STATE OF DELAWARE
FISH CONTAMINANT MONITORING AND HEALTH ADVISORY PROGRAM

Technical Procedures for Evaluating Human Health Risks Associated with the Consumption of Chemically Contaminated Fish and Shellfish

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1.0 INTRODUCTION

Surface water is the ultimate repository for many chemicals used and disposed of by society. Certain chemicals tend to concentrate ("bioaccumulate") in fish to levels thousands of times greater than the levels in the water itself. The resulting concentrations in fish and the attendant health risks to those individuals who consume the fish are of concern to various government agencies and the public. The U.S. Food and Drug Administration (FDA) carries the primary responsibility for regulating risks associated with fish sold in the interstate marketplace. At arriving at acceptable levels of contaminants in fish, the FDA considers national fish consumption habits and national fish contamination patterns. This approach is consistent with their mandate to ensure the overall safety of the nation's food supply. The FDA does not consider, however, the risks to recreational fishermen nor subsistence anglers who may consume substantially greater amounts of fish from a local or regional waterway which has become contaminated [Reinert, et.al., 1991]. Assessing and managing those risks is the responsibility of State environmental and health agencies.

In Delaware, the concern over fish contamination and the associated risks to recreational and subsistence anglers lead to the development of a Memorandum of Understanding (MOU) between the Department of Natural Resources and Environmental Control (DNREC) and the Department of Health and Social Services (DHSS). The Memorandum of Understanding, signed in June of 1993, established an interagency/interdisciplinary Fish Contaminants Committee and also set forth the steps the agencies intend to take to systematically assess, manage, and communicate the risks associated with fish contamination in Delaware, [DNREC/DHSS, 1993]. Among the tasks called for in the MOU was the development of procedures for assessing the human health risks associated with the consumption of chemically contaminated fish harvested from Delaware waters. Those procedures were first formalized in 1999. They were then updated in 2005 and 2016 to reflect incremental advances in knowledge and practice.

2.0 METHODS

The assessment of risk associated with the consumption of chemically contaminated fish begins with the collection of fish samples from a particular waterbody. Depending on available resources, Delaware prepares a sampling plan on an annual basis that specifies the locations to be sampled, the fish species targeted for collection, the portion to be tested, the parameters to be analyzed, and the field and laboratory methods. The list of chemical parameters typically tested in the tissue samples includes polychlorinated biphenyls (PCBs), chlorinated pesticides, dioxins/furans, polyaromatic hydrocarbons (PAHs), mercury, and lipid content. Appendix A presents the specific chemical contaminants currently included in Delaware's fish tissue monitoring program. In addition to the parameters listed in Appendix A, tissues have occasionally been analyzed for polybrominated diphenyl ethers (PBDEs), orthophosphorus (OP) pesticides, and inorganic arsenic.

Delaware's annual sampling plan incorporates screening level sampling, intensive sampling, and advisory follow-up sampling. Screening level sampling involves the collection of a single composite sample taken in an area with unknown contaminant conditions or where data are significantly outdated (e.g., >10 years old). When the screening sample indicates a potential contamination problem, intensive sampling is initiated. Intensive sampling involves the collection of several samples throughout an area (e.g., a watershed) to characterize the extent and magnitude of contamination. Depending on the specific situation, intensive sampling can entail testing of several individual fish or composite samples, testing of several species, and sometimes testing of different size classes of fish. Finally, advisory follow-up sampling is performed several years after an advisory has been issued (e.g., 5 years) to track changes and to determine whether adjustments are needed to the particular advisory.

The results of the screening samples are compared to "fish tissue screening values" to determine the need for and the priority of intensive sampling. Fish tissue screening values, which are developed and presented in this document, are contaminant concentrations in the fish which correspond to very low health risks (i.e., less than 10-5 cancer risk assuming consumption of 1 meal of recreationally-caught fish every week for 30 years). If the concentration of one or more contaminants in a screening sample clearly exceeds the corresponding fish tissue screening value(s), then an intensive survey is conducted as resources and priorities permit. Results of the intensive sampling are used, in turn, to determine if a fish consumption advisory might be needed. Risk assessment is the primary technical tool used to inform such determinations.

Because fish advisories are fundamentally a risk management action, factors in addition to the technical risk assessment are often considered prior to issuing an advisory. The responsibility for issuing an advisory in Delaware ultimately rests jointly with the Secretary of the DNREC and the Secretary of the DHSS. Experience has shown however that advisories are typically issued in those situations where consuming more than one meal of fish per week from the waterbody results in an aggregate lifetime cancer risk greater than 10-5 (1 additional possible cancer case in a population of 100,000 people). Aggregate risk in this case refers to the combined risk associated with all individual contaminants in the fish. Similarly, if the aggregate non-cancer health risk exceeds a hazard index of one when more than one meal of fish per week is consumed from the waterbody, then an advisory will normally be issued. Contaminants of concern (COCs) leading to the issuance of an advisory are those that contribute 10% or more to the aggregate cancer or non-cancer health risk or otherwise consistently exceed their associated fish tissue screening value in the waterbody. Identification of COCs can include and often does include consideration of supporting information such as surface water, sediment, and site data, plus relevant trend data if available. The terms used above and the foregoing discussion should become more clear after reviewing the entirety of this document.

2.1 Human Health Risk Assessment

In conducting a risk assessment for chemically contaminated fish, we seek to answer the following basic questions:

What contaminants are present in the fish and at what concentrations?

What types of health effects are associated with exposure to these contaminants?

How potent are the contaminants?

Who might consume the fish and how much do they consume?

What is the magnitude of health risks posed?

Risk assessment, as first proposed by the National Academy of Sciences [NAS, 1983], and refined over the years, is an orderly way of investigating and projecting future outcomes associated with "risky" situations. More formally, risk assessment is a scientifically-based procedure used to estimate the probability of adverse health effects under particular exposure conditions. As described by the NAS, risk assessment consists of four separate steps:

1. Hazard Identification;
2. Dose-Response Evaluation;
3. Exposure Assessment; and
4. Risk Characterization.

These steps are discussed in greater detail below.

2.1.1 Hazard Identification

Hazard identification is the qualitative determination of whether a substance causes or is likely to cause an increased incidence or severity of illness in the human population. This qualitative determination is based upon epidemiological evidence which links human exposure to actual observed illness in the human population as well as on results of laboratory tests conducted on experimental animals. These two primary forms of information are also supplemented by data on chemical structure, physical properties, and other assays.

Due to the general paucity of and difficulty in obtaining good epidemiologic data linking chemical exposure to illness in humans, the most common form of data used to support the hazard identification step comes from laboratory tests on experimental animals. Typically, these experiments involve the administration of moderate to high doses of a chemical agent to mice or other rodents over periods of months to years. Occasionally, higher mammals such as dogs or

primates are used. The primary objective of such experiments is to determine if continuous exposure to the chemical causes adverse health effects, what those health effects are, and what the nature of the dose-response curve is.

2.1.2 Dose-Response Evaluation

The purpose of the dose-response evaluation is to determine the relationship between the amount of a chemical administered, (deliberately, in the case of experimental animals, or accidentally, in the case of a human population) and an observed health effect in the exposed group. The manner in which the dose-response data are interpreted depends upon the toxicological endpoint being considered and whether the dose-response data were generated from human exposure data or from assays conducted on experimental animals. If the endpoint is cancer and sufficient dose-response data exists for a human population, a "best fit" line is drawn through the data and the slope is taken as a measure of the chemical's cancer potency.

In the case of animal carcinogenicity data, the moderate to high doses necessary to elicit a tumor response must be extrapolated back to the low dose region to be of practical use in evaluating the exposures typically experienced by the human population. This low dose extrapolation is performed using a mathematical model, typically the linearized multistage model. As noted in Johannsen [Johannsen, 1990], the linearized multistage model was originally proposed by Crump and others as a generalization of the Armitage-Doll multistage model of carcinogenesis. The linearized multistage model assumes that cancer results from a series of interactions between the carcinogenic agent and DNA, with the rate of interaction being linearly related at low dose [EPA, 1989a]. An important feature of this model is that it predicts some finite risk of cancer even at the lowest conceivable doses. Taken to the limit, the model assumes that risk is zero only if exposure is zero. The underlying hypothesis, therefore, is that cancer is a non-threshold phenomenon.

As in the case of human carcinogenicity data, the slope of the dose-response curve from an animal assay is an indication of the cancer potency of the chemical. In this case however, the upper 95th percent confidence limit on the slope in the low dose range, as computed through the multistage procedure, is typically used. This value is referred to alternatively as the cancer potency slope, slope factor, or simply q1* for short. Quantitatively, the slope factor represents the excess cancer risk per unit of exposure. As such, the units of q1* are the inverse of those of exposure. For instance, if the units of exposure are expressed as mg of pollutant ingested per body weight of the individual exposed per time (e.g. mg/kg/d), then the units of q1* will be 1/(mg/kg/d).

In contrast to carcinogenic hazard, non-cancer hazards assume that toxic effects only occur after exposure exceeds some threshold level. In other words, up to some particular level of exposure, the body's natural defense mechanisms are able to ensure that a toxic effect is not likely to occur. The so-called Reference Dose (RfD) is used as an estimate of the exposure that is assumed not to be associated with significant risk of non-cancer toxicity. More formally, the

RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime [Dourson and Clark, 1990]. The units of RfD are the same as the units of dose, mg of contaminant per body weight of human receptor per day (mg/kg/d). Operationally, the RfD is obtained by dividing either the highest dose of the chemical that did not produce a toxic effect in experimental studies (i.e. the No Observed Adverse Effect Level or NOAEL), or the lowest dose that did produce a toxic effect (i.e. the Lowest Observed Adverse Effect Level or LOAEL), by the product of an uncertainty factor and a modifying factor. The uncertainty factor accounts for differences in sensitivity to toxic effects within and between species, as well as differences in toxic effects between chronic and subchronic exposures. The modifying factor reflects the confidence in the quality of the animal assay data in predicting health effects in humans.

The principal sources of information that are consulted for potency slopes and reference doses are the U. S. EPA's Integrated Risk Information System [EPA, 2016], the U.S. EPA's HEAST database [EPA, 1997], and the U. S. Department of Health & Human Services' (DHHS) Toxicological Profiles. A list of potency slopes and reference doses for the most commonly detected fish contaminants appears in Appendix B of this document. These are the values currently used by the Delaware Fish Contaminants Committee.

2.1.3 Exposure Assessment

Exposure assessment is the estimation of the amount of a substance ingested, inhaled, or absorbed by a target population. When we are evaluating the potential for a chronic health effect such as cancer, the goal of the assessment is to obtain an estimate of the exposure integrated over long time periods. In contrast, non-cancer health effects can manifest themselves in much shorter time frames, and so the goal of the assessment is to estimate shorter-term exposure. The methods used to estimate long term and short term exposures are described below.

For long term exposure, an estimate of the lifetime average daily exposure rate (LADE) is computed from the following equation:

$$LADE\,(mg/d) = \frac{C_f \times MS \times MF \times ED \times (100 - RF)/100}{LT}$$

where the following variables are defined:

C_f = characteristic concentration of the contaminant in the edible portion of the fish, (mg/kg or ppm). This is typically an average or median depending on data distribution.
MS = meal size in ounces x 0.02835, (kg/meal)
MF = number of meals consumed per year divided by 365.25 days per year, (meals/d) ED = duration over which exposure is assumed to occur, (yrs)
LT = lifetime duration, (yrs)

RF = percent reduction in contaminant concentration in the fish due to trimming and cooking losses, (%)

When estimating exposure for non-cancer health effects, the equation above is modified by eliminating ED (exposure duration) and LT (lifetime duration). The resulting exposure is then simply an average daily exposure, rather than a lifetime average.

Aside from the concentration of the contaminant in the fish, the most important factors in the above equation are MS (meal size) and MF (meal frequency). The product of these two variables provides an estimate of fish ingestion in mass per unit of time. Clearly, the larger the portion and/or the more frequently one consumes fish, the greater the fish ingestion. On occasion, actual fish consumption data may be available for a particular waterbody. Such data can be used to estimate site-specific ingestion rates. In general, however, this type of information is not available. Consequently, reasonable assumptions must be made concerning meal size and frequency. As a default, a meal size of 8 ounces is assumed for the general adult population, while 6 ounces is assumed for women of child-bearing age, and finally 3 ounces is assumed for children. Furthermore, to reflect the fact that some people may consume fish from a waterbody frequently, while others only occasionally or rarely ever, a range of plausible meal frequency values is considered, rather than a single value. Meal frequencies ranging from 1 meal per week up to 1 meal per year are typically evaluated. Using a range of values not only provides an indication of how sensitive exposure and risk estimates are to the amount of fish consumed, it also makes the findings of the risk assessment ultimately easier for people to relate to in common terms, rather than when fish consumption is expressed in scientific units such as grams per day.

Exposure duration reflects the length of time an individual is expected to be exposed to a particular toxic agent from a given source. Information on exposure duration is typically derived from population mobility data. Because the population is quite mobile, exposure duration will vary from individual to individual, and from household to household. However, for purposes of risk assessment, exposure duration is typically assigned a value which reflects a reasonable worst-case of residence time. Consistent with that practice, the exposure duration used in risk assessments for carcinogens in fish flesh is taken as the 90th percentile value for the number of years adults reside in a given household. This value was computed by the EPA to be approximately 30 years [EPA, 1989b] and is based upon a survey conducted by the U.S. Bureau of the Census in 1983. The 90th percentile value contrasts with a median, (i.e. 50th percentile) value derived from the same study of roughly 9 years. An analysis using even more recent information suggests the average residence time for all U.S. households is closer to 4.6 years, and that the mean for the northeast region is roughly 7.4 years [Israeli and Nelson, 1992]. The procedures presented herein nevertheless use a 30-year exposure duration for adult receptors. Although this value may seem unreasonably conservative, especially in light of the more recent figures, the fact is that this value will underestimate exposure for those people who spend their entire lives in the same region, even if they do change their principal place of residence. In the case of a child receptor, exposure was assumed to occur over the entire first 6 years of life.

Based upon current data, the average life expectancy of the entire U.S. population is 74.7 years [EPA, 1989b]. For purposes of these procedures, a life expectancy of 75 years is assumed. This value aggregates males and females, blacks, whites, and others.

The final variable which appears in the above equation is RF, which is the percent reduction in contaminant concentration in the fish due to trimming and cooking losses. Based on studies conducted on Great Lakes fish, typical losses of PCBs and chlorinated pesticides resulting from proper trimming and cooking may be roughly one-third, (e.g., 33%), and in some cases, as high as 50% [Zabik et. al. 1993]. These figures, however, assume that the angler has followed trimming advice carefully and that the oils in the fish are allowed to drip away during cooking. Although it is indeed important to provide advice to anglers on how they might reduce their risk through proper trimming and cooking, there is little guarantee that they will follow that advice. For this reason, the reduction factor assumed in implementing these procedures is zero. This causes the reduction factor quotient in the above equation to default to a value of 1, which has no influence on the estimate of LADE.

After computing lifetime average daily exposure, an estimate of lifetime average daily dose (LADD) is then obtained using the equation below.

$$LADD\,(mg/kg/d) = \frac{LADE \times AF}{BW}$$

where the following additional variables are introduced:

AF = gastrointestinal absorption factor
BW = average body weight of the exposed population, (kg)

The equation for non-carcinogens is identical to that just presented except that LADE is replaced by average daily exposure. The resulting dose is referred to as average daily dose (ADD).

In the above equation, the gastrointestinal absorption factor is a value between 0 and 1 which reflects any known or expected differences between the efficiency at which the contaminant of interest is absorbed by bioassay animals verses humans. These procedures assume a default value of 1.

The other variable in the previous equation which must be specified is body weight. Body weight is an important factor because it influences dose in an inverse fashion. In other words, the lighter the individual being exposed to a given amount of a toxicant, the greater the dose. Conversely, the heavier the individual, the less the dose. This basic concept is taken into account within these procedures by specifying three separate receptor groups, each of which has its own characteristic weight. The three groups include adults of average weight, women of

child-bearing age, and children between the ages of 0 and 6 years old. The mean body weight of all adults between the ages of 18 and 75, men and women combined, is 71.8 kilograms, or roughly 158 pounds [EPA, 1989b]. The average body weight of women of child-bearing age is 63.6 kilograms, or approximately 140 pounds [EPA, 1989b]. This average includes all women between the ages of 18 and 45. Finally, the average weight of boys and girls combined between the ages of 0 and 6 years old is 14.5 kilograms, or 40 pounds [EPA, 1989b]. These procedures assume nominal weights of 70 kg, 64 kg, and 14.5 kg for the three groups.

Table 1 summarizes the various default exposure factors discussed above.

Table 1. Exposure Factors

	Receptor Group		
Exposure Factor	**Average Adult**	**Women of Child-Bearing Age**	**Children (0 - 6)**
MS, (ounces)	8	6	3
MF, (#/day)	variable	variable	variable
ED, (yrs)	30	30	6
LT, (yrs)	75	75	75
RF, (%)	0	0	0
AF, (decimal)	1	1	1
BW, (kg)	70	64	1

2.1.4 Risk Characterization

Risk characterization is the integration of the previous three steps (hazard identification, dose- response evaluation, and exposure assessment) to produce a concise description of the nature and magnitude of potential harm to the public. The risk characterization also identifies the major assumptions, scientific judgements, and, to the extent possible, estimates the uncertainties embodied in the assessment [EPA, 1986a]. A necessary step in defining the magnitude of potential harm is to calculate the cancer risk (in the case of carcinogens) and the hazard index (in the case of non-cancer endpoints). These procedures are presented below.

2.1.4.1 Carcinogenic Effects

Excess lifetime cancer risk is computed as the product of the lifetime average daily dose (LADD) and the cancer potency slope (q1*) as shown in the equation below. Because q1* is an upper bound estimate of the low-dose slope as determined through the multistage procedure, the equation below will yield estimates of risk that are conservative, representing a plausible upper limit for the cancer risk at the assumed exposure. Consequently, it is unlikely that the "true" or "actual" risk associated with a given exposure is higher than the risk predicted using this model.

$$RISK = LADD \times q_1^*$$

As discussed previously, LADD has units of mg/kg/d and q_1^* has units of 1/(mg/kg/d). The product of these two values, (i.e., risk) is therefore unitless. Risk, in fact, can assume any real value between 0 and 1. A risk of 0 corresponds to the absence of exposure, and a risk of 1 corresponds to certainty that exposure will result in a health effect. Most risk projections, of course, fall somewhere in between 0 and 1 and their meaning must therefore be interpreted within a probabilistic domain. Risk, by definition, is the probability of injury, disease, or death under specific circumstances.

By convention, excess lifetime cancer risks derived from the above equation are typically expressed in scientific notation. For example, a computed risk of 0.00004 is written as 4×10^{-5}. Alternatively, this same risk could be expressed as a lifetime rate. This is obtained simply by taking the reciprocal of the computed excess risk. For instance, a lifetime risk of 4×10^{-5} is the same as 1 additional cancer in 25,000 individuals over a 75 yr period, (i.e. 1/0.00004 = 25,000). Similarly, 1×10^{-6} is the oft-referenced 1 in a million cancer risk. Finally, risk values can be expressed as a standard rate per 100,000 individuals. Extending our original example, 1 in 25,000 could be written as 4 in 100,000. Risk projections produced by the Delaware Fish Contaminants Committee follow the first two conventions.

The final point to be made about the above equation is that it is written in terms of "excess" risk because risks associated with exposure to environmental contaminants are added to, or in "excess" of, cancer risks associated with other sources, (e.g. tobacco smoke, indoor radon, ambient air pollution, etc.).

The previous equation applies to the case where a person is consuming fish which contains a single contaminant. The more common situation, however, is that the person is simultaneously exposed to multiple pollutants in the fish. It is unknown at present whether the risk associated with multiple pollutants is greater than, less than, or equal to the sum of the risks for each pollutant taken individually. As a working hypothesis, simple additivity of risk is often assumed, as it has been by the Delaware Fish Contaminants Committee. This assumption, which is expressed in equation form below, is consistent with federal guidelines [EPA, 1986b]. This assumption is most reasonable in the case where the multiple contaminants are all associated with the same cancer site, (e.g., liver).

$$\frac{AGGREGATE\ LIFETIME}{CANCER\ RISK} = \sum_{i=1}^{n} (LIFETIME\ CANCER\ RISK)_i$$

Knowing the aggregate cancer risk from the equation above and cancer risk associated with each chemical individually, the proportion of risk attributable to each chemical is easily computed as follows:

$$\frac{\%\ of\ AGGREGATE\ RISK}{DUE\ to\ CHEMICAL\ i} = \frac{CANCER\ RISK\ FOR\ CHEMICAL\ i}{AGGREGATE\ CANCER\ RISK}\ x\ 100$$

2.1.4.2 Non-carcinogenic Effects

The magnitude of non-cancer health effects is determined by taking the ratio of the estimated average daily dose (ADD) to the RfD for the chemical of interest. This ratio is referred to as the Hazard Index. Hazard indices greater than 1 indicate that a potential non-cancer hazard exists. Hazard indices less than 1 are expected to be without appreciable risk of adverse effects.

$$H.I. = \frac{ADD}{RfD}$$

Similar to the approach used for cancer effects, an aggregate hazard index for chronic systemic health effects can also be computed along with the proportion of the hazard index attributable to each chemical in the mixture. The governing equations follow.

$$\frac{AGGREGATE}{HAZARD\ INDEX} = \sum_{i=1}^{n} (HAZARD\ INDEX)_i$$

$$\frac{\%\ of\ AGGREGATE\ H.I.}{DUE\ to\ CHEMICAL\ i} = \frac{HAZARD\ INDEX\ CHEMICAL\ i}{AGGREGATE\ HAZARD\ INDEX}\ x\ 100$$

2.2 Special Considerations for Selected Contaminants

2.2.1 Polychlorinated Biphenyls (PCBs) and Dioxins/Furans

One of the noteworthy elements of Delaware's fish tissue monitoring program is that it includes congener-specific PCB and dioxin/furan data. This testing began in 1992 with a subset of possible congeners selected based upon their molecular structure, toxicity, prevalence in the environment, prevalence in human tissue and blood, and their contribution as principal components in commercial Aroclor mixtures. Additional PCB congeners were added to our target list over time as experience, knowledge, and information increased. Beginning in 2003, Delaware began to target all 209 possible PCB congeners, which is the maximum number of congeners that theoretically exists.

In addition to the PCBs, all 2,3,7,8-substituted dioxins and furans, as well as all dioxin and furan homolog groups are also included in Delaware's fish tissue monitoring program. PCB congeners and dioxins/furans are quantified at the sub-parts per trillion level using high-resolution gas chromatography, high resolution mass spectrometry, (HRGC/HRMS). PCB congeners are analyzed by EPA Method 1668 and dioxins and furans are analyzed by EPA Method 1613B. All of this work is performance-based, being accompanied by extensive quality control, quality assurance data.

The highly detailed PCB and dioxin/furan data allows Delaware to take an in-depth approach to evaluating the human health risks associated with these contaminants. This section describes Delaware's approach for these contaminants.

Strong evidence exists to suggest that certain dioxins, furans, and coplanar PCBs follow a common receptor mediated physiological mechanism of toxicity (Safe 1990; Safe, 1994). This observation has led to the so-called "toxicity equivalency" approach for assessing the cumulative risk of these compounds. In applying the toxicity equivalency approach, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, (2,3,7,8- TCDD, or "dioxin" for short) is taken as the most toxic representative of the entire class of dioxin-like compounds. Other dioxin-like compounds in the class are assigned toxicities relative to that of 2,3,7,8-TCDD. As a means of normalizing the relative toxicities, 2,3,7,8-TCDD is assigned a toxicity equivalency factor (TEF) of 1 and the other dioxin-like compounds are assigned TEF values less than 1 based on the results of *in vivo* and *in vitro* studies. Operationally, the toxicity equivalents (or TEQs) of an environmental sample containing dioxins, furans, and dioxin-like PCBs is computed using the following equation:

$$TEQ = \sum_{i=1}^{n} (TEF_i \times C_i)$$

where:

TEF_i = Toxicity Equivalency Factor for the ith member, and
C_i = Concentration of the ith member

In evaluating its fish tissue data, Delaware uses the TEFs published by Van den Berg (Van den Berg, et.al., 2006). The TEFs for dioxins and furans are presented in Table 2 and the TEFs used for dioxin-like PCB congeners are reproduced in Table 3. Collectively, these TEFs are often referred to as the WHO 2005 TEFs based upon the June 2005 World Health Organization expert meeting where these TEFs were established. Prior to the release of WHO 2005 TEFs, Delaware used a combination of TEFs from different sources in the literature. For dioxins and furans, TEFs were taken from EPA's revised interim methods for estimating toxicity concentrations (Barnes and Bellin, 1989), then later from Van den Berg, et.al., 1998). For coplanar PCBs, Delaware had previously used TEFs recommended by an international panel of experts that met in December 1993, also as part of a consultation by the World Health Organization, (Ahlborg, et. al., 1994). Delaware has calculated TEQs for its fish samples using both the older TEFs and the recent TEFs and found there to very little practical difference. Researchers from the National Institute of Environmental Health Sciences and elsewhere published a study which supports the use of the TEF approach for dioxins, furans, and dioxin-like PCBs (Walker, et.al., 2005). The EPA has also recently recommended the TEQ approach in evaluating human health risks posed by dioxin and furan and dioxin-like PCB and has specifically recommended the use of the WHO 2005 TEFs (EPA, 2010).

Table 2. Toxicity Equivalency Factors (TEFs) for Tetra- through Octa-Chlorinated Dibenzo-*p*-Dioxins and Dibenzofurans

Analyte	TEF
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0003
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.03
2,3,4,7,8-PeCDF	0.30
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01

1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0003

Table 3. Toxicity Equivalency Factors (TEFs) for Dioxin-Like PCBs

Type	Congener		TEF
	IUPAC No.	Structure	
Non-*ortho*	77	3,3',4,4'-TetraCB	0.0001
	81	3,4,4',5-TetraCB	0.0003
	126	3,3',4,4',5-PeCB	0.1
	169	3,3',4,4',5,5'-HxCB	0.03
Mono-*ortho*	105	2,3,3',4,4'-PeCB	0.00003
	114	2,3,4,4',5-PeCB	0.00003
	118	2,3',4,4',5-PeCB	0.00003
	123	2',3,4,4',5-PeCB	0.00003
	156	2,3,3',4,4',5-HxCB	0.00003
	157	2,3,3',4,4',5'-HxCB	0.00003
	167	2,3',4,4',5,5'-HxCB	0.00003
	189	2,3,3',4,4',5,5'-HpCB	0.00003

Once the concentration of each of the dioxins and furans from Table 2 and each of the coplanar PCB congeners from Table 3 have been determined, those concentrations are multiplied by their respective TEFs. The resulting values are then added together per the previous equation to produce Total TEQs. The fraction of the Total TEQs associated with dioxins and furans is simply the sum of the TEQs from dioxins and furans divided by the Total TEQs. Similarly, the fraction of the Total TEQs from PCBs is the sum of the TEQs from PCBs divided by the Total TEQs.

Once Total TEQs have been computed, the resulting concentration is substituted as C_f into the equation presented previously for Lifetime Average Daily Exposure (LADE). Lifetime Average Daily Dose (LADD) is then computed from LADE, and finally, lifetime cancer risk is computed as the product of LADD and the cancer potency slope, q_1^*. In this case, the cancer potency slope used is 1.56+05 per mg/kg-d, which is the slope factor for 2,3,7,8-TCDD. Computing cancer risk in this way accounts for dioxins, furans, and dioxin-like PCBs. It does

not, however, account for the nondioxin- like fraction of the Total PCB. To address this fraction, we first subtract the concentration of the 12 dioxin-like PCB congeners listed in Table 3 from Total PCB. The resulting concentration, referred to as nondioxin-like PCB, is then substituted into the equation for LADE. As before, LADE is then used to compute LADD, which in turn is multiplied by a cancer potency slope to yield lifetime cancer risk. This time, the cancer potency slope used is 2 per mg/kg-d. This slope factor is EPA's recommended upper bound estimate for PCBs when the route of human exposure is through the aquatic food chain, (EPA, 1996).

The procedure used to address the noncancer health effects of dioxins, furans, and PCBs follows the same general approach described above. First, TEQ and nondioxin-like PCB are calculated exactly as previously described. Average daily dose (ADD) for TEQ and ADD for non-dioxin-like PCB are then calculated. Finally, those ADDs are divided by their respective RfDs to produce Hazard Index (H.I.) values.

2.2.2 DDT, DDD, and DDE

Delaware analyzes the *o,p* and *p,p* isomers of DDT, DDD, and DDE as a part of its fish tissue monitoring program. For purposes of risk assessments performed by the Delaware Fish Contaminants Committee, the concentrations of these six chemicals are lumped together to produce a single value referred to as Total DDT, or DDX. A single Reference Dose of 5E-04 mg/kg-d, and a single cancer potency slope of 0.34 per mg/kg-d, are applied to the lumped concentration. This is consistent with the approach recommended in EPA's fish tissue monitoring and advisory guidance documents (EPA, 2000a; EPA, 2000b).

2.2.3 Chlordane

Delaware's fish tissue monitoring program includes the *cis* and *trans* isomers of Chlordane, Oxychlordane (an important metabolite of Chlordane), and the *cis* and *trans* isomers of Nonachlor (which are impurities in Chlordane). Similar to the approach used for DDT, the various isomers of Chlordane and Nonachlor, plus Oxychlordane are added together for purposes of the exposure and risk assessment. The lumped concentration is referred to as Total Chlordane. A Reference Dose of 5E-04 mg/kg-d and a cancer potency slope of 0.35 per mg/kg-d are applied to the lumped concentration. The approach of adding the five components together is consistent with EPA guidance (EPA, 2000a; EPA, 2000b).

2.2.4 Polyaromatic Hydrocarbons (PAHs)

Beginning in 2013, Delaware began to regularly include PAHs in its fish tissue testing program. Prior to that, PAHs were tested on an as needed basis, typically in response to a spill or a special investigation. Testing since 2013 suggests that, although PAHs are ubiquitous in the environment, they rarely contribute significantly as risk drivers for fish consumption.

Nevertheless, Delaware will continue to gather data on PAHs in fish for the foreseeable future. For purposes of fish advisories, Delaware's approach for PAHs is similar to that used for dioxins, furans, and coplanar PCBs; namely, toxicity equivalents are computed for PAH compounds as a class. In this case, the reference compound, (i.e., the one for which a TEF of 1 is assigned) is taken as Benzo[a]Pyrene. Hence, Benzo[a]Pyrene toxicity equivalents, or B[a]P TEQs for short, are computed as the sum of the detected PAH compounds times their corresponding toxicity equivalency factor (TEF). Delaware uses the TEFs in Table 4 below, which are derived from Nisbet and LaGoy, 1992. This approach is also consistent with EPA guidance (EPA, 2000a; EPA, 2000b).

Table 4. Toxicity Equivalency Factors (TEFs) for Polyaromatic Hydrocarbons

Analyte	TEF
Dibenzo[a,h]anthracene	5
Benzo[a]pyrene	1
Benzo[a]anthracene	0.1
Benzo[a]fluoranthene	0.1
Benzo[k]fluoranthene	0.1
Indeno[1,2,3-cd]pyrene	0.1
Anthracene	0.01
Benzo[g,h,i]perylene	0.01
Chrysene	0.01
Acenaphthene	0.001
Acenaphthylene	0.001
Fluoranthene	0.001
Fluorene	0.001
2-Methylnaphthalene	0.001
Naphthalene	0.001
Phenanthrene	0.001
Pyrene	0.001

Once B[a]P TEQs are computed, the resulting concentration is substituted as C_f into the equation for Lifetime Average Daily Dose, LADE. Then, Lifetime Average Daily Dose (LADD) is computed from LADE, and finally lifetime cancer risk is estimated as the product

of LADD and a cancer potency slope, q_1^*. In this case, the cancer potency slope used is 1 per mg/kg-d, which is the slope factor for Benzo[a]Pyrene. The reference dose used for noncancer health risk is 3E-4 mg/kg-d. These are based on the January 19, 2017 update to IRIS.

2.2.5 Arsenic

Arsenic exists in fish in both organic and inorganic forms. Of these two forms, organic arsenic predominates. This is fortunate because inorganic arsenic is believed to represent a much greater health risk than organic arsenic, which is rapidly excreted upon ingestion. Accounting for the toxic (inorganic) fraction of arsenic in fish is critically important in risk assessment. Typical analytical methods however do not distinguish between the organic and inorganic forms. To avoid overstating the health risk associated with arsenic in fish, Delaware had traditionally applied a 10% adjustment factor to its total arsenic data to arrive at an estimate of the concentration of inorganic arsenic in the fish. This adjustment factor is based upon advice from the U.S. Food and Drug Administration (FDA, 1993). The resulting estimate of inorganic arsenic was then used for purposes of exposure and risk estimates. Based upon a fairly detailed study conducted by the DNREC in 2002 and 2003 in which inorganic arsenic was measured along with total arsenic and methyl arsenic compounds, we found that inorganic arsenic represents, on average, only 1.2% of total arsenic (Greene and Crecelius, 2006). Based on these findings, Delaware dropped the analysis of total arsenic from its routine fish tissue monitoring program. When arsenic testing is necessary in fish based upon some site-specific issue, Delaware will use the speciation approach in which inorganic arsenic is specifically tested. That fraction will be carried forward for exposure and risk assessment.

2.2.6 Mercury

Up until 2013, Delaware had measured total mercury as a part of its fish tissue monitoring program. Total mercury measures both inorganic and organic forms of mercury. Conventional wisdom holds that the vast majority (~95%) of mercury in fish exists in the organic form, specifically methyl mercury, which is a known toxin. For this reason, Delaware had used total mercury as an estimate of methyl mercury in the fish. Several contemporary studies have cast doubt on whether total mercury should universally be assumed to be equal to methylmercury. To eliminate this assumption, Delaware began testing both total mercury (by EPA method 1631E) and methylmercury (by EPA method 1630) in fish tissue in 2013. This not only allows the common assumption to be tested but more importantly it provides a direct measurement of methylmercury, which can be compared directly to the Delaware and EPA fish tissue methylmercury criterion of 300 ng/g (ppb).

2.2.7 Polybrominated Diphenyl Ethers (PBDEs)

Delaware measured PBDEs in fish tissue samples collected from several Delaware waters between 2003 and 2006. Delaware's data were merged and assessed along with similar regional data generated by the Delaware River Basin Commission (DRBC), the Academy of Natural Sciences (ANS), and the New Jersey Department of Environmental

Protection (NJDEP). Risk assessment calculations using worst case maximum observed PBDE concentrations showed low human health risk. The data summary and risk assessment were presented at the National Forum on Contaminants in Fish (Greene, 2007). Based on its findings, Delaware discontinued analysis of PBDEs in fish tissue. Delaware will continue to track developments regarding PBDEs and is prepared to reinstitute testing if justified.

2.3 Fish Tissue Screening Values

The equations presented in section 2.2 yield a quantitative estimate of human health risk associated with a specific fish tissue contaminant concentration. "Risk," in this formulation, is the "dependent" variable that depends on the "independent" variables, one of which is the concentration of the contaminant in the fish. In certain cases, it is desirable to rearrange the equations to make risk an "independent" variable and fish tissue concentration the "dependent" variable. For instance, we may be interested in knowing the PCB concentration in fish that is associated with a pre-specified, acceptable risk level such as 1-in-100,000. When tissue concentrations are computed in this way, the resulting values are often referred to as screening levels because they can be quickly compared to (i.e., "screened" against) field data on fish contaminant levels. Field data greater than the screening levels are worthy of further evaluation, possibly including additional data collection, detailed risk analysis, and potential risk management action.

It is important to note that fish tissue screening values are not intended to replace formal risk analysis. Rather, they help the assessor to decide whether a detailed risk analysis is even warranted and how to prioritize several analyses if screening values are exceeded at more than one location. This section outlines the procedures used by the Delaware Fish Contaminants Committee to compute fish tissue screening values.

The equations presented in section 2.2 can be rearranged and solved uniquely for the concentration of the contaminant in the fish. Doing so for the case of carcinogenic effects, we obtain:

$$\frac{C_f}{(ppm)} = \frac{Risk \times BW \times LT}{q_1^* \times AF\ MS \times MF \times ED \times (100 - RF)/100}$$

In a similar manner, we can derive the following equation for the case of non-carcinogenic health effects:

$$\frac{C_f}{(ppm)} = \frac{Hazard \times BW \times RfD}{AF \times MS \times MF \times (100 - RF)/100}$$

Appendix C presents tissue screening values for many common contaminants in fish. Screening values for the cancer endpoint are based on a target risk level of 1-in-100,000, while screening values for non-cancer effects are based on a target hazard index of 1.0. Screening values are presented for three separate exposure groups under the conservative assumption that each group consumes 1 fish meal per week.

Please note that the approach taken for mercury departs somewhat from the other chemicals in Appendix C. In July of 2004, the Department of Natural Resources and Environmental Control adopted a water quality criterion of 0.3 ppm (300 ppb) for methyl mercury in fish (DNREC, 2004). This criterion is based upon EPA's recommended water quality criterion for methylmercury (EPA, 2001), which assumes an RfD for methylmercury of 1E-4 mg/kg-d, a fish consumption rate of 17.5 g/d partitioned among three trophic levels, a body weight of 70 kg, and a relative source contribution of 2.7E-5 mg/kg-d to account for consumption of fully marine fish species.

The screening values in Appendix C are subject to change based upon updates to potency slopes, reference doses, and other relevant factors.

2.4 Maximum Number of Fish Meals Per Time

The concepts introduced in sections 2.2 and 2.3 can be extended to answer the basic question of how many meals of fish can be safely consumed from a given waterbody while maintaining health risk at a low level. To answer this question, we rearrange the equations presented previously, solving for meal frequency as the dependent variable. If we specify a maximum allowable cancer risk or maximum hazard index for a given set of exposure factors, then the resulting meal frequency is really the maximum number of meals per unit time.

For the general (and typical) case of multiple contaminants in fish, where each contaminant exhibits an ability to contribute to cancer risk, the equation describing the maximum number of meals per time is as follows:

$$\frac{Max\ No.\ of}{Meals\ /\ Year} = \frac{Aggregate\ Risk \times BW \times LT \times 365.25}{MS \times ED \times AF \times ((100 - RF)/100) \times [C_1(q_1^*)_1 + C_2(q_1^*)_2 + \ldots + C_n(q_1^*)_n]}$$

In the above equation, "Risk" is the target aggregate cancer risk level associated with all contaminants in the fish combined. For purposes of this exercise, this is set at some low, *de Minimus* risk level such as 1-in-100,000 (i.e., 10^{-5}). The assumption inherent in the above formulation is that the contaminants follow a similar mode of cancer action and that their combined effect is additive. If these assumptions are known not to be true, the above equation

can be decomposed into separate equations for each chemical or group of similar-acting chemicals. The other important assumption made in the above formulation is that gastrointestinal efficiency factor, AF, and the reduction factor for trimming and cooking, RF, are similar for all contaminants identified in the fish. If this is not the case and the assessor wishes to use different values for AF and RF for each chemical, then the above equation can be easily modified by placing separate factors inside the brackets of the denominator for each chemical.

In a similar manner, an equation can be derived that provides an estimate of the maximum number of fish meals per time at a pre-defined aggregate hazard index. In the general case of multiple contaminants that contribute to noncancer health risk, the resulting equation is as follows:

$$\frac{Max\ No.of}{Meals\ /\ Year} = \frac{Aggregate\ Hazard\ Index\ x\ BW\ x\ LT\ x\ 365.25}{MS\ x\ ED\ x\ AF\ x((100-RF)/100)\ x[(C_1/RfD_1)+(C_2/RfD_2)+...+(C_n/RfD_n)]}$$

Aggregate Hazard Index is set at a predefined acceptable level such as 1.0. The reduction factor for trimming and cooking, RF, as well as the GI adsorption efficiency factor, AF, can be handled as described above. Again, the limitations regarding similar mode of action and additivity of multiple contaminants also apply to the above equation for noncancer effects.

3.0 DISCUSSION

Fish contamination has become an issue of national importance over the past decade, not because the problem just recently materialized, but rather because the techniques for detecting the problem and interpreting the results have improved. The new techniques are generally more protective of the local angler than the previous approach of simply comparing tissue levels to FDA action and tolerance levels. The result has been a significant increase in the number of fish advisories that have been issued locally, regionally, and nationally.

It is important to note that the technical procedures described in this document are substantially equivalent to the national guidance cited herein. Delaware takes a risk-based approach as recommended in the national guidance, considering both potential cancer and non-cancer health risks. In addition, Delaware considers different exposure groups (including special at risk groups) as recommended in the national guidance. Delaware also relies upon the same toxicological data for hazard identification and dose-response as cited in the national guidance. Finally, as recommended in the national guidance, Delaware takes a tiered approach to fish tissue contamination investigations and considers tissue screening values to justify proceeding to a more intensive phase.

There are several minor differences between Delaware's overall approach and the approach outlined in the national guidance. Noteworthy differences include the use of a 30 year exposure period by Delaware for cancer risk to adults rather than 70 years as recommended in the national guidance. Delaware uses a 30 year exposure period to ensure consistency with the Federal and State Superfund Programs. It is those programs that are ultimately responsible for defining the level of cleanup needed for each contaminated waste site that releases or has the potential to release contaminants to surface waters. Since Superfund uses a 30 year exposure assumption for carcinogens, the Delaware fish contamination program believes it makes sense, from a cross-media and cross-programmatic perspective, to use this same exposure period. Another related difference is that Delaware assumes an average life expectancy of 75 years as opposed to 70 years as recommended by the EPA. Delaware believes the most recent vital statistics data clearly supports the 75 year assumption. EPA's use of 70 years appears to be rooted in their desire to be consistent with their own historic practice.

Other differences between the EPA fish contamination guidance and Delaware's approach are briefly discussed below.

- EPA recommends that mean contaminant concentrations in fish be used for purposes of evaluating the need for fish advisories. It has been Delaware's experience that contaminant levels in fish are not always normally distributed and hence means are not always the best measure of exposure concentration. When sufficient data are available, Delaware evaluates the distribution of the data first to decide the best measure of exposure concentration. Delaware uses the mean for normally distributed data and the median for non-normally distributed data.

- Delaware does not typically test for organophosphate (OP) pesticides, chlorophenoxy herbicides, or tributyltin (TBT) in fish tissues unless a specific needs exists. Limited testing that Delaware has performed on OP pesticides and chlorophenoxy herbicides in fish tissue has all been "non-detected." Under this circumstance, it is hard to justify the added expense of a separate analytical test.

- For the most intensive fish contamination studies, EPA recommends that 3 separate size classes of 2 separate fish species be captured for analysis. It has been Delaware's experience that it is extremely labor intensive to capture 3 separate size classes of 1 fish species, let alone 2 species. This challenge becomes even more extreme when sampling must be performed at several locations. Delaware feels its current approach of obtaining single composite samples for screening studies and replicate samples at multiple stations in intensive studies is a reasonable approach to field collection. Delaware does collect and analyze different size fish when the specific need arises

- Delaware takes a much more detailed approach to PCBs in fish tissue than is recommended in the national guidance. Delaware, through contract assistance,

analyzes PCBs at the congener level using a state-of-the-science, performance-based method. Risk analyses performed by Delaware for PCBs consider and differentiate between dioxin-like PCBs and nondioxin-like PCBs, again exceeding expectations in the national guidance.

Delaware has allocated an estimated $50,000 to $100,000 per year in generating and reviewing high quality fish tissue data since the MOU was signed back in 1993, while annual budgets prior to that time were generally less than $10,000. The allocation of resources to monitoring, coupled with greater focus on cleaning up remaining sources of contamination, has allowed Delaware to track long-term improvements in contaminant levels in local fish. Despite the improvements, much work remains and so there will be a need to continue monitoring and to adjust advisories as appropriate for the foreseeable future.

A final point made here relates to advisory awareness. Once an advisory is issued, a key objective is to make sure the public is aware of the advisory so they can make an informed decision concerning their catch. Delaware attempts to increase awareness of fish advisories through a variety of means, including:

- Issuing a press release when a new advisory is issued. These releases are nearly always picked up by local and regional news outlets, including newspapers, radio stations and TV stations.
- Posting of multi-lingual advisory signs at known fishing access locations.
- A special section on fish advisories within Delaware's Annual Fishing Guide. Approximately 100,000 hard copies of the fishing guide are distributed annually through various retail outlets (e.g., bait and tackle shops; sporting goods stores), State parks, and other venues. The Fishing Guide is also available on DNREC's web site.
- A special 'Fish Smart, Eat Smart' trifold brochure was designed to highlight Delaware's fish advisories. Funding for the brochure came from Delaware's Cancer Consortium. Thousands of copies were printed in English and Spanish and were provided to Public Health Clinics throughout the State. They were also distributed at various public events including the Delaware State Fair and the University of Delaware Coast Day. The brochure also appears on DNREC's web site.
- Presentations at various national, regional, and State conferences (including Delaware's Oncology and Hematology Conference, intended to increase awareness among the medical community).
- Direct response to phone and written inquiries.

Not only have we used the above techniques to get the word out, we have also conducted research to find out what the level of awareness is among recreational anglers regarding Delaware's fish advisories. That research indicates that roughly half of the

interviewed anglers were aware of Delaware's advisories, with residents having a much higher level of awareness than non-residents (62.4% vs 31.7%). The awareness rate among Delaware resident anglers is comparable to that among avid Great Lakes recreational anglers (70%), who have been saturated with health advisory messages for over 20 years. Delaware's rate of awareness is therefore considered good but not great and suggests we need to continue to get the word out.

The Delaware Fish Contamination Committee will update this document from time to time as necessary to reflect any major changes in the technical procedures or to clarify the existing approach.

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APPENDIX A

Target Chemical Contaminants in Delaware's Fish Tissue Monitoring Program and Associated Detection Limits

Target PCB Congeners, Homologs and Total (analysis by EPA Method 1668A)

PCB-1	PCB-52	PCB-118	PCB-176
PCB-2	PCB-54	PCB-120	PCB-177
PCB-3	PCB-55	PCB-121	PCB-178
PCB-4	PCB-56	PCB-122	PCB-179
PCB-5	PCB-57	PCB-123	PCB-180 + 193
PCB-6	PCB-58	PCB-126	PCB-181
PCB-7	PCB-59 + 62 + 75	PCB-127	PCB-182
PCB-8	PCB-60	PCB-128 + 166	PCB-183 + 185
PCB-9	PCB-61 + 70 + 74 + 76	PCB-129 + 138 + 160 + 163	PCB-184
PCB-10	PCB-63	PCB-130	PCB-186
PCB-11	PCB-64	PCB-131	PCB-187
PCB-12 + 13	PCB-66	PCB-132	PCB-188
PCB-14	PCB-67	PCB-133	PCB-189
PCB-15	PCB-68	PCB-134 + 143	PCB-190
PCB-16	PCB-72	PCB-135 + 151 + 154	PCB-191
PCB-17	PCB-73	PCB-136	PCB-192
PCB-18 + 30	PCB-77	PCB-137	PCB-194
PCB-19	PCB-78	PCB-139 + 140	PCB-195
PCB-20 + 28	PCB-79	PCB-141	PCB-196
PCB-21 + 33	PCB-80	PCB-142	PCB-197 + 200
PCB-22	PCB-81	PCB-144	PCB-198 + 199
PCB-23	PCB-82	PCB-145	PCB-201
PCB-24	PCB-83 + 99	PCB-146	PCB-202
PCB-25	PCB-84	PCB-147 + 149	PCB-203
PCB-26 + 29	PCB-85 + 116 + 117	PCB-148	PCB-204
PCB-27	PCB-86 + 87 + 97 + 108 + 119 + 125	PCB-150	PCB-205
PCB-31	PCB-88 + 91	PCB-152	PCB-206
PCB-32	PCB-89	PCB-153 + 168	PCB-207
PCB-34	PCB-90 + 101 + 113	PCB-155	PCB-208
PCB-35	PCB-92	PCB-156 + 157	PCB-209
PCB-36	PCB-93 + 95 + 98 + 100 + 102	PCB-158	Total Monochloro Biphenyls
PCB-37	PCB-94	PCB-159	Total Dichloro Biphenyls
PCB-38	PCB-96	PCB-161	Total Trichloro Biphenyls
PCB-39	PCB-103	PCB-162	Total Tetrachloro Biphenyls
PCB-40 + 41 + 71	PCB-104	PCB-164	Total Pentachloro Biphenyls
PCB-42	PCB-105	PCB-165	Total Hexachloro Biphenyls
PCB-43	PCB-106	PCB-167	Total Heptachloro Biphenyls
PCB-44 + 47 + 65	PCB-107 + 124	PCB-169	Total Octachloro Biphenyls
PCB-45 + 51	PCB-109	PCB-170	Total Nonachloro Biphenyls
PCB-46	PCB-110 + 115	PCB-171 + 173	Decachloro Biphenyl
PCB-48	PCB-111	PCB-172	TOTAL PCBs
PCB-49 + 69	PCB-112	PCB-174	
PCB-50 + 53	PCB-114	PCB-175	

Target List of 2,3,7,8-Substituted Dioxins and Furans and Dioxin and Furan Homologs (analysis by EPA Method 1613B)

2,3,7,8-TCDD	TOTAL TETRA-DIOXINS
1,2,3,7,8-PECDD	TOTAL PENTA-DIOXINS
1,2,3,4,7,8-HXCDD	TOTAL HEXA-DIOXINS
1,2,3,6,7,8-HXCDD	TOTAL HEPTA-DIOXINS
1,2,3,7,8,9-HXCDD	TOTAL TETRA-FURANS
1,2,3,4,6,7,8-HPCDD	TOTAL PENTA-FURANS
OCDD	TOTAL HEXA-FURANS
2,3,7,8-TCDF	TOTAL HEPTA-FURANS
1,2,3,7,8-PECDF	
2,3,4,7,8-PECDF	
1,2,3,4,7,8-HXCDF	
1,2,3,6,7,8-HXCDF	
1,2,3,7,8,9-HXCDF	
2,3,4,6,7,8-HXCDF	
1,2,3,4,6,7,8-HPCDF	
1,2,3,4,7,8,9-HPCDF	
OCDF	

Target Organochlorine Pesticides (Lists 1, 2 and 3)
(analysis by modified EPA Method 8081)

List 1 (E1)	**List 2 (E2)**	**List 3**
Hexachlorobenzene	HCH, delta	Technical Toxaphene
HCH, alpha	Heptachlor Epoxide	Methoxychlor
HCH, beta	alpha-Endosulfan	
HCH, gamma	Dieldrin	
Heptachlor	Endrin	
Aldrin	beta-Endosulfan	
Chlordane, oxy-	Endosulfan Sulfate	
Chlordane, gamma (trans)	Endrin Aldehyde	
Chlordane, alpha (cis)	Endrin Ketone	
Nonachlor, trans-		
Nonachlor, cis-		
2,4’-DDD		
4,4’-DDD		
2,4’-DDE		
4,4’-DDE		
2,4’-DDT		
4,4’-DDT		
Mirex		

Target Polyaromatic Hydrocarbons (PAHs) Lists 1, 2 and 3 (analysis by HRGC/LRMS)

List 1 – Standard PAH Parents and Select Alkylated PAHs	List 2 – Additional Alkylated PAHs	List 3 – Extended Alkylated PAHs
Naphthalene	1-methylnaphthalene	C1-Biphenyls
Acenaphthylene	C1-Naphthalenes	C2-Biphenyls
Acenaphthene	1,2-Dimethylnaphthalene	C1-Acenaphthenes
Fluorene	C2-Naphthalenes	2-Methylfluorene
Phenanthrene	2,3,6-Trimethylnaphthalene	C1-Fluorenes
Anthracene	C3-Naphthalenes [1]	1,7-Dimethylfluorene
Fluoranthene	1,4,6,7-Tetramethylnaphthalene	C2-Fluorenes
Pyrene	C4-Naphthalenes	C3-Fluorenes
Benz(a)anthracene	2-Methylphenanthrene	2/3-Methyldibenzothiophenes
Chrysene	3-Methylphenanthrene	C1-Dibenzothiophene
Benzo(b)fluoranthene	9/4-Methylphenanthrenes	2,4-Dimethyldibenzothiophene
Benzo(j/k)fluoranthenes	2-Methylanthracene	C2-Dibenzothiophene
Benzofluoranthenes	C1-Phenanthrenes/Anthracenes	C3-Dibenzothiophene
Benzo(e)pyrene	1,7-Dimethylphenanthrene	C4-Dibenzothiophene
Benzo(a)pyrene	1,8-Dimethylphenanthrene	3-Methylfluoranthene/Benzo(a)fluorene
Perylene	2,6-Dimethylphenanthrene	C1-Fluoranthenes/Pyrenes
Dibenzo(ah)anthracene	3,6-Dimethylphenanthrene	C2-Fluoranthenes/Pyrenes
Indeno(1,2,3-cd)pyrene	C2-Phenanthrenes/Anthracenes	C3-Fluoranthenes/Pyrenes
Benzo(ghi)perylene	1,2,6-Trimethylphenanthrene	C4-Fluoranthenes/Pyrenes
2-Methylnaphthalene	C3-Phenanthrenes/Anthracenes	1-Methylchrysene
2,6-Dimethylnaphthalene	Retene	5/6-Methylchrysenes
2,3,5-Trimethylnaphthalene	C4-Phenanthrenes/Anthracenes	C1-Benz(a)anthracenes/Chrysenes
1-Methylphenanthrene	Biphenyl	5,9-Dimethylchrysene
Dibenzothiophene		C2-Benz(a)anthracenes/Chrysenes
		C3-Benz(a)anthracenes/Chrysenes
		C4-Benz(a)anthracenes/Chrysenes
		7-Methylbenzo(a)pyrene
		C1-Benzofluoranthenes/Benzopyrenes
		C2-Benzofluoranthenes/Benzopyrenes

Target Metals (total mercury analyzed by EPA Method 1631E; methylmercury by EPA Method 1630)

Total Mercury
Methylmercury

Other Measurements

Percent Moisture
Percent Lipid

Target Detection Limits (Range) by Analyte Group

Analyte Group	DL Range	Units
209 PCB Congeners	0.1 - 0.2	pg/g
2,3,7,8-Substituted Dioxins and Furans	0.05 - 0.10	pg/g
Organochlorine Pesticides List 1	10 - 20	pg/g
Organochlorine Pesticides List 2	50	pg/g
Organochlorine Pesticides List 3	100	pg/g
Polyaromatic Hydrocarbons	100 - 200	pg/g
Total Mercury	0.36 - 0.64	ug/kg
Methylmercury	3	ug/kg

APPENDIX B

Reference Doses and Cancer Potency Slopes for Common Fish Contaminants

Contaminant	Reference Dose, RfD (mg/kg-d)	Cancer Potency Slope, q_1^* (1/(mg/kg-d))
Metals		
Arsenic (inorganic)	3E-04	1.5
Cadmium	1E-03	-
Mercury (methyl)	1E-04	-
Selenium	5E-03	-
Organochlorine Pesticides		
Total Chlordane	5E-04	0.35
Total DDT	5E-04	0.34
Dicofol	1E-03	-
Dieldrin	5E-05	16
Endosulfan (I and II)	6E-03	-
Endrin	3E-04	-
Heptachlor epoxide	1.3E-05	9.1
Hexachlorobenzene	8E-04	1.6
Lindane	3E-04	1.3
Mirex	2E-04	-
Toxaphene	2.5E-04	1.1
Polyaromatic Hydrocarbons		
B[a]P TEQs	3E-04	1
Polychlorinated Biphenyls		
Nondioxin-Like PCBs	2E-05	2.0
Dioxin-Like PCB TEQs	7E-10	1.56E+05
TCDD TEQs		
2,3,7,8-Dioxins & Furans TEQs	7E-10	1.56E+05

APPENDIX C

Delaware Screening Values for Common Chemical Contaminants in Fish Tissue

I. Exposure Factors

	Ave Adult	Woman	Child
Body Weight (Kg)	70	64	14.5
Meal Size (ounces)	8	6	3
Meal Frequency (#/week)	1	1	1
Exposure Duration (years)	30	30	6
Lifetime Duration (years)	75	75	75
Reduction for Cooking/Cleaning, %	0	0	0
GI Absorption Efficiency, %	100	100	100

II. Target Cancer Risk Level and Hazard Index

Target Risk Level =	0.00001
Target Harzard Index =	1

III. Toxicological Data:

Contaminant	Reference Dose (mg/kg-d)	Cancer Potency 1/(mg/kg-d)
Arsenic (inorganic)	3.0E-04	1.5
Cadmium	1.0E-03	-
Mercury (methyl)	1.0E-04	-
Selenium	5.0E-03	-
Total Chlordane	5.0E-04	0.35
Total DDT	5.0E-04	0.34
Dicofol	1.0E-03	-
Dieldrin	5.0E-05	16
Endosulfan (I and II)	6.0E-03	-
Endrin	3.0E-04	-
Heptachlor epoxide	1.3E-05	9.1
Hexachlorobenzene	8.0E-04	1.6
Lindane	3.0E-04	1.3
Mirex	2.0E-04	-
Toxaphene	2.5E-04	1.1
Benzo[a]pyrene TEQs	3.0E-04	1
Polychlorinated Biphenyls	2.0E-05	2
2,3,7,8-Substituted Dioxin & Furan TEQs	7.0E-10	1.56E+05

IV. Fish Tissue Screening Values (ppb)

	SCREENING VALUES FOR INDICATED GROUP Noncancer Endpoint			SCREENING VALUES FOR INDICATED GROUP Cancer Endpoint		
Contaminant	Average Adult	Women of Child-Bearing Age	Children	Average Adult	Women of Child-Bearing Age	Children
Arsenic	648	790	358	36	44	99
Cadmium	2161	2634	1193	na	na	na
Mercury*	300	300	300	na	na	na
Selenium	10803	13169	5967	na	na	na
Total Chlordane	1080	1317	597	154	188	426
Total DDT	1080	1317	597	159	194	439
Dicofol	2161	2634	1193	na	na	na
Dieldrin	108	132	60	3	4	9
Endosulfan (I/II)	12963	15803	7161	na	na	na
Endrin	648	790	358	na	na	na
Heptachlor epoxide	28	34	16	6	7	16
Hexachlorobenzene	1728	2107	955	34	41	93
Lindane	648	790	358	42	51	115
Mirex	432	527	239	na	na	na
Toxaphene	540	658	298	49	60	136
Benzo[a]pyrene TEQs	648	790	358	54	66	149
Polychlorinated Biphenyls	43	53	24	27	33	75
2,3,7,8-Substituted Dioxin & Furan TEQs	0.00151	0.00184	0.00084	0.00035	0.00042	0.00096

*Based on EPA and DE water quality criterion for methylmercury in fish