return, while improving the health and lives of Washington citizens. Please make the health of our children, neighbors, and communities a foremost priority, and restore budget funding for the Department of Ecology's PBT program.

Sincerely,

Jean Marshall MD

Jean H. Marshall, MD
President
Washington Academy of Family Physicians

18530 156th Ave NE Suite 100, Woodinville WA 98072

[i] "Environmental Health Perspectives," July 2002, Volume 110, Number 7
"Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities. Philip J. Landigran, Clyde B. Schechter, Jeffrey M. Lipton, Marianne C. Fahs, and Joel Schwartz.
http://ehpnet1.niehs.nih.gov/docs/2002/110p721-728landigran/abstract.html

[ii] Mortality data from "Cancer Facts and Figures, 2002", American Cancer Society. Cost derived from cost and projected incidence data in "Cancer Facts and Figures, 2002" and "Cancer Facts and Figures 2003", American Cancer Society.

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Washington Association of Churches

The Rev. John C. Boonstra, Executive Minister
419 Occidental Avenue South, Suite 201 Seattle, Washington 98104-2886

voice 206.625.9790 fax 206.625.9791 wac@thewac.org www.thewac.org

Cheri Peele
Dept. of Ecology
P.O. Box 47600
Olympia, WA 98504.

RECEIVED
OCT 29 2004

Dear Cheri Peele,

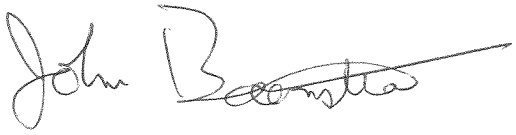
The Washington Association of Churches represents 1,200 member congregations. Protecting God's creation from harmful toxins in wildlife to newborn babies is of high priority to our members. I applaud the Dept. of Ecology and Dept. of Health's Draft Plan to ban PBDE's. I am writing to express concern though regarding the 2008 deadline for banned production of these toxins. **I urge you to push up the date in your final plan to January 1, 2006**. There is no reason to wait when the evidence is so clear and the stakes are so high regarding the health of our environment and children. PBDE's persist in the environment, build up in the food chain and our bodies, and are highly toxic. At very low levels, they have been shown to impair memory and learning in lab animals.

Several studies have found that these toxic flame retardants are ubiquitous and unsafe in our environment. A study by Northwest Environment Watch found toxic flame retardants in breast milk donated by Puget Sound women at levels 20 to 40 times higher than those found in European and Japanese women. And a form that industries once thought to be safe, deca-BDE (deca), has been found in the blood and house dust of U.S. citizens. Deca has been shown to degrade rapidly in the environment to more harmful forms (penta and octa).

There is enough evidence that we need to act now to protect ourselves from a lasting legacy of these chemicals. Governments in the United States and around the world are beginning to take swift action, and Washington State should join this growing list of leaders to address the serious problems caused by PBDEs.

Thank you again for your work in this Draft Plan. Now I urge you make a stronger statement regarding the phase-out of these chemicals.

Thank you,



Rev. John Boonstra

November 11, 2004

Cheri Peele
Department of Ecology
PO Box 47600
Olympia, WA 98504

Dear Ms. Peele:

Thank you for this opportunity to comment on the draft Washington State PBDE Chemical Action Plan. Washington Citizens for Resource Conservation (WCRC) is a non-profit advocacy group working to keep Washington a leader in recycling and waste reduction. For the past few years, WCRC has focused on electronics recycling and extended producer responsibility, and we have become increasingly concerned about the use of brominated flame retardants in electronics and other products.

As a member of the Computer TakeBack Campaign (CTBC), WCRC participated in CTBC and Clean Production Action's analysis of dust samples wiped from computers. Brominated flame retardants, including Deca-BDE, were found in all of the 16 samples including the two from college computer labs in Washington State.

WCRC appreciates the thoughtful approach the Department of Ecology and the Department of Health have taken in drafting the PBDE Plan. We ask that the Plan be further strengthened in order to protect human and environmental health. The following are WCRC's specific comments and recommendations:

1) WCRC strongly supports your recommendation to **ban the manufacture, distribution, or sale of new products containing Penta-BDE and Octa-BDE** in Washington State by 2006.

2) The Departments of Ecology and Health should recommend a **ban on the manufacture, distribution and sale of <u>all</u> new products containing Deca-BDE** in Washington State.

WCRC believes that your current recommendation to ban Deca-BDE in electronics and electrical equipment and upholstered fabric should be expanded to all products. As stated in the draft Plan, "There is a weight of evidence suggesting that Deca-BDE breaks down into more bioaccumulative compounds" (page 60). It is also building up in wildlife, our food, and our bodies. This evidence raises serious concerns regarding Deca-BDE and points to the need for a comprehensive ban.

While WCRC appreciates your consideration of whether safer alternatives to the use of Deca-BDE are available, industry's response to the European Union's Restriction on Hazardous Substances (RoHS) Directive clearly shows that bans drive innovation on alternatives and material substitution. RoHS created the level playing field necessary for companies to research and develop alternatives to PBDEs in electronics. A ban on Deca-BDE in all products in Washington State would help catalyze the research and development of alternatives to Deca-BDE in all uses.

3) The **ban on Deca-BDE should become effective by 2006**. As stated in the Plan, "Each additional year that PBDE products are produced and sold will extend that timetable – and any related costs – by a decade or more" (page iv).

4) As stated above, WCRC believes there should be a comprehensive ban on Deca-BDE in all products. However, if Ecology and DOH decide to limit their recommendation on Deca to electronic and electrical equipment and upholstered fabric, we recommend that **the list of covered electronic and electrical products be the list adopted by the European Union**.

5) WCRC supports the recommendation to establish a process to **examine current disposal and recycling practices and determine reasonable end-of-life procedures that are protective of human health and the environment**. However, we would like to see this process begin immediately and be completed on a quicker timeline. End-of-life issues are extremely important and have not yet been adequately examined. Steps must be taken to ensure that materials containing PBDEs are separated at end-of-life and not put into new products. Information is needed on how best to isolate and contain PBDEs.

6) Products containing PBDEs should not be incinerated. PBDEs are similar to PCBs, which cannot be burned in solid waste incinerators. Burning PBDEs can create dioxins and furans, chemicals that are known to cause health problems in humans and wildlife. Even state-of-the-art incinerators can have equipment failures and temperature control problems. We therefore recommend that the Plan include a recommendation to **ban incineration of products containing PBDEs**.

7) If Deca-BDE is not banned in all uses by 2006, **products containing Deca should be labeled**. Labeling would provide information needed for consumers to make informed choices about purchasing products until a ban is in place. Labeling would also assist electronics recyclers, who face significant challenges in identifying the presence of PBDEs in plastics.

8) The State should lead by example. **General Administration and other state agencies should specify that goods purchased on state contracts not contain any PBDEs, including Deca-BDE**.

9) The monitoring and research section should include a recommendation to **research the fate of PBDEs in solid waste incinerators and other burn facilities**. This should include testing incinerator air emissions and ash for PBDEs as well as brominated dioxins and furans.

10) An earlier list of possible actions included **establishing an institute in the Washington State university system for research and development of "clean" product design and production**. WCRC strongly supports including this recommendation in the Plan. As we move away from throwing products into "graves" and toward "cradle-to-cradle" thinking, the focus will move from wastes to products. Technical and research expertise on product design, including safe alternatives to toxic and persistent chemicals, will be needed.

11) In the section of the draft Plan on end-of-life recommendations (p. 57), the fourth bullet under #3 could be interpreted to mean that solid waste disposal facilities safely contain PBDEs. However the draft Plan states that, "Under WAC . . . most products containing PBDEs would probably be considered hazardous waste at end-of-life" and "It is unknown whether the current system for disposing of and recycling products containing PBDEs adequately protects human health and the environment" (page 56). The purpose of recommendation #3 is to evaluate and recommend end-of-life management that protects human health and the environment. We therefore recommend changing the fourth bullet to read: "Allowing the disposal of products containing PBDEs in waste disposal if and where it is determined that they will be safely contained."

12) The draft Plan states, "Results for the latter three watersheds probably represent background for PBDEs in local freshwater fish" (page 29, second paragraph). Since PBDEs do not exist naturally, what is meant by "background" in this statement?

Thank you for this opportunity to comment and for your good work on the Plan. Please feel free to contact me if you need any clarifications.

Suellen Mele,
WCRC Program Director

Washington Citizens for Resource Conservation
2021 Third Ave.
Seattle, WA 98121
www.wastenotwashington.org
206-441-1790

November 9, 2004

Cheri Peele
Washington State Department of Ecology
PO Box 47600
Olympia, WA 98504

RE: Comments to the Draft PBDE Chemical Action Plan

Dear Ms. Peele:

Washington Physicians for Social Responsibility commends the Washington State Departments of Ecology and Health for your thoughtful and thorough work on the PBDE chemical action plan. Taking action to phase out polybrominated diphenyl ethers is a crucial step in protecting children from this preventable source of harm. As members of the health community throughout Washington State, we urge you to make children's health your top priority and finalize a PBDE chemical action plan that phases out all forms of PBDEs.

PBDEs, which share chemical properties with banned PCBs, are ubiquitous in our environment, and enter our food, homes, our bodies, our children, and mothers' breast milk. Studies have linked these chemicals to serious health effects including memory impairment, and learning and behavioral problems in laboratory animals at very low levels. They have also been associated with disruption of thyroid hormone balance, non-Hodgkin's lymphoma in humans, and a variety of cancers in rodents. These chemicals have been put into commerce, products, the environment and our bodies without proof of their safety, and pose a burden our children should not be required to bear.

WPSR supports the recommendations in the draft PBDE chemical action plan. We strongly support the plan's ban on the import and use of Penta-PBDE and Octa-PBDE in Washington State. As noted in the plan, this is necessary for insuring that manufacturers do not reintroduce these chemicals, which are currently undergoing voluntary phase out. We also support the plan's recommendation to convey messages that breastfeeding remains the best feeding option for infants.

Our requests to expand and strengthen the recommendations in the plan are noted in the points provided below:

Deca-PBDE Elimination

- **It is vital that the draft plan's inclusion of deca-PBDE phase out in electronics and upholstery products be maintained in the final plan, with expansion to include all applications of deca-PBDE.** Due to deca's breakdown properties, this is necessary in order to reduce and eventually eliminate PBDEs

from our environment, and from human exposures and risks of harm to human health.

Another area for strengthening deca-PBDE phase out is in the plan's state purchasing guidelines. Please amend this portion of the plan to recommend that products for state purchase do not contain penta, octa or deca-PBDE.

Timeline for Phase Out

While we recognize the need for appropriate time allowances to provide industry with adequate lead time, we support a shorter timeframe than the 2008 timeline provided in the draft plan. This would allow another four years for PBDEs to enter the environment, and exposes our most vulnerable populations – infants and young children -- to these chemicals for an unnecessarily lengthy duration. **With alternative products and processes already in use, we support a 2006 timeline for phase out.**

Labeling: Right-to-Know

We request that you add labeling requirements for products containing deca-PBDE while these products remain on the market until the ban takes place. This provides consumers with essential information they need to make an informed choice about products they bring into their homes and offices.

Human Health Monitoring

We strongly support your recommendations for human health monitoring and particularly recommend that regional monitoring not only be explored, but is needed, and recommend that Washington DOH undertake state biomonitoring. Biomonitoring provides vital information for assessing exposure levels regionally, and as noted in the plan, for identifying at-risk populations. Given this, we also recommend consideration of methods to monitor that would yield the best results for at-risk populations

Removing PBDEs from manufacture and use, and from our environment, is essential for making Washington State a place in which our children can learn, play and grow without risk from these toxic exposures. With mounting scientific evidence of these chemicals' harmful effects, we have both the ability and the responsibility to take action now to protect public health. **Please finalize a strong PBDE chemical action plan which includes your current recommendations as well as the additional requests provided in this comment letter.**

Thank you very much for your time, work and commitment to human health and the environment.

Sincerely,

Steven G. Gilbert, PhD, DABT
Board Member and Co-Chair Environment & Human Health Committee, WPSR

Margaret Kitchell, MD
Co-Chair, Environment & Human Health Committee, WPSR

Charles E. Weems, MD
Vice President, WPSR

Richard Grady, MD
Laura Hart, MD
Sally Goodwin, MD
Mary Ann O'Hara, MD, MPH
John Roberts
Nancy Dickeman, MA
Toxics Coordinator, WPSR

cc: Mary Selecky, Secretary of Health, Washington State Department of Health
Linda Hoffman, Director, Washington State Department of Ecology
Janice Adair, Assistant Secretary, Environmental Health, DOH
Robert Duff, Director, Office of Environmental Health Assessments, DOH

From: Duff, Robert
Sent: Friday, October 29, 2004 5:47 PM
To: Sorlie, Greg
Cc: Adair, Janice; Dunning, Mike (DOH); Sturdevant, Ted (ECY); Peele, Cheri LaFlamme, Denise; Prado, Joanne
Subject: FW: Touching Base
Nice work Greg!!!!!!

Robert Duff
Director
Office of Environmental Health Assessments
Washington State Department of Health
P.O. Box 47846
Olympia, WA 98504-7846
Phone: 360-236-3181
Fax: 360-236-2251
E-mail: robert.duff@doh.wa.gov
http://www.doh.wa.gov/ehp/oehas/default.htm

-----Original Message-----
From: Anjela.Foster@wsp.wa.gov [mailto:Anjela.Foster@wsp.wa.gov]
Sent: Friday, October 29, 2004 5:34 PM
To: gsor461@ECY.WA.GOV
Cc: Robert.Duff@DOH.WA.GOV; Ron.Shultz@OFM.WA.GOV; CHEP461@ECY.WA.GOV; Samuel.Pierre@wsp.wa.gov; Ellen.Tombleson@wsp.wa.gov; Fred.Fakkema@wsp.wa.gov; Dawn.Wilkes@wsp.wa.gov
Subject: RE: Touching Base

Greg,
Our new State Fire Marshal, Samuel Pierre, is currently out of state until next week. However I have solicited feedback from the fire service and are supportive of the recommendation that you outline in your report and supporting documentation. We believe there will be alternative flame retardant materials that are currently in development and beginning to enter the market place and look forward to continued research and development of these materials. We also recognize the associated health and environmental hazards that exist in the current materials being offered and would like to support a move towards decreasing these risks and hazards.

Based on this information there is no opposition from the Office of the State Fire Marshal in terms of your report - we strive for the same purpose of protecting lives and the environment and support the direction Governor Locke is providing towards reducing the use of these specific flame retardant chemicals.

Upon Samuel's return I will request that his secretary contact you and schedule a meeting to further discuss this issue. I have also forwarded this information to our Agency Government and Media Relations Office.

Please contact me if I can be of further assistance.

Sincerely,
Anjela Foster

Washington State Patrol - Office of the State Fire Marshal
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-----Original Message-----
From: Sorlie, Greg [mailto:gsor461@ECY.WA.GOV]
Sent: Friday, October 29, 2004 4:20 PM
To: Foster, Anjela (WSP)
Cc: Duff, Robert; Shultz, Ron; Peele, Cheri
Subject: Touching Base

Hello Anjela

Thanks again for the opportunity to meet with you about the PBDE Chemical Action Plan. I understand there is a new permanent State Fire Marshall now. We think it would be a good idea to have a brief meeting with him to help get him up to speed on the issues. The comment period for the plan ends November 11 and then we will be developing the final plan for the Advisory Committee meeting December 2nd. As you know our common goal is to not lower fire safety standards or adversely impact human safety in any way, so it would be important to emphasize this point in the plan. Is there a good time to met with the new person and can you tell me whom I might call? It would be helpful if you were present as well. Thanks.

Greg Sorlie
Special Assistant for Regulatory Improvement
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Washington State Public Health Association
Resolution
Submittal: **October 2004 Annual Meeting**

Title: **Protecting Public Health by Phasing Out Polybrominated Diphenyl Ethers (PBDEs) – Toxic Flame Retardants**

Whereas: Polybrominated diphenyl ethers (PBDEs) accumulate in the food chain, and in the blood and breast milk of nursing mothers, and in the blood of infants;[i] and

Whereas: PBDEs are linked to serious health effects including memory impairment, and learning and behavioral problems in laboratory animals at very low levels, and they have also been associated with disruption of thyroid hormone balance, non-Hodgkin's lymphoma in humans, and a variety of cancers in rodents; [ii] and

Whereas: PBDEs are similar in chemical structure to polychlorinated biphenyls (PCBs), a highly toxic chemical banned in the United States for production in 1977, and for distribution in 1978; [iii] and

Whereas: Recent scientific studies have documented rapidly rising levels of PBDEs in human breast milk, with levels in Puget Sound women documented at levels from 20 to 40 times higher than their European and Japanese counterparts;[iv] and

Whereas: PBDE levels are rising in fish, building up in sediment and other aquatic organisms, and fish consumption constitutes a major exposure pathway for humans[v]; and

Whereas: Tests for PBDEs in household dust in US homes, including Washington State samples, have revealed high levels of PBDEs in homes[vi].

Whereas: PBDEs can enter the environment during the production and disposal of materials containing PBDE flame-retardants, as well as during the lifetime of PBDE-containing products[vii]; and

Whereas: Since PBDEs are not chemically bound to plastics, they can evaporate into indoor air or outdoor environments[viii]; and

Whereas: PBDEs are widely used in upholstered furniture, electronics, automotive interiors, and plastics to slow the spread of fire[ix]; and

Whereas: Alternative measures including use of less hazardous flame retardant chemicals, and use of less flammable materials in manufacturing are available, and have been adopted for use by companies including IKEA, Intel, and others;[x] and

Whereas: Maine and Hawaii banned the use of PBDEs in 2004, and California and Europe banned the use of PBDEs in 2003, in these instances citing the safe, cost-effective alternatives to PBDEs for use as flame retardants[xi]; and

Whereas: Governor Locke signed an Executive Order in 2004 directing the Washington State Department of Ecology to develop a phase out plan for PBDEs, as part of its larger strategy to phase out persistent bioaccumulative toxic chemicals[xii]; and

Whereas: Phasing out use of potentially toxic chemicals is an essential step in reducing these exposures to infants through lactation, and in reducing these exposures to general populations through environmental, including fish, contamination[xiii];

Therefore, Be It

Resolved, that: The Washington State Public Health Association (WSPHA) supports the phase out and elimination of the use of PBDEs in Washington State while maintaining existing fire safety standards; and be it further

Resolved, that: The WSPHA supports monitoring of PBDE levels in the people and environment of Washington State; and be it further

Resolved, that: The WSPHA encourages further development of safe, cost-effective alternative products to PBDEs; and be it further

Resolved, that: The WSPHA reiterates its commitment to promotion of breast milk as the 'healthiest choice' for babies while supporting this reduction of toxicants to human breast milk.

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[i] Hooper, K. and T.A. McDonald. The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs, Environmental Health Perspectives Journal 2000; 108(5): p. 387-392. http://ehp.niehs.nih.gov/docs/2000/108p387-392hooper/abstract.html

[ii] Darnerud, P.O., *et al.* Polybrominated Diphenyl Ethers: Occurrence, Dietary Exposure, and Toxicology, Environmental Health Perspectives Journal 2001; 109(Supplement 1): p. 49-68 http://ehp.niehs.nih.gov/docs/2001/suppl-1/49-68darnerud/abstract.html

[iii] Hooper, K. and T.A. McDonald. The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs, Environmental Health Perspectives Journal 2000; 108(5): p. 387-392. http://ehp.niehs.nih.gov/docs/2000/108p387-392hooper/abstract.html

[iv] Northwest Environmental Watch: Flame Retardants in Puget Sound Residents, First Round of Results from a Study on Toxic Body Burdens; February 2004: p. 2 http://www.northwestwatch.org/pollution/

[v] She, Jianwen, et al. PBDEs in the San Francisco Bay Area: measurements in harbor seal blubber and human breast adipose tissue, Chemosphere 46 (2002) p. 697-707

[vi] Environmental Working Group: In the Dust: Toxic Fire Retardants in American Homes; p. 2 http://www.ewg.org/reports/inthedust/part1.php

[vii] Hooper, K. and T.A. McDonald. The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs, Environmental Health Perspectives Journal 2000; 108(5): p. 387-392. http://ehp.niehs.nih.gov/docs/2000/108p387-392hooper/abstract.html

[viii] Schecter, Arnold, MD, Birnbaum, Linda, et.al. Polybrominated Ethers (PBDEs) in US Mothers' Milk Environmental Health Perspectives Volume 111 Number 14 November 2003, p: 1723-1729 http://ehp.niehs.nih.gov/members/2003/6466/6466.html

[ix] Belliveau, Mike, Press Release, " Maine Leads Nation with Ban on Toxic Fire Retardants", April 8, 2004

[x] Belliveau, Mike, Press Release, " Maine Leads Nation with Ban on Toxic Fire Retardants", April 8, 2004

[xi] Washington State Department of Ecology, News Release, "Governor Locke Signs Executive Order to Protect the Public from Toxic Chemicals", January 28, 2004 http://www.ecy.wa.gov/../news/2004news/2004-026.html

[xii] Washington State Department of Ecology, News Release, "Governor Locke Signs Executive Order to Protect the Public from Toxic Chemicals", January 28, 2004 http://www.ecy.wa.gov/../news/2004news/2004-026.html

[xiii] Madsen, Travis and et al. Growing Threats: Toxic Flame Retardants and Children's Health, 2003 http://www.environmentcalifornia.org/reports/GrowingThreats03.pdf ; and Schecter, Arnold, MD, Birnbaum, Linda, et.al. Polybrominated Ethers (PBDEs) in US Mothers' Milk Environmental Health Perspectives Volume 111 Number 14 November 2003, p: 1723-1729 http://ehp.niehs.nih.gov/members/2003/6466/6466.html

November 11, 2004

Dear Cheri:

We appreciate the opportunity to comment on the draft PBDE phaseout plan and would like to thank you for all your work to put together a plan that will begin to address the serious health threats posed by PBDEs. The plan is a good start, but there are some key places that the plan should be strengthened to better protect our children's health and keep all PBDEs out of our food supply, wildlife, and our bodies. Specifically, the recommendations in the draft plan should be strengthened to include:

- A ban on the manufacture and sale of products containing **all** forms of PBDEs, including penta-BDE, octa-BDE, and deca-BDE by January 1, 2006.
- A requirement for labeling products that contain halogenated flame-retardants. If deca-BDE is not banned in all uses by 2006, deca-BDE containing products should be labeled.
- A shorter time-line (early 2006) for determining a plan for disposal and recycling of PBDE-containing products.
- A requirement for state agencies to purchase PBDE-free products—not just products free of penta-BDE and octa-BDE—including computers, electronics, and carpets.
- The creation of an institute whose purpose it is to research alternatives to PBDEs and other persistent toxic chemicals (PBTs) that will help Washington companies make PBDE-free and PBT-free products that are more competitive in the global marketplace.

General Comments

Overall, the plan makes an excellent case from a scientific and policy perspective that a phaseout of all forms of PBDEs, including penta-BDE, octa-BDE, and deca-BDE is necessary and urgent.

Levels of PBDEs are rising in our breast milk, wildlife, and food supply. The examples are staggering:

- In the Northwest, a recent study of 40 women found levels of PBDEs in breast milk that were 20-40 times higher than Europe or Japan.

- In the Columbia River system, levels of PBDEs in fish (whitefish) doubled in a mere 1.6 years. A recent scientific study found levels of PBDEs in Puget Sound orca whales that were 2-10 times higher than levels found in other whales around the world. High levels of PBDEs have also been documented in studies of salmon, peregrine falcons, terns, osprey, bald eagle (eggs) and other wildlife.

- ❖ PBDEs contaminate everyday foods bought at the supermarket, including certain meat, dairy, and fish products. Levels found in U.S. food are higher than levels found in Spain and Japan.

- ❖ PBDEs are also found widely in house dust and indoor air. The Environmental Working Group (EWG) study on dust discussed in the plan found average level of PBDEs in dust from nine homes was more than 4,600 parts per billion (ppb), well above the average level found in other studies. Studies have also found higher levels in indoor air compared to outdoor air. Since we spend much of our time indoors, this is particularly important.

What is particularly disturbing is that PBDEs are used in scores of consumer products that are difficult if not impossible to avoid in homes, schools, or workplaces. PBDEs are in mattresses, couches, chairs, computers, televisions, carpets, building materials, and many other products. Some of these products may even be considered hazardous waste due to the significant amount of PBDEs they contain. In fact, Washington State Dangerous Waste Regulations (WAC 173-303-100) say that certain solid wastes must be treated as hazardous waste if they contain .01% or more halogenated organic compounds (HOCs). Because PBDEs are considered HOCs, many products that are in our homes, schools, and workplaces would probably be considered dangerous waste when they are discarded as products can contain upwards of 30% PBDEs by weight.

Due to the mounting scientific evidence, pervasiveness of these chemicals, and the widespread and heavy use in consumer products, it is reasonable to treat all PBDEs as a family of compounds that must be phased out. While two of the three forms (penta-BDE and octa-BDE) will be phased out by the end of 2004, the third most heavily used form, deca-BDE, will continue to be used in the United States for consumer products, although Europe has taken action to ban deca-BDE in consumer electronics by 2006. EPA has not taken specific action to phase out deca-BDE, which leaves it up to the states to protect the public health and the environment from rising levels of these persistent toxic chemicals.

The draft PBDE plan rightly states that there is sufficient evidence to warrant concern over deca-BDE and it provides a strong case for a deca-BDE phaseout. Some of the most compelling reasons for a deca-BDE (deca) phaseout, some of which were documented in the plan, include:

1. Deca is used in massive quantities. An estimated 49 million pounds were used in the U.S. in 2001 and this use is expected to grow by 2% a year. Approximately 500 million pounds of deca is already in consumer products that are in our homes, offices, schools, or landfills (Hooper, BFR 2004). **The amount already in our environment should be included in the report.**

2. Deca breaks down into more toxic and bioaccumulative compounds. Studies show that deca breaks down into lower brominated forms when exposed to sunlight and when

metabolized by fish and other biota. These forms are found in people, wildlife, and food. This is not a new fact, but old facts are being supported by new science. The PBDE plan should clearly state this and specifically cite this paragraph from a study done in 1973 by Dow Chemical Company (Norris, 1973):

> *"The stepwise photoreduction of DBDPO (deca) and OBBP (octa) in xylene leads to the formation of lower brominated diphenyl oxides and biphenyl oxides and biphenyls which may be more stable to UV light than the parent compounds and cause toxicological and environmental problems in their own right."*

The plan should include more information on the biological transformation of deca including: humans (Jakobsson 2003); rats (Morck 2003); anerobic sludge (Gerecke 2004); and, Detroit River fish (Letcher 2003)

As the plan points out, there is also evidence that deca can form brominated dioxins and furans in some natural processes and when burned. Also, while we have identified some of the compounds that deca breaks down into and have found them in the food chain, wildlife, and people, we have not identified all of them and characterized their persistence, bioaccumulation, and toxicity. This is also cause for great concern and a reason to end the use of deca.

3. Deca has been found in people, food and wildlife. Deca is found in the breast milk and blood of people in the United States and Europe. This is important since the chemical industry had long held that deca is not bioavailable in humans or wildlife. The PBDE plan should more clearly state that this is being proven false by new scientific evidence and that the levels are quite high in some cases. For example:

- In 2004, in a bio-monitoring survey, World Wildlife Fund found that members of the public can be exposed to levels of deca much higher than expected. The study found median levels 10 times higher than previous studies had found in occupationally exposed individuals.
- In the Northwest Environment Watch breast milk study deca was detected in 24 of the 40 women and the highest levels found were comparable to those found in Swedish electronics workers. Other breast milk studies (Schecter 2003; Lunder 2003; She 2004) show that 25-85% of the women have deca contamination.

The food supply is also contaminated with deca. This should be further described in the plan (p.11), as this may be an important pathway for human exposure to deca. In Schecter, et al 2004, deca was found in products ranging from meat and cheese to soy formula. The plan should report the findings of this study, particularly since deca was the dominant form of PBDE found in calf liver, soy instant formula, cheese, and margarine. Also, forms that deca is known to break down into (BDE-153 and BDE-154) were found present in several food samples in ratios that differ somewhat from ratios found in the commercial mixtures.

Deca is now being found in wildlife, including peregrine falcon eggs (Sweden), grey

heron (U.K.), harbor seals (North Sea) and fish (bream, pike). It is even being dispersed to far reaches of the Arctic and a remote Baltic island. Studies of gulls in polar regions have shown extensive deca contamination in livers, plasma and eggs. Also, recent findings show that polar bears are contaminated with deca.

It would be very helpful if a chart were included in the human health section of the report showing the levels of deca found in the food chain and people.

4. Deca is found at high levels in the environment. Numerous studies show that deca is the dominant congener in sediments, contributing the largest percentage to overall PBDE levels (Sellstrom, 1999; Sawal, et al, BFR 2004; Khan, et al, BFR 2004; Song, W., et al., 2004, Environmental Science & Technology, Vol 38, Issue 12, pp3286-3293). This should be reflected in the plan. Given that deca is the predominant congener in sediment and it has the ability to break down into more bioaccumulative forms there is great concern that over time these forms could be found in fish and wildlife (and people) in increasing concentrations.

Deca is also found in sewage sludge, which as the plan points out is used extensively in Washington to fertilize food crops, forests, and other things. This may be an important route for PBDEs getting into the environment and food supply. A calculation should be included in the report (based on Hale's studies) on the potential quantity of PBDEs being land applied as fertilizer or incinerated. We are particularly concerned with sludge that may be applied to grazing land because, for example, a large portion of a cows diet is dirt. This means that PBDEs that are deposited onto soils through sludge application (or air deposition) could be directly ingested.

5. **Deca is often the most predominant form found in dust.** A recent study conducted by Clean Production Action on levels of toxic flame-retardants in computer dust found deca in every sample. This study should be included in the environment section of the plan. The Environmental Working Group (EWG) study on dust discussed in the plan found average level of PBDEs in dust from nine homes was more than 4,600 parts per billion (ppb). It should be noted that this level is well above the average in any previous U.S. dust study. In half of the homes EWG sampled, the predominant PBDE present was the type found only in deca.

6. Toxicity concerns of deca also continue to grow. In 2003, Viberg showed irreversible motor and behavioral effects in mice that worsen with age when exposed to deca during brain growth spurt. These were the same effects caused by banned PCBs and PBDEs being phased out. The deca toxicity section should also include information about the toxicity of the degradation products (brominated dioxins/furans, penta, and octa) as well as the fact that other forms and other chemicals (PCBs, mercury) found in the environment and our bodies also have neurotoxic effects. We would like to see a more thorough explanation of deca toxicity in the final plan. We would also like some of the qualifiers removed. For example, on the Viberg deca study (p.19) the statement that the

study was criticized should be removed unless the plan presents an analysis of whether and why the criticisms are valid.

All of these facts are enough to take precautionary measures now to keep deca out of products, the environment, and our bodies.

Alternatives

It is very important that fire safety standards are met and that our families, friends, and loved ones are protected. We are not suggesting that product be less safe in order to protect the environment and our health. We do however want companies to get a clear, strong, message from this plan on PBDEs and other persistent toxic chemicals. The message is that it is unacceptable to use toxic chemicals that build up in our wildlife, food supply, bodies and breast milk.

We understand that some companies may exchange one bad chemical for another. This plan should send a clear signal that Washington and other states and countries have policies to eliminate persistent toxic chemicals so it is a poor economic choice to substitute with another persistent toxic chemicals. Because of failures of chemical policy at the federal level, the public cannot be assured that new flame-retardants will be safe. But through this PBDE plan our state can send a strong signal to the marketplace about what is acceptable in Washington. This includes the following:

- Redesign products to reduce or avoid chemical flame-retardants.
- Use naturally flame-resistant materials such as wool and leather, plastics containing sulfur, preceramic polymers, and, aramide blends (like Kevlar).
- Use safer flame-retardant chemicals that are not persistent, bioaccumulative and toxic such as: aluminum trihydroxide; ammonium polyphosphate; and red phosphorus.

Many companies have begun to move in this direction, particularly in light of new more stringent European regulations on chemicals. We should send the same signals to our companies in Washington so that they can be more competitive in the global marketplace.

It would also be good to include criteria, consistent with the PBT strategy, PBT rule and Beyond Waste plan that companies can use to avoid certain products. These criteria would include:

• Accumulation potential in environmental media (occurrences in humans, wildlife, and environment);
• Persistence;
• Toxicity: carcinogenicity, neurotoxicity, endocrine disruption, mutagenicity, reproductive toxicity; acute toxicity; aquatic or eco-toxicity; and,
• PBT potential of breakdown products

Specific Comments on Plan

We urge the Department of Ecology to strengthen the plan for eliminating PBDE pollution in Washington. We ask that the plan be revised to include the following recommendations and actions:

1) **Bans on penta-BDE and octa-BDE.** The draft recommendation is that the Washington State Legislature should ban the sale of new products containing penta-BDE and octa-BDE in Washington State by July 2006. We support this recommendation, but ask that the date be changed to January 2006 to ensure that products containing penta-BDE and octa-BDE are not imported for sale from other countries into Washington. This is consistent with other states including Maine, California, New York, and Hawaii and is a necessary step in ensuring that the contamination from these chemicals ends.

2) **Ban on deca-BDE (deca) in products manufactured or sold in Washington State.** The draft recommendation is for the legislature to ban deca in electronics and certain upholstery fabric by July 2008. We ask that this recommendation be strengthened to ban the manufacture and sale of all new products containing deca by January 2006. While we support a ban on these uses, it does not go far enough. Specifically,

   - ❖ <u>The timeline is too long.</u> The largest electronics manufacturers are already poised to meet the European Restriction on Hazardous Substance (RoHS) ban on deca in electronics that goes into effect in 2006. Washington would merely be playing catch up with what is already happening in the marketplace. Washington should level the playing field for all manufacturers and ban deca in electronics by 2006.

     For textiles, the new flammability regulations that the Consumer Product Safety Commission is working on that could result in the increased use of deca will most likely be in effect sooner than 2008. It makes sense to put this ban in place as soon as possible since companies haven't yet chosen deca and there are alternative flame-retardants and materials that they could choose that will meet the standard. Putting a ban in place now, will prevent companies from investing in deca now and having to switch down the road.

   - ❖ <u>Without a ban on deca in all products, there is no driver in place for manufacturers to find alternatives to deca in other applications.</u> The lesson learned from Europe is that the deca ban drove innovation on alternatives and material substitution in electronics (plastic to other more inherently flame resistant materials such as metal, non-halogenated flame-retardants, etc.). A ban on the manufacture, sale, and use of all products containing deca including transportation, wiring and cable, etc., would provide the

driver for innovation and ensure that the use of deca will not continue and potentially expand into other applications.

Please include a recommendation in the final chemical action plan to ban the manufacture and sale of all new products containing deca by January 2006.

3) Labeling of products containing halogenated flame-retardants and deca.

To be consistent with the state dangerous waste regulations, labeling should be required for all products containing halogenated flame-retardants. This would provide consumers with important information and help ensure proper waste disposal. While this plan only deals with PBDEs, there is concern with all halogenated chemicals as they are more likely to persist in the environment and build up in the food chain and our bodies. Also, there is no recommendation in the draft plan for labeling products containing deca, despite the extended 2008 timeline for the phaseout. If a longer timeline is put in place for the ban on deca-containing products consumers have a right to know what products contain deca.

4) Disposal of PBDE containing products.

The recommendation to examine current disposal and recycling practices and determine reasonable end-of-life procedures that are protective of human health and the environment makes sense. While it is reasonable to complete such an investigation and come up with a more comprehensive approach to PBDE disposal and recycling, it is **unreasonable** for this to take 3 years. Ecology has an obligation to enforce the current hazardous waste requirements for PBDE containing products, which you have stated in the draft plan would probably be considered hazardous waste at the end of life.

In 2005, Ecology should identify the products containing the largest quantities of PBDEs and ensure that their disposal or recycling is protective of human health and the environment. Ecology should use existing law (Pollution Disclosure Act of 1971, RCW 90.52.010) to obtain information on PBDEs used in Washington state businesses that discharges waste into waters of the state (or sewer systems that discharge to waterways). Ecology should also require that manufacturers providing products to the state report the use of PBDEs and other halogenated flame- retardants. This is one way we will begin to get a handle on the use of these toxic chemicals in products.

In addition, we ask that Ecology more immediately ban the incineration of PBDE-containing solid wastes. Our concerns regarding incineration include:

- PBDEs are chemical cousins of PCBs, which cannot be burned in solid waste incinerators. PBDEs can make up a substantial portion of a product, in some cases up to 30% of the product by weight.

- Solid waste incinerators cannot legally burn hazardous waste. It is possible that certain PBDE containing products would trigger current dangerous waste regulations due to persistence and toxicity criteria.
- Burning PBDEs can create brominated dioxins and furans, which pose additional environmental health threats. Pollution control devices and temperature control do not prevent dioxin and furans from ending up in the air emissions and ash. Even incinerators that claim to be "state of the art" can regularly have equipment failures, temperature control problems, and by-pass incidents (where pollution control devices are avoided). Furthermore, incinerators do not monitor these emissions continuously so we have no idea how much dioxin and other pollution they actually release.
- Incineration creates waste by-products (ash) that must be disposed of in landfills. Thus, incineration does not solve the problem of landfilling and the problems are potentially increased due to the presence of brominated dioxins and furans in the ash.

While landfills are far from an ideal disposal option, they do not result in the creation of new persistent toxic chemicals (dioxins and furans) that we will have to deal with at some point in the future.

With respect to recycling, in order to permanently retire PBDEs and prevent them from entering new products, Ecology/Health should recommend that companies be required to separate sort these products out at the end-of-life and for electronics electronic products brominated (and other halogenated chemicals) should be separated out.

5) State Purchasing of PBDE-free products

In order to be consistent with the recommendations to ban penta, octa, and deca as well as the Executive Order on persistent toxic chemicals, Ecology/Health should amend the draft recommendation to require that General Administration immediately specify that goods purchased on state contracts should not contain Penta-BDE, Octa-BDE **or Deca-BDE.**

6) Institute for Clean Product and Design

The preliminary plan included a recommendation to establish an institute in the Washington public university system with the goal of making Washington an international and national leader in research and development of "clean" product design and production. We would like to see this recommendation be put back in the plan and have the focus of the institute be eliminating toxic chemicals (particularly PBTs) from products at the design stage, using green chemistry.

7) National Chemicals Policy

We also supported the recommendation from the earlier draft to revise federal chemical policy, however, more information on chemicals is not the only change that needs to occur. We would like Ecology/Health to support the following principles with respect to reforming national chemicals policy as part of the PBDE plan:

a) Require Safer Substitutes and Solutions -- seek to eliminate hazardous chemical use and emissions by altering production processes, substituting safer chemicals, redesigning products and systems, and rewarding innovation. Safer substitution includes an obligation on the part of the public and private sectors to invest in research and development for sustainable chemicals, products, materials, and processes.

b) Phase-out Persistent, Bioaccumulative, or Highly Toxic Chemicals -- prioritize for elimination chemicals that are slow to degrade, accumulate in fatty tissues, **or** are highly hazardous to humans or the environment.

c) Give the Public and Workers the Full Right-To-Know -- label products that contain hazardous chemicals, list quantities of hazardous chemicals used in agriculture and in manufacturing facilities, and provide public access to safety data on chemicals. Also require manufacturers to report the amount of hazardous chemicals they use each year.

d) Act on Early Warnings -- act to prevent harm when credible evidence exists that harm is occurring or is likely to occur, even when some uncertainty remains regarding the exact nature and magnitude of the harm.

e) Require Comprehensive Safety Data for All Chemicals -- assume that a chemical is highly hazardous unless comprehensive safety data are available for the chemical and require manufacturers to provide this data by 2015 for a chemical to remain on the market -- this is the principle of "No Data, No Market."

f) Take Immediate Action to Protect Communities and Workers -- when communities and workers are exposed to levels of chemicals that pose an immediate health hazard, immediate action is necessary to eliminate these exposures.

Finally, we would like Ecology/Health to evaluate and suggest specific regulatory actions that can be take at the state level to fill the serious gaps in federal policy (specifically with respect to testing and data) which are resulting in significant problems in Washington.

8) Monitoring and research

We support DOH seeking resources and establishing bio-monitoring for PBDEs in Washington State. This is important for establishing a baseline and measure progress as steps are implemented to phase out PBDEs. We also support the worker exposure study of PBDEs in collaboration with CDC.

It makes sense to maximize resources and use existing programs to monitor for PBDEs in the environment. However, it is particularly urgent that WA conducts extensive fish tissue monitoring for PBDEs and other PBTs since this may be an important route of exposure. We urge DOH/Ecology to establish a much more comprehensive fish-monitoring program so that the State can acquire basic information about potential health threats, allowing us to better protect all Washington residents, particularly women and children, from exposure to PBDEs and other persistent toxic chemicals.

In addition to fish monitoring, the monitoring programs should include testing of a variety of wildlife, including marine mammals, and sediments to identify sites that need to be cleaned up and to monitor our progress in eliminating PBDEs from the environment.

We supported the earlier recommendation to test incinerator emissions for PBDEs, and requested that brominated dioxins and furans also be tested. Ecology should immediately use its authority to immediately require this testing by the Spokane incinerator and the Tacoma incinerator (if it proposes to re-open). Finally, we support testing sludge for PBDEs as well as brominated dioxins and furans.

Conclusion

We greatly appreciate the hard work and thought that have gone into this PBDE phaseout plan. As we have stated, it provides a strong case for the phaseout of all forms of PBDEs. We urge Ecology/Health to strengthen the plan by including our suggestions.

Thank you again for this opportunity to provide input. We look forward to the final plan.

Sincerely,

Laurie Valeriano
Policy Director

WASHINGTON RETAIL ASSOCIATION
"THE EYES, EARS AND VOICE OF RETAILERS"

November 5, 2004

PBDE External Advisory Panel
c/o the Washington State Department of Ecology
Washington State Department of Ecology
PO Box 47600
Olympia, WA 98504-7600

Dear Advisory Panel,

The Washington Retail Association (WRA) and its 2,800 storefronts oppose any actions by the State of Washington to ban or restrict the use of the flame retardant known as Deca bromodiphenyl ether (Deca) in consumer products. Deca is one of the most common PBDE's used in the U.S. and has been proven by extensive scientific studies to be safe. To discontinue the use of Deca would be an unnecessary, arduous task that would only lead to increased costs to consumers, retailers, manufacturers and add to the anti-business atmosphere in the state of Washington. This is the second letter that the WRA has written in opposition to the phase out of Deca.

Deca has been approved as a safe product by a number of world agencies. Included in these is:

- The European Union, on May 26, 2004, reaffirmed that Deca is safe for continued use in all its approved applications. This **concluded a 10-year, multi-million dollar Risk Assessment** process.

- In 2003, the Voluntary Children's Chemical Evaluation Panel (VCCEP) Risk Assessment submitted by industry (and later reviewed by a panel of independent experts) found that Deca poses no significant risk to children's health.

- The National Academy of Sciences reported in 2000 that the use of Deca in textile applications was acceptable and posed little risk.

- The World Health Organization concluded of Deca: *"Risk to the general population from [Deca] is considered to be insignificant."*

- The U.S. Consumer Products Safety Commission concluded: *"[Deca] is not likely to present a hazard to consumers."*

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Instituting a state law that would effectively ban the use of the most commonly used flame retardant (Deca) into the state of WA and ONLY the state of WA, would be placing WA retailers at a serious disadvantage competitively and would expose them to costly litigation. In addition what about on-line retailers? How is this ban going to apply to them, where the seller had no physical presence in the state? This law only impacts sellers within the state of WA, increasing the cost of goods in the state, in turn drives consumers to the internet or to neighboring states to save money. Online sales would still contain Deca and there would be no way to police these sales.

Retailers that sell second hand goods would be exposed to lawsuits. There would be no way of knowing which products contained Deca and which didn't for second hand retailers. Other retailers will face litigation as well since there will be issues surrounding labeling that would expose the retailer to a lawsuit.

WRA urges all participants involved to examine the clear benefits of Deca, including saving lives from fire and the positive effects of Deca used in plastics that are consumed by fire as compared to those same plastics without Deca treatment all with no harm to the people of the state of Washington.

Thank you for your consideration on this matter.

Sincerely,

Jan Teague, President/CEO
Washington Retail Association

cc: Jan Gee, Contract Lobbyist

Pierce County Recycling, Composting and Disposal, LLC dba LRI
17925 MERIDIAN STREET EAST • PUYALLUP, WASHINGTON 98375
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November 8, 2004

Ms. Cheri Peele
Environmental Assessment Program
Department of Ecology
P O Box 47600
Olympia, WA 98504-7600

Delivered via email to chep461@ecy.wa.gov

RE: Washington State PBDE Chemical Action Plan

Dear Ms. Peele:

Thank you for the opportunity to review and provide comment on the Washington State Draft PBDE Chemical Action Plan. Although, I applaud the Departments of Ecology and Health for identifying actions the state may take to reduce threats posed by PBDEs. I have concerns over several fundamental statements and approaches.

In the Executive Summary, the statement is made that the departments of Ecology and Health recommend a strategy that guides the handling and disposal of existing PBDE products and reduces the manufacture and sale of new PBDE products. As I review the list of participants I find a list of manufactures, retailers, environmental groups, local, state and federal governments representation, however, although many of the strategies focus on " handing and disposal" I do not see a representative from the solid waste industry that is actually responsible for the handling and disposal of these materials. Such a representative could provide valuable insight on this matter.

In Section III under the heading of Products Containing PBDE's at End-of-Life, the statement is made "while pathways for PBDEs from products to the environment is unknown, it is thought that much of the substance is likely released at the time of disposal". In the executive summary it is stated "PBDEs have been detected in everything from food to house dust to indoor air, exactly how people are exposed to PBDEs is an area of ongoing study". It seams to me that we are leading the reader to the conclusion that the pathway to the environment is at the time of disposal even though it was previously stated it is found in house dust and indoor air etc. Is it not as likely that the environment can be receiving PBDEs from this route also?

Under the section entitled Landfills the statement is made "most PBDEs are probably landfilled in Washington. The fate of PBDEs in the landfill environment is unknown." I find it unfortunate

that the LRI landfill is singled out due to the receipt of Auto fluff even though PBDEs are found in so many waste streams (as indicated in the document) and accepted by every landfill.

Section IV PBDEs and the Regulatory Environment states, "Under this criteria (WAC 173-303) many products containing PBDEs would probably be considered dangerous waste at end-of-life". Is this where we really want to end up designating carpet, chairs, foam, and interiors of cars as dangerous waste? Creating a "special waste" designation will be costly for the consumer. Do we want to start manifesting large quantities of consumer household products at the end-of-life? Would this possibly lead to and even bigger problem, the increase of illegal dumping and wide spread contamination rather than controlling the destination in an environment built specifically to control such releases.

Washington State has always been a leader in solid waste handling with high aspirations of reducing waste, reusing and recycling materials. This draft PBDE plan has the potential of sending the State in the opposite direction if we are to work with charities and businesses to "minimize the resale of upholstered furniture" and "remove materials from the recycling stream…" We need to focus on the beginning of life.

Once again thank you for the opportunity to respond to the Draft PBDE Chemical Action Plan. If you have any questions regarding this correspondence I can be reached at 253-927-6710.

Sincerely,

Jody L Snyder
Director of Regulatory Services

Cc: Eddie Westmoreland, Vice President, LRI
Norman LeMay, Treasurer, LRI
Cullen Stevenson, Department of Ecology
John Sherman, Tacoma Pierce County Health Department
Steve Wamback, Pierce County Solid Waste

Comments on the Washington State PBDE Chemical Action Plan, October 11, 2004 Draft

-Thomas A. McDonald, M.P.H., Ph.D.
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The Department of Ecology and the Department of Health have produced a very good document that summarizes the uses, environmental and health concerns, alternatives, and the policy recommended for the PBDEs. Some of my comments are technical; most are for clarification.

Exec Summary, Page iii. You summarize studies that suggest that 90 % of PBDEs come from the diet. It would be more exact to say that recent studies suggest that for most individuals that the diet is the major pathway of exposure, but other studies suggest that for a smaller fraction of the population, indoor exposures may predominate (see Wilford et al. 2004 ES&T, on line version).

Exec Summary, page iv, end of first paragraph: The paper states that each year that PBDE products are sold will extend the timetable and costs by decades. This does not make sense to me. There is a lag time from production and use until PBDEs reach the top of the food chain, but this lag time should not be different from products sold last year from products sold next year.

Page 10 second paragraph: ". . . PBDE levels were not correlated with age, except for infants." This sentence does not make sense as written. Maybe delete the phrase "except in infants."

Page 11 ref 52: author should be spelled "Luksemburg"

Page 17, Table 5. last row. Delete "(0.86 – 2.4 ug/day)", use the intake values in the same units as the others in the table. And Footnote b: in McDonald 2004, the default female body weight used was 62 kg, not 70 kg. Recalculate the daily intake value in the table accordingly.

Page 17 and 18, Penta-BDE and Octa-BDE: Please note that Viberg et al. 2003 (Toxicol Appl Pharmacol 192(2):95-106) also found that postnatal exposure to PBDE-153 caused neurodevelopmental effects in mice. PBDE-153 is present in both the Penta-BDE and Octa-BDE technical mixtures. Other (penta-related) studies you may want to cite include a study of PBDE-99 (Viberg et al. 2004 Toxicol Sci 81(2):344-53) and one on the Penta-BDE tech. (Stoker et al. 2004 Toxicol Sci. 78(1):144-55).

Page 18. Reference 116 is unnecessary.

Page 20, Table 6. Some studies relate to the technical mixtures, while others relate to specific congeners, which is not apparent from the Table. For example, the LOEL for the reproductive effects shown in the table 0.06 mg/kg are for PBDE-99, whereas the LOEL for developmental reproductive effects from the Penta-BDE tech mixture was 30 mg/kg (Stoker et al. 2004 ref given above). Also, the "reproductive effects" are best characterized as developmental reproductive effects.

Page 31. Table 7 – a square box appears were the "μ" symbol should be. Also, since the document is for Washington State, you should consider organizing the table into subheadings, one for data related to the Pacific Northwest and a separate heading for other regions in N America.

Page 35 Federal overview: you may want to consider mentioning that a bill has been introduced into the U.S. House of Representatives (HR 4076, Solis, DeGette and Woolsey) which would phase out and require labeling for all forms of the PBDEs. It is unclear whether this bill will go anywhere, though.

Page 37, California: Please note that California passed into law in 2004 AB2587 which moves the date of the California ban from 2008 to June 1, 2006. See: http://www.leginfo.ca.gov/pub/bill/asm/ab_2551-2600/ab_2587_bill_20040921_chaptered.pdf for the text of the law.

Page 45, Chapter on Alternatives. This chapter is very good, with lots of good information. However, I really would like to see a greater emphasis given to non-chemical solutions in the tables and text. For example, increasing the density of polyurethane foam reduces dramatically the need for flame retardants. Also, some have proposed using low-flammability barrier layers between the fabric and foam of furniture, thus bypassing the need to flame retard the foam. The non-chemical solutions may include the use of other materials, such as some natural fibers (wool or heavy cotton) that may not need to be chemically treated; use of wood and metal instead of plastic; and use mineral wool insulation instead of rigid polyurethane foam insulation.

Table 11 and elsewhere in the text: Please include the computer company NEC's alternatives solution. NEC developed and uses in its electronic equipment a plastic called polylactic acid, which is both non-flammable and biodegradable.

Table 11. Delete 2 occurrences of "No non-halogenated alternatives identified in commercial use" which is redundant with the "non-identified" in the last column of the table.

Page 55. I believe San Francisco also has similar purchasing requirements. I can dig around for contact name if you wish to explore this.

Page 61. State purchasing: specifying that products do not contain penta or octa in purchasing agreements would be unnecessary if they become banned in the State of Wash.

Page 65. Reference section. You can delete the references, as they have already been cited as footnotes throughout the text.

Page 68: Reference for McDonald 2004 is incomplete.

Page 83. I think IBM, IKEA, Intel, and Motorola have, or are trying to, phase out PBDE use.

Nice work; it was a pleasure to read.

Seattle public meeting comments on PBDEs

- **Elise Miller, exec. Director, Institute for Children's Health –** It's clear you know that PBDEs persist in the environment and can impair memory in lab animals and that lab tests can under-predict effects and that children and infants are particularly vulnerable. What you may not know is that … I work nationally with people in groups concerned with the rise in learning difficulties and emotional and social costs, economic costs, things like lost income, special education and even incarceration ... social, nutritional and genetic factors … but the factor that is least acknowledge and is most preventable is environmental. We learned that with lead, PCBs, etc. We're not going to stand for this any more. It's not ok to expose our children to neurotoxins. If the cornerstone of public health is prevention, why are we waiting? Not only can we save the emotional costs of kids with learning disabilities, we can save the economic costs. (A study) conservatively estimated we spend $4.6 billion annually on learning disorders that are environmentally contributed. When there's a political will to act, we can create a healthier environment for our children. Let's have Washington State lead the country by phasing out all PBDEs.

- **Doreen Smith, salesperson at a natural bedding store** – Every day people come in and talk about PBDEs. God, that's 500 people right there. Some days that's all we talk about. No one wants to die in a fire, but people come to me desperate, with a doctor's note for something without flame retardants. Some folks are ok with other beds, and a PBDE alternative is boric acid powder. The numbers of people I'm talking to astounds me. They're pretty well educated, but they're scared. My hope is we can turn the industry around, because there is demand out there.

- **Nancy Evans, health consultant for breast cancer fund in San Francisco, 14-year breast cancer survivor** – We help to identify and advocate for environmental causes of breast cancer. We support your excellent draft report. Scientists don't know whether exposure causes breast cancer, but they do know PBDEs are increasing in women's bodies. Other countries are way ahead of us. Sweden has done a remarkable job. We urge you to resist industry pressure to weaken your report and your recommendations. We don't have proof that these chemicals cause cancer but we have evidence. We are bodies of evidence (as in the bodies of women who have been diagnosed with breast cancer).

- **John Abbots, NW Environmental Watch** – At the end of September, you reported on high levels of PBDEs in women in the Northwest. We commend the state agencies' commitment and work; we wish to submit detailed analytical data.

- **David Hayworth, Wash. Physicians for Social Responsibility** – We urge Ecology to phase out all forms of PBDEs. There are alternatives adopted already by about 20 corporations. (SUBMITS letter singed by about 60 health professionals.) Excerpt: PBDEs share properties with banned PCBs, including learning behavior problems with lab animals, these chemicals are ubiquitous and higher levels than elsewhere. Steady reducing and removing these chemicals from the environment is fundamental to children's health.

- **Elizabeth Davis, League of Women Voters** – The league strongly supports ecology's effort, notes several league studies, including "Early Intervention and Prevention of Children at Risk," that promote well-being, encourage development and ensure the safety of all children. The league notes the increase of PBDEs in breast milk, orcas, polar bears, indoor and outdoor air and food and considers it horrendous that the fetus and growing child is exposed to persistent and bio-accumulative chemicals that can have such permanent effects. This will surely prevent children from reaching their full potential. We support Ecology's plan, with the following changes: treat PBDEs as hazardous waste; consider earlier phase-out of Penta and Octa; move up the date for the ban on Deca to July 2006. As a personal note, as a breast cancer survivor with no family history, genetic markers or exposures, I say 'enough.' It is not enough to treat cancer in people, we must reduce exposure to chemicals. It is the right thing to do.

- **Jim Mulligan, Earth Ministry** – As a member of a multi-denominational denomination, I speak on behalf of the membership, who are not technical experts but endorse all concerns/comments and commend Ecology for its good work and taking seriously these risks. Our constituency sees the need for these steps as moral responsibility as citizens and as persons of faith. Thank you for the thoroughness with which you're looking into this. I spent the last five years working off and on on the Duwamish River, where PCBs are on bottom of the river. If we had people like you (doing what you're doing today) years ago, we might not need to be doing that work. On behalf of unborn members, I encourage you to take strongest possible stand.

- **Amy Hirsch, law student** – Fewer people are smoking in bed, industrialized countries worldwide are developing alternatives, so why is inflammability being forced upon us? Ecology is not moving fast enough. The sooner PBDEs are banned, the better not only for our environmental health but for the local and export economy. Keeping PBDEs out of products will enhance marketability overseas, and the sooner we clean up the food chain, we enhance the exportability of food products, including fish.

- **Ann (?), mother/mom** – I urge Ecology to adopt strong a phase-out plan for all PBDEs.

- **Matthew Cachow, Healthy Building Network** – I call for environmental justice for where we work and play. Concerned by high levels of PBDEs in orcas, concentrations in Columbia River doubling every 4 years, high breast cancer rates. Successful industry in green building, attracts innovators, architects and builders are knowledgeable about toxic effects. An editorial in the Environmental Building News recommends the elimination of toxic chemicals with highest priority given to PBDEs. Many building professionals are disappointed that not all companies are committed to health concerns. Support sustainable building. Ban all PBDEs by 2006. All state agencies should purchase PBDE-free materials. Building market demand for safer alternatives will build a robust economy for Washington State now and in future.

- **Tracy Hendershot, health care worker** – More lives will be lost than saved in using these chemicals. I can smell odor from my home pc and it permeates other materials. My

old metal-encased computer does not have this effect. Another thing: stop torturing and killing animals to find out if these things are harmful, instead just ending their use. Humans are eventually the study group for these chemicals inasmuch as we allow them to be used today. I hope I'm not back here in the future to discuss the replacement chemical.

- **Bobbi Morgan, Bainbridge retired speech language pathologist** – Worked 25 years with students with learning disabilities. I'd like to speak to you tonight as the spirit of Rachel Carson, who died researching PCBs. "Here we are again – my work is not done. Problems of PBTs persist. Untested chemicals arrive unasked for in products around us. We are all subjects in experiments, no permission was granted. I speak for women who would do anything to breast-feed with toxic-free milk. I sound a warning even more imperative now; now we should know better about allowing PBTs to build up in bodies. Have we not learned the lesson of DDT, which is still found contaminating breast milk? Do we not appreciate the delicate balance of the endocrine system that is wrecked by these chemicals? Ban the manufacture and sale of all toxic flame retardants and then all PBTs. Even after they're banned, their legacy of damage lingers for so long. Make sure chemicals are safe before they are released into our delicately balanced world. Hold fast under industry pressure, hold strong. I speak for life."

- **Jennifer Cropack, Burien, Washington Toxics Coalition, Audubon Society** – I want source control at the industry level. I bought a hybrid car, had to off-gas it for months before could drive it. I paid more for my decisions, paid for my decision, want Washington to put action behind the health of Washington residents. I saw the graph (in the presentation) where Sweden decreased PBDE levels; I want the same here. At a time when people think government is for corporate profits and greed, Washington should stand up and say it cares about the health of its citizens and indicator species like salmon and bald eagles. I had a 50-yearold friend die, my dad has five forms cancer, now terminal … the individual family and social costs of these illnesses goes out beyond $4.6 billion dollars in health care costs (into) second mortgages on homes, trying to care for loved ones. I want Deca banned sooner, not later. I'm concerned about Deca creep and how many other things can happen. I bought a special bed as an educated consumer, I think we need to educate consumers, but also stop it at the source. We consumers can only do so much.

- **Cindy Chowdry, mother of 2** – As a breast-feeding advocate, I hear moms concerned about children, also Duwalup, it's insane to think women are doubting their breasts on any level. Appalling they have to waste time and energy talking about these kinds of things and that they're going to question what they're doing as a mother. My husband sits in front of 3-4 monitors every day on the job, how confident (of safety from PBDEs) can you be? I urge you to ban PBDEs as soon as possible; make the first breath and first drink of milk as fresh as possible.

- **Kelly Faye, mother, toxicology student** – I went into my field out of concern for pollutants, and I urge you to ban all PBDEs. I also want to know, do we have assurance that alternatives are going to be better? Will adequate risk assessment be done? I want assurance before we jump into making alternatives that might be more harmful.

- **Megan Blankwise, WashPIRG** – I thank Ecology for recognizing the need for phasing out PBDEs. I'm here for 20,000 members to support the phase-out of all PBDEs. Fish PBDE levels are up, breast milk levels are higher than elsewhere. These chemicals are a threat to public health, are toxic, government must protect the environment and children. Since Deca is the most used PBDE, any plan that doesn't include it won't be effective. We must make sure any alternatives aren't persistent or toxic. Stand up to industry pressure and ban all PBDEs and monitor levels and develop programs to encourage business to develop PBDE-free products.

- **Beth Seltzer, with son** – I ask you to phase out all PBDEs. We only get one chance to develop our brains and we want them to develop the right way. It's hard to express how awful it is that PBDEs are so present, I'm happy you're acting but want a sooner phase-out of Deca.

- **Sarah Augustine** – I heard about this on the radio today, said 'Gosh. Why are we phasing out in 2008? Why wait?' No one wants to wait. If there's a health risk, act now.

- **Eldon Wall** – I remember 30 years back , Dow Corning, or Union Carbide, come company coined the phrase 'better living through chemistry.' Dupont, (other audience member) remembers. Sitting here listening, I started thinking about sometime back in history class we were told when the U.S. Congress required drug companies to test before products go on the market. If we can do that for the drug companies, and they make the biggest profits, let's do it for the chemical companies. As for when … the Legislature meets in January, let's get it done by the time the Legislature convenes, which is probably in May, so it should be phased out by May.

- **Nancy Dickman, Physicians for Social Responsibility** – We urge Ecology to phase out all PBDEs. PBDEs are linked to serious health impacts, accumulating in the environment, and are ubiquitous, creating exposures over which we have no control. There's no way to shield children, and a continued risk of harm to children's health and development. We encourage Washington businesses to join other companies already developing PBDE-free products, at the same time producing products valued for safety to the environment and human health. Take the precautionary approach and act now. Prevention is the only cure.

- **Ivy Sager-Rosenthal, People for Puget Sound** – We support the phase-out of all PBDEs, we don't want a toxic legacy like PCBs. The evidence shows that PBDEs should be our concern. We need to stop now, phase them out, not spend millions and millions of dollars cleaning up a mess, so don't have species like orcas on the decline. And clean up Puget Sound. Deca is on the increase, just as dangerous as Penta and Octa. I'm now a mother and the issue has become even more personal. I want to give my child the cleanest, safest environment possible. It's unfair that industries can put these products to market but I can't do anything about it. I can't do anything about computers and rugs in my house. It's not fair for (my child). I can take the preventative measures described by Health, but that's not the solution. The solution is phase-out.

- **Lindsey Datelund, Seattle resident** – It's wonderful to speak, to have my words heard, but I hope my words aren't outweighed by industry forces, I hope there's not a power of differential there. I strongly urge, beg you, demand of you as a future breast-feeder: 2008 is not enough. I've been excited about having kids all my life, and learning at 26 what is in my body makes me so sad. Watching sister breast-feed, my sister was so excited and saying how she was only eating organic, and I had just learned about PBDEs and I couldn't tell her; my heart was breaking. I hope our health and our future is what guides us, not industry.

- **Mary Ann O'Hara, family physician and PBDE Advisory Committee member** – I appreciate how evidence-based the process has been. All major physicians' groups in the state have passed resolutions urging Ecology to ban all PBDEs. (She lists them, including the state medical association.) The amount of evidence is persuasive, especially in light of other toxins and how their toxicity became more apparent when more tests were done. Sine we only get to be a fetus once, environmental health is crucial. In utero exposure and house dust are far worse to children than breast milk. The terror is already out there; women and physicians need to know how to put this in context. So it's important it be done in a way that doesn't jeopardize breast-feeding, which offsets toxins and promotes neurodevelopment. What is your challenge, and how can we help you succeed?

- **Laurie Valeriano, Toxics Coalition** – I am a breast-feeding mother of twins, and I breast-fed a third, and I thank you for your courage in drafting the plan in the face of intense opposition from the bromine industry. I'm angry that my rights were violated, that my ability to grow children in a toxic-free womb and feed them toxin-free breast milk was taken away by a chemical industry that brought us PCBs and failed to properly test these chemicals before putting them in our homes. I am hopeful the plan will propose strong action and take problem the seriously, putting resources into it and making proposals that will make a difference. I support the recommendations but ask for improvements as well. There is too much Deca already out there; phase out deca across the board, in all products. If we know enough to act on electronics and textiles, we know enough to act on all uses. Dow knew in 1973 that Deca breaks down into more harmful forms, so (the industry has known) for many years. It's time to phase it out now. By 2005.

- **Sybil Diver, Toxic-Free Legacy Coalition** – We have 48 members representing other groups, a truly broad-based effort demonstrating community support. There is mounting evidence that Deca is a problem. Ecology's plan indicates recent studies show Deca building up in humans and infants, who ingest more dust. The plan has strong evidence Deca breaks down into more harmful forms, and it recognizes its use in large quantities. Given these determinations, Washington State needs a stronger phase-out plan for Deca. Ecology and Health should support a ban on Deca for all new products; Ecology and Health should recommend a consistent timeline for the phase-out of all PBDE products by 2006 or sooner; Ecology and Health should (recommend) state contracts not buy Deca products as well as Penta and Octa. We need to shift the market to safer substitutes.

- **John Staltfuss, Snow Coalition** – I love the gifts of life and health, and I'm disappointed with the corporate and governmental willingness to compromise our lives and health. Thanks for your effort to work in the gift of life all of us share. (Tells story about cutting bruises from apples before giving them to friends.) PBDEs aren't apple bruises, and I'm really glad you and others are working to cut out these toxins from products we use.

- **Linda Boyd** – I didn't intend to speak. I want to see a public information campaign, a full-page ad in The Seattle Times, or a campaign through the schools to inform parents. Can we change the regulations for schools and child cares for PBDEs (i.e. flame retardant materials)? It's hard to find out whether this product or that product contains it. Thank you for your good work.

Spokane public meeting comments on PBDEs

- **Nicole Lee, Washington Toxics Coalition –**
- **Jenny Greenwood, parent** – My son has gone from mildly autistic to gifted because I keylated him from mercury. Now I'm hearing this is something else to be worried about. Is there anything out there that can decrease the levels once they're in your blood or whatever? Also, what resources are manufacturers bringing to the table, since they're putting this in our environment? I would like to encourage everyone to think outside of the box. Besides being a mother who healed my kid, I work with special-ed kids and I have to tell you the population is growing right now. I see this as an epidemic (developmental disabilities). There has to be a lot of collaboration on the behalf of our kids.

- **Debbie Boswell, Lands Council, mother of two children** – I'm very concerned about my kids' children and I support the ban on the manufacture and sale of these items. I'd like to see this process sped up so there will be hope for my children.

- **Michael Abbier** – I'm glad the mother brought up the mercury content issue. The current White House administration is deregulating so much, such as Clean Skies. I used to work with children that were severely disabled, and those institutions just do not exist any more. It's come to a point where those children who are hospitalized 24/7 no longer exist. As long as this stuff keeps going on, your children, your descendants, there will be no help, we'll just die. This is not very nice, this doesn't feel very good. Thank you.

- **Mike Peterseon, Lands Council, 1400 members** – I thank the Department of Ecology for moving forward on this emerging issue. I congratulate you for getting on top of this one. These are very serious issues when you have toxic chemicals in furniture… I believe there's evidence that the Deca form turns into other PBDE forms. We should ban all those chemicals by 2006, and state agencies should take the lead and advertise that they're purchasing products that do not contain PBDEs. I believe we have a legacy of PBDEs in furniture, electronic devices. When they're sitting at home or end up in a landfill, we need to look at existing contamination and deal with that right away. We need to monitor and track levels in eastern Washington State. I would like to see our

manufacturers, including Boeing, who I think is fighting this, step up and takes the lead in finding solutions to this thing.

- **Linda Greene** – I thank you for what you've done. It's really incredible the amount of time and work and enthusiasm you have for this thing. Ban all forms of PBDEs by Jan. 1, 2006.

Other, unidentified speakers:

- Thank you for coming tonight and talking to us. I support the ban on these chemicals.
- I support this ban. My reservation is that it doesn't go far enough.