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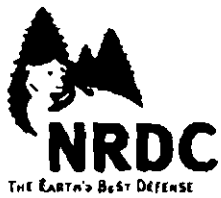
DATE: June 14, 2004

TO: Arthur G. Baggett, Chair and Board Members
State Water Resources Control Board

FROM: David Beckman

FAX NUMBER: (916) 341-5620

MESSAGE:



NATURAL RESOURCES DEFENSE COUNCIL

June 14, 2004

VIA FACSIMILE (916-341-5620) AND U.S. MAIL

Arthur G. Baggett, Chair and Board Members
State Water Resources Control Board
1001 I Street
Sacramento, CA 95814

Re: Comments on "Notice of Public Solicitation of Water Quality Data and Information – 2004 Clean Water Act Section 303(d) List"

Dear Chairman Baggett and Board Members:

On behalf of the Natural Resources Defense Council, we are submitting these comments on the "Notice of Public Solicitation of Water Quality Data and Information – 2004 Clean Water Act Section 303(d) List" (Solicitation Notice). We also are including additional information and renewing our request that the San Francisco Bay be listed for a number of chemicals generally referred to as PBDEs.

I. Listing Solicitation and Board's 2004 Listing Process

In summary, we have significant concerns with regard to the legality of the Solicitation Notice, specifically with respect to its lack of compliance with Clean Water Act Section 303(d) and its implementing regulations. The failure of the Solicitation Notice to comply with these legal mandates makes it inherently flawed with respect to gathering information that could be relevant to decisions on the quality of the state's waters. As the agency charged with protecting the health of the waters of the state and cleaning up waters that fall through the cracks, the SWRCB should be particularly careful to comply with all statutory and regulatory mandates to cast a wide net to gather and use all existing and readily available information.

Legal Mandates

As the Solicitation Notice acknowledges, the SWRCB is required by Clean Water Act Section 303(d) and 40 C.F.R. § 130.7 to develop a list of water quality limited segments. Specifically, Section 303(d)(1) states that "[e]ach State shall identify those waters within its boundaries for which the effluent limitations required by section 1311(b)(1)(A) and section 1311(b)(1)(B) of this title are not stringent enough to implement any water quality standard applicable to such waters." (33 U.S.C.

§ 1313(d)(1)(A).) *Pronsolino*[1] made clear that Section 303(d)(1)(A) "appl[ies] to all waters in the state, not only to the subset covered by certain kinds of effluent controls," interpreting "not stringent enough" in Section 303(d)(1)(A) to mean "not adequate for" or "inapplicable to."[2]

The regulations at 40 C.F.R. § 130.7(b)(5) add that:

> Each State shall assemble and evaluate all existing and readily available water quality-related data and information to develop the list required by §§130.7(b)(1) and 130.7(b)(2). At a minimum "all existing and readily available water quality-related data and information" includes but is not limited to all of the existing and readily available data and information about the following categories of waters:
>
> (i) Waters identified by the State in its most recent section 305(b) report as "partially meeting" or "not meeting" designated uses or as "threatened";
> (ii) Waters for which dilution calculations or predictive models indicate nonattainment of applicable water quality standards;
> (iii) Waters for which water quality problems have been reported by local, state, or federal agencies; members of the public; or academic institutions. These organizations and groups should be actively solicited for research they may be conducting or reporting. For example, university researchers, the United States Department of Agriculture, the National Oceanic and Atmospheric Administration, the United States Geological Survey, and the United States Fish and Wildlife Service are good sources of field data; and
> (iv) Waters identified by the State as impaired or threatened in a nonpoint assessment submitted to EPA under section 319 of the CWA or in any updates of the assessment.

(Emphasis added.) In addition, 40 C.F.R. § 130.7(b)(6) requires California to provide documentation to the EPA Region IX to support the State's determination to list or not to list its waters. This documentation must include a "rationale for any decision to not use any existing and readily available data and information for any one of the categories of waters as described in §130.7(b)(5)." (40 C.F.R. § 130.7(b)(6)(iii).) In other words, the state must explain why it did not seek out and assemble existing and readily available information.

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[1] *Pronsolino v. Nastri*, No. 00-16026, at 7929 (9th Cir., May 31, 2002)

[2] *Id.* at 7928; *see also Dioxin/Organochlorine Center v. Clarke*, 57 F.3d 1517, 1528 (9th Cir. 1995) ("since best practical technology effluent limitations do not apply to toxic pollutants, those limitations are, as a matter of law, 'not stringent enough' to meet water quality standards")

Inconsistencies Between Solicitation Notice and Legal Mandates

In a number of places, the Solicitation Notice sets limitations on the solicitation process such that the Notice violates the basic requirement to "assemble and evaluate all existing and readily available information" to develop the required list of "water quality-limited segments." (40 C.F.R. §§ 130.7 (b)(1), (b)(2) and (b)(5).) These include, but are not limited to, the following:

- The Solicitation Notice asks for information to "assess the State's water bodies for possible inclusion on or removal from the existing section 303(d) list," and then defines the list as including only those waters exhibiting "deleterious impacts from a pollutant or pollutants." However, nothing in Clean Water Act Section 303(d)(1)(A), which defines the scope of the list, or in the regulations limits the application of the listing requirement to only waters in which water quality standards are not met because of the presence of a "pollutant." The list must include all waters in which water quality standards are not achieved despite the application of effluent limitations, regardless of whether a pollutant is causing this failure to achieve water quality standards. Limitation of the Solicitation Notice in this way illegally limits the amount of information being solicited below the "all existing and readily available" threshold.

- The Solicitation Notice states that "[r]equirements for data and information from the Listing Policy – including those for quality control and assurance, temporal and spatial characteristics, and minimum sample sizes – will be followed when reviewing data and information." EPA Region IX's February 18, 2004 letter from Alexis Strauss to Art Baggett on the draft Listing Guidance makes clear that though "'high quality' data should be accorded the greatest weight . . . all data and information must be considered (see EPA, 1997a and EPA, 2003)" for listing decisions. (*See also* U.S. EPA, *Guidance for 2004 Assessment, Listing and Reporting Requirements Pursuant to Sections 303(d) and 305(b) of the Clean Water Act* (July 21, 2003) p. 25 (stating in response to the question "How should a State address data and information quantity?" that "All existing and readily available data and information must be considered during the assessment process") [hereafter "July 2003 Guidance"].)

- The Notice states that "[a]ll available data and assessment information generated since May 15, 2001 will be considered." This artificially short time constraint eliminates many potentially valuable pieces of information and again conflicts with the "all existing and readily available" standard. As EPA reiterated in July 2003 Guidance "[d]ata should not be excluded from consideration solely on the basis of age. . . . A State should consider all data and information." (July 2003 Guidance p. 25.) There are many situations in which information from before the last listing cycle would be submitted, including but not limited to: older data that

recently became relevant due to new scientific understandings about the relationships between the constituents at issue and impairment of beneficial uses, and older information that is meaningful and important in combination with more recent data.

- In paragraphs 6 through 9, the Notice states that "[a]ll" data and information submitted should be accompanied by numerous additional pieces of information and additional evaluations. Some of these additional requirements are simply unnecessary to the SWRCB's decision on whether a water body is impaired or threatened, and some represent tasks that even the regional water boards and SWRCB cannot currently perform. More importantly, virtually none, if any, of the additional information and evaluations called for in the Solicitation Notice is required under the broad "all existing and readily available" standard. Again, the end result is to severely discourage organizations and people from submitting what could be useful information, an extremely short-sighted decision given the paucity of SWRCB-collected and -organized data.[3] The SWRCB should instead indicate that such accompanying information and evaluations would be "welcome and useful," rather than require such additional information or evaluations or create the perception that such information and evaluations are required.

Finally, the Solicitation Notice states that the "final list will be based on data and information available to SWRCB" no later than June 14, 2004. (Emphasis added.) This language, which focuses updating the 303(d) list only on information *made available* to the SWRCB, makes it sound as if all that will be reviewed is the information handed to the SWRCB as part of the solicitation process. This, however, limits the data and information in a way that violates federal requirements and ignores the state's responsibility under federal regulations to seek out and use the myriad sources of information on water quality that are "existing and readily available." As set forth in those regulations, the State must base the 303(d) list on all existing and readily available data and information *that it has assembled*. (40 C.F.R. § 130.7(b)(5) "each state *shall assemble* . . . all existing and readily available . . . data and information" (emphasis added).) As such, the State is under a mandatory duty to collect, assemble and use all

[3] EPA Region IX commented on a similar approach in the state's draft Listing Policy, finding that "[t]he policy's minimum sample size and high quality data provisions and supporting rationale do not provide a 'good cause' rationale for excluding data and information from consideration (see 40 CFR 130.7(b)). These regulatory provisions create a rebuttable presumption that all readily available data and information will be used in the assessment process. A great deal of useful data from STORET, academic and agency reports, and volunteer monitoring groups would appear to be excluded from consideration under the proposed rule, an outcome which appears inconsistent with the federal requirements." (Letter from Alexis Strauss to Art Baggett, February 18, 2004.) EPA also noted in this letter that "the proposed policy appears to set a higher burden of proof than typically used in California's administrative proceedings." (Citations.) The onerous responsibilities for submitting information that the Solicitation Notice places on the public, many of which the SWRCB does not place on even itself, similarly appear to be more stringent that the principles governing the admissibility of evidence and opportunities for public participation typically used in California administrative proceedings.

readily available data and information. (*See Forest Guardians v. Babbitt* (10th Cir. 1999) 174 F.3d 1178, 1187 ("'shall means shall,' which imposes "a mandatory duty upon the subject of the command").)

It is insufficient, therefore, for the State to base the final 303(d) list merely on data and information that it has been handed. Rather, the State must complete its mandate and *actively gather and collect* all existing and readily available information from all potential sources, many of which are readily obvious to members of the public (who do not have the resources to do the state's job for them) and should be similarly obvious to the SWRCB. These include but are not limited to USGS data, DPR data, Monterey Bay Sanctuary data, DHS's Source Water Assessment database, and numerous other data sources, some of which are included in the state's draft Listing Policy. In its February 18th letter on the Policy, EPA Region IX specifically called on the state to "include all EPA monitoring data (not just EMAP) as well as other agencies that operate high quality sampling programs (e.g., U.S. Fish and Wildlife Service, US Department of Agriculture, US Army Corps of Engineers, and National Oceanic and Atmospheric Administration)."

The apparent self-restriction on SWRCB data collection assembly activities is particularly problematic in light of the SWRCB's refusal to support the funding and implementation of a meaningful ambient monitoring program, or to effectively integrate the myriad databases that exist and that contain useful information. We would appreciate additional details from the SWRCB on its and the regional boards' activities to collect, assemble on their own initiative, and use to develop the 303(d) list "all existing and readily available information," over and above that provided as a result of the Solicitation Notice (40 C.F.R. § 130.7 (b)(5).)

* * *

It is our understanding that the SWRCB is cutting $1.4 million in contract funds from the Surface Water Ambient Monitoring Program (SWAMP), which already is seriously under-funded. This is not the first time that this important program has been in jeopardy of near-collapse. The SWRCB must place monitoring information at a much higher priority if it is to adequately protect the health of the waters on which we all depend. Artificial and illegal constraints on the amount of information sought as part of the 2004 solicitation process, and continued assaults on SWAMP, appear to indicate that the SWRCB places a low value on obtaining the monitoring data its needs to do its job.

II. San Francisco Bay and Tributaries

NRDC requests that the SWRCB list the following chemicals under Section 303(d):

Chemical Name:	CAS Number:
Dibromobiphenyl Ether	2050-47-7
Tribromobiphenyl Ether	49690-94-
Tetrabromobiphenyl Ether	40088-47-9
Pentabromobiphenyl Ether	32534-81-9
Heptabromobiphenyl Ether	68928-80-3
Hexabromobiphenyl Ether	36483-60-0
Octabromobiphenyl Ether	32536-52-0
Nonabromobiphenyl Ether	63936-56-1
Decabromodiphenyl Ether	1163-19-5
Polybrominated Diphenyl Ether**	
Polybrominated Diphenyl Oxide**	

**Synonyms: Polybrominated Biphenyl Ether(s) = Polybrominated Biphenyl Oxide(s) = Polybrominated Diphenyl Ether(s) = Polybrominated Diphenyl Oxide(s)

The listing these compounds (collectively referred to as PBDEs) is justified due to an ongoing exponential increase of these substances in the water environment as evidenced by continuing increases in levels in bird eggs, fish, and seals. Over the past year, while the PBDEs have remained on the RMP analyte list, evidence has accumulated that concentrations are increasing in the Estuary: in water, in bird eggs, in fish, and in seals. Humans and wildlife in the Bay Area have some of the highest reported concentrations of these substances in the world.

In 2002, the RMP collected its 1st year of PBDE data in San Francisco Estuary water, sediments, and bivalve samples. Time series data for these three media are not yet available so it is not yet possible to determine temporal trends. The 2002 individual congener and total PBDE concentrations and distributions in San Francisco Estuary water, sediments, and bivalves are presented in the RMP's 2002 Annual Monitoring Results report.

Reports of PBDEs in environmental samples date back to the early 1980's in Sweden. Contamination by these chemicals has been reported in sediment, sewage sludge, pike, eel, sea trout, and human breast milk from locations throughout the world. Most researchers believe that a major exposure pathway of humans to these chemicals is through fish consumption.

Although the data on PBDE levels in the United States are less complete, the evidence shows that these chemicals are found in the environment and in human tissues at levels considerably higher than those reported in other countries. The Hazardous Materials Laboratory of the California Department of Toxic Substances Control has been tracking PBDE levels in harbor seal blubber from the San Francisco Bay, and breast and abdominal fat samples from women living in the San Francisco Bay Area.[1]

Thirty-four seal blubber samples were collected between 1989 and 1998 from eleven stranded, dead harbor seals found along the San Francisco Bay shoreline. Overall, levels of total PBDEs increased by nearly 100-fold during the two decades studied, implying a doubling of concentration every 1.8 years. Levels of total PBDEs in seals averaged 1730 ng/g fat with a range from 88 ng/g fat to 8325 ng/g fat. These levels are among the highest reported anywhere in the world.

The human fat levels were significantly higher than any levels previously reported anywhere in the world. The average level in San Francisco Bay area women was 86 ng/g fat. This level is ten times higher than the average level reported in Germany and Canada, three times higher than levels reported in Sweden, and twenty-five times higher than levels reported in human tissue in Spain. Levels of PBDEs tended to be higher in younger (premenopausal) women, implying that the exposures are likely to be a fairly recent phenomenon.

To further investigate the extent of contamination of the Bay area with PBDEs, this same group of researchers analyzed the eggs of four species of fish-eating shorebirds for levels of PBDEs. Shorebirds are an important ecosystem indicator due to their location at a high trophic level on the marine food chain, and the fact that they tend to breed in the same location every year. 53 individual eggs of four species (Least Tern, Clapper Rail, Forsters Tern, and Caspian Tern) and multiple nesting sites were tested. The results were compared with eggs gathered from these species in Washington state. The total PBDE levels measured in the California egg samples averaged 6.2 ppm (fat based), with a range of 0.30 to 62 ppm. Five PBDE congeners (PBDE 47, PBDE 99, PBDE 100, PBDE 153, and PBDE 154), were found in all egg samples from SF Bay and Washington state. When compared, the average level in eggs from San Francisco Bay birds was over 1000 ng/g fat higher than the average level measured in eggs from Washington state birds. As shown in Figure 1 below, the PBDE levels in bird eggs, seals, and humans all showed a similar congener pattern.

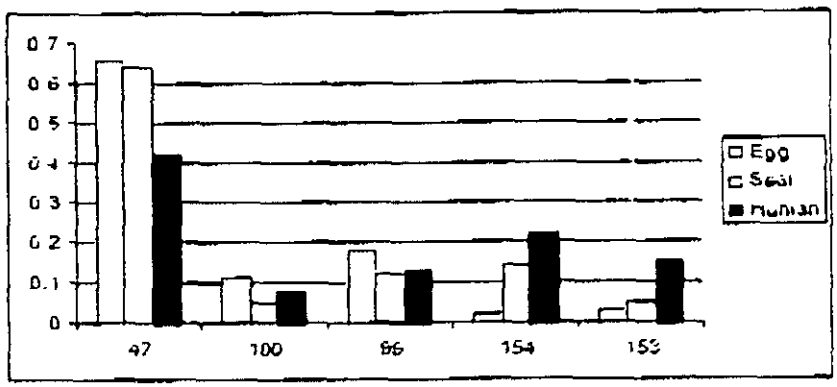


Figure 1. Relative congener patterns of PBDE in cormorant eggs, seals and samples of human breast adipose tissue from the San Francisco Bay Area

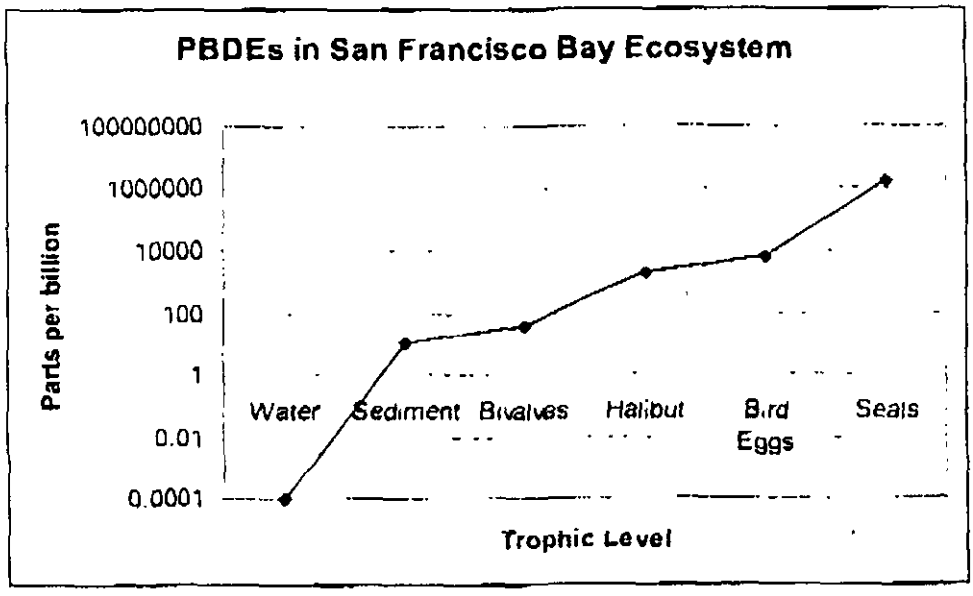
Data collected by the San Francisco Estuary Institute (SFEI) in 2001 on PBDE levels in bird eggs (double-crested cormorants and Samuels song sparrows), showed that these chemicals were present in all samples analyzed at levels ranging from 0-245 ug/kg depending on the congener, location, and species. (http://www.sfei.org/cmr/data/birdeggtables/CISNET_Egg_pbde.htm) SFEI also collected data on bivalves in the San Francisco Bay, detecting levels that averaged about 40 ppb in these animals. (http://www.sfei.org/rmp/2002/CSV%20FILES/PBDE%20TISSUE.csv) In addition, SFEI collected sediment samples from the San Francisco Bay in 2002, detecting BDE 99 in most of the samples tested, and reporting numerous detections of other congeners as well. (http://www.sfei.org/rmp/2002/CSV%20FILES/PBDE%20SEDIMENT.csv) As expected, detections of dissolved PBDEs in the water column were at far lower levels than the levels reported in sediment. (http://www.sfei.org/rmp/2002/CSV%20FILES/PBDE%20DISSOLVED.csv)

These data allow us to estimate the bioconcentration of the PBDEs in the San Francisco Bay. Table 1 below shows that these chemicals may concentrate by as much as 12 orders of magnitude from the water column to the blubber of seals.

Table 1: Bioconcentration of PBDEs in the San Francisco Bay Ecosystem

Trophic Level	Average PBDEs (2002)	Source:
Water column	0.000103 ug/L (ppb)	SFEI
Sediment	11.8 ug/kg (ppb)	SFEI
Bivalves	40.1 ug/kg (ppb)	SFEI
Halibut	2,000 ppb	EWG
Bird Eggs	6200 pg/g fat (ppb)	She et al.
Seals	1,730,000 pg/g fat (ppb)	She et al.

Figure 2



PBDEs Chemical Structure and Use

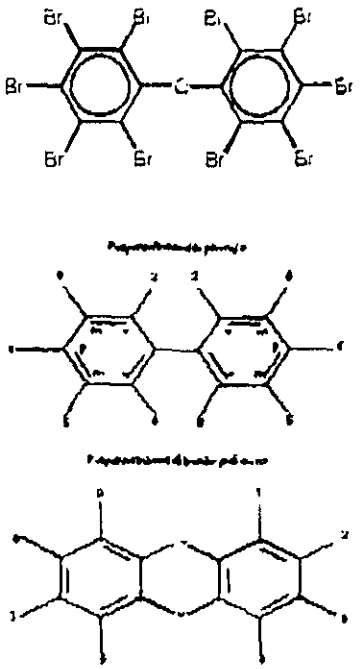
The polybrominated diphenyl ethers (PBDEs) are brominated organic compounds with chemical structures similar to dioxins and PCBs. Global production of these chemicals is approximately 40,000 tons per year for use as fire retardants in plastics and textiles. These chemicals are of major toxicological concern due to their environmental persistence and bioaccumulation, and due to available data on their toxicity. Environmental and human exposure studies worldwide are identifying dramatic increases in PBDE levels in sediments, biota, and mammalian tissues. The increases are generally logarithmic, indicating potential for significant health and environmental impairment in the relatively near future if the pollution from these chemicals is not addressed very soon. We are convinced that these chemicals will pose a threat to water on a par with the PCBs. Accordingly we ask the SWRCB to move rapidly to list these chemicals under Section 303(d) and immediately begin enhanced monitoring programs, identification of discharges, and pollution reduction activities. Waiting and watching will simply allow the health and environmental threat to grow.

PBDEs in current widespread use have somewhat different environmental persistence and toxicity profiles. Deca-BDE accounts for the largest percentage of the market. However, the lower brominated congeners (hexa-BDE and below) are more readily absorbed by animals and their half-lives in living organisms are comparable to that of 2,3,7,8-TCDD (dioxin). In some test systems, tetra-and penta-BDE had higher bioaccumulation potential than the PCBs. Strategies aimed only at the penta-BDEs may

not solve the problem, because there is evidence that in the presence of sunlight, the higher brominated forms may degrade to the more readily absorbed, persistent and bioaccumulative lower-brominated forms.

Figure 3: Comparative Structures of PBDEs, PCBs, and Dioxins

Polybrominated diphenyl ethers



PBDEs Toxicity Summary

Dioxin-Like and PCB-Like Activity

The PBDEs are structurally very similar to the PCBs and the dioxins. The toxicological evidence to date indicates that these chemicals also share many common traits. The tetra- to hexa-BDEs are strong inducers of liver enzymes in the rodent. In particular, these chemicals induce the cytochrome p-450 (CYP) enzyme system in the liver, including CYP1A1 as assessed by the standard test using ethoxyresorufin-o-deethylase (EROD). EROD activity is considered a hallmark of dioxin-like compounds, and penta-BDE is a more powerful inducer of EROD activity than commercial PCB mixtures."

PBDEs also interact with the aryl hydrocarbon (Ah) receptor, another hallmark of dioxin-like activity. In a study of 17 PBDE congeners, seven acted as Ah receptor

agonists and nine acted as antagonists when co-treated with 2,3,7,8-TCDD. The potencies of the PBDE agonists were similar to some PCBs. When PBDEs and PCBs were administered together, the effects were additive, suggesting a similar mechanism of action."[iii]

Immune Suppression

Dioxins and PCBs are known to cause immunosuppression in laboratory animals, and probably in humans. In standardized tests of immune response, a mixture of PBDEs resulted in immune suppression in mice.[iv] In addition to reduction in immune function, cellular changes were observed in organs critical to immune function such as the spleen and thymus. The immunosuppressive effects of PBDEs have been reported to exceed the effects of PCBs in laboratory animals. Suppressed production of IgG antibodies after stimulation with chemicals that would normally enhance IgG production indicates that these chemicals may have significant adverse effects on immune function.[v] Negative findings reported from some *in vitro* standardized immunotoxicity tests are probably due to the fact that the immune effects are indirect and require interaction with other systems such as the Ah receptor. Simple *in vitro* systems fail to reflect the real effects of these chemicals in the body. This hypothesis is supported by the failure of these same systems to detect the known immunotoxic effects of the PCBs.[vi]

PBDEs and Cancer

Swedish hospital patients with non-Hodgkin's lymphoma were reported in one study to have higher concentrations of tetra-BDE in their fat.[vii] There are no other relevant cancer data from human epidemiologic studies. Mutagenicity studies have shown conflicting results, with several studies failing to show cell mutations in *in vitro* test systems, whereas other studies have shown evidence of an epoxide intermediate (a metabolite that would be expected to cause mutations) and of genotoxicity.[viii ix]

There is a major gap in the cancer toxicology database for the PBDEs. Only the deca-BDE form, which is poorly absorbed and rapidly eliminated, has been tested for carcinogenicity in the rodent. Nonetheless, in studies performed by the National Toxicology Program (NTP), deca-BDE produced statistically significant increases in hepatocellular adenomas and carcinomas in male mice, and marginal increases in thyroid follicular cell adenomas and carcinomas in both male and female mice. In rats, statistically significant dose-related increases in liver adenomas were seen in both males and females, and significantly increased numbers of pancreatic adenomas were seen in the males.[x]

Endocrine Disruption

The hormonal effects of the PBDEs, are of great toxicological concern. It is clear that the lower-brominated PBDEs disrupt thyroid hormone. In rats, penta-BDE reduces

thyroid hormone levels and increases thyroid hyperplasia even at the lowest doses tested - 2 mg/kg/day.[xi] In mice, one single dose of penta-BDE at only 0.8 mg/kg caused continued suppression of thyroid hormone levels more than a week later.[xii] When animals were exposed to both PBDEs and PCBs, the effect on thyroid hormone suppression was additive.[xiii]

The hormonal effects of PBDEs are not expected to be peculiar to laboratory rats. In fact, men working in the production of deca-BDE were found to have a higher than expected rate of hypothyroidism. Four out of 35 exposed workers had clinically-significant hypothyroidism whereas no cases of thyroid dysfunction were identified among 89 age and sex-matched workers who were not exposed to these chemicals.[xiv]

Neuro-Behavioral and Developmental Effects

In standard developmental toxicology studies, the PBDEs cause increased fetal death, abnormal formation of the skull, enlarged heart, and subcutaneous edema. The doses that caused fetal toxicity were lower than the doses that affected the mothers.[xiv xv] It should be noted, however, that the standard developmental toxicology studies detect only obvious birth defects and toxicity, and are unable to detect more subtle alterations in neurologic function and behavior.

Single low doses of penta-BDE (only 0.8 mg/kg), administered to mice during the vulnerable period for brain development just after birth, resulted in permanent neurological dysfunction. These mice were permanently more sluggish and had decreased spontaneous activity levels throughout their lives, worsening with age, and permanent reductions in learning and memory.[xv] These important findings have been confirmed in several different studies, and may be related to alterations in the cholinergic system or to suppression of thyroid hormone during a critical period of brain development.[xvi xvii xix] Thyroid suppression during development has been shown to cause permanent subtle impairment of neurological function.[xx]

Summary

The PBDEs are of serious toxicological concern on the basis of their environmental properties, their chemical structure, and the toxicological evidence to date. These chemicals are environmentally persistent and are known to bioaccumulate. The chemical structure of PBDEs is similar to the PCBs, dioxins, and other organohalogen compounds of serious concern. Although the toxicological database is incomplete, there is evidence that these chemicals have dioxin-like and PCB-like properties, including interference with enzyme systems and with the aryl hydrocarbon receptor. The effects of greatest concern to date are anti-thyroid effects and adverse effects on neurological development from early life exposures.

The levels of the PBDEs in biota in the San Francisco Bay is a serious cause for concern. The fact that the concentrations are among the highest reported anywhere in the world, combined with the evidence that the concentrations are increasing logarithmically, means that it is imperative to act quickly. A Section 303(d) listing will allow action to reduce exposures before the toxicity becomes severe enough to cause serious damage to the entire ecosystem in the San Francisco Bay.

* * * *

Thank you for the opportunity to provide these comments. Please do not hesitate to call if you have any questions.

Sincerely,

David S. Beckman
Senior Attorney and Director,
Coastal Water Quality Project

cc: Celeste Cantu, Executive Director, SWRCB
Craig Wilson, Chief Counsel, SWRCB
Craig J. Wilson, Chief, Monitoring and TMDL Listing Unit, SWRCB
Alexis Strauss, U.S. EPA, Region IX

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[i] She J, Petreas M, Winkler J, Visita P, McKinney M, Kopec D. PBDEs in the San Francisco Bay Area: measurements in harbor seal blubber and human breast adipose tissue. Chemosphere 46:697-707, 2002.

[ii] Von Meyerinck L, Hufnagel B, Schmoldt A, Benthe HF. Induction of rat liver microsomal cytochrome P-450 by the pentabromo diphenyl ether Bromkal 70 and half-lives of its components in the adipose tissue. Toxicology 61(3): 259-274, 1990.

[iii] Hallgren S, Darnerud PO. Effects of polybrominated diphenyle ethers (PBDEs), polychlorinated biphenyls (PCBs), and chlorinated paraffins (CPs) on thyroid hormone levels and enzyme activities in rats. Organohalogen Compounds 35:391-394, 1998.

[iv] Fowles JR, Fairbrother A, Baecher-Steppan L, Kerkvliet NI. Immunologic and endocrine effects of the flame-retardant pentabromodiphenyl ether (DE-71) in C57BL/6J mice. Toxicology 86:49-61, 1994.

[v] Darnerud PO, Thuvander A. Effects of polybrominated diphenyl ether (PBDE) and polychlorinated biphenyl (PCB) on some immunological parameters after oral exposure in rats and mice. Toxicol Environ Chem 70.229-242, 1999.

[vi] Darnerud PO, Eriksen GS, Johannesson T, Larsen PB, Viluksela M. Polybrominated diphenyl ethers: Occurrence, dietary exposure, and toxicology. Environ Health Perspect 109(Suppl 1) 49-68, 2001.

[vii] Hardell L, Lindstrom G, van Bavel B, Wingfors H, Sundelin E, Liljegren G. Concentrations of the flame retardant 2,2', 4,4'-tetrabrominated diphenyl ether in human adipose tissue in Swedish persons and the risk for non-Hodgkin lymphoma. Oncol Res 10(8):429-432, 1998.

[viii] Orn U, Klasson-Wehler E. Metabolism of 2,2', 4,4'-tetrabromodiphenyl ether in rat and mouse. Xenobiotica 28(2): 199-211, 1998.

[xi] Helleday T, Tuominen KL, Bergman A, Jenssen D. Brominated flame retardants induce intragenetic recombination in mammalian cells. Mutat Res 439(2):137-147, 1999.
[xii] NTP. Toxicology and carcinogenesis studies of decabromodiphenyl oxide (CAS No. 1163-19-5) in F344/N rats and B6C3F1 mice (feed studies). TR 309. Research Triangle Park, NC: National Toxicology Program, 1986.
[xiii] U.S. EPA TSCA Section 8e report numbers 04760A, 04856A, 05420A.
[xiv] Fowles JR, Fairbrother A, Baecher-Steppan L, Kerkvliet NI. Immunologic and endocrine effects of the flame-retardant pentabromodiphenyl ether (DE-71) in C57BL/6J mice. Toxicology 86(1-2): 49-61, 1994.
[xv] Bahn AK, Mills JL, Snyder PJ, Gann PH, Houten L, Bialik O, et al. Hypothyroidism in workers exposed to polybrominated biphenyls. N Engl J Med 302(1):31-33, 1980.
[xvi] WHO. Brominated diphenyl ethers. IPCS Environmental Health Criteria 162. Geneva: World Health Organization, 1994.
[xvii] U.S. EPA. Brominated diphenyl ethers. Chemical Hazard Information Profile. Washington, DC, 1986.
[xviii] Eriksson P, Jakobsson E, Fredriksson A. Developmental neurotoxicity of brominated flame-retardants, polybrominated diphenyl ethers and tetrabromo-bisphenol A. Organohalogen Compounds 35:375-377, 1998.
[xix] Eriksson P, Viberg H, Jakobssen E, Orn U, Fredriksson A. PBDE 2,2',4,4'-pentabromodiphenylether causes permanent neurotoxic effects during a defined period of neonatal brain development. Organohalogen Compounds 40:333-336, 1999.
[xx] Viberg H, Fredriksson A, Jacobsson E, Ohrn U, Eriksson P. Developmental neurotoxic effects of 2,2',4,4',5,5'-*pentabromodiphenyl ether (PBDE 99) in the neonatal mouse*. Toxicologist 54(1):290, 2000.
[xxi] Eriksson P, Jakobsson E, Fredriksson A. Brominated flame retardants: A novel class of developmental neurotoxicants in our environment? Environ Health Perspect 109(9):903-908, 2001.
[xxii] Haddow JE, Palomaki GE, Allan WC, Williams JR, Knight GJ, Gagnon J, et al. Maternal thyroid deficiency during pregnancy and subsequent psychological development in the child. N Engl J Med 341:549-555, 1999.