RISK-BASED APPROACH FOR PCB's FISH CONSUMPTION ADVISORIES IN CONNECTICUT

MAY, 1999

BY
Brian Toal, M.S.P. H.
Director of Toxic Hazards Assessment
&
Gary Ginsberg, Ph.D. Toxicologist

Department of Public Health
Environmental Epidemiology & Occupational Health
(860)509-7742

Risk-Based Approach for PCBs Fish Consumption Advisories in Connecticut

1.0 Introduction

The Connecticut Department of Public Health (DPH), in conjuction with the Connecticut Department of Environmental Protection (DEP), have issued fish consumption advisories due to a variety of contaminatants in fish (including PCBs) for over a decade. The basis for the PCBs advisory program from its inception in Connecticut has been the Food and Drug Administration (FDA) Tolerance Level for PCBs in fish (2.0 ppm). USEPA has strongly endorsed a more risk-based methodology and has jointly issued a letter with FDA which states: "While FDA's action levels ensure a safe food supply for consumers of commercial fish, they may not be appropriate levels for ensuring the safety of those who consume locally caught fish." A number of other states have recently developed more risk-based approaches that rely upon toxicity values similar or identical to those from USEPA's IRIS system for PCBs. Table 1 summarizes the approaches used for PCBs advisories in the Great Lakes States, the northeast, and several mid-Atlantic states.

The goal of this document is to evaluate the existing FDA-based approach used in Connecticut, as well as the more recent risk-based approaches to determine the need to update DPH's advisory program for PCBs. Given that the FDA Tolerance Level for PCBs has not changed in the past 17 years while advances have been made in the areas of PCBs toxicology in animals (e.g., carcinogenicity, endocrine/developmental effects, immune system effects) and epidemiology, re-evaluation of the DPH advisory program is important to make sure it incorporates the latest science. In addition, DPH moved from a FDA-based to a risk-based fish advisory program for mercury in 1996. Therefore,to the extent possible,a goal is to make the PCBs and mercury advisory programs consistent with respect to how they evaluate health risks from fish consumption.

The current PCBs advisory program lists 6 waterbodies (Long Island Sound, Housatonic River, Lake Housatonic, Quinnipiac River, Eight Mile River, Connecticut River) and a variety of species within these waterbodies as needing advisories due to PCBs contamination. This document provides a risk-based approach for evaluating the significance of PCB fish concentrations, and then utilizes this approach to determine whether the existing advice is still appropriate for fish caught in Connecticut water bodies.

2.0 Existing Advisory Program for PCBs

DPH currently uses an adaptation of the FDA Tolerance Level first developed in Wisconsin to set PCB consumption advisories. As seen in Table 1, Wisconsin has since changed to the risk-based, Great Lakes Protocol.

Table 1. Summary of PCB Advisory Approaches in Other States[1]

Listing Based Upon Whether States Follow the
Great Lakes Protocol (GLP)[1], the FDA Tolerance Level[2], or other Approaches

Great Lake States	
(MN, WI, OH, IL, PA):	GLP
(NY, MI):	GLP for their portion of Great Lakes; FDA approach for inland waterbodies
New Jersey:	FDA, but goal to switch to risk-based
Virginia, Maryland, Delaware:	Recently switched to GLP; Delaware - no 50% cooking reduction
Maine:	Cancer risk-based approach beginning at fish conc. of 0.01ppm
Vermont:	FDA, but very little PCB data in fish

[1]Table summarizes telephone conversation with Jeff Bigler, USEPA, 3/99
[2]Great Lakes Protocol: risk-based approach that limits PCB exposures to 0.05 ug/kg/d.
[3]FDA Tolerance Level: 2 ppm cutoff above which fish cannot enter commercial markets

The modified FDA Tolerance Level approach that currently underlies Connecticut's advisories is as follows:

- If all fish in a waterbody have PCB levels below 2 ppm, no advice needed;
- If ≥10% of fish in a waterbody exceed 2 ppm:
  - high risk individuals (pregnant women, women planning to become pregnant, nursing mothers, young children) advised not to eat the fish;
  - everyone else advised to eat no more than two meals per month;
- If ≥50% of fish in a waterbody exceed 2 ppm, "do not eat" advice for everyone.

This approach has required individual rather than composite sampling of fish to determine the percentage of fish exceeding 2 ppm in a given waterbody. The basis for the 2 ppm FDA Tolerance level is described in the next section.

2.1 Basis for FDA Tolerance Level

FDA summarized the derivation of their PCB Tolerance Level of 2 ppm in a 1982 publication (Cordle, et al., 1982). The publication provided an exposure assessment for the U.S. population which suggested that the major PCB-containing fish species had concentrations between 0.25-1.7 ppm. Based upon the amounts of these species eaten by fish consumers in a 1978-79 survey of nearly 26,000 individuals, FDA estimated a 50% consumption rate of 8.25g /d and a 90th percentile rate of 23 g/d. This yielded daily exposure estimates at a 2 ppm Tolerance Level that ranged between 0.08 to 0.21 ug/kg/d for a 70 kg adult. These exposure levels were below an acceptable PCBs exposure level of 1 ug/kg/d derived by FDA for infants and young children based upon the Japanese Yusho incident. Therefore, a 2 ppm tolerance appeared to be health protective.

The 1 ug/kg/d exposure level was derived from the Yusho incident in which humans were poisoned by cooking oil contaminated by PCBs. A key FDA assumption was that exposure in the U.S. population would be significant for only 1000 days (2.7 years) from the time of their analysis (1982) due to the expectation that PCB fish concentrations would drop below levels of concern in that time. FDA also utilized a 10 fold safety factor, thus setting the acceptable exposure level 10 fold below that experienced in the Yusho incident. It should be noted that the Japanese group exposed to the contaminated cooking oil experienced overt health effects including chloracne, neurological disorders (visual disturbances, numbness and weakness in limbs) and disturbances in liver function. This population also had an increased cancer risk and offspring had skin pigmentation abnormalities and multiple neurobehavioral effects that persisted for years. When extrapolating from such marked effects in humans, a 10 fold safety factor does not provide assurance that that some degree of toxicity would not be experienced in the general population or that effects wouldn't occur in sensitive individuals.

However, the larger issue with the Yusho dataset is that it is not a considered to be valid for PCB risk assessment (ATSDR, 1998). A major limitation is that the incident involved a mixed exposure to PCBs and chlorinated dibenzofurans (CDFs). CDFs became part of the exposure both due to the heating of the PCBs before they entered the cooking oil as well due to a likely conversion of PCBs to CDFs during the cooking process. Since the role of CDFs and PCBs cannot easily be separated in this case, the Yusho incident is not optimal for dose-response assessment of PCB effects in humans (ATSDR, 1998).

Therefore, the risk assessment approach that supports the FDA 2 ppm PCB Tolerance Level is limited in the following ways:

- It is quite dated, not taking into consideration the more recent monkey studies showing low dose effects on immune function, reproduction, and fetal development (Tryphonas, 1989; Arnold, 1995). The FDA Tolerance also does not take into consideration the recent epidemiologic investigations that are supportive of neurobehavioral effects in offspring of women who ate PCB-contaminated fish during pregnancy in Michigan (Jacobson and Jacobson, 1996), North Carolina (Rogan et al., 1986), and in the Netherlands (Patandin, et al., 1999).

- It relies primarily upon the Yusho incident to develop an acceptable exposure level. This incident showed marked health effects but its relevance to setting acceptable exposure levels for PCBs is decreased by several factors, the most important being the likely contribution of CDFs to the toxicity seen. FDA discussed some of the other data available at the time (early rat and monkey studies) which suggested that health effects might be possible below the 1 ug/kg/d exposure level. However, FDA did not strongly consider these findings because of several uncertainties and since monkeys appeared to be more sensitive than rodents or humans.

- It developed an acceptable exposure level under the assumption that exposure would not be chronic, but limited to 2.7 years on the basis that PCB levels in foods were expected to decline. However, PCBs are very persistent and they continue to enter the environment from a variety of old industrial sites (ATSDR, 1998). For the purpose of risk assessment it is prudent to consider current exposures from fish consumption to be chronic rather than set allowable exposures on the high end because in the future PCB exposures may decline, thus offsetting some of the chronic risks.

- It is based upon national average levels of fish consumption which do not reflect the amounts of fish consumption possible in sport or subsistence fisher families or in other high end fish consumers. Further, the consumption survey data are from the 1970's which may underestimate current levels of fish consumption. A recent survey across a broad spectrum of Connecticut residents (Balcom, 1999) found fish consumption rates to be considerably higher than the national average fish

consumption data used by FDA (Cordle, et al., 1982). For example, the mean fish consumption rate in CT's general population was 28g/day, with mean rates in sport fishing, minority, and Southeast Asian immigrant families ranging between 40 to 60g/day (90th percentile rates over 100g/d). FDA assessed PCBs exposure based upon a range of fish consumption of 8.25 (mean) to 23g/d (90th percentile) for the top 20 PCBs-containing species. Given that many of these species are commonly caught or available in Connecticut, much of the Connecticut fish-eating population are likely to consume greater amounts of PCBs-contaminated fish than the 1970s national estimates used by FDA.

- Perhaps most importantly, it is based upon the premise of a single bright-line cutoff for fish consumption, not recognizing that risks vary depending upon the fish concentration and frequency of meal consumption.

These factors, plus the recent development of lower acceptable exposure levels for PCBs by USEPA (RfD of 0.02 to 0.07 ug/kg/d) (USEPA, IRIS), ATSDR (Draft Minimum Risk Level of 0.02 ug/kg/d) (ATSDR, 1998) and the Great Lakes Protocol (Health Protection Value of 0.05 ug/kg/d) (GLSFATF, 1993) weigh against the continued use of the FDA Tolerance Level as the sole determinant in deriving fish consumption advisories in Connecticut.

3.0 Review of the Health Effects of PCBs

PCBs are a mixture of chlorine-bearing biphenyls, each congener in the mixture having a unique number and/or arrangement of chlorines on the biphenyl ring. The more chlorine atoms on the biphenyl ring, the more environmentally stable, less volatile, and more bioaccumulative the overall molecule becomes. In toxicity testing, PCBs have traditionally been tested in the form of commercially available Aroclor mixtures, with lower chlorination mixtures (e.g., Aroclor 1016 - 16% chlorine) being somewhat less toxic and carcinogenic than higher chlorination Aroclors (e.g., 1254 - 54% chlorine). Recent studies have focused on certain PCB congeners in terms of their ability to mimic the toxicity of dioxin (coplanar congeners most active), and to alter hormone status (oxygenated metabolites are estrogenic and impair thyroid function; coplanar congeners are anti-estrogenic - similar to dioxin). However, long-term health effects studies are generally not available for individual congeners and a dioxin-like toxicity equivalency factor (TEF) approach is somewhat controversial. NIEHS/NTP has plans to test a number of PCB congeners to determine if predictive methods for developing TEFs are borne out in 2 year cancer bioassays (Bucher, 1998). Currently, most agencies utilize toxicology data for the Aroclor mixtures rather than individual congeners in developing health-protective standards.

The following sections provide a brief summary of the toxicology of PCBs in animals and humans, providing an indication of the most sensitive endpoints.

3.1 PCB Effects in Animals

PCBs have been shown to cause a diverse array of biochemical, hormonal, and toxic effects in animals (ATSDR, 1998). PCBs have a long half-life in mammals (months to years) which can lead to rising concentrations in blood and tissues (particularly fatty tissues) if exposure is on a frequent basis. This buildup may, in part, be responsible for the much greater potency of PCBs in long-term studies (effects at doses as low as 0.005 mg/kg/d) as compared to short-term/acute studies (effects not seen below 1 mg/kg/d). Monkeys are generally more susceptible than rodents, although in terms of cancer potency, the only data are from rat studies. This is because lifetime cancer bioassays are not commonly conducted in primate species. The following points highlight the major effects found, with some indication of the exposure levels needed to produce these effects.

- Liver: PCBs are powerful inducers of mixed function oxidases, which will change the way other xenobiotics are metabolized; relatively high doses in monkeys have caused liver necrosis and gall bladder hypertrophy (0.2 mg/kg/d) with increased liver weight as low as 0.08 mg/kg/d; PCBs have also caused porphyria (dysfunction of heme synthesis in liver) and lipid accumulation.

- Immune System: PCBs have decreased antibody production post antigenic stimulation as documented by decreased IgM and IgG titers in the sheep red blood cell assay (a test that is generally predictive of immunotoxic compounds); this effect occurred at 0.005 mg/kg/d in a 1989 monkey study (Tryphonas, 1989), while a similar effect occurred in an earlier (1978) monkey study that showed a NOAEL for this effect of 0.1 mg/kg/d. This decreased antibody response after exposure to PCBs may be responsible for the increased susceptibility to infection seen in 2 monkeys exposed to 0.1-0.2 mg/kg/d (Barsotti, 1976) and in mice exposed to 22 mg/kg/d.

- Developmental: the most sensitive endpoint from in utero exposure is neurobehavioral development. Decreased birth weight and subsequent learning deficits were found in monkeys from chronic maternal exposure to 0.03 mg/kg/d with the developmental NOAEL identified at 0.007 mg/kg/d; similar learning deficits have been seen in rats, although those studies used somewhat higher doses and shorter exposure periods; PCBs can be teratogenic and fetotoxic at still higher doses (i.e. 8 to 244 mg/kg/d) as found in mice and rats exposed for from one to several days during gestation.

- Reproductive: a variety of adverse reproductive effects have been seen across several species including decreased fertility (males and females both affected), prolonged estrus, and prolonged menstruation; monkeys and mink appear most sensitive, with effects on reproduction in the range of 0.1-1mg/kg/d.

- Endocrine: the thyroid gland is an important target which may mediate PCB effects on body weight, growth, and reproduction. Doses as low as 0.09 mg/kg/d lowered

serum thyroid hormone levels in rats: research is ongoing regarding estrogenic/anti-estrogenic effects.

- Blood: anemia has been shown in monkeys at doses as low as 0.2 mg/kg/d with less dramatic hematological effects (decreased platelets) as low as 0.02 mg/kg/d.

- Skin: monkeys experienced a variety of dermal and cutical effects in chronic studies including chloracne (keratin clogging sebaceuous gland pores, comedomes and inflammatory folliculits) at 0.1 mg/kg/d, lost, cracked or otherwise altered nails and nailbeds at 0.005 mg/kg/d. and facial swelling, especially swollen eyelids, and hair loss at 0.1 mg/kg/d.

- Cancer: higher chlorination PCBs, particularly Aroclor 1260 is carcinogenic in rat liver, as found in 3 separate bioassays; doses between 1 and 5 mg/kg/d produced rat liver tumors in these studies. A gender difference is suggested by higher tumor rates in females in two of the studies. EPA, IARC, and NTP consider PCBs as probable human carcinogens with EPA's IRIS database provides different cancer slope factors for higher chlorination vs. lower chlorination mixtures (see Section 4.2).

3.2 Human Studies

3.2.1 Worker Studies

Studies of PCB-exposed capacitor and transformer maintenance workers showed some of the effects seen in animal studies, while others were not evident (ATSDR, 1998). The exposure levels in worker studies were not sufficiently well-defined to establish dose response relationships, although some comparison is possible across species on a blood concentration/body burden basis. The difference in route of exposure between the workplace (inhalation and dermal exposures) and the animal toxicology database (predominantly oral studies) may account for some of the differences in effects found to date.

Similar to the animal database, hepatic and dermal effects have been documented in workers, with a suggestion that the endocrine system may also have been affected.. Hepatomegaly, increases in liver enzymes in serum, and increased urinary excretion of porphryins have been seen in workers exposed by inhalation to 0.048 to 0.275 mg/m^3 (0.007 to 0.04 mg/kg/d) for 12 years. Increased serum enzymes and other indices of liver effects were found in capacitor workers exposed for 17 years at a mean concentration of 0.69 mg/m^3. Chloracne and skin rashes have been found in occupational studies of workers exposed to 0.1 mg/m^3 (0.014mg/kg/d) or higher, with other studies showing pigmentation of skin and nails and skin thickening at an estimated exposure of 0.003 mg/m^3 for over 5 years. However, the contribution of dermal contact to the overall dose to the skin is not known from such studies.

Occupational studies have also examined whether exposure to PCBs is linked to excess cancer risk. While a number of cohort studies have shown higher rates of liver/biliary or other cancers (hematologic neoplasms, kidney carcinoma), limits in the ability to reconstruct the PCBs exposure dose and exposure to other chemicals, combined with conflicting results from different studies, makes interpretation of the human cancer data difficult. Therefore, the human data can be seen as generally supportive of the PCBs classification as "probable human carcinogen" with a need to rely upon the animal dose-response data to project risk to humans.

3.2 Studies of Fish-Eating Populations

3.2.1 Neurobehavioral Effects

Maternal exposure to PCBs in fish has been correlated to adverse neurobehavioral effects in children in three major cohort studies to date. The Michigan Maternal Infant Chort Study was initiated in the mid-1980s, involving 242 infants from mothers who had moderate to high intake of Lake Michigan fish during pregnancy and for the preceding 6 years. The control group of 71infants were from mothers who had not eaten Lake Michigan fish. The fish-eating cohort tested at birth had statistically decreased gestational age (avg. decrease of 4.9 days), birth weight (160-190 g), and head circumference (0.6 cm). Follow-up at 5-7 months of age indicated depressed responsiveness, impaired visual recognition, and poorer short-term memory in the PCBs group. Further follow-up at 4 and 11years of age showed a continuing pattern of neurobehavioral and cognitive deficits in the PCBs/fish cohort. Exposure during the in utero rather than post-natal period (e.g., thru breast milk or later in life) appeared to be the exposure pathway conferring the most risk. Overall, this longitudinal study is limited by not measuring or controlling for in utero exposures to mercury or other organochlorines for which concomitant exposure with PCBs can be expected. Out of a large number of potential confounders evaluated, maternal consumption of alcohol, caffeine, and cold medicines cannot be ruled out as contributing to the effects seen. Dose reconstruction for the Michigan PCBs/fish cohort has been attempted based upon two separate approaches (Tilson, 1990; ATSDR, 1998; Minnesota DOH,1990; Minnesota DOH, 1992):

1) Maternal body burden of PCBs and PCBs half-life to estimate long-term daily dose - mean PCBs concentrations in breast milk fat were in the range of 1 to 3.4 ug/g fat in the cohort of fish eaters whose offspring showed evidence of neurobehavioral effects. Based upon an assumed body fat content of 25% and a body weight of 60 kg, the total PCBs body burden would be 15 to 51 mg; this is then assumed to have accumulated slowly over the mother's life (assumed age of 25 years) with an estimated PCB half-life in vivo of 1 to 4.8 years (Minnesota DOH, 1992; ATSDR, 1998). This approach yields a chronic human LOAEL in the affected Michigan cohort of 0.0003 to 0.001 mg/kg/d.

2) Estimates of maternal fish consumption and PCBs concentrations in fish - Minnesota DOH (1990, 1992) utilized fish consumption survey information for the Michigan fish-eating cohort to estimate maternal Lake Michigan fish consumption and PCB exposures. They estimated that effects were seen in women eating as little as 2.0-3.4 kg of lake trout per year for 6 years. The midpoint of this range corresponds to a daily ingestion rate of 7.4 g/d, which Minnesota combined with an estimate of lake trout PCBs concentrations (1980 survey data from Michigan, mean = 4.12 ppm). This yielded a human LOAEL of 0.0005 mg/kg/d.

The two approaches yielded approximately the same LOAEL for the Michigan cohort; further, as discussed below, this LOAEL is approximately 10 fold lower than the monkey LOAEL for developmental effects (0.005 mg/kg/d), which fits with standard risk assessment practice in using a 10 fold safety factor in extrapolating non-cancer effects across species.

Other studies of in utero exposure to PCBs through maternal fish consumption are supportive of the findings in the Michigan cohort. The North Carolina Breast Milk and Formula Project enrolled 858 women for the assessment of in utero and post-natal exposures to organochlorines and health outcomes from birth thru 1 year. In utero exposure to PCBs was evaluated based upon maternal and cord blood samples and placental samples. Similar to the Michigan cohort, milk fat PCB concentrations in the low ppm range were associated with pyschomotor deficits in the first year of life, but birth parameters (body weight, head circumference) were not affected in the North Carolina study. Follow-up testing found lasting effects through age 2, but not in testing done in later years. Once again, in utero rather than post-natal exposure appeared to be most important for developmental risks from PCBs. This study also was limited by not collecting data on exposure to other chemicals in fish which may have affected the neurological endpoints.

The recent PCB/Dioxin series of publications from the Netherlands (e.g, Huisman, et al., 1995; Patandin, et al., 1999) also suggest that in utero exposure to PCBs affects neurological development. The study evaluated 418 newborns in 2 cities with maternal plasma and breast milk concentrations of PCBs, dioxins, and furans used to determine the degree of in utero and post-natal exposure. The study population was not especially exposed to fish or PCBs, with the median breast milk PCBs concentration being 0.4 ug/g fat. Neurological testing conducted 10 to 21 days post-partum indicated an association between PCBs in utero exposure and hypotonia and other neurological deficits. Similar results were seen at 18 months while no evidence of adverse neurological effects were found at 42 months. A battery of cognitive/psychomotor tests conducted at three months indicated lower scores due to in utero but not postnatal (breastfeeding) exposure. This pattern continued out to 42 months where overall cognitive functioning and short and long-term memory tasks were affected (Patandin, 1999). However, the relative contribution of PCBs vs. dioxins/furans or other related chemicals was not clearly established. Again, in utero exposure to mercury was not investigated.

Support for the neurobehavioral findings described above comes from a cohort of 536 newborns whose mothers consumed Lake Ontario fish during pregnancy. Newborns examined within the first 48 hours after birth were found to have a greater number of abnormal reflexes and wereless attracted to external stimuli if they were from mothers who consumed high amounts of PCB contaminated fish (>40 pounds) (EPA/ATSDR, 1998).

3.2.2 Endocrine and Immune System Effects

Suggestive correlations between PCB exposure and endocrine and immune system changes are consistent with evidence from animal studies. However, the dose response relationships and clinical relevance of these effects are not well established. The Dutch PCBs/dioxins study found that levels of these organochlorines in breast milk correlated with lower T3 and thyroxine levels in mother's serum, higher TSH levels in offspring 2 weeks and 3 months post-partum, and lower thyroxine in offspring at week 2. Various biochemical indices of immune status have shown alterations in PCB-exposed or fatty fish eating populations (EPA/ATSDR, 1998). These findings include decreased NK cells in fish-eating populations in Sweden and in men eating fatty fish from the Baltic sea, altered monocyte and granulocyte levels at 3 months of age and increased cytotoxic T-cells and total T-cells at 18 months in the Dutch PCBs/dioxins study, and altered T-cell ratios in Inuit 6 and 12 month old children whose mothers had elevated exposure to PCBs. Yu-Cheng and Yusho populations have also exhibited altered immune status.

3.3 Summary of Animal and Human Evidence

Animal and human data are in general concordance on the qualitative aspects of the PCBs effects profile. At high doses, dermal and hepatic effects are manifest, with the most sensitive endpoints at low doses appearing to be immune function and neurobehavioral development. There is also limited support from occupational studies that the positive carcinogenicity findings in rats may translate into elevated cancer risks in PCBs-exposed workers. While the human data are confounded by the likely exposure to other chemicals in fish or in the workplace, the general concordance between human and animal data suggest that PCBs were important etiologic agents in the human studies. This is because the animal studies were not confounded by exposure to other chemicals indicating the ability of PCBs on their own to cause these effects. Where a quantitative estimate of PCBs potency in humans has been made (developmental endpoints in the Michigan cohort), the human-based RfD is in general agreement with the animal-based RfD and MRL. Overall, the studies point towards an increasing risk of developmental, immune, and cancer effects beginning at levels of exposure that are near the background rate of PCB exposure (e.g., Patandin, 1999; Rogan, et al., 1986). Ingestion of PCBs in fish comprises a substantial part of the background exposure, with studies showing that PCBs body burdens increase in relation to consumption of fish from contaminated water bodies. Therefore, fish consumption advisories are needed that steer fisherman and their families.

especially women who are or may become pregnant, away from fish that have elevated PCBs concentrations.

4.0 Toxicity Values from USEPA and ATSDR

The variety of toxicity potency values (benchmarks) derived for Aroclors is summarized in Table 2. These values are briefly described in Sections 4 and 5.

Table 2. Summary of PCBs Toxicity Benchmarks

PCB Mixture	Benchmark	Potency	Critical Effect	Species
Aroclor 1016	RfD[1]	0.07 ug/kg/d	Birth Weight	Monkey
Aroclor 1254	RfD[1]	0.02 ug/kg/d	Immune/Dermal	Monkey
Aroclor 1254	Chronic MRL[2]	0.02 ug/kg/d	Immune/Dermal	Monkey
Bioaccumulative, higher chorination	Oral Slope Factor[1]	2.0/mg/kg/d	Female liver tumors	Rats
More water soluble and volatile PCBs	Oral Slope Factor[1]	0.4/mg/kg/d	Female liver tumors	Rats
Low chlorination PCBs	Oral Slope Factor[1]	0.07/mg/kg/d	Female liver tumors	Rats
PCBs in Fish	Health Protection Value[3]	0.05 ug/kg/d	Weight of evidence from mulitple studies	Monkeys and Humans

[1]RfD is the daily oral dose that should not be exceeded as obtained from USEPA/IRIS database.
[2]Minimum Risk Level (MRL) is the daily oral dose that should not be exceeded as obtained from ATSDR Draft Toxicological Profile.
[3]Health Protection Value from 1993 Great Lakes Protocol.

4.1 Non-Cancer

USEPA has developed oral RfDs for two PCB mixtures: Aroclor 1254 and Aroclor 1016, with RfD development for other mixtures limited by insufficient data. The Agency derived an RfD of 0.02 ug/kg/d Aroclor 1254 based upon a LOAEL in monkey studies of 5 ug/kg/d for both an immune system endpoint (decreased antibody response in vitro) and a dermal response (swollen Meibomian gland in eye, altered nails). A 300 fold uncertainty factor was applied to this LOAEL (3 fold for inter-species extrapolation, 10 fold for sensitive individuals, 3 fold to extrapolate from a minimal LOAEL to a NOAEL, 3 fold to adjust from subchronic - 55 month - exposure to chronic RfD) to yield an RfD of 0.02 ug/kg/d. This RfD is compatible with developmental effects data for Aroclor 1254 which showed a LOAEL (swollen Meibomian gland in eye, nail changes) of 5

ug/kg/d from chronic maternal exposure. Assuming that similar uncertainty factors are applied to the developmental LOAEL as used above, the developmental RfD would be the same as the chronic RfD of 0.02 ug/kg/d.

The oral RfD for Aroclor 1016 is 0.07 ug/kg/d based upon a developmental LOAEL in monkeys whose mothers were exposed to this Aroclor for 7 months prior to delivery. The LOAEL for reduced birth weight (80% of control) was 0.028 mg/kg/d with the NOAEL determined to be 7 ug/kg/d. An overall uncertainty factor of 100 [3 fold for interspecies extrapolation , 3 fold for sensitive individuals, 3 fold to adjust subchronic to chronic) was applied to yield a RfD of 0.07 ug/kg/d.

In its Draft Toxicological Profile, ATSDR developed a chronic oral Minimum Risk Level (MRL) of 0.02 ug/kg/d for Aroclor 1254 based upon the same study and endpoints in monkeys as that used by USEPA in its Aroclor 1254 RfD. ATSDR used the same overall uncertainty factor (300 fold) but it was constructed differently (10x for LOAEL to NOAEL, 3x for animal to human extrapolation, 10x for sensitive individuals).

4.2 Cancer

USEPA has developed a range of oral cancer slope factors which corresponds to the range of PCB mixtures tested in a 1996 bioassay series. Relying principally upon findings of liver tumors in female rats receiving lifetime dietary exposure, the summarized data show similar potency in the higher chlorinated Aroclor mixtures (1254 and 1260), somewhat lower potency with Aroclor 1242, and lower potency again for Aroclor 1016. Based upon this pattern of decreasing potency with decreasing chlorination, and based upon the environmental fate of PCBs (higher chlorination mixtures tend to have greater environmental persistence and bioaccumulation in fish and other foods), the cancer slope factor for PCBs found in the food chain was set at 2.0/mg/kg/d, the slope factor for more water soluble and volatile (lower chlorination) PCBS was set at 0.4/mg/kg/d, and the slope factor for the lowest chlorination mixtures was set at 0.07 ugkg/d. This information is summarized in Table 2.

5.0 The Great Lakes Protocol

Health departments and natural resource departments from the eight Great Lake States convened a task force in the early 1990's to develop a consistent framework for risk-based fish consumption advisories for the Great Lakes. This resulted in the 1993 "Protocol for a Uniform Great Lakes Sport Fish Consumption Advisory", a document which in addition to describing a general framework, also provided a risk assessment focus on PCBs in fish (GLSFATF, 1993). The task force reviewed the toxicology and epidemiology literature for PCBs and rather than settling upon a key endpoint or study, they used a composite weight-of-evidence approach spanning a number of endpoints in monkeys (immunological, endocrine) and humans (developmental) for non-cancer effects. The task force also reviewed the basis for PCBs health benchmarks developed by

ATSDR, EPA/IRIS, the National Wildlife Federation, the World Health Organization, the Tennessee Valley Authority, and the Ohio River Valley Sanitation Commission. The result of their composite analysis was the development of a Health Protection Value (HPV) of 0.05 ug/kg/d. Their document shows that the animal and human data provide good support for this value and it is within the range of values derived other bodies for PCBs.

Fish consumption advice for PCBs was then described based upon this HPV and other key assumptions: average meal size for 70 kg of one-half pound (227 grams); 50% reduction in fish fillet PCBs content (skin on, scales off fillet) through trimming and cooking losses of fatty portions of the fish. The goal of the advisory program was to limit PCBs exposure from fish to the HPV (0.05 ug/kg/d * 70 kg = 3.5 ug/d), with less frequent meals needed to limit exposure to 3.5 ug/day as PCBs fish concentrations rise.

The risk-based PCBs fish concentration cutoffs for different meal frequencies developed in the protocol were as follows:

- Unrestricted fish consumption for fish concentrations ≤ 0.05 ppm (assuming unrestricted is 1 meal every 1.6 days or 140g fish/day)
- One meal per week for concentrations 0.06 to 0.2 ppm
- One meal per month for concentrations 0.21 to 1.0 ppm
- One meal every other month for concentrations 1.1-1.9 ppm
- No consumption > 1.9 ppm.

This protocol thus involves advice to moderate fish consumption beginning at fish concentrations of 0.06 ppm, a rather low starting point for advice given that fish concentrations below 0.1ppm are detectable but difficult to accurately quantitate (personal communication, Russ Spencer, DPH Laboratory). The cutoff between unrestricted and once per week consumption advice is somewhat arbitrary, depending upon the amount of PCBs-contaminated fish one might chronically eat per week. The protocol assumes 4.32 meals per week in the unrestricted case. If one assumed unrestricted meant 2 or 3 meals per week, then fish concentrations greater than 0.11 or 0.072 ppm, respectively, would trigger a once per week advisory. Therefore, a possible modification to the Great Lakes Protocol is to consider unrestricted fish consumption below 0.1 ppm. 1 meal/week between 0.1 and 0.2 ppm, one meal per month between 0.21 and 1.0 ppm. and less frequent than this above 1.0 ppm.

The protocol used a 50% loss of PCBs from fish due to trimming and cooking in their calculations of PCB advisory levels. The 50% PCBs loss was derived from studies in six

different species showing a range of trimming losses of organochlorine contaminants between 43 to 64%. A number of studies examining the effects of various cooking methods on organochlorine fish content were also reviewed. Overall, cooking did not materially change fillet concentration of PCBs (decreases in lipid content parallel decreases in water content from cooking) although the overall amount present in the meal was reduced. This becomes a factor if one assumes the average meal size prior to cooking is 227 grams and becomes smaller post-cooking. However, most risk assessments assume the ingested meal portion is 227 grams. In that case, cooking would not materially affect the risk assessment. On the basis that most anglers trim their catch and that PCB-based advisories stress trimming to reduce exposure, the protocol adopted a 50% reduction in PCBs from the amount available in the raw fillet.

6.0 Recommendations for CT's Advisory Program

6.1 General Recommendation

Given that Connecticut's current advisory program for PCBs is based upon a eat/don't eat tolerance level set by FDA in the 1970's, it is current or fully risk-based and thus not necessarily protective of public health. As summarized in Table 1, a number of states have now adopted more risk-based approaches, with most basing the advisory on non-cancer health effects. The Great Lakes Protocol is a well-thought out approach, relying upon a Health Protection Value (HPV) (0.05 ug/kg/d) that is consistent with determinations made independently by ATSDR (draft MRL), USEPA (RfDs), and by regional, national and international bodies. Use of this health protection value is prudent to mimimize the risks for the variety of health effects seen at low doses in monkeys (immunological, dermal, reproductive/developmental) and humans (primarily developmental).

6.2 Comparison Across FDA Tolerance Level, Great Lakes Protocol and CTDPH Recommended Approach

The major difference between the Great Lakes Protocol and the FDA Tolerance Level is in terms of the overall purpose of the program. FDA's regulatory charge is to determine which foods can and cannot be sold in the marketplace, with a single cut-off concentration approach used to determine what can be sold. In contrast, the Great Lakes Protocol provides the public with advice regarding how much fish can be eaten without appreciable risk, thus informing consumers of locally caught fish how often they can safely eat specific species from specific waterbodies. Table 3 illustrates the different approaches, with FDA (and the modified FDA approach currently used in CT) having one bright line cutoff, and the Great Lakes Protocol having a similar "do not eat" cutoff but recognizing that below this level there is still some risk depending upon the amount of fish consumption (and PCBs intake). The Great Lakes Protocol allows unlimited consumption at fish concentrations that are so low that they could not deliver enough

PCBs to supercede the HPV dose (on a 227 gram meal size basis). At higher fish concentrations (0.06 to 1.9 ppm) moderate levels of consumption (one meal per week to two meals per month) are advised. Above 1.9 ppm, the HPV would be exceeded even if fish consumption were very infrequent (less than once per 2 months). Thus, the Great Lakes Protocol establishes meal frequencies specific to different fish concentrations that maintain the daily PCBs dose to the HPV.

Table 3. Summary of FDA vs. Risk-Based Approaches

FDA: > 2 ppm Do Not Eat
< 2 ppm Unlimited Consumption

Modified FDA Approach for CT (Current CTDPH Approach):
10% > 2 ppm - high risk - Do Not Eat: low risk - 2 Meals per Month
50% > 2 ppm - Do Not Eat for everyone

Great Lakes Protocol:

< 0.05	Unlimited Consumption
0.06 - 0.20	One meal per week
0.21 - 1.0	One meal per month
1.1 - 1.9	One meal every 2 months
> 1.9 ppm	Do Not Eat

CTDPH Draft Approach (Modified Great Lakes Protocol):

<0.1	Unlimited Consumption
0.1-0.2	One meal per week
0.21 - 1.0	One meal per month
1.1 - 1.9	One meal every 2 months (high risk group - do not eat)
> 1.9 ppm	Do Not Eat (everyone)

Table 3 also shows a recommendation to modify the Great Lakes Protocol for Connecticut advisories to take into account detection limit issues (see Section 5) and the somewhat greater concern for higher risk individuals (pregnant women, women planning pregnancy). The draft approach would allow unlimited consumption at fish concentrations below 0.1 ppm, the point where quantitation of PCBs in fish becomes certain. In this range, it is possible but not highly likely that one would receive PCB exposures above the HPV (more than 2 meals/week of local fish on a regular basis).

Regarding the issue of higher risk individuals, the animal toxicology database supports a RfD/HPV that is in the same range for reproductive and other (immunological, dermal) endpoints. This suggests that in utero development is no more sensitive to PCBs than are endpoints seen in adult animals. However, the evidence of low dose effects in humans is strongest for in utero effects (CNS development). This creates a somewhat greater concern for pregnant women and women planning pregnancy. An additional factor is that

while the cumulative PCB dose from long-term exposure may be the most critical determinant for immunological or dermal effects, the period of exposure needed for in utero effects is uncertain. The monkey studies which show low dose PCBs effects involved pre-pregnancy exposure over several years. Therefore, it is not clear whether build-up of maternal PCB body burden prior to and during pregnancy is critical or whether a relatively short exposure period (during pregnancy) could also produce low dose developmental effects. It is noteworthy that two shorter term studies in rats and mink did find low dose developmental effects (ATSDR, 1999). This suggests that, while uncertain, there may be a greater sensitivity during in utero exposure such that a few recent exposures that don't involve a cumulative body burden (which is important to adult toxicity) could produce an adverse effect. This uncertainty over PCBs pharmacokinetics and developmental outcomes supports a prudent avoidance (do not eat) approach for pregnant women for markedly elevated PCBs concentrations (e.g., over 1 ppm).

6.3 Consideration of Background Exposures

It is important to place the HPV dose into the perspective of background exposures to PCBs. Exposures to PCBs from dietary, non-fish sources are somewhat difficult to quantify because sampling of the food supply for PCBs is limited and there is always uncertainty in estimating how much consumption there is of different foods. However, it appears that PCBs concentrations in a variety of food products have decreased substantially from the 0.05 to 0.1 ppm concentrations found in certain foods (milk, eggs) in the early 1970s. FDA's most recent data indicate that there are very few detections of PCBs in commercial food products (detection limit 0.05 ppm) suggesting that background (non-fish) exposures to PCBs are not generally significant (M. Bolger, FDA Food Contaminants Branch, personal communication). Thus, ingestion of PCBs from certain fish species in Long Island Sound (bluefish, striped bass) and from PCB-affected waterbodies (e.g., Housatonic, Quinnipiac, Connecticut Rivers) present the greatest potential for dietary exposure to PCBs in CT.

6.4 Consideration of Cancer Risks

The HPV establishes a target exposure rate protective against non-cancer health effects. However, as summarized in Section 4.2, PCBs have shown carcinogenic activity in rat liver in numerous oral studies, with a composite cancer slope factor 2.0/mg/kg/d for PCBs that are present in the food chain. In combination with an allowable daily exposure (HPV) of 0.05 ug/kg/d (5E-05 mg/kg/d) leads to a chronic cancer risk of 1E-04 or 1 in 10,000. This suggests that a cancer risk at the upper end of the generally acceptable range (1E-06 to 1E-04) would exist if consumers follow the Great Lakes Protocol. While it would be ideal to reduce the cancer risk further, a risk management consideration is the weighing of the documented benefits of fish consumption (high protein, low cost, beneficial fish oils) against the theoretical cancer risk associated with PCBs in fish. Setting fish limits based upon cancer risk concerns would lead to virtually no fish consumption (local or commercial) due to the widespread occurrence of low levels of PCBs in fish. This would cause the benefits of fish consumption to be lost in the interests

of minimizing cancer risks. Given that the number of avid consumers of locally caught fish in CT may not be large, the theoretical 1 in 10,000 cancer risk level is of less concern than if this were a population-wide exposure. Therefore, the recommendation is to focus on prevention of the non-cancer health effects of PCBs, which the Great Lakes Protocol (and modified version for CT) accomplishes.

6.5 Specific Recommendations for Connecticut Waterbodies and Fish

PCB concentrations in CT fish and the associated fish consumption advice are listed in the appendix table. That table indicates how the risk-based approach might change the advice for specific fish. Given the value of keeping the overall advice simple and easy to follow, a risk management strategy is needed to integrate the waterbody-specific advice for PCBs into broader messages (e.g., a single advisory for entire Housatonic River or for all carp in CT?) that also take into account the statewide freshwater advisory for methylmercury (one meal/month - high risk group; one meal/week - all others). Table 4 summarizes the proposed changes to this year's advisory; it should be noted that CTDPH may recommend additional PCBs monitoring to better define the need for consumption advisories in certain waterbodies. Aside from the waterbodies and species mentioned below, fish in CT waters tend to be have PCBs concentrations <0.2 ppm (once a week to unlimited consumption range) and thus there does not appear to be a need for a general statewide advisory for PCBs. The methyl mercury statewide advisory is protective of the potential for PCB exposures from fish with low levels (<0.2 ppm).

Table 4. Draft Changes to CT Fish Consumption Advisories for PCBs

Waterbody	Species	Current Advice	New Advice
L.I.Sound	Striped Bass	hi risk - do not eat low risk - 2x/month	hi risk - do not eat low risk - 1x/2month
L.I.Sound	Bluefish < 25" > 25"	unlimited consumpt. same as striped bass	one meal/month same as striped bass
L.I.Sound	Lobster Heptopanc.	Do not eat	hi risk - do not eat low risk - 1x/2month
Housatonic River	All Species	Do not eat with some exceptions	no change now; new data this year
CT River	Carp	hi risk - do not eat low risk - 2x/month	hi risk - do not eat low risk - 1x/2month
CT River	Catfish	No PCB advice; but statewide Hg advice	hi risk - do not eat low risk - 1x/2month
Quinn. Gorge & Hanover Pond	All Species	No PCB advice: but statewide Hg advice	One meal per month
Union Pond[1]	Carp,Catfish,Bass	No PCB advice; but statewide Hg advice	Do not eat

[1]Advisory for Union Pond due to elevated chlordane concentrations.

7.0 References

Arnold, D.L., Bryce, F., McGuire, P.F., et al. (1995) Toxicological consequences of Aroclor 1254 ingestion by female rhesus monkeys. Part 2. Reproduction and infant findings. Food Chem. Toxicol. 33: 457-474.

ATSDR (Agency for Toxic Substances and Disease Registry) (1998) Draft Toxicological Profile for Polychlorinated Biphenyls (PCBs).

Balcom, N.C., Capacchione, C.M., and Hirsch, D.W. (1999) Quantification of fish and seafood consumption rates for Connecticut. Report submitted to CTDEP/OLISP

Barsotti, D.A., Marlar, R.J., and Allen, J.R. (1976) Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14: 99-103.

Bucher, J.R. and Lucier, G. (1998) Current approaches toward chemical mixture studies at the National Institute of Environmental Health Sciences and the U.S. National Toxicology Program. Env. Health Perspectives 106, Suppl. 6: 1295-1298.

Cordle, F., Locke, R., Springer, J. (1982) Risk assessment in a federal regulatory agency: an assessment of risk associated with the human consumption of some species of fish contaminated with PCBs. Environ. Health Perspectives 45: 171-182.

GLSFATF (Great Lakes Sport Fish Advisory Task Force) (1993) Protocol for a Uniform Great Lakes Sport Fish Consumption Advisory.

Huisman, M., Koopman-Esseboom, C., Fidler, V., et al. (1995) Perinatal exposure to polychlorinated biphenyls and dioxins and its effect on neonatal neurological development. Early Hum. Dev. 41: 111-127.

Jacobson, J.L. and Jacobson, S.W. (1996) Intellectual impairment in children exposed to polychlorinated biphenyls in utero. N. Engl. J. Med. 335: 783-789.

Minnesota Dept. of Health (1990) Minnesota Analysis and RfD Development - Assessing Risks to Human Health from PCB-contaminated Fish: risk assessment based on epidemiology studies.

Minnesota Dept. of Health (1992) PCB Reference Dose for Effects on Fetal Development: Development of a Reference Dose based on Human Exposure Data. HRA series 92-FSH-002.

Patandin, S., Lanting, C.I., Mulder, P.G.H., et al. (1999) Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age. J. Pediatrics 134: 33-41.

Rogan, W.J., Gladen, B.C., McKinney, J.D., et al. (1986) Neonatal effects of transplacental exposure to PCBs and DDE. J. Pediatrics 109: 335-341.

Tilson, H.A., Jacobson, J.L., and Rogan, W.J. (1990) Polychlorinated-biphenyls and the developing nervouse system: cross-species comparisons. Neurotoxicol Teratol. 12: 239-248.

Tryphonas, H., Hayward, S., O'Grady, L., et al. (1989) Immunotoxicity studies of PCB (Aroclor 1254) in the adult rhesus monkey - preliminary report. Int. J. Immunopharmacol 11: 199-206.

USEPA/ATSDR (1998) Public Health Implications of Exposure to Polychlorinated Biphenyls (PCBs).

APPENDIX

PCBs SUMMARY IN CONNECTICUT WATERS

PCBs Summary in CT Waters (4/99)

Waterbody	Species	Date; Sample Size[1]	Avg PCB (ppm)	PCB Range (ppm)	Current Advice	New Advice
L.I.Sound	Striped Bass	'94; 0 x 303	1.18	17% > 2 ppm	No consump (hi risk); 2x/mth (others)	No cons (hi risk), 1x/2 mth (others)
L.I.Sound	Bluefish	'98; 0 x 57	0.89	<25" = 0.35 >25" = 1.26	<25" - unlimited consumption >25" - same as striped bass	<25" - One meal / month >25" - No cons / 1x per 2 month
L.I.Sound	Flounder	'85; 6 x 6	0.029	0.01 - 0.043	Unlimited consumption	No change
L.I.Sound	Blackfish	'85; 6 x 6	0.050	0.007 - 0.16	Unlimited consumption	No change
L.I.Sound	Lobster	'85; 6 x 6	0.004	0.01 - 0.043	Unlimited consumption	No change
L.I.Sound	Lobster hepatopn	'85; 6 x 6	2.90	0.11 -12	Do not eat	No cons (hi risk), 1x/2 mth (others)
Hous/Cornwall	Brown Trout	'96; 0 x 20	2.42	0.11 - 8.64	Do not eat	No change
Hous/Cornwall	Rainbw Trout	'88;	2.63		Do not eat	No change
Hous/Cornwall	Smllmth Bass	'96; 0 x 5	1.00	0.61 - 1.4	Do not eat	No change
Hous/Bull's Br	Smllmth Bass	'96; 0 x 5	0.99	0.86 - 1.15	Do not eat	No change
Hous/Bull's Br	Largmth Bass	'88;	2.09		Do not eat	No change
Hous/Bull's Br	Carp	'88;	5.17		Do not eat	No change
Hous/Bull's Br	Brn Bullhead	'88;	1.68		Do not eat	No change
Hous/Bull's Br	Yellow Perch	'92;	0.56		No PCBs advice; Hg advice	No change (change in future?)
Hous/Bull's Br	Bluegill	'88;	1.85		Do not eat	No change
Hous/Bull's Br	RdbrstSunfsh	'88;	1.66		Do not eat	No change
Hous/Bull's Br	Pumpkinseed	'88;	0.27		Do not eat	No change (change in future?)

PCBs Summary in CT Waters - pg 2

Waterbody	Species	Date; Sample Size[1]	Avg PCB (ppm)	PCB Range (ppm)	Current Advice	New Advice
Hous/Lillinona	Smllmth Bass	'96; 0 x 5	0.30	0.21 - 0.49	Do not eat	No change (change in future?)
Hous/Lillinona	Largmth Bass	'88;	1.15		Do not eat	No change
Hous/Lillinona	Carp	'88;	5.61		Do not eat	No change
Hous/Lillinona	BrnBullhead	'88;	1.42		Do not eat	No change
Hous/Lillinona	Catfish	'88;	4.33		Do not eat	No change
Hous/Lillinona	White Perch	'88;	1.53		Do not eat	No change
Hous/Lillinona	Yellow Perch	'92;	0.32		No PCBs advice; Hg advice[2]	No change (change in future?)
Hous/Lillinona	Bluegill	'92;	0.45		No PCBs advice; Hg advice[2]	No change (change in future?)
Hous/Lillinona	RdbrstSunfsh	'92;	0.47		No PCBs advice; Hg advice[2]	No change (change in future?)
Hous/Lillinona	Pumpkinseed	'92;	0.18		No PCBs advice; Hg advice[2]	No change (change in future?)
Hous/Zoar	Smllmth Bass	'96; 0 x 5	0.48	0.34 - 0.75	Do not eat	No change (change in future?)
Hous/Zoar	Largmth Bass	'88;	1.15		Do not eat	No change
Hous/Zoar	Carp	'88;	12.07		Do not eat	No change
Hous/Zoar	Brn Bullhead	'88;	0.62		Do not eat	No change (change in future?)
Hous/Zoar	Catfish	'88;	3.4		Do not eat	No change
Hous/Zoar	White Perch	'92;	1.01		No PCBs advice; Hg advice[2]	No change (change in future?)
Hous/Zoar	Yellow Perch	'92;	0.26		No PCBs advice; Hg advice[2]	No change
Hous/Zoar	Bluegill	'92;	0.25		No PCBs advice; Hg advice[2]	No change
Hous/Zoar	RdbrstSunfsh	'92;	0.24		No PCBs advice; Hg advice[2]	No change

PCBs Summary in CT Waters - pg 4

Waterbody	Species	Date; Sample Size[1]	Avg PCB (ppm)	PCB Range (ppm)	Current Advice	New Advice
QRiv/Gorge	Rainbw Trout	'97; 0 x 10 (2x)	0.20 - 0.8		No PCBs advice; Hg advice[2]	One meal / month
QRiv/Gorge	White Sucker	'97; 0 x 10 (2x)	0.23 - 0.46		No PCBs advice; Hg advice[2]	One meal / month
QRv/Hnovr Pd	Carp	'97; 0 x 10 (2x)	0.61 - 1.34		No PCBs advice; Hg advice[2]	Do not eat
QRiv/Gorge	Yellow Perch	'97; 0 x 10	0.15		No PCBs advice; Hg advice[2]	One meal / week
QRiv/Meriden	Brook Trout	'90; 1 x 3	0.46		No PCBs advice; Hg advice[2]	One meal / month
Sodom Brook Meriden	White Sucker	'90; 1 x 5	0.42		No PCBs advice; Hg advice[2]	One meal / month
8 Mile River	Brown Trout	'96; 0 x 10	2.7		Do not eat	Do not eat
8 Mile River	White Sucker	'96; 0 x 10 (2x)	0.027 - 0.029		Do not eat	*No PCBs advice; Hg advice*[2]
Thames River	White Sucker	'91; 0 x 3	0.41	ND - 0.70	No PCBs advice; Hg advice[2]	No change (need new data)
MillRiv -Fairfd	White Sucker	'91; 4 x 4	0.16	0.05 - 0.29	No PCBs advice; Hg advice[2]	No change
MillRiv -Fairfd	Brown Trout	'90; 1 x 4	4.13		No PCBs advice; Hg advice[2]	No change (need new data)
Naugatuck Riv	White Sucker	'92; 5 x 4	0.18	0.025 - 0.63	No PCBs advice; Hg advice[2]	No change
NorotnR Darien	White Sucker	'90; 1 x 5	0.26		No PCBs advice; Hg advice[2]	No change
All Other CT Rivers	Various	'90 - '92	<0.2		No PCBs advice; Hg advice[2]	No PCBs advice; Hg advice[2]