ANALYSIS OF RISKS FROM CONSUMPTION OF QUINCY BAY FISH AND SHELLFISH

TASK IV REPORT

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I. Introduction

This report is one of a series of studies being conducted by the U.S. Environmental Protection Agency, Region I, to investigate the types and concentrations of pollutants in sediment deposits in Quincy Bay, Massachusetts and the potential public health implications of consumption of seafood exposed to these deposits.

This series of studies was mandated by Report 99-731 of the 99th Congress, 2nd Session, U.S. House of Representatives, relative to appropriations, on page 30. Other reports in the series which have been completed include the following:

- *Types and Concentrations of Pollutants and Extent of Sludge Deposits in Quincy Bay, Massachusetts* - Draft Report by Metcalf & Eddy to U.S. EPA Region I, October, 1987.

- *A Histopathological and Chemical Assessment of Winter Flounder, Lobster, and Soft-shelled Clams Indigenous to Quincy Bay, Boston Harbor and an In Situ Evaluation of Oysters including Sediment (surface and cores) Chemistry* - Report by George R. Gardner and Richard J. Pruell, U.S. Environmental Protection Agency, Environmental Research Laboratory, Narragansett, Rhode Island to U.S. EPA Region I, December 1, 1987.

These reports provided a summary of available historical data on sediment and biological residues of contaminants in Quincy Bay and the results of field and laboratory investigations of concentrations of contaminants in Quincy Bay sediments and biota conducted by the U.S. EPA in 1987. Together, these two reports represent the results of Phases I, II, and III of the five phases of the required studies. This report presents the results of Phase IV, the analysis of risks of consuming seafood which originates in Quincy Bay. As described in more detail below, the report is based on the use of measured values of seafood contamination obtained in the Phase II and III work (Gardner and Pruell. 1987) in a quantitative risk assessment conducted according to the most recently available EPA guidance (PTI. 1987). The results of this and the previous studies are integrated in the Phase V/Task V Summary Report.

II. Approach

The general approach used in the conduct of this study involved use of the data on tissue concentrations of contaminants in Quincy Bay seafood obtained by EPA in 1987 (Gardner and Pruell. 1987) in a quantitative risk assessment following the latest available EPA guidance for studies of this type (PTI. 1987). Specific aspects of the approach to components of the risk assessment are described below.

Hazard Identification

Identification of contaminants of concern for this task was based on inclusion of those chemical species for which residue concentrations were documented in Quincy Bay seafood. These included the organic and metal compounds measured by EPA in 1987 (Gardner and Pruell. 1987). The contaminants chosen for study had the following characteristics:

- corresponding data were available for sediment and fish tissue concentrations;

- the contaminants were those for which either an EPA Carcinogenic Potency Factor (CPF) or a Reference Dose (RfD) or a U.S. Food and Drug Administration (FDA) Action Level had been published.

As recommended by PTI, 1987, the latest available compilations by the EPA of reference doses, carcinogenic potency factors, and toxicity profiles were used. We relied primarily on the EPA's Integrated Risk Information System (IRIS) data base, but supplemented it as necessary (as described in Section III below).

Dose-Response Assessment

As suggested in PTI, 1987, two forms of dose-response information were used. The first was the Carcinogenic Potency Factor (CPF), which attempts to quantify the implied finite risk of cancer at various doses of a chemical. The second, for non-carcinogens, was the reference dose (RfD), defined as the highest average daily exposure over a lifetime that would not be expected to produce adverse effects. With the exception of a congener-specific CPF for the mix of PCBs found in the 1987 Quincy Bay seafood samples, no new data in either category were developed. This CPF was developed in the manner documented in Appendix C by EPA's Office of Health and Environmental Assessment in Washington, D.C. (USEPA. 1988a).

Exposure Assessment

The Guidance Manual (PTI.1987) suggests that two forms of exposure assessment are appropriate, depending upon the level of available information. Consistent with those suggestions and the level of available information, we used the following basis for exposure assumptions:

- A dual basis for consideration of detection limit values in fish tissue, first assuming that values below the detection limit represent zero concentration and secondly assuming that the values are equal to the detection limit.

- Evaluation of risks due to consumption of three species of seafood from Quincy Bay: lobsters, flounder and soft-shelled clams.

- Use of a standard consumption rate from among those contained in the manual (PTI. 1987) for the hypothetical maximally exposed individual. Other, potentially more typical seafood consumption patterns were developed on the basis of historical surveys of fisheries consumption in New England (Penn State. 1985) and field interviews with persons familiar with the Quincy Bay fishery.

- Assumption that the ingested dose is equal to the absorbed dose of the pollutants of interest.

- Initial assumption of zero background concentration of the pollutants in other ingested items, such as drinking water and other foods. This is consistent with the overall methodology for carcinogens, which assesses incremental risk above background.

- Use of other standard assumptions for an integrated exposure analysis, including exposure over a 70-year lifetime and a body weight of the exposed individual of 70 kilograms.

- Assumption that cooking has no effect on the contaminants (as noted in Section VI, this assumption may or may not be conservative).

Risk Characterization

Based on the guidance of PTI, 1987, two measures of risk were examined:

1. The plausible upper limit to excess lifetime risk of cancer;

2. The summary of non-carcinogenic risk represented by the ratios of the estimated exposure doses to the Reference Doses for the studied chemicals.

As suggested by the Manual in its discussion of chemical mixtures, we evaluated the additive risks of the several contaminants present in the seafood as follows:

- Arithmetic summation of upper limit risks for carcinogens; and

- Arithmetic summation of the ratios of exposure dose to RfD for only those non-carcinogens acting on the same target organs.

III. Hazard Identification

To focus the public health assessment on those contaminants likely to represent the greatest risks, the 1987 Quincy Bay analytical data collected under Tasks II and III of this study and toxicity information were reviewed, including analytical data from Tasks II and III as available in the December 1, 1987 draft report (Gardner and Pruell. 1987). Maximum and mean concentrations of contaminants detected in each sediment and in each of the different seafood species tissues were summarized (Table 1). The mean concentrations represent the average with concentrations below the detection limit assumed to be equal to the detection limit, and were used in the public health assessment. A second mean was also calculated with contaminant concentrations below the detection limit assumed to equal zero. These values are included in Appendix B.

Three of the references used extensively to obtain toxicity data were (1) the *Integrated Risk Information System* (USEPA. 1986a-b; 1987d-h), an EPA-maintained computer database currently available in hard copy, (2) *Health Effects Assessment Documents* (USEPA. 1984a-j) and (3) the *Superfund Public Health Evaluation Manual* (USEPA. 1986c). The availability of data from the first two sources was also summarized (Table 1). In the *Superfund Public Health Evaluation Manual*, the Carcinogenic Potency Factor (CPF) is defined as an upper 95 percent confidence limit on the probability of carcinogenic response per unit intake of a chemical over a lifetime. The 95 percent confidence limit

TABLE 1 Summarized Contaminant Levels and Hazard Identification (a)

CHEMICAL IDENTIFIED	IRIS AVAIL (b)	HEA AVAIL (c)	FDA Limits (ppm) (d)	CPF Carcinogenic Potency Factor (mg/kg/day) 1 (e) Oral	CPF Carcinogenic Potency Factor (mg/kg/day) 1 (e) Inhaled	EPA Weight of Evidence (f)	RfD Reference Dose (mg/kg/day) (g)	REF (h)	Toxicity Rating (i)	1987 EPA Data CLAMS (Soft-shell) (ug/g wet) MAX	MEAN	LCD (j)	1987 EPA Data LOBSTER (Tissue) (ug/g wet) MAX	MEAN	LCD	1987 EPA Data LOBSTER (Hepatopancreas) (ug/g wet) MAX	MEAN	LCD	1987 EPA Data FLOUNDER (Tissue) (ug/g wet) MAX	MEAN	LCD
ELEMENTS/METALS																					
Cadmium	Y	Y	-	-	6.10E+00	B1	2.90E-04	SFUND	10	2.50E-02	2.10E-02	1.70E-02	5.00E-03	2.00E-03	1.00E-03	2.23E+00	1.31E+00	6.93E-01	9.00E-03	1.00E-03	1.00E-03
Chromium	Y	Y	-	-	4.10E+01	A	5.00E-03	SFUND	8	2.45E-01	2.06E-01	1.67E-01	2.60E 01	2.40E-02	2.00E-03	2.38E+00	7.20E-01	1.03E-01	3.77E-01	2.90E-02	0.00E+00
Copper	N	Y	-	-	-	D	3.70E-02	SFUND	5	1.95E+00	1.85E+00	1.76E+00	6.22E+00	4.06E+00	2.67E+00	2.79E+02	1.37E+02	1.77E+01	2.15E-01	1.09E-01	3.60E-02
Mercury (k)	N	Y	1.0	-	-	D	2.00E-03	IRIS	7	2.00E 03	2.00E-03	-	1.68E-01	8.50E-02	2.20E-02	1.12E-01	6.50E-02	2.00E-03	8.60E-02	3.00E-02	6.00E-03
Lead (k)	N	Y	-	-	-	C	1.40E-03	IRIS	10	4.60E-01	4.50E-01	4.40E-01	2.07E-01	1.69E-01	1.21E-01	7.00E-01	3.35E-01	1.20E-01	4.30E-02	1.50E-02	0.00E+00
ORGANIC COMPOUNDS																					
Chlordane (total) (l)	Y	Y	0.3	1.30E+00	-	B2	5.00E-05	IRIS	2	3.48E-03	2.88E-03	2.28E-03	6.07E-04	3.76E-04	1.72E-04	2.40E-01	9.75E-02	2.80E-02	3.00E-02	3.15E-03	1.63E-04
a-Chlordane	Y	Y	0.3	1.30E+00	-	B2	5.00E-05	IRIS	2	1.56E 03	1.26E-03	9.61E-04	1.92E-04	1.66E-04	1.09E-04	8.76E-02	3.18E-02	1.30E-02	7.67E-03	9.14E-04	1.09E-04
g Chlordane	Y	Y	0.3	1.30E+00	-	B2	5.00E-05	IRIS	2	1.92E 03	1.62E-03	1.32E-03	4.15E-04	2.10E-04	6.26E-05	1.52E-01	6.57E-02	1.50E-02	2.23E-02	2.24E-03	5.40E-05
PP DDD	N	N	5.0	3.40E-01	-	-	5.00E-04	SFUND	2	1.42E-03	1.23E-03	1.04E-03	6.64E-04	5.28E-04	5.78E-05	3.12E-01	1.80E-01	1.82E-02	1.33E-02	1.58E-03	1.87E-04
PP DDE	N	N	5.0	3.40E-01	-	B2	5.00E-04	SFUND	2	4.76E-03	4.26E-03	3.77E-03	7.46E-03	5.03E-03	2.83E-03	1.89E+00	1.30E+00	6.58E-01	1.59E-02	5.19E-03	1.51E-03
PP DDT	N	N	5.0	3.40E-01	-	B2	5.00E-04	SFUND	2	3.37E-04	3.03E-04	2.70E-04	6.12E-04	5.45E-04	3.99E-04	7.34E-02	2.95E-02	4.34E-03	4.97E-03	8.55E-04	4.16E-04
Hexachlorobenzene (HCB)	N	Y	-	1.69E+00	-	B2	8.00E-04	SFUND	3	1.03E-04	1.02E-04	1.01E-04	2.19E-04	1.33E-04	8.41E-05	1.92E-02	1.37E-02	8.64E-03	2.52E-04	1.27E-04	4.90E-05
Hexachlorocyclohexane (HCH)	Y	Y				B2															
a-HCH			-	6.30E+00	-	B2	3.00E-04	SFUND	2	1.28E-04	1.19E-04	1.10E-04	1.83E-04	1.63E-04	1.19E-04	3.37E-02	1.84E-02	4.09E-03	8.93E-04	1.82E-04	6.70E-05
g-HCH (Lindane)	Y	N	-	1.33E+00	-	B2/C	3.00E-04	SFUND	2	1.18E-04	1.17E-04	1.16E-04	1.81E-04	1.22E-04	2.68E-05	3.18E-03	1.78E-03	8.30E-04	1.70E-04	1.56E-04	1.41E-04
PAH (m)	Y	Y	-	1.15E+01	6.11E+00	-	-	-	-	4.51E-02	4.35E-02	4.19E-02	7.43E-02	5.19E-02	3.58E-02	4.78E+00	3.37E+00	2.26E+00	2.61E-04	2.45E-04	2.26E-04
PCBs (n)	N	N	2.0	2.60E+00	-	B2	1.00E-04	APP.C	-	1.53E-01	1.51E-01	1.49E-01	3.82E-01	2.37E-01	1.43E-01	6.18E+01	4.39E+01	2.28E+01	7.43E-01	2.73E-01	6.12E-02
Aroclor 1242	N	Y	-	-	-	-	-	-	-	1.99E-02	1.44E-02	8.94E-03	3.68E-03	1.97E-03	1.69E-03	2.27E+00	1.50E+00	6.53E-01	1.77E-02	3.61E-03	1.39E+03
Aroclor 1254	N	N	-	-	-	-	-	-	-	1.40E-01	1.36E-01	1.33E-01	3.80E-01	2.37E-01	1.43E-01	5.96E+01	4.24E+01	2.22E+01	7.26E-01	2.71E-01	6.12E-02

(a) = Data are presented as ug/g wet weight, converted from Gardner and Pruell, 1987. Means were calculated using detection limits for undetected observations. For data results using zero for undetected observations, see Table B-10 in Appendix B.

(b) = IRIS stands for Integrated Risk Information System. Health risk assessment information on chemicals is included in IRIS only after a comprehensive review of chronic toxicity data is performed by USEPA scientists from several EPA Program Offices. Y= data available. N= data unavailable.

(c) = HEA stands for Health Effects Assessment. These documents present a brief, quantitatively oriented scientific summary of health effects data from many toxic compounds. Y= data available. N= data unavailable.

(d) = FDA stands for Food and Drug Administration. These limit values (ppm) correspond to several priority pollutants and pesticides in fish and fishery products in the United States (Tetra Tech Inc. 1986).

(e) = CPF stands for Carcinogenic Potency Factor. These factors are used to estimate the potential carcinogenic risk. They represent the 95% confidence limit of the dose response curve.

(f) = EPA Weight of Evidence is the rating which qualifies the level of evidence that supports designating a chemical a human carcinogen. See Table A-1, Appendix A. Weight of evidence classifications are made without regard to the route of exposure. Route specific information is considered when determining the EPA

(g) = RfD stands for Reference Dose or Acceptable Intake-Chronic (AIC) level which is the long term acceptable intake level for non-carcinogenic effects. Values were obtained from the Superfund Public Health Evaluation Manual (USEPA, 1986b) and USEPA Integrated Risk Information System chemical file for the specific metal or compound. (USEPA, 1986a).

(h) = Reference sources for the RfD values are the following: IRIS- USEPA, 1987d.; SFUND- USEPA, 1986b.; APP.C- USEPA, 1988a.

(i) = Toxicity Ratings are unitless integers ranging from 1 to 10 and corresponding to various severity levels of effects. Refer to Table A-2 in Appendix A.

(j) = Lowest Concentration Detected.

(k) = Data correspond to inorganic compound values.

(l) = Same CPF value used for both chlordane isomer.

(m) = Total Polycyclic Aromatic Hydrocarbons.

(n) = Total Polychlorinated Biphenyls (PCBs). See Appendix C for documentation of the derivation of the CPF for PCBs.

conventionally referred to implies a greater degree of accuracy than is currently available given the uncertainty associated with calculating CPF values. However, the CPF is generally considered the plausible upper bound value. The CPF is the generally accepted approach to convert estimated intake levels directly to estimated plausible upper bound incremental risk as described in Section V of this report. CPFs are presented for those chemicals considered by the EPA to be human carcinogens or probable human carcinogens (USEPA. 1986a-b; 1986c; 1987d-h). The EPA weight of evidence (Table 1) refers to evidence of carcinogenicity, with Group A signifying a known human carcinogen and Group D signifying no classification. Group B signifies probable human carcinogenicity based on animal studies, while Group C signifies possible human carcinogenicity. The weight of evidence classifications are described in more detail in Appendix A. In general, the weight of evidence is classified by EPA without regard to route of exposure, and route specific information is included in the CPF determination. In Table 1, for metals, different classifications have been made for inhalation and oral routes. A classification of Group D was input for the oral route where no evidence of carcinogenicity by the oral route of exposure is available.

The values for reference dose (RfD) are generated by the EPA based on the assumption that threshold levels exist for noncarcinogenic health effects (USEPA. 1986c). The RfD is considered to be the level unlikely to cause significant adverse

health effects associated with a threshold mechanism of action in humans exposed for a lifetime (USEPA. 1986a-b; 1986c; 1987d-h). The RfD is used for comparison with calculated intake levels as discussed in Section V. The EPA toxicity rating (Table 1) is associated with noncarcinogenic health effects where 1 is associated with small changes due to contaminant exposures and 10 is associated with death or pronounced life shortening and teratogenic effects. The basis for the toxicity ratings is presented in more detail in Appendix A.

Indicator Chemicals

The majority of those contaminants recently analyzed by EPA and found in Quincy Bay sediments and seafood (Gardner and Pruell. 1987) have been included as indicator chemicals in the public health evaluation. In some cases the contaminants are grouped based on availability of toxicological information, and on similarity of chemical properties and toxicological effects. The subset of indicator metals and compounds considered in the public health evaluation (Table 2) are shown with the CPFs, RfDs and critical effects for each. Toxicity profiles for the organic compounds and metals found in Appendix A and excerpted here focus on chronic exposure by ingestion. While some of the metals are considered possible or probable human carcinogens, where there is no evidence of carcinogenicity by ingestion, no CPF or weight of evidence values are provided in Table 2. The RfD values for an oral exposure to metals as well as the critical effect (Table 2)

TABLE 2. TOXICITY VALUES FOR INDICATOR CHEMICALS

Metal/Compound	Oral Carcinogenic Potency Factor(CPF) $(mg/kg/day)^{-1}$	Weight of Evidence[e]	Oral Ref. Dose (RfD) (mg/kg/day)	Critical Effect[a]
Cadmium	-	-	$2.90 * 10^{-4}$	Renal dysfunction[5]
Chromium	-	-	$5.00 * 10^{-3}$	NOEL, renal dysfunction[1][2
Copper	-	-	$3.70 * 10^{-2}$	GI symptoms [7]
Lead	-	-	$1.40 * 10^{-3}$	-, renal effects[4]
Mercury	-	-	$2.00 * 10^{-3}$	-, renal effects[5]
Chlordane	1.3	B2	$5.00 * 10^{-5}$	Liver necrosis[1
Dichlorodiphenyl trichloroethane (DDT)	0.34	B2	$5.00 * 10^{-4}$	Liver lesions[1]
Hexachlorobenzene (HCB)	1.69	B2	$8.0 * 10^{-4}$	-, liver changes, teratogenic effects [3][9]
Hexachlorocyclohexane (HCH)	6.3[b] 1.33[c]	B2 B2/C	$3.0 * 10^{-4}$	Liver hypertrophy[6]
Polycyclic aromatic hydrocarbons (PAHs)	11.53	(d)	NA	-
Polychlorinated biphenyls (PCBs)	2.6	B2	$1.0 * 10^{-4}$	Reduced size of offspring[8]

(a) The critical effect is the effect seen in the studies from which th RfD is developed. The RfD is set at a level where the critica effect is unlikely to occur. Where the study used to set the Rf indicates a NOEL (no observable effect level), the most common observed effect is also noted. A "-" indicates the information i the specific study defining the RfD is not included in this report and the critical effects reported from other studies are included.
(b) Alpha HCH.
(c) Gamma HCH.
(d) See Table 3 for weight of evidence for PAHs
(e) For explanation of weight of evidence see Appendix A Table A1.

References:

1. USEPA. 1986a, and 1987e;g.
2. USEPA. 1984c.
3. USEPA. 1984f.
4. USEPA. 1984g.
5. USEPA. 1987a.
6. USEPA. 1987b.
7. USEPA. 1984d.
8. USPHS. 1987 for RFD. USEPA. 1988a for CPF.
9. USEPA. 1987c.

document the health effects seen at the lowest exposure concentrations. The RfD is set by the EPA at a level where the critical health effect is judged unlikely to occur.

Chlordane is considered here as total chlordane without distinguishing between the alpha and gamma isomers measured by Gardner and Pruell (1987). Many of the toxicity studies in the referenced database were performed utilizing a technical grade chlordane which includes both isomers. RfDs and CPFs were only available for total chlordane (Table 2). The weight of evidence, B2, indicates that the evidence of carcinogenicity in humans is inadequate to consider the compound a known human carcinogen, however, due to sufficient evidence of carcinogenicity in animals, chlordane is considered a probable human carcinogen. Toxicity profiles for chlordane and other organic chemicals are provided in Appendix A.

Technical DDT (dichlorodiphenyltrichloroethane) is generally a mixture of p,p-DDT, o, p-DDT, p,p-DDD, and traces of other materials. Metabolites of DDT include p,p-DDE and o,p-DDD. DDT isomers and metabolites are often found together and have similar properties, therefore, they have been considered together as a chemical class (Clement. 1985). The analytical data for p,p-DDD, p,p-DDE, and p,p-DDT are presented separately in the public health evaluation, however, the same RfD and CPF values provided by the EPA, for DDT as a class, are used for all three compounds.

Teratogenic and carcinogenic effects have been documented for exposure to hexachlorobenzene (HCB). Both CPF and RfD values

are available for HCB. Liver effects such as hepatomegaly have been noted in the literature. CPF values for hexachlorocyclohexane (HCH) are available for both the alpha and gamma isomers and are both used in this public health evaluation. The RfD developed for the gamma isomer (lindane) was used for both isomers. Lindane is considered the most acutely toxic isomer, while no RfD is available for the alpha isomer.

There is one CPF value available for polycyclic aromatic hydrocarbons (PAHs) based on the carcinogenicity of benzo(a)pyrene (Table 2). Not all PAHs are known carcinogens. PAHs evaluated in Quincy Bay seafood tissues (Table 3), have varying amounts of evidence that they are carcinogenic in animals. The individual PAHs have been grouped for evaluation in the public health assessment as total PAHs. Evaluating all PAHs as carcinogens is a standard conservative approach, which will tend to overestimate increased lifetime cancer risk. No RfD for PAHs was found during the literature search.

Polychlorinated biphenyl (PCBs) contamination was also evaluated by grouping the data and evaluating total PCBs. EPA determined that there is positive evidence for carcinogenicity in animals for Aroclor 1254, Aroclor 1260, and some other PCBs. Because any PCB mixture can contain appreciable amounts of carcinogenic PCBs and because of the variability of PCB mixtures, EPA has recommended that all commercial PCB mixtures be considered to have a similar carcinogenic potential and classified all PCB mixtures as Group B2 - Probable Human

TABLE 3. EVIDENCE OF CARCINOGENICITY IN ANIMALS

Polycyclic-aromatic Hydrocarbons	Sufficient Evidence	Limited Evidence	Inadequate Evidence	No Evidence
Fluorene			X	
Phenanthrene			X	
Anthracene				X
Fluoranthene				X
Pyrene				X
Benzo(a)anthracene	X			
Chrysene		X		
Benzofluoranthenes	X			
Benzo(e)pyrene			X	
Benzo(a)pyrene	X			
Perylene			X	
Indeno(1,2,3-cd)pyrene	X			
Benzo(ghi)perylene			X	
Dibenz(a,h)anthracene	X			
Corene			X	

Source: Clement. 1985.

Carcinogens, based on sufficient evidence of carcinogenicity in animal studies (USPHS. 1987). A CPF of 4.34 $(mg/kg/day)^{-1}$ has been used in risk assessments in the recent past as the generally accepted value. A new CPF of 7.7 $(mg/kg/day)^{-1}$ based on carcinogenicity data for Aroclor 1260 has been proposed in a draft report (USPHS. 1987). Work by the US EPA Exposure Assessment Group indicates that based on the thirteen congeners

measured in Quincy Bay seafood the mixture more closely resembles Aroclor 1254 than 1260. (USEPA. 1988b) An upper bound CPF of 2.6 $(mg/kg/day)^{-1}$ was developed by the EPA Carcinogen Assessment Group for Aroclor 1254, and is used in this evaluation. Appendix C documents the development of this CPF. A sensitivity analysis was performed as part of the results and conclusions in Section VI to determine the effect on plausible upper bound increased lifetime cancer risk given the use of different CPF values for PCBs. The RfD for non-cancer risks for PCBs proposed in the 1987 USPHS draft document has been used in this public health evaluation at the suggestion of USEPA-OHEA.

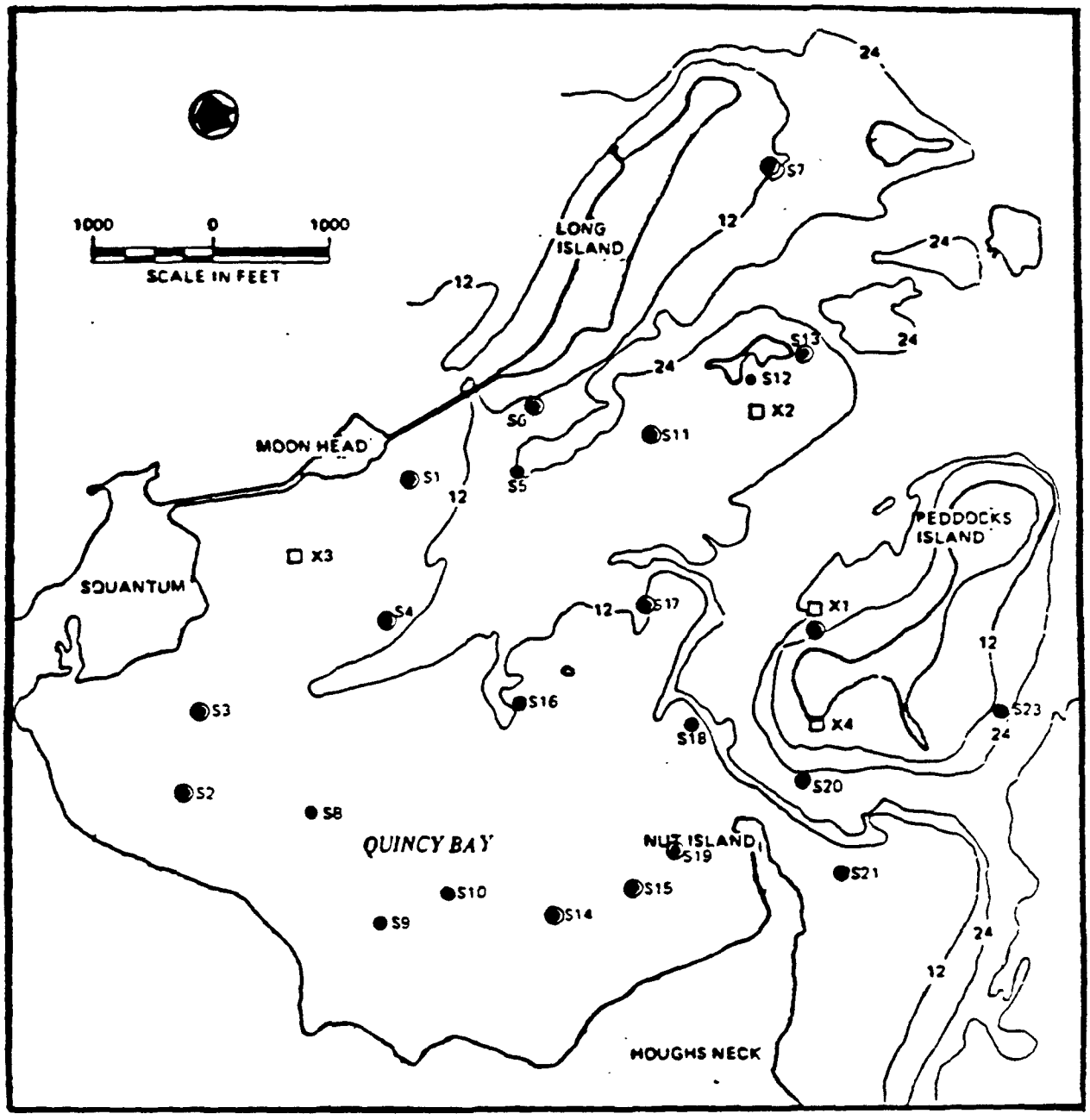
IV. Exposure Assessment

Portions of the data developed by Gardner and Pruell (1987) were used to define exposure estimates in risk assessment scenarios related to the extent of contamination of sediments and selected biota in Quincy Bay. This section discusses the selection of the chemical data, consumption data and population characteristics required for the exposure portion of the Public Health Assessment below.

A. Species Selection and Characteristics

Based on the guidance provided in the Report 99-731 of the 99th Congress, sampling for this study was conducted in Quincy Bay during early spring and summer, 1987, for sediments, winter flounder (*Pseudopleuronectes americanus*), soft shelled clams (*Mya arenaria*), and the American lobster (*Homarus americanus*). Additionally, 400 oysters (*Crassostrea virginica*) were suspended at four locations: three in Quincy Bay and one at the Graves in Massachusetts Bay.

Surface sediments were collected at 22 locations in Quincy Bay and core samples were collected at four locations (see Figure 1). Inorganic contaminant levels were measured in all samples, and selected organics were measured in the core samples and 14 of the surface sediment samples. Sediment sampling and analyses methodologies are discussed in detail by Gardner and Pruell (1987). Levels of contaminants at many locations throughout the Bay were elevated beyond the levels generally



LEGEND

- SURFACE SEDIMENT SAMPLING LOCATIONS INORGANIC ANALYSIS ONLY
- SURFACE SEDIMENT SAMPLING LOCATIONS INORGANIC AND ORGANIC CHEMICAL ANALYSIS
- CORE SEDIMENT SAMPLING LOCATION INORGANIC AND ORGANIC CHEMICAL ANALYSIS

FIGURE 1. QUINCY BAY SAMPLING AREA. LOCATION OF SEDIMENT SAMPLING SITES.

SOURCE: US EPA. 1987.

reported by others for Boston Harbor (see the Task I report for details). Some organics (e.g. PCBs, DDE, PAHs) and some inorganics were found at the highest levels offshore of Moon Head and Long Island in the vicinity of sewer system discharges, and around Peddocks Island and Nut Island near the discharges from the Nut Island wastewater treatment facility. While contaminants from these sediments may be released to surrounding water and may be linked to contaminant levels in organisms, there is no generally accepted method for directly quantifying the importance of marine sediment contaminant levels to human health risks from ingestion of contaminated seafood. Thus, sediment contamination is not directly included in the computations of the exposure assessment. Possible implications of the measured sediment contamination levels are discussed further in the Task V report.

The biological sampling and analyses by Gardner and Pruell (1987) included collection and evaluation of histopathological condition and chemical contamination in three species of indigenous marine organisms of high commercial and/or recreational value in Quincy Bay: Winter flounder, American lobster, and soft-shelled clam. Oysters brought in from Cotuit populations were also placed in the Bay to allow in situ evaluations of contaminant uptake after a 40 day exposure to Quincy Bay conditions. Since this species is not commercially or recreationally harvested from Quincy Bay, these results were not included in the public health assessment of exposure to the Quincy Bay fishery.

Gardner and Pruell (1987) indicate that histopathological evaluations provide strong evidence that Quincy Bay populations of Winter flounder and soft-shelled clams are in poor health (See Task V Report). At present, theoretical or analytical methods for correlating the histopathological results with any potential effects in humans do not exist. The exposure assessment was thus limited to evaluation of the ingestion by humans of the residues of chemicals contained in lobster, flounder, and soft-shelled clams from Quincy Bay.

One hundred Winter flounder were collected by otter trawls from four transect locations in the Bay (Figure 2). An additional transect from Moon Head to the eastern end of Long Island was eliminated due to lack of fishing success. Lobster collections occurred at nine locations in the Quincy Bay study area (Figure 3). Collections of specimens later analyzed were made by traps. Seven sites were chosen for soft-shelled clam collection, but the organisms were present only at Moonhead and Moon Islands (Figure 4).

The three species chosen for chemical contamination evaluation from Quincy Bay are the more commercially/ recreationally significant species harvested from the Bay. In addition, each was determined to be sufficiently narrow-ranging to be considered indigenous to the area. Soft-shelled clams are essentially sedentary as adults. Winter flounder do move. However, an extensive tagging study conducted in the early 1960's (Howe & Coates. 1975) suggested that winter flounder in the

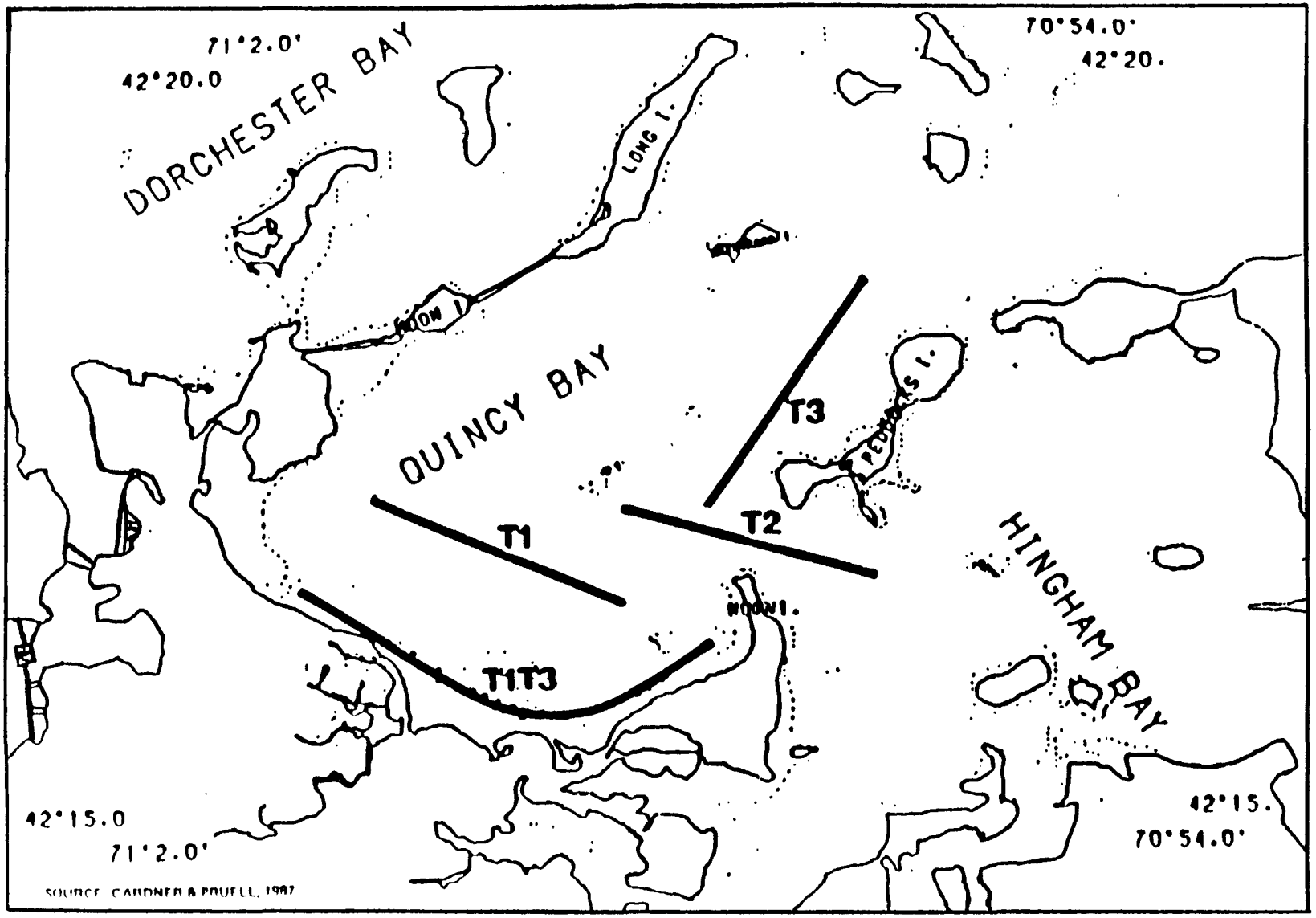


FIGURE 2. LOCATIONS OF OTTER TRAWL FISHING TRANSECTS FOR WINTER FLOUNDER.

SOURCE: US EPA, 1987.

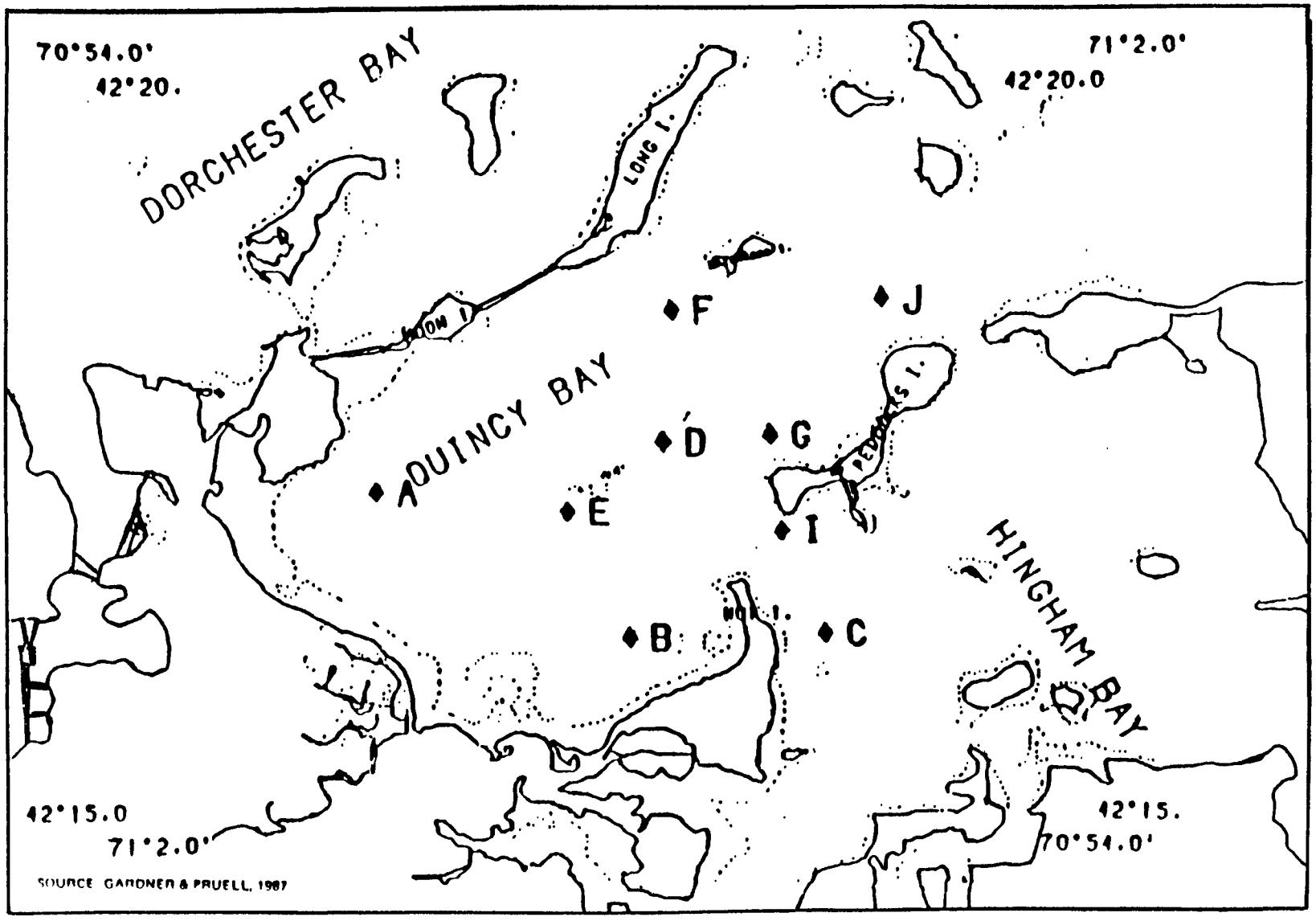


FIGURE 3. LOCATIONS OF LOBSTER COLLECTIONS.

SOURCE: US EPA. 1987.

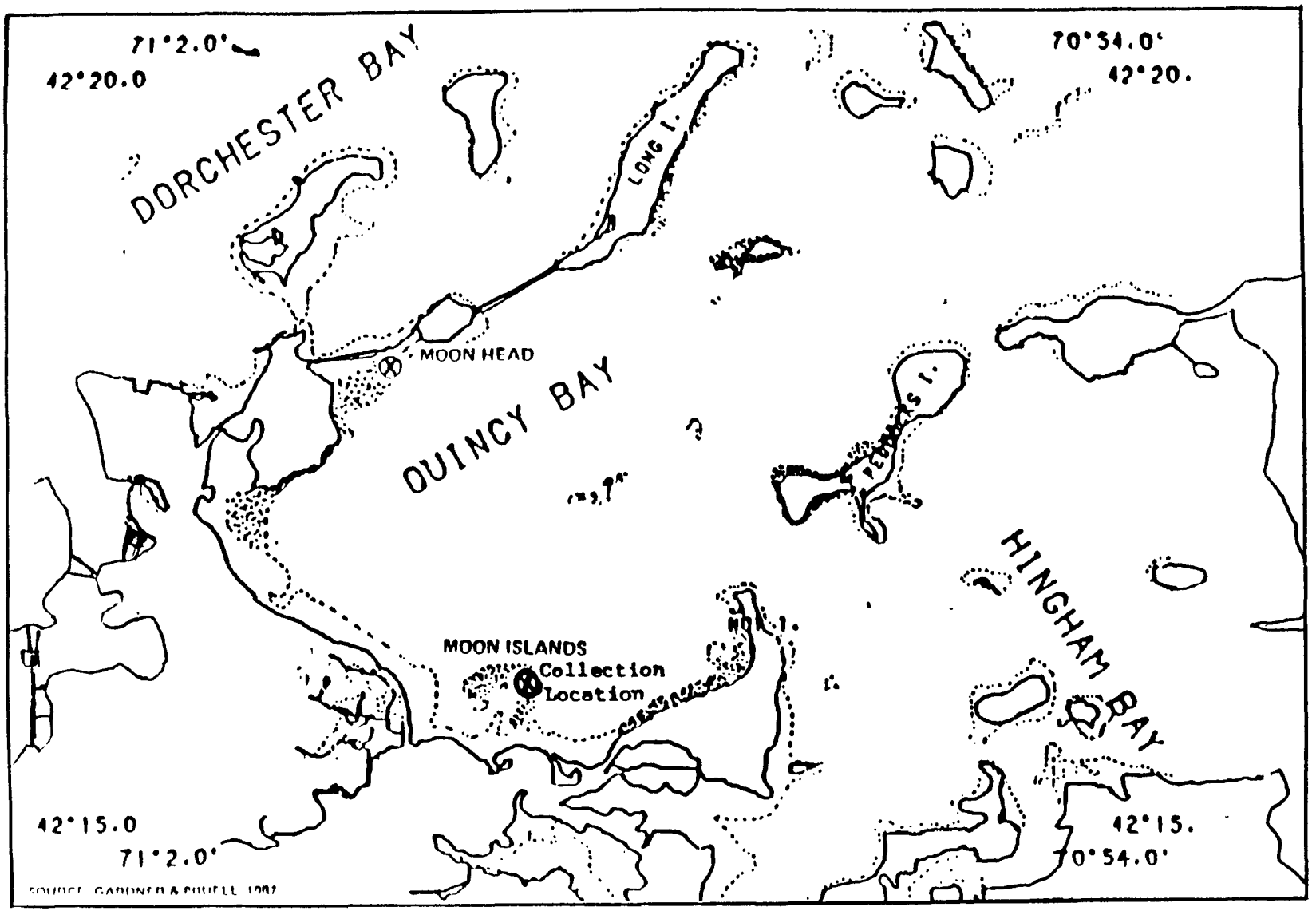


FIGURE 4. FIELD SAMPLING LOCATIONS FOR SOFT - SHELLED CLAMS.

SOURCE: US EPA. 1987.

Boston Harbor area showed only limited movement from inshore release areas. Specifically, sexually mature fish moved in to shallow water to spawn during winter and spring. Many remained near spawning areas. Some migrated to deeper waters near the harbor mouth and farther in warmer months. Howe and Coates (1975) provide sufficient evidence of very high local recapture rates to allow the simplifying assumption that the flounder caught by trawl in Quincy Bay during May, 1987 and those caught and consumed by fishermen had been in the study area for at least several months preceding their capture.

A similar simplifying assumption was made concerning lobster caught in Quincy Bay. Lobster fishermen trap near shore in the spring when mature lobsters are in shallow water to spawn. They follow lobster movement to deeper water through summer and fall months (Jones. 1987). Fishermen believe this suggests movement of lobster populations that is temperature related. There is additional evidence, according to the State Division of Marine Fisheries (Estrella. 1987) that such movement may occur in older lobsters, with juvenile populations being less migratory. The DMF also indicates that there is evidence to suggest that up to 95 percent of the legal size inshore population is cropped by fishing pressure. At legal size, a number of captured lobsters may not be sexually mature (Estrella. 1987). In conclusion, it is possible but not verifiable that many of the captured Quincy Bay lobsters in the fishery and for the sampling and analysis in this study may have

spent long enough to have become contaminated (i.e. at least several months) in the Quincy Bay environment.

B. Contaminant Characterization

As may be seen from the narrow ranges of inorganic and organic contaminant levels found in soft-shelled clams (Table 1), residues from both the Moon Head and Moon Island locations were very similar. Sediment samples were not collected near enough to the clam collection locations to provide a basis for comparison.

The differences in inorganic and organic levels in lobster tissues and the lobster hepatopancreas from different sampling locations were not large (around 2X) and did not follow any clear geographic pattern. The sample of lobsters was small and it is likely that movement was sufficient to preclude definitive conclusions concerning the relationship between lobster and sediment contamination in this study. The significant difference between lobster muscle tissue (tail) and hepatopancreas concentrations, however, requires special consideration in this assessment. Specifically, consumers of lobster "tomalley" (hepatopancreas) may have a much higher exposure to the studied contaminants than would those who only consume lobster meat.

Similarly, while there is some variability among contaminant residues in individual flounder samples, geographic patterns of tissue contamination that might be associated with differing sediment contaminant levels within the Bay can not be established in view of the lengths of the collection trawl

transects (covering both more and less contaminated sediments) and the opportunities for either or both the fish and sediments to move. The Task V Summary Report discusses potential sediment/organism contaminant relationships on a broader basis, including discussion of Quincy Bay data versus data from other locations.

Given the above results, potential consumer exposure was assumed to be best represented for the analysis by the maximum and the mean concentrations of contaminants found in each of the three types of organisms analyzed (Table 1).

C. Estimates of Seafood Consumption

1. Commercial Catch

Seafood consumption estimates for risk assessment include assumptions about the amount of seafood consumed as well as its source. EPA guidance (PTI. 1987) recommends against attempting to quantify the inherently variable commercial catch to consumer distribution patterns for a risk assessment. As discussed below, the distribution pattern for commercially harvested seafood from Quincy Bay is typically irregular, and supports the guidance.

The scope for this study limits the number of species considered in the consumption estimates to three. On the basis of available harvest data and interviews with fisheries industry participants, it is believed that clams, flounder and lobster do in fact constitute the great majority of the consumed significant catch from Quincy Bay. Other species seasonally harvested from

the Bay in minor but measurable amounts include bluefish, eel and smelt, all of which are migrant visitors.

Soft-shelled clams are in theory represented by only a commercial fishery in Quincy Bay, as only "Master Diggers" are legally permitted to harvest clams. The clams must then go through the state's shellfish depuration plant (along with clams from other areas in the state) prior to distribution. Reportedly, local clam harvest makes up about 15 percent of the local demand in metropolitan Boston (Kennedy. 1987). The remainder of Boston's demand for soft-shelled clams is filled with imports from areas such as Maryland. It would likely be impossible to accurately trace Quincy Bay clams through the local distribution system to ultimate consumers as destinations change daily and sources are not well tracked (Connerty. 1987). Additionally, individuals can hold "bait licenses" for clams. It is believed that some (perhaps many) of these individuals and others who may or may not hold licenses are clamming for personal consumption (Ayers. 1987).

Over 12 million pounds of lobster were taken from the coastal waters of Massachusetts in 1986 (Hoopes. 1986). The coastal lobster permit reporting area that includes Boston Harbor and Quincy Bay has been the most productive according to reports for the last three years (Hoopes. 1985; Hoopes. 1986; Nash. 1984). This reporting system tracks the home port of vessels and general reporting of areas harvested, but does not provide overall harvest from an area that corresponds to the geographic

boundaries of Quincy Bay. Lobster fishermen sell to a number of different distributors. As with clams, it would be practically impossible to track commercial catch from one area in sufficient detail to generate a commercial Quincy Bay lobster distribution to consumption profile over a long period of time.

There is no commercial winter flounder fishery in Quincy Bay, although some flounder taken in the Bay are sold by recreational fishermen (Ayers. 1987; Jones. 1987).

2. Recreational Catch

The most recent EPA guidelines for seafood consumption risk assessment (PTI. 1987) suggest that quantitative considerations of recreational harvest and distribution to consumption patterns may be appropriate for risk assessment, depending on the quality of available data. These were investigated for the three target species in this assessment.

The recreational flounder fishery in Quincy Bay has been renowned for many years, with as many as 17,000 estimated annual boat trips in the mid 1960's (Jerome. 1966). The fishery is reportedly in decline due to publicity concerning water quality (Childs. 1987). The state plans an updated recreational survey but such numbers are not available at this time. Several marinas rent boats in the area. On a summer day with good weather, several independent estimates by local fishermen suggest that up to 1,000 boats may be on the bay. The number of these boats engaged in fishing is not known. A large number of recreational

flounder fishermen are from out-of-state. Many evidently come a number of weekends every year, returning home with large amounts (e.g. 50 pounds or more) of flounder. It is reported that some of these individuals sell some of their catch dockside and/or out-of-state. However, it is known that some also keep considerable amounts for regular personal consumption throughout the year. Local fishermen can and do also keep enough flounder for regular consumption, and such has been assumed here and tabulated later in this chapter. Some local fishermen also fish for striped bass and bluefish in the summer, and/or smelt in the winter along with flounder, and fish areas outside Quincy Bay. The data available at this time limit the consideration of finfish consumption risks in this study to flounder.

Approximately 250 Quincy residents hold 10 pot (recreational) licenses for lobster (MDMF. 1987. Unpublished data). It is assumed that many of these individuals likely fish Quincy Bay or its environs at least some of the time. In addition, an unquantified number of license holders from other nearby areas likely harvest the bay as well. (Reporting of harvest location is not required for these license holders). Local commercial lobstermen would anticipate five or six "keepers" per set per 10 pot string of baited pots (Jones. 1987). Using the unpublished 1987 DMF License data (MDMF. 1987), the average catch per license holder was over 38 lobsters/year. These data provide a basis for judging the

reasonableness of lobster consumption estimates from recreational catch as tabulated below.

As noted above, soft-shelled clams are only legally available for consumption by purchase following depuration. However, some individuals are believed to consume Quincy Bay clams that are collected illegally or with a bait license. This is a form of illegal activity for which no records are available although some estimates have been made below.

3. Consumption Estimates

The above assessment of the fishery makes it clear that risk should be assessed for several different exposure assumptions for the Quincy Bay fishery. The data also document that while it is possible to generate a range of consumption profiles, the fishery data are not adequate for definitively assigning the population sizes that fit each profile.

Several levels of Quincy Bay seafood consumption were developed for the risk assessment. These numbers were derived using published surveys of a range of seafood consumption, along with the approach recommended in the risk assessment guidance manual (PTI. 1987).

According to PTI (1987) the standard value for maximum consumption estimates in risk assessment is based on the approximately 0.1 percent of the U.S. population which reportedly consumes 165 grams/day of seafood. This is a slightly more than 1/3 lb. serving of fish per day on average. On the basis of

local interviews, it has been assumed that there is a small percentage of the "local" population of Quincy area residents that consume this much Quincy Bay fish or seafood, although the actual population size was not estimated. In the absence of more definitive, site-specific data, the consumer of this amount of seafood, in various combinations (see below) is considered the "Maximally Exposed Individual", (MEI), for this study.

Regional data for seafood consumption by species were available for New England, so that consumption levels could be estimated for "typical" consumers without relying on the Guidance Manual default value.

Several national consumer surveys place New England residents among the highest consumers of fish and shellfish. The consumption estimates for "typical Quincy area resident" were based on the survey data for New England consumers reviewed in a study for the National Marine Fisheries Service (Penn State. 1985). Data from three surveys were cited in summaries of regional consumption patterns. One represented a year (1969-70) of survey results. The other two (1973-74; 1977-78) represented more recent surveys of greater numbers of individuals, but were conducted over shorter time periods of 3 days to one month. Each survey represented a different bias. The differences reported for average yearly flounder consumption in New England were 0.618 lbs per capita to 1.005 lbs per capita, and for average yearly lobster consumption were 0.601 lbs per capita to 1.895 lbs per capita. The choice was made to rely more heavily on the year

long (1969-70) survey and to modify the averages, slightly up in the case of flounder and slightly down in the case of lobster, based on the data from the later surveys. The differences among the averages in these surveys is small. From these data, then, it is assumed that the typical local consumers with access to the recreational fish harvest from Quincy Bay could consume a long-term average of 1 gram/day (0.8 lbs/year) of Quincy Bay flounder and 2.1 grams/day (1.71 lbs/year) of lobster.

Both of these figures appeared reasonable considering the apparent recreational lobster and flounder harvest levels and the exposure that could be associated with commercially distributed catch. They were used to provide a departure point for comparison with the "maximally exposed individual". Again the number of individuals in the consuming population was not estimated, but there is reason to believe that it could be relatively large, given the catch volumes.

These above estimates resulted in four separate consumption profiles, which are discussed below and summarized in Table 4.

TABLE 4. SUMMARY OF ASSUMED LIFETIME CONSUMPTION LEVELS

	Maximally Exposed Individual		Typical Local Consumer	
	Mixed Diet	Flounder Only	Mixed Diet	Mixed Diet
Quincy Bay Clams	16 g/day	--	--	--
Quincy Bay Flounder	113 g/day	165 g/day	1 g/day	1 g/day
Quincy Bay Lobster[a]				
Tissue	30 g/day	--	2.1 g/day	1.7 g/day
Tomalley	6 g/day	--	--	0.4 g/day

(a) Breakdown of tomalley versus other edible lobster tissue based on MDMF, unpublished data.

1a. Maximally Exposed Individual, Mixed Seafood Diet from Quincy Bay.

This represents a potential group of local residents (likely small) who consume an average of 165 g/day of locally caught seafood. This group typically would include individuals who, for economic reasons catch a large amount of seafood for home consumption. A local individual could catch and consume this amount of flounder, Quincy Bay clams (illegally dug) and Quincy Bay lobster as a recreational fisherman in the normal course of the typical fishing seasons. The whole lobster, including tomalley, is assumed to be eaten in this diet. It is assumed to be available and consumed within the practical limits of reported catch rates (see above) and season imposed by a 10-pot license. The distribution among the three seafood

categories reflect the understanding gained concerning potential recreational catch levels for all species. Because lobster tissue residues for the study chemicals were higher than those in the other two species, the amount of lobster assigned to this mixed diet was estimated first, based on assumptions of availability and catch success within the practical limits of the reported catch rates and season imposed by a 10 pot license (see above). Next, the assignment of clam consumption levels was based on some discussion of maximum consumption of this species with local fishermen and health officials. Finally, the remainder of the 165 g/day total was assumed to consist of flounder, on the basis of interviews with local residents indicating that such a level of consumption likely took place.

1b. Maximally Exposed Individual, flounder only diet from Quincy Bay.

This represents a group of individuals (likely small) who consume an average of the 99.9 percentile value of 165 g/day of seafood, in this case, of Quincy Bay flounder. This could be represented by either local or out-of-state flounder fishermen who keep large enough amounts of caught flounder for year-round home consumption.

2a. Typical Local Consumer

This group represents those metropolitan Boston area residents who consume the regional averages of 1 g/day of Quincy Bay flounder and 2.1 grams/day of lobster meat, in this case assuming that both came consistently from Quincy Bay. It is first assumed that these consumers eat lobster without tomalley, as many individuals do not consume this organ. This typical local consumer is assumed to have no access to the small number of Quincy Bay clams that may be available in the area.

2b. Typical Local Consumer

This group would be the same as (2a) above, except these individuals do consume the lobster tomalley as well.

Clearly any of the above groups could consume fish from other sources, or other species from the bay. A more detailed survey of recreational fishing and local consumption patterns conducted over a full year would allow some estimate of the size of each of the populations affected and could allow a better sensitivity analysis based on the more typical consumption patterns. In the absence of such data, the figures in Section VI were developed to illustrate sensitivity of some of the risk estimates to the assumptions about the amounts of seafood consumed.

V. Public Health Evaluation

To determine whether adverse health effects are likely from exposure to contaminated Quincy Bay Seafood, exposure scenarios for maximally exposed individuals and for typical Quincy area residents, who ingest average amounts of seafood, have been developed. The method used to calculate dose, hazard index, and the plausible upper limit of excess cancer risk follows guidance provided by PTI (1987).

A. Dose Calculation

The human dose of a specific chemical from ingestion of Quincy Bay seafood is calculated as:

$$\frac{(Cij)\ (CRj)}{BW} = Dij$$

Where,

Cij = Concentration of contaminant i in species j (units: µg/gram tissue, wet weight)

CRj = Consumption rate for species j (units: grams seafood/day)

BW = Average American body weight (units: kilograms)

Dij = dose of contaminant (i) from ingestion of species j (units: µg/kg/day)

The concentrations of chemicals in seafood were obtained from the EPA study of Quincy Bay chemistry results (Gardner and Pruell. 1987). Maximum and mean contaminant levels detected were used to calculate dose. Following the EPA Guidance,

(PTI. 1987) the mean concentrations were calculated assuming the detection limit for undetected observations, and recalculated assuming a value of zero where the concentration was below the limit of detection. The results where zeros were used to calculate means differed very little from those where the detection limits were used, and are presented in Appendix B (Tables B-5 through B-8, and B-10).

The consumption rates used to calculate dose are presented in Section IV. Different consumption rates were assumed for the maximally exposed and average exposed individuals. The dose calculations were made utilizing the standard assumptions for an integrated risk analysis, including exposure over an entire 70-year lifetime and a 70 kilogram body weight for an average American adult male. In addition, it was assumed in accordance with EPA Guidance (PTI. 1987) that the ingested dose is equal to the absorbed contaminant dose, and that cooking has no effect on the contaminants.

B. Risk Characterization

To calculate the plausible upper bound to excess lifetime risk of cancer, the contaminant-specific dose is multiplied by the carcinogenic potency factor (CPF) for oral exposures. This equation is considered valid only at low risk levels where it is assumed that the slope of the dose response curve is linear and equal to the CPF. To indicate the level of non-carcinogenic risk, the ratio of calculated contaminant-specific dose to the

reference dose (RfD) is presented. In addition, in some cases, a sum of the hazard ratios for similarly acting chemicals is calculated. The CPF and RfD values used in this assessment are values established by the EPA (Table 2) and described in greater detail in Section III.

The plausible upperbound excess lifetime risk of cancer associated with the estimated exposure is expressed as a fraction (e.g. $1 * 10^{-6}$ or 1 in 1,000,000). It represents the estimated incrementally increased risk in an individual's lifetime of developing cancer attributable to the exposure. In this assessment, incremental excess lifetime cancer risks from the various seafood contaminants were assumed to be numerically additive in accordance with the Guidance Manual (PTI. 1987). Chemical-specific cancer risks were thus used to calculate total plausible, upperbound excess lifetime cancer risks adding across species and species-specific cancer risks were totalled across chemicals. Taken together, these provided the basis for estimating total plausible, upperbound excess lifetime cancer risks from exposure to Quincy Bay seafood.

The hazard ratio is a ratio of calculated dose to reference dose. Hazard ratios are summed across similarly acting chemicals. Since the reference dose is defined as the level unlikely to cause significant adverse health effects associated with a threshold mechanism of action in humans exposed for a lifetime, a sum of hazard ratios of less than one indicates that overall the calculated dose is less than the RfD, and adverse

health effects from this exposure are not likely. A sum of hazard ratios of greater than one indicates that adverse health effects may occur from the exposure, however, does not by itself indicate that adverse effects will occur as there are margins of safety and/or uncertainty in the derivation of the RfDs upon which the ratios are based. Margins of safety or safety factors are generally multiples of 10, each representing a specific area of uncertainty in the available data. Three types of uncertainty to which a factor of 10 are often applied are: (1) expected differences in responsiveness between humans and animals in prolonged exposure studies, (2) the variability among individuals within the human population, (3) incomplete data (USEPA. 1986a).

Following the Guidance Manual (PTI. 1987) and generally accepted practice, chemical-specific hazard ratios were assumed additive only where the contaminants act on the same target organ. Species-specific hazard ratios are additive for the same contaminant, so a hazard ratio for a given chemical in flounder can be added to a hazard ratio for the same chemical in lobster to determine the total hazard ratio for one chemical from ingestion of both flounder and lobster.

C. Maximally Exposed Individual

Exposure scenarios were developed to evaluate the risk from eating Quincy Bay seafood by two types of maximally exposed individuals (MEI). The first is a person who consumes an average of 165 grams of Quincy Bay seafood each day which consists of

flounder, clams, and lobster. This MEI would eat both the lobster tissue and the hepatopancreas or tomalley. Calculations of dose, hazard ratio, and plausible upperbound increased lifetime cancer risk for each of the different seafoods consumed are presented in Appendix B, Tables B-1, B-2, B-3, and B-4. A summary of hazard ratio and plausible upper bound increased cancer risk values (Table 5) documents that the only individual species- and chemical-specific hazard ratio that exceeds one is the hazard ratio for PCBs. When the species specific hazard ratios are summed, the hazard ratio for exposure to the maximum concentration of chlordane is also larger than one and the hazard ratio for exposure to maximum and mean concentrations of PCBs are 67 and 43 respectively. Most (79 percent) of the calculated PCB hazard is associated with exposure to the lobster hepatopancreas. Since the critical effect for PCBs is reduced size of offspring (Table 2), and no other indicator chemical in this study has a similar critical effect, this hazard ratio also serves as a hazard index.

The largest part (69.2 percent) of the chlordane hazard (Table 5) comes from exposure to the flounder portion of the diet. The critical effect for establishing the RfD for chlordane is liver necrosis. Since some of the other chlorinated organics also affect the liver, the hazard ratios for chlordane, DDT, HCB and HCH were added. The RfDs for the metals and PCBs are not based on adverse effects on the liver. Thus, the hazard ratios for metals and PCBs are not included in this hazard ratio

TABLE 5. RISK CHARACTERIZATION FOR A MAXIMALLY EXPOSED INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER, CLAMS, LOBSTER AND HEPATOPANCREAS (a)(b)

	FLOUNDER HAZARD RATIO		CLAMS HAZARD RATIO		LOBSTER HAZARD RATIO		HEPATOPANCREAS HAZARD RATIO		TOTAL HAZARD RATIO		FLOUNDER UPPER BOUND INCREASED CANCER RISK		CLAMS UPPER BOUND INCREASED CANCER RISK		LOBSTER UPPER BOUND INCREASED CANCER RISK		HEPATOPANCREAS UPPER BOUND INCREASED CANCER RISK		TOTAL UPPER BOUND INCREASED CANCER RISK	
CHEMICAL IDENTIFIED	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean
ELEMENTS/METALS																				
Cadmium	0.05	0.01	0.02	0.02	0.01	0.00	0.66	0.39	0.74	0.41	-	-	-	-	-	-	-	-	-	-
Chromium	0.12	0.01	0.01	0.01	0.02	0.00	0.04	0.01	0.20	0.03	-	-	-	-	-	-	-	-	-	-
Copper	0.01	0.00	0.01	0.01	0.07	0.05	0.65	0.32	0.74	0.38	-	-	-	-	-	-	-	-	-	-
Lead	0.05	0.02	0.08	0.07	0.06	0.05	0.04	0.02	0.23	0.16	-	-	-	-	-	-	-	-	-	-
Mercury	0.07	0.02	0.00	-	0.04	0.02	0.00	0.00	0.11	0.05	-	-	-	-	-	-	-	-	-	-
ORGANIC COMPOUNDS																				
Chlordane (total)	0.97	0.10	0.02	0.01	0.01	0.00	0.41	0.17	1.40	0.29	6.3E-05	6.6E-06	1.0E-06	8.6E-07	3.4E-07	2.1E-07	2.7E-05	1.1E-05	9.1E-05	1.9E-05
PP DDD	0.04	0.01	0.00	0.00	0.00	0.00	0.05	0.02	0.10	0.02	7.3E-06	8.7E-07	1.1E-07	9.6E-08	9.7E-08	7.7E-08	9.1E-06	2.9E-06	1.7E-05	4.0E-06
PP DDE	0.05	0.02	0.00	0.00	0.01	0.00	0.32	0.22	0.38	0.25	8.7E-06	2.0E-06	3.7E-07	3.3E-07	1.1E-06	7.3E-07	5.5E-05	3.8E-05	6.5E-05	4.2E-05
PP-DDT	0.02	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.03	0.01	2.7E-06	4.7E-07	2.6E-08	2.4E-08	8.9E-08	7.9E-08	2.1E-06	8.6E-07	5.0E-06	1.4E-06
Hexachlorobenzene (HCB)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6.9E-07	3.5E-07	4.0E-08	3.9E-08	1.6E-07	9.6E-08	2.8E-06	2.0E-06	3.7E-06	2.5E-06
Hexachlorocyclohexane (HCH)																				
a-HCH	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	9.1E-06	1.9E-06	1.8E-07	1.7E-07	4.9E-07	4.4E-07	1.8E-05	9.9E-06	2.8E-05	1.2E-05
g-HCH (lindane)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3.6E-07	3.3E-07	3.6E-08	3.6E-08	1.0E-07	7.0E-08	3.6E-07	2.0E-07	8.7E-07	6.4E-07
PAH (total)	-	-	-	-	-	-	-	-	-	-	4.9E-06	4.6E-06	1.2E-04	1.1E-04	3.7E-04	2.6E-04	4.7E-03	3.3E-03	5.2E-03	3.7E-03
PCBs (total)	11.99	4.41	0.35	0.34	1.64	1.02	52.98	37.63	66.96	43.40	3.1E-03	1.1E-03	9.1E-05	8.9E-05	4.3E-04	2.6E-04	1.4E-02	9.8E-03	1.7E-02	1.1E-02
											3.2E-03	1.2E-03	2.1E-04	2.1E-04	8.0E-04	5.2E-04	1.9E-02	1.3E-02	2.3E-02	1.5E-02

Ingestion rates:	grams/day
Flounder	113
Clams	16
Lobster	30
Hepatopancreas	6
TOTAL	165

(a): Mean calculated using detection limits for undetected observations.
See Appendix Table B-5 for calculations using zero instead of detection limits.

(b): See Table 2 for weight of evidence classification for carcinogens.

total. Summing the maximum ratios across the organic compounds listed above results in a total hazard ratio sum of 1.92. The sum of the mean hazard ratios for organic compounds is less than one. Exposure to seafood contaminated at the maximum level detected in Quincy Bay samples may result in adverse non-carcinogenic health effects to the maximally exposed individual if the dose received not only exceeds the reference dose but actually reaches a level corresponding to a health effects threshold. The RfD for PCBs of $1 * 10^{-4}$ mg/kg/day is based on a no observable adverse effects level (NOAEL) in monkeys with an intake of 0.01 mg/kg/day divided by an uncertainty factor of 100 made up of uncertainty factors for interspecies (10) and intraspecies (10) extrapolation. The critical effect of smaller offspring size (Table 2) was seen in monkeys with an intake of 0.4 mg/kg/day. The uncertainty associated with the determination of this RfD (LOAEL/RFD=4,000) indicates that while exceeding the RfD by the amount indicated in Table 2 (i.e. by a factor of between 43 and 67 times) may increase the probability of an adverse health effect, there is no basis for expectation of a specific adverse non-carcinogenic response.

The range of estimated total upper limit increased cancer risk for this maximally exposed individual (Table 5) is $1.5 * 10^{-2}$ to $2.3 * 10^{-2}$, based on exposure to mean and maximum concentrations of contaminants. These numbers are estimates of the plausible upper bound of lifetime cancer risk and may not represent the actual risk. The largest increased lifetime cancer

risks are primarily (82 percent) associated with consumption of the tomalley (hepatopancreas) and secondarily the flounder (14 percent) and lobster meat (3.4 percent). As shown in Table 6, the contaminants contributing the largest portion (approximately 75 percent) of the excess cancer risk are polychlorinated biphenyls (PCBs), followed by polycyclic aromatic hydrocarbons (PAHs) (about 20 to 25 percent).

The hazard ratios and cancer risks calculated for the mean levels where undetected observations are assumed equal to zero (Appendix B, Table B-5) are essentially the same as the values calculated where undetected observations are set equal to the detection limit (Table 5).

The risk characterization for a maximally exposed individual consuming 165 grams per day of Quincy Bay flounder and no other Quincy Bay seafood (Table 7 and Table B-6 in Appendix B) indicates that both the hazard ratios for ingesting flounder contaminated with chlordane and PCBs exceed one. Summing the indices across chlordane, DDT, HCB, and HCH as discussed previously, results in a hazard ratio total or index of 1.58 associated with adverse health effects on the liver for the maximum contaminant level and 0.18 for the average contaminant level. The hazard ratios associated with exposure to PCBs are 17.51 and 6.44 for maximum and mean concentrations respectively, indicating that adverse noncarcinogenic health effects may occur from exposure to the level of contamination detected in Quincy Bay flounder.

TABLE 6. PERCENT CONTRIBUTION TO UPPER BOUND CANCER RISK BY EACH INDICATOR CHEMICAL

CHEMICAL IDENTIFIED	PERCENT CONTRIBUTION FOR MEI FROM INGESTION OF QUINCY BAY FLOUNDER, CLAMS, LOBSTER AND HEPATOPANCREAS(a)(b)		PERCENT CONTRIBUTION FOR MEI FROM INGESTION OF QUINCY BAY FLOUNDER ONLY(c)		PERCENT CONTRIBUTION FOR A TYPICAL QUINCY AREA INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER AND LOBSTER(d)		PERCENT CONTRIBUTION FOR A TYPICAL QUINCY AREA INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER, LOBSTER, AND HEPATOPANCREAS(e)	
	max	mean	max	mean	max	mean	max	mean
ELEMENTS/METALS								
Cadmium	-	-	-	-	-	-	-	-
Chromium	-	-	-	-	-	-	-	-
Copper	-	-	-	-	-	-	-	-
Lead	-	-	-	-	-	-	-	-
Mercury	-	-	-	-	-	-	-	-
ORGANIC COMPOUNDS								
Chlordane (total)	0.40	0.12	1.96	0.57	0.69	0.16	0.18	0.09
PP-DDD	0.07	0.03	0.23	0.07	0.08	0.03	0.05	0.02
PP-DDE	0.29	0.28	0.27	0.24	0.18	0.16	0.29	0.28
PP-DDT	0.02	0.01	0.08	0.04	0.04	0.02	0.01	0.01
Hexachlorobenzene (HCB)	0.02	0.02	0.02	0.03	0.02	0.02	0.02	0.02
Hexachlorocyclohexane (HCH)								
a-HCH	0.12	0.08	0.28	0.16	0.14	0.10	0.10	0.08
g-HCH (lindane)	0.00	0.00	0.01	0.03	0.01	0.02	0.00	0.00
PAH (total)	22.84	24.59	0.15	0.39	30.61	38.40	25.55	25.76
PCBs (total)	76.24	74.87	96.99	98.46	68.22	61.09	73.80	73.75
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

FOOTNOTES:

(a): MEI= Maximally Exposed Individual.
(b): See Table 5 for further detail.
(c): See Table 7 for further detail.
(d): See Table 8 for further detail.
(e): See Table 9 for further detail.

TABLE 7 LIFETIME RISK CHARACTERIZATION FOR A MAXIMUM EXPOSED INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER ONLY (a)(b)
(using wet weight data)

CHEMICAL IDENTIFIED	FDA LIMITS (ppm)	CPF Carcinogenic Potency Factor (mg/kg/day) 1 Oral	REF	RfD Reference Dose (mg/kg/day)	REF	1987 Data FLOUNDER (Flesh) (ug/g) max	mean	FLOUNDER DOSE (c) (ug/kg/day) max	mean	HAZARD RATIO (d) max	mean	INCREASED UPPER BOUND CANCER RISK (e) max	mean	PERCENT CONTRIBUT FOR MEI FROM INGES OF QUINCY BAY FLOU ONLY(c) max	mean
ELEMENTS/METALS															
Cadmium		NA		2.90E-04	1	9.00E-03	1.00E-03	2.12E-02	2.36E-03	0.07	0.01	-	-	-	-
Chromium		NA		5.00E-03	3	3.77E-01	2.90E-02	8.89E-01	6.84E-02	0.18	0.01	-	-	-	-
Copper		NA		3.70E-02	3	2.15E-01	1.09E-01	5.07E-01	2.57E-01	0.01	0.01	-	-	-	-
Lead *		NA		1.40E-03	3	4.30E-02	1.50E-02	1.01E-01	3.54E-02	0.07	0.03	-	-	-	-
Mercury *		NA		2.00E-03	3	8.60E-02	3.00E-02	2.03E-01	7.07E-02	0.10	0.04	-	-	-	-
ORGANIC COMPOUNDS															
Chlordane (total)	0.3	1.3	1	5.00E-05	1	3.00E-02	3.15E-03	7.07E-02	7.43E-03	1.41	0.15	9.2E-05	9.7E-06	1.96	0.57
PP DDD	5.0	0.34	3	5.00E-04	3	1.33E-02	1.58E-03	3.12E-02	3.72E-03	0.06	0.01	1.1E-05	1.3E-06	0.23	0.07
PP DDE	5.0	0.34	3	5.00E-04	3	1.59E-02	5.19E-03	3.75E-02	1.22E-02	0.08	0.02	1.3E-05	4.2E-06	0.27	0.24
PP-DDT	5.0	0.34	3	5.00E-04	3	4.97E-03	8.55E-04	1.17E-02	2.02E-03	0.02	0.00	4.0E-06	6.9E-07	0.08	0.04
Hexachlorobenzene (HCB)		1.69	3	8.00E-04	4	2.52E-04	1.27E-04	5.94E-04	2.99E-04	0.00	0.00	1.0E-06	5.1E-07	0.02	0.03
Hexachlorocyclohexane (HCH)															
a HCH		6.3	1	3.00E-04	3	8.93E-04	1.82E-04	2.11E-03	4.29E-04	0.01	0.00	1.3E-05	2.7E-06	0.28	0.16
g HCH (lindane)		1.33	2	3.00E-04	3	1.70E-04	1.56E-04	4.01E-04	3.68E-04	0.00	0.00	5.3E-07	4.9E-07	0.01	0.03
PAH (f)		11.53	2	NA		2.61E-04	2.45E-04	6.15E-04	5.77E-04	-	-	7.1E-06	6.7E-06	0.15	0.39
PCBs (g)	2.0	2.6	2	1.00E-04	5	7.43E-01	2.73E-01	1.75E+00	6.44E-01	17.51	6.44	4.6E-03	1.7E-03	96.99	98.46
												4.7E-03	1.7E-03	100.00	100.00

FOOTNOTES:
* = Data correspond to inorganic compounds.
NA: Not available

(a): Mean calculated using detection limits for undetected observations.
See Appendix Table B-6 for calculations using zero instead of detection limits.

(b): See Table 2 for weight of evidence classification for carcinogens.

(c): Calculated dose= contaminant concentration (ug/g) x 165 grams of fish ingested /day/70 kilograms body weight.

(d): Hazard Ratio= Calculated Dose(ug/kg/day)/[Reference Dose(mg/kg/day)*1000ug/mg].

(e): Increased Upper Bound Cancer Risk= [Calculated Dose(ug/kg/day)*0.001mg/ug] * Carcinogenic Potency Factor (mg/kg/day)-1.

(f): Total Polycyclic Aromatic Hydrocarbons.

(g): Total Polychlorinated Biphenyls.

REFERENCES

(1) Integrated Risk Information System Chemical Files

(2) Health Effects Assessment Documents

(3) Superfund Public Health Evaluation Manual

(4) Health Advisories for 25 Organics.

(5) USEPA. 1988a, b and c.

For this MEI consumer of flounder only, total plausible upperbound increased estimated cancer risks range from $1.7 * 10^{-3}$ to $4.7 * 10^{-3}$ for mean and maximum contaminant levels respectively. Comparison with the values of Table 5 shows that the MEI flounder-only diet leads to a projection of between about 10 percent and about 30 percent of the estimated upper bound cancer risk of the MEI mixed diet.

D. Typical Quincy Area Resident

Risk characterizations are presented for two types of typical Quincy area residents (Tables 8 and 9 and B-7 and B-8 in Appendix B). One case was based on the assumption was that the resident regularly consumes locally caught flounder and lobster in average amounts (Table 8) without eating the lobster tomalley (hepatopancreas). The second case (Table 9) was for the resident who consumes flounder, lobster and tomalley.

None of the hazard ratios associated with typical ingestion of flounder and lobster without the tomalley (Table 8 or 9) are larger than 0.22, indicating that non-carcinogenic health effects are not likely from ingesting seafood at the levels suggested in the first scenario. The estimated upper bound increased lifetime cancer risks range from $4.7 * 10^{-5}$ to $8.4 * 10^{-5}$ for exposure to mean and maximum levels of contamination for individuals who do not eat tomalley.

For the typical Quincy resident who eats flounder, lobster and tomalley, the hazard ratios (Table 9) associated with maximum

TABLE 8. RISK CHARACTERIZATION FOR A TYPICAL QUINCY AREA INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER AND LOBSTER (a)(b)

CHEMICAL IDENTIFIED	FLOUNDER HAZARD RATIO max	FLOUNDER HAZARD RATIO mean	LOBSTER HAZARD RATIO max	LOBSTER HAZARD RATIO mean	TOTAL HAZARD RATIO max	TOTAL HAZARD RATIO mean	FLOUNDER UPPER BOUND INCREASED CANCER RISK max	FLOUNDER UPPER BOUND INCREASED CANCER RISK mean	LOBSTER UPPER BOUND INCREASED CANCER RISK max	LOBSTER UPPER BOUND INCREASED CANCER RISK mean	TOTAL UPPER BOUND INCREASED CANCER RISK max	TOTAL UPPER BOUND INCREASED CANCER RISK mean
ELEMENTS/METALS												
Cadmium	0.00	0.00	0.00	0.00	0.00	0.00	-	-	-	-	-	-
Chromium	0.00	0.00	0.00	0.00	0.00	0.00	-	-	-	-	-	-
Copper	0.00	0.00	0.01	0.00	0.01	0.00	-	-	-	-	-	-
Lead	0.00	0.00	0.00	0.00	0.00	0.00	-	-	-	-	-	-
Mercury	0.00	0.00	0.00	0.00	0.00	0.00	-	-	-	-	-	-
ORGANIC COMPOUNDS												
Chlordane (total)	0.01	0.00	0.00	0.00	0.01	0.00	5.6E-07	5.9E-08	2.4E-08	1.5E-08	5.8E-07	7.3E-08
PP-DDD	0.00	0.00	0.00	0.00	0.00	0.00	6.4E-08	7.7E-09	6.8E-09	5.4E-09	7.1E-08	1.3E-08
PP-DDE	0.00	0.00	0.00	0.00	0.00	0.00	7.7E-08	2.5E-08	7.6E-08	5.1E-08	1.5E-07	7.7E-08
PP-DDT	0.00	0.00	0.00	0.00	0.00	0.00	2.4E-08	4.2E-09	6.2E-09	5.6E-09	3.0E-08	9.7E-09
Hexachlorobenzene (HCB)	0.00	0.00	0.00	0.00	0.00	0.00	6.1E-09	3.1E-09	1.1E-08	6.7E-09	1.7E-08	9.8E-09
Hexachlorocyclohexane (HCH)												
a-HCH	0.00	0.00	0.00	0.00	0.00	0.00	8.0E-08	1.6E-08	3.5E-08	3.1E-08	1.1E-07	4.7E-08
g-HCH (lindane)	0.00	0.00	0.00	0.00	0.00	0.00	3.2E-09	3.0E-09	7.2E-09	4.9E-09	1.0E-08	7.8E-09
PAH (total)	-	-	-	-	-	-	4.3E-08	4.0E-08	2.6E-05	1.8E-05	2.6E-05	1.8E-05
PCBs (total)	0.11	0.04	0.11	0.07	0.22	0.11	2.8E-05	1.0E-05	3.0E-05	1.8E-05	5.7E-05	2.9E-05
							2.8E-05	1.0E-05	5.6E-05	3.7E-05	8.4E-05	4.7E-05

Ingestion rates:	grams/day
Flounder	1
Clams	0
Lobster	2.1
Hepatopancreas	0.0
TOTAL	3.1

(a): Mean calculated using detection limits for undetected observations.
See Appendix Table B-7 for calculations using zero instead of detection limits.

(b): See Table 2 for weight of evidence classification for carcinogens.

TABLE 9. RISK CHARACTERIZATION FOR A TYPICAL QUINCY AREA INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER, LOBSTER AND HEPATOPANCREAS (a)(b)

CHEMICAL IDENTIFIED	FLOUNDER HAZARD RATIO		LOBSTER HAZARD RATIO		HEPATOPANCREAS HAZARD RATIO		TOTAL HAZARD RATIO		FLOUNDER UPPER BOUND INCREASED CANCER RISK		LOBSTER UPPER BOUND INCREASED CANCER RISK		HEPATOPANCREAS UPPER BOUND INCREASED CANCER RISK		TOTAL UPPER BOUND INCREASED CANCER RISK	
	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean
ELEMENTS/METALS																
Cadmium	0.00	0.00	0.00	0.00	0.04	0.03	0.04	0.03	-	-	-	-	-	-	-	-
Chromium	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	-	-	-	-	-	-	-	-
Copper	0.00	0.00	0.00	0.00	0.04	0.02	0.05	0.02	-	-	-	-	-	-	-	-
Lead	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	-	-	-	-	-	-	-	-
Mercury	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-	-	-	-	-	-	-	-
ORGANIC COMPOUNDS																
Chlordane (total)	0.01	0.00	0.00	0.00	0.03	0.01	0.04	0.01	5.6E-07	5.9E-08	1.9E-08	1.2E-08	1.8E-06	7.2E-07	2.4E-06	7.9E-07
PP-DDD	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6.4E-08	7.7E-09	5.5E-09	4.4E-09	6.1E-07	1.9E-07	6.8E-07	2.1E-07
PP-DDE	0.00	0.00	0.00	0.00	0.02	0.01	0.02	0.02	7.7E-08	2.5E-08	6.2E-08	4.2E-08	3.7E-06	2.5E-06	3.8E-06	2.6E-06
PP-DDT	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	2.4E-08	4.2E-09	5.1E-09	4.5E-09	1.4E-07	5.7E-08	1.7E-07	6.6E-08
Hexachlorobenzene (HCB)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6.1E-09	3.1E-09	9.0E-09	5.5E-09	1.9E-07	1.3E-07	2.0E-07	1.4E-07
Hexachlorocyclohexane (HCH)																
a-HCH	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	8.0E-08	1.6E-08	2.8E-08	2.5E-08	1.2E-06	6.6E-07	1.3E-06	7.0E-07
g-HCH (lindane)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3.2E-09	3.0E-09	5.8E-09	3.9E-09	2.4E-08	1.4E-08	3.3E-08	2.0E-08
PAH (total)	-	-	-	-	-	-	-	-	4.3E-08	4.0E-08	2.1E-05	1.5E-05	3.1E-04	2.2E-04	3.4E-04	2.4E-04
PCBs (total)	0.11	0.04	0.09	0.06	3.53	2.51	3.73	2.61	2.8E-05	1.0E-05	2.4E-05	1.5E-05	9.2E-04	6.5E-04	9.7E-04	6.8E-04
									2.8E-05	1.0E-05	4.5E-05	3.0E-05	1.2E-03	8.8E-04	1.3E-03	9.2E-04

Ingestion rates:	grams/day
Flounder	1
Clams	0
Lobster	1.7
Hepatopancreas	0.4
TOTAL	3.1

(a) Mean calculated using detection limits for undetected observations.
See Appendix Table B-8 for calculations using zero instead of detection limits.

(b) See Table 2 for weight of evidence classification for carcinogens.

and mean PCB contaminant levels are 3.73 and 2.71 respectively, indicating non-carcinogenic health effects may occur from exposure. The largest portion (approximately 95 percent) of the hazard ratio comes from consuming the tomalley. The plausible upper bound increased lifetime cancer risk levels are $9.2 * 10^{-4}$ to $1.3 * 10^{-3}$, with PCBs contributing approximately 74 percent of the risk.

VI. Conclusions and Uncertainties

Risk Comparison

Tabel 10 shows a summary comparison of the total upperbound estimated cancer risks in this study by consumption scenario and by type of seafood. A comparison of the estimated upperbound increased lifetime cancer risks of consuming Quincy Bay seafood with other estimated lifetime cancer risks from eating and drinking (Table 11) shows that some of the cases analyzed in this study result in risk estimates considerably higher than those estimated by others from other types of activities. In particular, the estimated incremental risk (plausible upper bound) for the hypothetical Maximally Exposed Individual eating a mixed diet of Quincy Bay seafood is about 50-100 times higher than the estimates for any of the other typical eating and drinking activities shown on the table. The estimated risk (plausible upper bound) for just consumption of Quincy Bay Winter flounder is about 10 times higher than the levels estimated for the other eating and drinking activities.

TABLE 10. UPPER BOUND ESTIMATED LIFE TIME CANCER RISKS FROM CONSUMPTION OF QUINCY BAY SEAFOOD

	Maximally Exposed Individual		Typical Exposed Individual	
	Mixed Diet	Flounder	Mixed Diet	Mixed Diet
Clams	$2.1*10^{-4}$ (1%)	-	-	-
Flounder	$3.2*10^{-3}$ (14%)	$4.7*10^{-3}$ (100%)	$2.8*10^{-5}$ (33%)	$2.8*10^{-5}$ (2.2%)
Lobster Meat	$8.0*10^{-4}$ (3.4%)	-	$5.6*10^{-5}$ (67%)	$4.5*10^{-5}$ (3.5%)
Tomalley	$1.9*10^{-2}$ (82.6%)	-	-	$1.2*10^{-3}$ (92.3%)
Total Risk	$2.3*10^{-2}$	$4.7*10^{-3}$	$8.4*10^{-5}$	$1\ 3*10^{-3}$

TABLE 11. COMPARISON OF ESTIMATED LIFETIME CANCER RISKS (PLAUSIBLE UPPER LIMIT)

Lifetime Cancer Risks From Eating and Drinking Activities	Estimated Lifetime Risks(a)
• Maximally Exposed Individual - mixed diet of Quincy Bay seafood	1.5 to $2.3 * 10^{-2}$
• Maximally Exposed Individual - diet of Quincy Bay winter flounder	1.7 to $4.7 * 10^{-3}$
• Typical Quincy area resident - mixed diet of Quincy Bay seafood, including lobster tomalley	$9.2 * 10^{-4}$ to $1.3 * 10^{-3}$
• Four Tablespoons peanut butter per day	$5.6 * 10^{-4}$
• One 12 1/2 ounce diet drink per day (6)	$7.0 * 10^{-4}$
• Average saccharin consumption in the United States	$1.4 * 10^{-4}$
• One pint milk per day(b)	$1.4 * 10^{-4}$
• Typical Quincy area resident - mixed diet of Quincy Bay seafood without lobster tomalley	$4.7 * 10^{-5}$ to $8.4 * 10^{-5}$
• Miami or New Orleans drinking water	$7.0 * 10^{-5}$
• 1/2 lb. charcoal broiled steak per week (cancer risk only; heart attack and other risks additional)	$2.1 * 10^{-7}$

(a) Except for Quincy Bay seafood consumption estimates for sub-populations, all other estimates are averaged over the whole population of the United States, assuming a 70 year lifetime.

(b) Based on human data for aflatoxin carcinogenicity. Note that it is assumed that the measured aflatoxins are aflatoxin B, the most potent. If some corresponds to other aflatoxins, these estimated risks should be reduced.

Sources: modified from Meta Systems, Inc. 1986.

Note: Meta Systems Inc., (1986), modified the original annual risk estimates from Crouch and Wilson, (1982), to represent estimated lifetime risks.

The estimated lifetime risk for the hypothetical "typical" local resident consumer of a mixed Quincy Bay seafood diet including lobster tomalley is about two to ten times higher than the estimate for the other eating and drinking activities. Note that without lobster tomalley, the estimated risks for the hypothetical typical Quincy area consumer of Quincy Bay seafood drop into the 10^{-5} range corresponding to the risks of the other illustrated eating and drinking activities.

In work done by the Canadian Government, (Environment Canada. 1987), the estimated dietary intake of PCBs was calculated for a variety of food items. The calculations were based on a mixture of measured PCB residues for most food items and the assumed presence of maximum allowed PCB residues in fish. These data are presented in Table 12 with the data used in this public health evaluation to provide a comparison of how PCB intake from fish compares with PCB intake from other food sources. Under any of the consumption scenarios documented by the Penn State (1985) report, the Canadian studies, or otherwise assumed in this study, more than half of the total exposure to PCBs comes from seafood consumption. In the case of the MEI for this study, estimated PCB exposure from seafood is more than 20 times higher than that estimated by the Canadian data from all other dietary sources combined.

TABLE 12. SOURCES OF INTAKE OF PCBs

Food	Food Intake(a) (g/person-day)	Maximum Residue Level (µg/g)(h)	PCB Intake (µg/person-day)
Canadian data (b)			
dairy	32.8	0.2(d)	6.6
meat	48	0.2(d)	9.6
poultry	3.6	0.5(d)	1.8
eggs	34	0.1(e)	3.4
fish	20	2(f)	40
Quincy Bay seafood			
maximally exposed individual	165	(g)	470
typical Quincy area resident	3.1	(g)	26

(a) Based on statistics Canada uses for disappearance of foods from the marketplace.
(b) Reference for Canadian data: Environment Canada. 1987.
(c) Includes milk, cheese, and butter.
(d) Fat basis.
(e) Whole weight minus shell.
(f) Edible portion, assumed based on maximum residue level allowed
(g) Varies by different kind of seafood, see Table 1.
(h) Based on measured residues for all Canadian data except fish. Fish value based on maximum allowed.

Uncertainties

Extreme caution must be exercised in the interpretation and use of any risk data due to a variety of uncertainties. Sources of uncertainty in this risk assessment are discussed individually below.

1. *Representativeness of the measured values for contaminants in seafood.* Comparisons of the PCB values obtained by Gardner and Pruell (1987) with other data for the same species in Quincy Bay and other parts of Boston Harbor (Table 13) suggest that the 1987 EPA values are representative for Quincy Bay, given the differences in sample locations and analytical methodologies of the various studies. Preliminary results of ongoing studies involving inter-laboratory calibration of EPA, MDMF and FDA methods of determining PCB concentrations in various edible portions of lobster indicate that differences among the agency analytical techniques are likely not significant.

2. *Use of standard risk assessment assumptions.* Many of the assumptions used in this risk assessment are standard risk assessment assumptions chosen to be

TABLE 13. COMPARISON OF PCB LEVELS MEASURED IN ORGANISMS SAMPLED FROM QUINCY BAY AND BOSTON HARBOR (µg/g, WET WEIGHT)

	Quincy Bay EPA, 1987(a)	No.	Boston Harbor Including Quincy Bay Mass DMF(b)	No.	Boston Harbor NOAA. 1984(c)	No.
Lobster	MAX - 10.69 MEAN - 7.61	(8)	1983 MAX - N.R. MEAN - 4.00 1985 MAX - 1.60 MEAN - 0.90 1986 MAX - 2.19 MEAN - 1.17	(10) (15) (5)		
Flounder	MAX - 0.74 MEAN - 0.27	(25)	1983 MAX - 0.30 MEAN - 0.20 1984 MAX - 1.00 MEAN - 0.39 1986 MAX - 1.30 MEAN - 0.63	(10) (10) (18)	MAX - 0.14 MEAN - 0.10	(4)
Clam	MAX - 0.15 MEAN - 0.15	(2)	1986 MAX - N.R. MEAN - 0.14	2 Samples, 24 organisms		

(a) Source: Gardner and Pruell. 1987. Lobster values represent calculated weighted average of separately measured values for tomalley (17% by weight) and other edible tissue (83% by weight) for comparison purposes. These values were not used in the risk assessment. Percentages from MDMF (unpublished data)

(b) Source: Mass. Division of Marine Fisheries (MDMF), unpublished data and Schwartz, 1987.

(c) Source: Boehm et al. 1984.

N.R. = Not reported.

conservative, albeit uncertain. These include the following assumptions: (1) Probable Human Carcinogens, (Group B2, where human evidence of carcinogenicity is limited or inadequate but animal evidence of carcinogenicity is available), contribute to estimated increased cancer risks; (2) related compounds such as PAHs or PCB congeners, can be evaluated by the assumed toxicity of the more toxic compounds for which data are available (e.g. B(a)P for the PAHs) and (3) ingested doses of contaminants are totally absorbed. Since these assumptions generally apply to the other types of contaminant risk assessments conducted by the EPA, their use was considered valid as an initial reference point for this study. It is acknowledged that not all PAHs are known carcinogens (see section III). However, all 5 of the PAHs rated as having sufficient evidence of animal carcinogenicity (Table 3) were detected in Quincy Bay seafood in this study. These five compounds comprised up to about ten percent of the total PAHs in some of the organisms analyzed (Gardner and Pruell. 1987). Given this detection and the lack of importance of PAHs versus PCBs in the total risk calculation (see table 6) the effect of treating all PAHs as carcinogens in this study was minor.

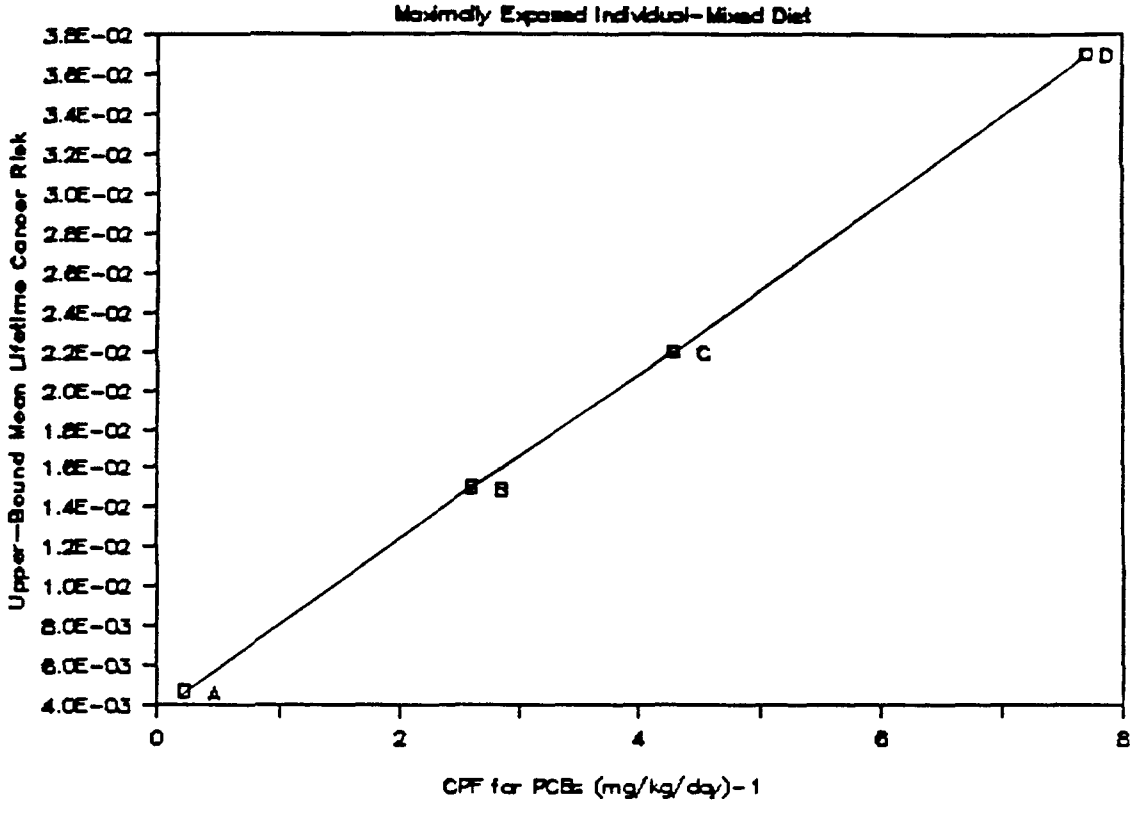
Note that for PCBs there are different CPFs used by different agencies. Agencies have recognized the need

for congener-specific CPFs for PCBs and are working towards development of such numbers. The CPF used in this assessment was developed by EPA based on laboratory experiments using Aroclor 1254. The CPF for Aroclor 1254 was used because the congener mix detected in Quincy Bay seafood more closely resembled Aroclor 1254 than Aroclor 1260. Appendix C documents this derivation.

A sensitivity analysis was performed to determine the effect of varying the CPF for PCBs, from 0.22 $(mg/kg/day)^{-1}$ to 7.7 $(mg/kg/day)^{-1}$ on the calculation of plausible upper bound lifetime cancer risk. The lower CPF value cited has been used by New York State to evaluate PCB levels in fish (Bro. 1987). The 7.7 value is proposed by EPA to replace the current US EPA CPF value of 4.34 based on the carcinogenicity of Aroclor 1260. Figures 5 and 6 show the effect on the risk calculations of using different CPF assumptions for PCBs. For the maximally exposed individual the plausible upper bound increased lifetime cancer risk is 4.7×10^{-3} using a CPF of 0.22 $(mg/kg/day)^{-1}$ and average contaminant levels, and 3.7×10^{-2} for a CPF of 7.7 $(mg/kg/day)^{-1}$.

3. *Assumption that cooking does not change contaminant levels.* This assumption is recommended in EPA guidance (PTI. 1987) for seafood consumption risk assessments. The same authors acknowledge that the assumption may not

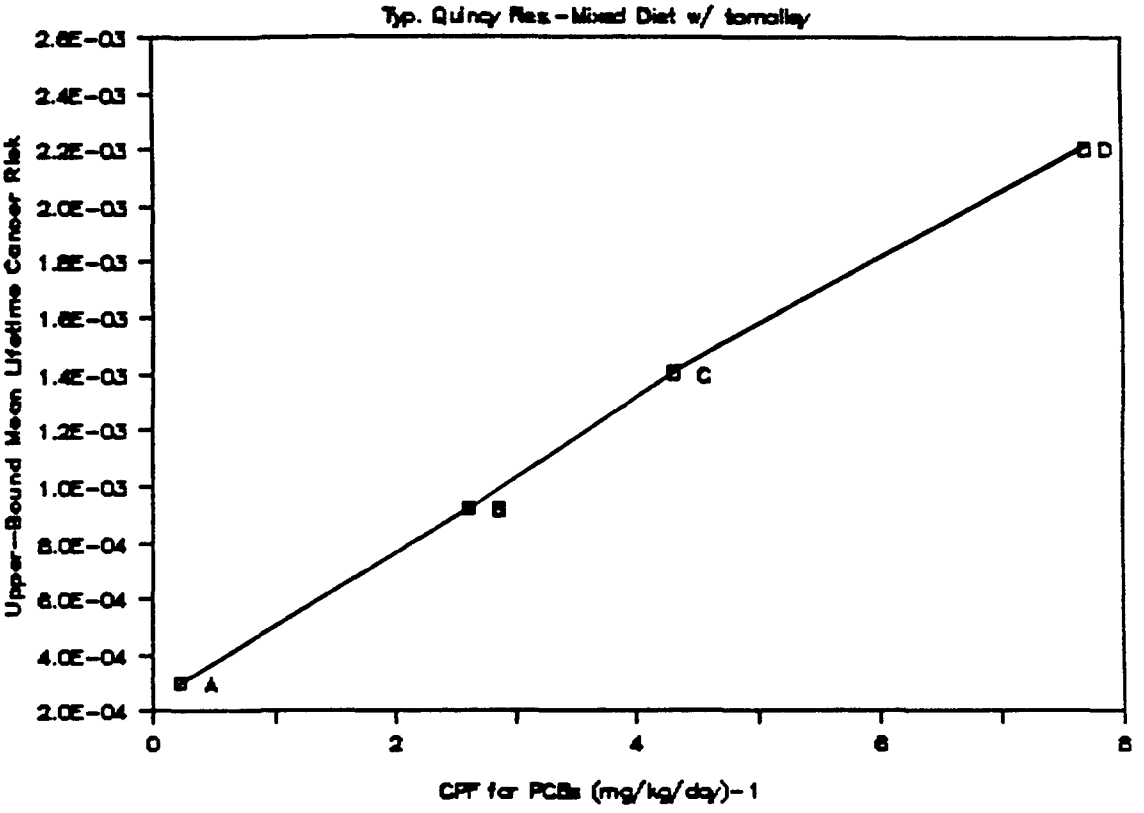
Fig. 5. EFFECT of CPFs for PCBs



KEY

A. Used by N.Y. State in a study on PCBs in fish (Bro et al. 1987).
B. USEPA OHEA developed this value for this study (USEPA. 1988a).
C. From the Superfund Public Health Evaluation Manual (USEPA.1986b).
D. Value developed by USEPA based on the carcinogenicity of Aroclor 1260 (USPHS. 1987).

Fig. 6. EFFECT of CPFs for PCBs



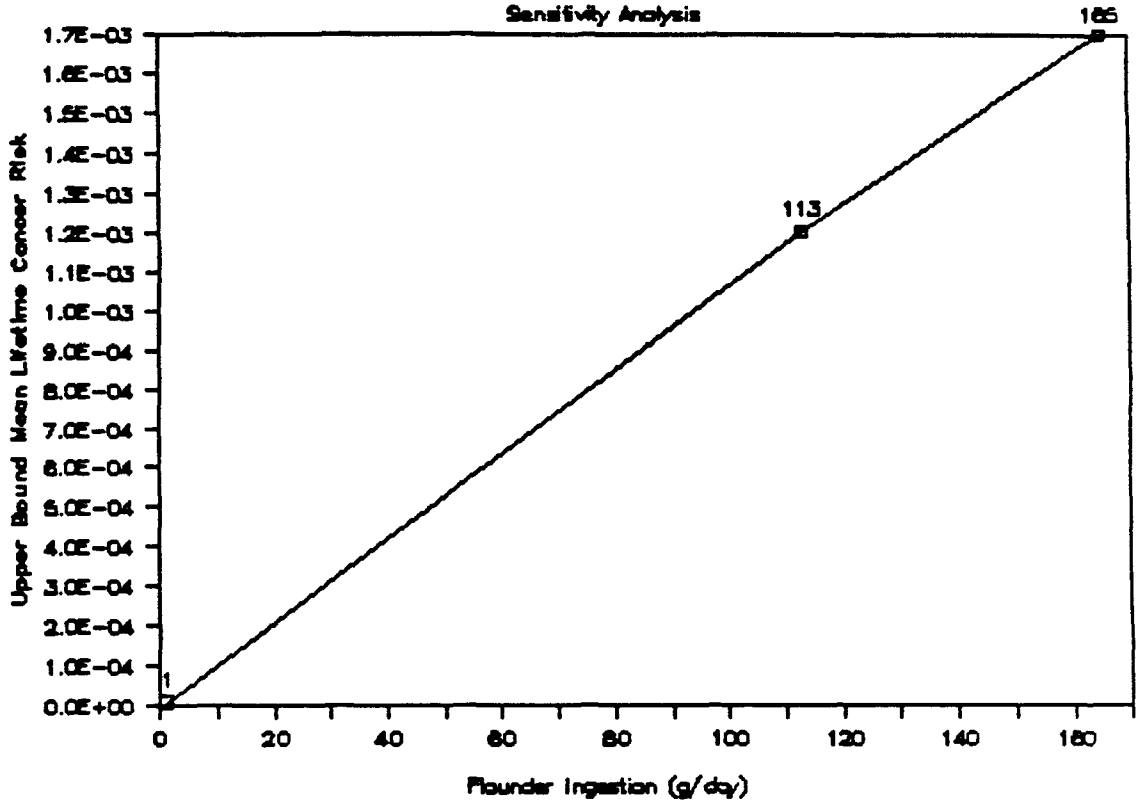
KEY

A. Used by N.Y. State in a study on PCBs in fish (Bro et al. 1987).
B. USEPA OHEA developed this value for this study (USEPA. 1988a).
C. From the Superfund Public Health Evaluation Manual (USEPA.1986b).
D. Value developed by USEPA based on the carcinogenicity of Aroclor 1260 (USPHS. 1987).

be valid in all cases, citing, for example, that there have been discussions of possible decreases in concentrations of PCBs in cooked versus uncooked samples of Great Lakes salmonids. However, there is no evidence to support alternative assumptions in this case. Also of interest is the possibility that some of the contaminants in lobster tomalley may be released by cooking, thereby becoming potentially available to affect (increase) the concentration in other edible lobster tissue being cooked in the same vessel. Further investigation of this uncertainty by sampling and analysis of uncooked and cooked lobsters would resolve this uncertanity.

4. *Affected population size and consumption patterns.* As noted in Section IV of this report, estimates of the actual size of affected populations were not made in this study due to the necessary reliance on a fall-winter study period.. Some of the data obtained from earlier consumption surveys (Penn State. 1985) may need to be checked. For example, the reported average regional lobster consumption values may be high if the respondents described their consumption in terms of whole lobster rather than edible lobster tissue, and if the researchers failed to adjust the reported values. Figures 7, 8 and 9 show the sensitivity of the upper-bound increased cancer risk calculations to the assumed

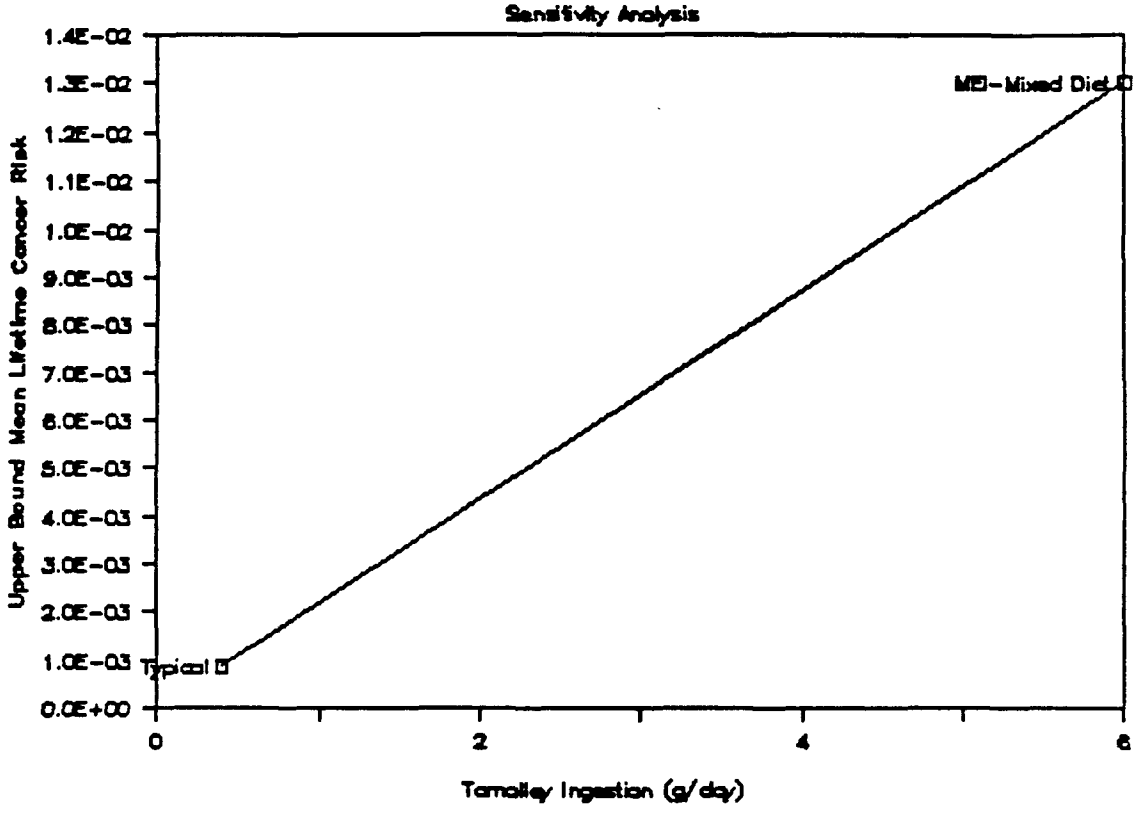
FIG. 7. FLOUNDER CONSUMPTION EFFECTS



FLOUNDER INGESTION

Consumption Scenario	Grams/Day	Mean Risk
Typical Local Consumer	1	1.0E-05
Max. Exp. Ind. - Mixed Diet	113	1.2E-03
Max. Exp. Ind. - Flounder Only	165	1.7E-03

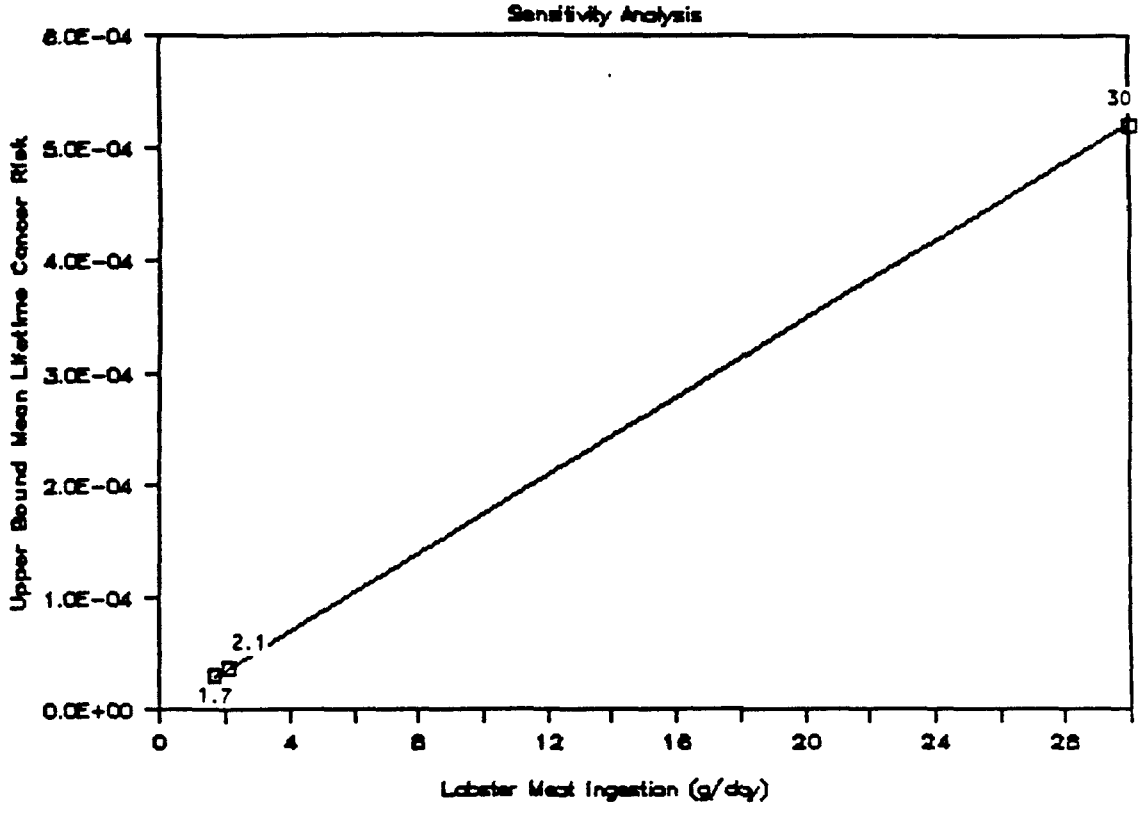
FIG. 8. TOMALLEY CONSUMPTION EFFECTS



HEPATOPANCREAS INGESTION

Consumption Scenario	Grams/Day	Mean Risk
Typical Local Consumer	0.4	8.8E-04
Maximally Exposed Individual	.6	1.3E-02

FIG. 9. LOBSTER CONSUMPTION EFFECTS



LOBSTER MEAT CONSUMPTION

Consumption Scenario	Grams/Day	Mean Risk
Typical Local Consumer (With Tomalley)	1.7	3.0E-05
Typical Local Consumer (Without Tomalley)	2.1	3.7E-05
Max. Exp. Ind. - Mixed Diet	30	5.2E-04

comsumption rates for flounder, lobster tissue and lobster tomalley.

5. *Other sources of the same contaminants.* The study results (Tables 5, 7, 8 and 9) indicate that PCB and/or chlordane residues in Quincy Bay flounder may constitute a significant fraction of threshold-based, non-carcinogenic health risks if taken in combination with other sources of exposure of the same chemicals. Estimation of total risks due to PCB and chlordane exposure would require a specially focused investigation, but is feasible.

In summary, several areas of uncertainty remain, some of which have been assessed above by sensitivity analysis. The results of the sensitivity analyses do not change the conclusions stated earlier regarding human health risks.

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b)	Chlordane.	PB86-134343
c)	Chromium.	PB86-134301
d)	Copper	PB86-134368
e)	DDT.	PB86-134368
f)	Hexachlorobenzene.	PB86-134285
g)	Lead.	PB86-134665
h)	Lindane.	PB86-134673
i)	Mercury	PB86-134533
j)	Polycyclic Aromatic Hydrocarbons.	PB86-134244
k)	Polychlorinated Biphenyls.	PB86-134512

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a)	Chromium III	CAS No.: 16065-83-1
b)	Lindane	CAS No.: 58-89-9.

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d)	Cadmium	CAS No.: 7440-43-9
e)	Chlordane	CAS No.: 57-74-9
f)	Chromium VI	CAS No.: 7440-47-3
g)	DDT	CAS No.: 50-29-3
h)	Lindane	CAS No.: 58-89-9.

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Appendix A

Toxicity Profiles

TABLE A1. EPA WEIGHT-OF-EVIDENCE CATEGORIES FOR POTENTIAL CARCINOGENS

EPA Category	Description of Group	Description of Evidence
Group A	Human Carcinogen	Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer
Group B1	Probable Human Carcinogen	Limited evidence of carcinogenicity in humans from epidemiologic studies
Group B2	Probable Human Carcinogen	Sufficient evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans
Group C	Possible Human Carcinogen	Limited evidence of carcinogenicity in animals
Group D	Not Classified	Inadequate evidence of carcinogenicity in animals
Group E	No Evidence of Carcinogenicity in Humans	No evidence for carcinogenicity in at least two adequate animal tests or in both epidemiologic and animal studies

Source: USEPA. 1986 (a).

TABLE A2. RATING CONSTANTS (RVs) FOR NONCARCINOGENS[a]

Effect	Severity Rating (RVs)
Enzyme induction or other biochemical change with no pathologic changes and no change in organ weights.	1
Enzyme induction and subcellular proliferation or other changes in organelles but no other apparent effects.	2
Hyperplasia, hypertrophy or atrophy, but no change in organ weights.	3
Hyperplasia, hypertrophy or atrophy with changes in organ weights.	4
Reversible cellular changes: cloudy swelling, hydropic change, or fatty changes.	5
Necrosis, or metaplasia with no apparent decrement of organ function. Any neuropathy without apparent behavioral, sensory, or physiologic changes.	6
Necrosis, atrophy, hypertrophy, or metaplasia with a detectable decrement of organ functions. Any neuropathy with a measurable change in behavioral, sensory, or physiologic activity.	7
Necrosis, atrophy, hypertrophy, or metaplasia with definitive organ dysfunction. Any neuropathy with gross changes in behavior, sensory, or motor performance, Any decrease in reproductive capacity, any evidence of fetotoxicity.	8
Pronounced pathologic changes with severe organ dysfunction. Any neuropathy with loss of behavioral or motor control or loss of sensory ability. Reproductive dysfunction. Any teratogenic effect with maternal toxicity.	9
Death or pronounced life-shortening. Any teratogenic effect without signs of maternal toxicity.	10

[a] Rating scale identical to that used by EPA in the RQ adjustment process, as described in US EPA (1983).

Source: USEPA. 1986 (a).

TOXICITY PROFILE FOR: CADMIUM

BACKGROUND INFORMATION

Cadmium is a soft metal, and is found naturally occurring in zinc ores. This element often serves as a constituent of easily feasible alloys, amalgam in dentistry, electrodes for cadmium vapor lamp, batteries, color pigment, electroplating and photometry of ultraviolet rays (Merck. 1983).

Cases of acute industrial cadmium poisoning date as far back as the 1920's. The first definite reports of chronic effects due to industrial cadmium exposure date to the late 1940's. It was not until the 1960's that health effects were noted due to cadmium associated with environmental pollution, when the Itai-Itai disease complex in Japan was linked to rice paddy contamination by smelter wastes (USEPA. 1980a).

The population, in general, is exposed to cadmium through drinking water and food. For the vast majority of the U.S. population, ambient air is not a significant source of cadmium exposure (USEPA. 1980a). A major non-occupational source of cadmium exposure is derived from cigarettes (Klaassen. 1986).

TRANSPORT & FATE

Cadmium reaches surface water in municipal effluents, and effluents from pigment, plastics, alloys and other manufacture. Fallout from air is also a source of cadmium in water (USEPA. 1980a).

Cadmium is relatively mobile in water, compared with other metals, and may be transported as hydrated cations or as organic or inorganic complexes. In saltwater (typical salinity) the number of probable cadmium species is reduced to a few, with cadmium chloride complexes likely predominant (USEPA. 1980a).

Cadmium is strongly adsorbed to clays, muds, humic and organic materials. In polluted waters, cadmium complexing with organic materials is an important fate/transport process. Sorption processes account for removal of dissolved cadmium to sediments (USEPA. 1980a).

Cadmium does bioaccumulate in aquatic organisms and evidently is eliminated slowly. A high degree of variability exists among the BCFs reported for saltwater organisms. Fish and most shellfish bioconcentration factors were generally lower than the uptake factors for bivalves examined. The latter organisms however, are noted as not reaching a steady-state with water concentrations (USEPA. 1980a). The visceral meat of terrestrial organisms (liver, kidney, pancreas) are noted as organs that bioconcentrate cadmium (USEPA. 1980a). Lobster hepatopancreas (analogous structure) in this study had higher residues of cadmium than muscle tissue.

HEALTH EFFECTS

The major non-occupational routes of human exposure to cadmium are through food and tobacco smoke. It is estimated that approximately 5% of cadmium is absorbed by the human gastro-intestinal tract. This is less efficient than uptake across pulmonary membranes.

The major effects of long-term oral exposure to cadmium in humans include: increased proteinuria and renal dysfunction, which results in kidney stone formation and mineral metabolism disturbances (USEPA. 1984a). The US EPA (1980) estimated a Lowest Observed Effect Level (LOEL) of 228 µg Cd/day, based upon the human dietary intake of contaminated rice from areas of Japan in which itai-itai disease is prevalent. Since chronic renal dysfunction occurs approximately at this intake level, the kidneys are the critically affected organ (USEPA. 1984a).

This element has been demonstrated to be teratogenic and can reduce fertility, following intravenous, intraperitoneal, and subcutaneous administration in rats (USEPA. 1984a).

The calculated reference dose for cadmium is 2.9×10^{-4} (mg/kg/day) (USEPA. 1984a).

Based on exposure to cadmium by inhalation, cadmium has been classified as a Group B1, Probable Human Carcinogen. There is no conclusive evidence that cadmium is carcinogenic following oral exposures.

TOXICITY PROFILE FOR: CHROMIUM

BACKGROUND INFORMATION

Chromium is a metal that exists in four naturally occurring isotopes (Merck. 1983). It is a relatively rare element which occurs naturally in the earth's crust.

Among the uses of Chromium VI are the manufacture of chrome-steel or chrome-nickel-steel alloys (stainless steel). Chromium salts are also contained in paints and pigments, and are utilized in the plating and leather tanning industry (USEPA. 1987d).

The adverse effects on skin of high level exposure to chromium in industrial exposure have been known for a century (USEPA. 1980b). The known harmful effects of chromium have been predominantly associated with exposure to the hexavelent state (Chromium VI) of this element (Klaassen. 1986).

TRANSPORT & FATE

Although chromium is widely distributed, it is rarely found in significant concentrations in natural waters or air in non-urban areas. Much of the chromium detected in air and water is presumably derived from industrial processes. Chromium may enter waterbodies in discharges or as fallout from airborne sources (USEPA. 1980b).

The trivalent (CrIII) and hexavalent (CrVI) forms of chromium are the most environmentally and biologically significant forms. Hexavalent chromium (more widely used in industry) is very soluble in water as a component of a complex anion. These are readily reduced to the more insoluble trivalent chromium compound sulfur dioxide or organic reducing matter (USEPA. 1980b). Chromium III only slowly oxidizes to Chromium VI. The hexavalent form is relatively more stable in neutral or alkaline solutions

and traces can be found. Trivalent Chromium has low solubility in saltwater, and tends to precipitate out, being associated with the sediments (USEPA. 1980b).

The evidence for bioconcentration of Chromium VI in fish muscle appears to be at or below 1.0. Bivalves, on the other hand, apparently bioconcentrate CrIII and/or CrVI. Thus shellfish consumption may become a source of chromium in human consumption.

HEALTH EFFECTS

Chromium plays a role in human nutrition and is generally considered essential in small amounts. Chromium levels found to have adverse effects on humans or other test organisms are several orders of magnitude higher than those recommended as safe in consumed sources, including drinking water (USEPA. 1980b).

Hexavalent chromium is more toxic than trivalent chromium and more readily taken up by cells than trivalent chromium. Adsorption of chromium from the gut is generally poor. Once inside cells, chromium VI is likely reduced to the trivalent state (USEPA. 1980b).

The major acute effects from ingested chromium include renal tubular necrosis (Klaassen. 1986). Chromium VI (chromic acid and its salts) have a corrosive action on the skin and mucous membranes.

Mutagenic effects by chromium have been documented. It has been suggested that the chromium mutagenesis causative agent is trivalent chromium bound to genetic material after its reduction from the hexavalent form (Klaassen. 1986).

There is inadequate evidence of chromium carcinogenicity by oral exposure and it has not been classified as a carcinogen by this exposure route. Chromium carcinogenicity has only been shown

through the occupational inhalation of chromium VI, where its effects are observed in the human respiratory passages and in the lungs (USEPA. 1987d).

TOXICITY PROFILE FOR: COPPER

BACKGROUND INFORMATION

Copper is a soft heavy metal. Elemented copper is very reactive with organic or mineral acids that contain or act as oxidizing agents. Copper has two oxidation states, the cuprous and cupric. The cuprous state is unstable in aerated water over the pH range of most natural waters (6 to 8) and will oxidize to the cupric state (USEPA. 1980c).

Many copper containing compounds are used as fungicides. Medicinally, copper sulfate is used as an emetic (Klaassen. 1986).

TRANSPORT & FATE

Copper is ubiquitous in rocks and minerals of the earth's crust and these sources are responsible for background levels of copper in water typically below 20 µg/l. Higher levels are likely from corrosion of brass/copper pipe, effluent and fallout from industry and sewage treatment plant effluents (USEPA. 1980c).

Some copper compounds are highly soluble in water (copper sulfate, chloride, nitrate), while others may precipitate out of solution more readily (basic copper carbonate, cupric hydroxide, oxide, or sulfide). Cupric ions are adsorbed by clays, sediments and organic particles, or may form complexes with a number of inorganic compounds (USEPA. 1980c).

The levels of copper in water are dependent upon water chemistry, including pH, temperature, alkalinity and the concentration of bicarbonate, sulfide and organic ligands. Acid conditions and low concentrations of complexing agents favor ionic copper solubility. Alkalinity and complexing agents reduce levels of

cupric ions in water. Many of the various copper complexes and precipitates appear to be largely non-toxic (USEPA. 1980c).

Copper is an essential element, especially in plant and crustaceans. Bivalves bioconcentrate copper to the highest levels but the highest observed are not known harmful to man (USEPA. 1980c).

HEALTH EFFECTS

Copper is an essential element in humans. There are two inherited diseases that represent abnormal copper metabolism. In Menke's disease there is reduced absorption of copper, resulting in symptoms resembling copper deficiency. In Wilson's disease, copper accumulates in the liver and brain, resulting in copper toxicosis (USEPA. 1980c).

Copper has toxic effects at high dose levels and is an essential element in lower levels. Excessive ingestion of copper salts (i.e. copper sulfate) may result in acute poisoning and eventually produce death. Symptoms such as vomiting, hypotension, coma and jaundice are particular to acute copper poisoning. The use of copper containing dialysis equipment and burn treatment with copper compounds has also produced hemolytic anemia (Klaassen. 1986).

No evidence of human teratogenesis associated with oral exposure has been reported by the US EPA. Data regarding the carcinogenicity of copper were not sufficient to rate this element adequately, therefore, it was categorized by EPA's Carcinogen Assessment Group as a group D, Not Classified substance.

TOXICITY PROFILE FOR: LEAD

BACKGROUND INFORMATION

Lead is a ubiquitous soft gray acid-soluble metal that exists in three oxidation states. Lead is widely used in industry because of its high density, softness, resistance to corrosion and radiation. It has often been used in electroplating, metallurgy, the manufacture of construction materials, radiation protection devices, plastics, and electronics equipment, as a gasoline additive and as a pigment in paint. (USEPA. 1980d).

Unlike many contaminants where exposures may be related to a specific route or situation, substantial "background" lead exposure occurs, primarily through food and water. Lead gasoline combustion has also been a major source of environmental exposure (USEPA. 1984g).

TRANSPORT & FATE

Lead reaches the aquatic environment through precipitation, fallout of lead dust, sheet runoff, and both industrial and municipal waste water discharges. (USEPA. 1980d).

Inorganic lead compounds are most stable in the +2 valence state, while the organic lead compounds are most stable in the +4 valence state. Neither metallic lead nor the common lead minerals are considered soluble in water. They can be solubilized by some acids. However, some of the lead compounds produced in industry are considered water soluble. Natural lead compounds typically become adsorbed by ferric hydroxide or combined with carbonate or sulfate ions. These are insoluble in water. The solubility of lead compounds in natural waters depends heavily on pH. It ranges from 10,000,000 µg/liter at pH of 5.5 to 1 µg/l at pH of 9 (USEPA. 1980d).

A few available studies have shown that lead can be bioaccumulated. The range of bioconcentration factors for species examined was 17.5 to 2,570. The species were largely bivalves. No saltwater fish species were examined in these studies. (USEPA. 1980d).

HEALTH EFFECTS

Approximately 8% of the lead ingested by adults is absorbed by the gastrointestinal tract (USEPA. 1984g). Age has a major influence on the extent of lead absorption. It has been observed that absorption of lead in infant rats was considerably greater than in adults. Similar results have been seen in humans (USEPA. 1984g). Lead is a cumulative poison which most directly affects the blood cells (Merck. 1983). Lead tends to produce a brittleness within the red blood cells thus causing intensified fragility of the tissue. This phenomenon results in a faster destruction of cells, leading to anemic symptoms (USEPA. 1984g).

Neurological effects are most common in those children having direct contact and exposure to lead contents in paint films. Chronic exposures to low levels of lead can cause subtle learning disabilities in children. Among the neurological effects caused by lead poisoning in children are alterations in cognitive functioning, inappropriate social behavior and the inability to focus attention (Klaassen. 1986). IQ decrements and EEG brain wave pattern alterations were observed among those children exposed to lead, with an average blood lead level ranging from 30-50 µg/dl (USEPA. 1984g). They also showed weight loss, weakness and anemia (Merck. 1983).

In a multigenerational study of rats, histological changes in kidney were noted as a sensitive indicator of liver toxicity. Data concerning the carcinogenic potential of lead to humans after oral exposure proved inconclusive (Clement. 1985). There

is some animal evidence that several lead salts are carcinogenic. Lead and lead compounds were classified by the US EPA as a Group C, Possible Human Carcinogen.

TOXICITY PROFILE FOR: MERCURY

BACKGROUND INFORMATION

Mercury is a silver-white metal that exists in three oxidation states: elemental, mercurous and mercuric. It can be part of both organic and inorganic compounds. Mercurous salts are much less soluble than mercuric salts and are much less toxic than the mercuric forms. (USEPA. 1980e, 1984i).

Natural degassing of the earth's crust releases mercury, although mining, smelting and industrial discharge have contributed greatly to the environmental pollution from mercury (Klaassen. 1986).

Mercury is used in the manufacture of mercury and incandescent lamps, in amalgams with copper, tin, silver and gold, in photography, paints and as a fungicide (Merck. 1983).

TRANSPORT & FATE

The atmosphere is the major pathway for distribution of mercury. The main input is from natural sources, although input from industry is significant. Mercury is removed from the atmosphere mainly through precipitation. Mercury is also added to aquatic systems through runoff and discharges (USEPA. 1980e).

At one time elemental mercury was considered relatively inert, and was thought to settle to the bottom of a water body and remain inoccuous. It is now known that elemental mercury can be oxidized in sediments to divalent mercury. Both aerobic and anaerobic bacteria can methylate divalent mercury in sediments, with the reverse reaction occurring very slowly. Evidently, the slime coat and intestines of fish can methylate mercury. Methyl mercury is both directly toxic and bioaccumulates. It is more

toxic to mammals than inorganic mercury. Uptake of methyl mercury is extremely rapid. These compounds rapidly cross cell membranes and bind to ligands in tissue - importantly, in muscle tissue (the part of fish consumed by man). Depuration by excretion evidently requires demethylation, a slow process. This is evidently responsible for mercury's biological half-life of 2-3 years and high bioconcentration (up to 40,000X reported for oyster) (USEPA. 1980e).

HEALTH EFFECTS

The main sources of human mercury exposure are methylmercury compounds in the food supply and mercury vapor in the atmosphere of occupational settings.

Metallic mercury (inorganic form) appears to be poorly absorbed from the GI tract as demonstrated by a study in which animals who ingested gram quantities of mercury only absorbed 0.01 percent of the element. Methylmercury (alkyl form of mercury), however, was essentially completely absorbed in volunteers who consumed tuna contaminated with the compound (USEPA. 1984i).

After oral ingestion of inorganic mercury and mercuric salts, microscopic evaluation of the kidneys in exposed rats was performed and showed various degrees of damage to the proximal convulated tubules (PCT) and the glomeruli. Other portions of the tubule were affected in later stages (USEPA. 1984i).

The acute and chronic effects of methylmercury (an alkyl mercury) have been observed in the central nervous system in poisoning incidents, including the well-documented case of seafood contamination in residents of the area around Minimata Bay, Japan. Effects such as visual and hearing impairment, ataxia and loss of sensation in the extremities and around the mouth have been recorded in man and seem related to cortical neuron destruction (USEPA. 1984i).

Data regarding teratogenicity could not be located for inorganic mercury, however, several investigators have reported embryotoxic and teratogenic effects in animals treated with methylmercury (alkyl mercury) (USEPA. 1984i). Neurological defects were the most common effect noted but an increased frequency of cleft palate in mice was also documented at doses of 0.1 mg/kg/day of methylmercury. In humans, brain damage has been reported in incidents of methylmercury poisoning (USEPA. 1984i).

TOXICITY PROFILE FOR: CHLORDANE

BACKGROUND INFORMATION

Chlordane is a complex mixture that includes two isomers of chlordane, heptachlor, and two isomers of nonachlor (Clement. 1985). This compound has a high chlorine content ranging between 64-67% (Merck. 1983).

Chlordane's solubility ranges from 0.56 to 1.85 mg/liter at 25 C° and is miscible in aliphatic and aromatic solvents (Clement. 1985). Although relatively insoluble in water, this compound loses chlorine content in the presence of alkaline reagents. With the exception of its use through subsurface ground insertion (as a pesticide for termite control and dipping of roots or tops of non-food plants) the USEPA has cancelled registrations of pesticides which contain this toxic compound (Merck. 1983). It previously served as an agricultural home & garden pesticide or insecticide (USEPA. 1987g).

TRANSPORT & FATE

Atmospheric transport of vapors and contaminated dust particles from soil application sites can occur.

Chlordane, however, is a compound with a high resistance to chemical and biological degradation making it very persistent in the environment. Chlordane is somewhat volatile in clear water, and this may be a loss process. Adsorption to organic particles in water is likely. Sorption to sediments is also a likely important mechanism for removal of chlordane from the water column. Residue concentrations in sediment are often much higher than in the water. (Clement. 1985).

Chlordane degradation to photoisomers, (i.e. photo-cis-chlordane) occurs under natural environmental conditions. These can be even more toxic to certain animals and can bioaccumulate to a much higher degree (USEPA. 1987).

Chlordane accumulates in tissues of aquatic organisms to levels higher than in the water. Bioconcentration factors thousands of times greater than water concentrations have been observed in a wide variety of aquatic organisms. (Clement. 1985).

HEALTH EFFECTS

Chlordane has been found to be poisonous to humans by ingestion, inhalation, intravenous and percutaneous absorption. Chlordane has been determined to be a CNS stimulant whose exact mode of action, although unknown, may involve some microsomal enzyme stimulation (Sax. 1987).

The fatal chlordane dose to humans has been estimated to range from 6 to 60 grams (.2 to 2 ounces) (Sax. 1987). Low oral chlordane doses showed severe chronic fatty degeneration of the liver. This phenomenon is corroborated by the results of numerous laboratory studies in which chlordane exposed animals show degenerative changes in the liver and kidney tubules (Sax. 1987). Chlordane is associated also with reproductive and metabolic disorders as observed in exposed laboratory mice (Clement. 1985).

The reference dose for chlordane has been determined to be 5×10^{-5} mg/kg/day based on a 1983 study, where the LOEL was 1 ppm in the diet for chlordane exposed rats. The critical effect was liver necrosis (USEPA. 1987c).

Several oral cancer bioassays have been conducted. Data indicate increased incidence in hepatocellular carcinomas in chlordane

exposed mice and rats. From these studies, a human carcinogenic potency risk factor of 1.3 (mg/kg/day)-1 was computed (USEPA. 1987c). Chlordane was categorized by EPA's Carcinogen Assessment Group as a B2 group compound, Probable Human Carcinogen (USEPA. 1986c).

TOXICITY PROFILE FOR: DDT

BACKGROUND INFORMATION

DDT is a colorless crystal or a white to slightly off-white powder and is odorless or with a slightly aromatic odor. Technical DDT (Dichlorodiphenyltrichloroethane) is generally a mixture of p,p'-DDT, o,p'-DDT, p,p'-DDD, and traces of other materials. Metabolites of DDT include p,p'-DDE and o,p'-DDD. DDT isomers and metabolites are often found together and have similar properties. (Clement. 1985).

DDT is the best known of all the synthetic insecticides. This compound was synthesized in 1874, albeit it wasn't until 1939 that its insecticidal effectiveness was discovered and later patented in 1942. During World War II, DDT was directly applied to humans for the control of lice and other insects. It was one of the most widely used agricultural insecticides in the United States and other countries from 1946 to 1972 (Klaassen. 1986).

TRANSPORT & FATE

Due to its high molecular stability, DDT, along with all its metabolites, is very persistent in the environment. DDT's primary transport from application sites was probably volatilization from soil and water. Isomers of DDT, however, are most often transported via sorption on sediments and bioaccumulation (Clement. 1985). This compound's half-life in water has been determined to range from 56 to 110 years in lake water, and from 3-15 years in soil (Sax. 1987).

DDT is unusually stable in the environment due to its very low solubility in water and its resistance to destruction by light and oxidation. (Merck. 1983).

Bioaccumulation of DDT is well documented, and is a particularly important fate process for this compound in aquatic systems. Analysis of environmental samples indicate that direct uptake, sorption to biota, and biomagnification in food chains result from DDT contamination (USEPA. 1984e).

HEALTH EFFECTS

While DDT is classified as a neuropoison, no unequivocal reports of fatal human poisoning have been recorded despite widespread use of the substance for 30 to 40 years (Klaassen. 1986). A dose of 200 mg/kg of DDT has been determined to be highly dangerous though not fatal to man (Sax. 1987). Chronic exposures to DDT, DDD and DDE in humans lead to accumulation of the chemical in fatty tissues. DDT's location of primary toxic action is the sensory, motor nerve fibers and the motor cortex (Klaassen. 1986).

Most toxicological data are based on oral exposures. Acute oral exposures can lead to symptoms of burning or prickling of the tongue, lips and face, apprehension, irritability, dizziness and tremors (Klaassen. 1986). Chronic oral exposures resulted in liver lesions at all doses tested, the lowest of which was 10 ppm in food or 0.5 mg/kg/day. Additional animal studies showed increased incidence of tumors and increased mortality of offspring in a six generation study with an exposure of 100 ppm (13 mg/kg/day). Oral exposures of 2.5 mg/kg/day of DDT ingested by pregnant mice proved embryotoxic and fetotoxic (USEPA. 1984e). DDT has consistently caused a decrease in the reproductive capacity of organisms tested.

DDT and all its metabolites are compounds with a capacity to bioconcentrate, typically in the adipose issues of most animals. Toxic doses produce vomiting, muscle weakness, disturbance of equilibrium, and finally chronic or asphyxial

convulsions, followed by death from respiratory failure or ventribular fibrillation (Clayton. 1981). The RfD of 5.0×10^{-4} mg/kg/day was derived from a study of rats fed commercial grade DDT, where hepatocellular hypertrophy were observed at some doses, and a NOEL was shown to be 0.05 mg/kg/day (USEPA. 1987h).

There is evidence of carcinogenicity in animals with exposures to DDT. Exposures to DDT and its metabolites have lead to liver tumors in mice (USEPA. 1984e). Exposures to DDT have also shown to develop hepatomas in rats and lymphomas and lung cancers in mice. DDT is classified as a Group B2, Probable Human Carcinogen by the US EPA (USEPA. 1986c). Results from six animal studies were used to develop a q_1^*, carcinogenic potency value of 0.34 $(mg/kg/day)^{-1}$ (USEPA. 1984e).

TOXICITY PROFILE FOR: HEXACHLOROBENZENE (HCB)

BACKGROUND INFORMATION

HCB is an intermediate in dye manufacture and issued as a wood preservative. HCB is a very stable, unreactive compound that when exposed to heat emits highly toxic chlorides (Sax. 1987). In its physical state, HCB consists of white needles or monoclinic prisms, and is insoluble in water (Merck. 1983). Since 1978, HCB is no longer manufactured in the U.S. (Klaassen. 1986).

TRANSPORT & FATE

Although a half-life value cannot be determined, HCB's detection in remote areas may suggest that it could be a long one, due to evidence of long distance transport (USEPA. 1984f). Aerial dispersion of this compound at HCB manufacturing plants is the major entry pathway of this compound into the environment (Clayton. 1981). Rainout and dry deposition are effective mechanisms for the atmospheric removal of HCB and consequent entry into the aquatic environment (USEPA. 1984f). Photodecomposition is extremely slow and rarely observed. Excessively high temperatures will destroy this compound. An aromatic hydrocarbon, HCB degrades very slowly and is persistent in the environment. It is a hydrophilic compound and as such is expected to bioaccumulate in aquatic organisms. Depuration occurs over time and HCB levels can decrease in biological organisms, once removed from the exposure sources.

HEALTH EFFECTS

A classified fungicide, hexachlorobenzene, produced numerous cases of acquired porphyria cutanea tarda, (PCT), which is characterized by symptoms such as pigmentary changes, deep

scarring, hepatomegaly, permanent loss of hair, skin atrophy and death. Accidental exposure was traced to the consumption of feed grains treated with this compound. In this case, ninety-five percent of the infants of the mothers that had PCT died within a year of birth and others acquired the disorder known as "pink sore" from their HCB affected mother. The presence of HCB in the mother's milk suggested that "pink sore" was a resulting effect due to lactation as an exposure route rather than HCB placental transfer (Klaassen. 1986; USEPA. 1984f). Teratogenic and reproductive effects, however, have been found to be minimal in experimental animals (USEPA. 1984f).

Hexachlorobenzene has been demonstrated to be carcinogenic in rodents (rats, mice, and hamsters), following oral exposure. Data for humans is not available at this point (USEPA. 1984f). A carcinogenic potency value of 1.688 (mg/kg/day)-1 was derived by the US EPA in 1980 based on the incidence of hepatomas in male Syrian Golden hamsters. Hexachlorobenze has been categorized as a group B2, Probable Human Carcinogen, by the US EPA Carcinogen Assessment Group (USEPA. 1986c).

TOXICITY PROFILE FOR: HEXACHLOROCYCLOHEXANE (HCH)

BACKGROUND INFORMATION

HCH is the common name for the family of isomers of hexachlorocyclohexane. Technical HCH contains approximately 64% alpha, 10% beta, 13% gamma, 9% delta and 1% epsilon isomers of 1,2,3,4,5,6-hexachlorocyclohexane (Sax. 1987). HCHs are the chlorination products of benzene. All the isomers are crystals with melting points ranging from 112 to 309 degrees Celsius (Merck. 1983). These compounds exhibit very low volatility and are slightly soluble in water.

Technical hexachlorocyclohexane is used as an insecticide for the control of insects on cotton, fruits and vegetables. Lindane, the gamma isomer, is more often used in insect control on both livestock and pets (Clayton. 1981). Lindane is presently imported into the U.S. and according to a 1970 import level report, less than one million pounds were imported in that year (USEPA. 1984h).

TRANSPORT & FATE

Adsorption to sediments seems to be a major transport mechanism in the aquatic environment (USEPA. 1984h). A low mobility in soil has been recorded for lindane, although surface runoff could represent a transport mechanism for surface water. Based on the saturation vapor pressure data, lindane may not be absorbed onto particulate matter in the air. Nevertheless, in this media, rainout has been the demonstrated removal mechanism.

HEALTH EFFECTS

The alpha and gamma (lindane) HCH isomers have been recorded as convulsant poisons, while the beta and gamma isomers are central

nervous system depressant. The epsilon isomer appears to have no observable effects on our system (Klaassen. 1986). Toxicity studies have been complicated by the fact that each of the isomers has its own characteristic toxic effect(s) (USEPA. 1984h).

Lindane, along with the other four HCH isomers, has been associated with aplastic anemia and paramyeloblastic leukemia. A study in which technical grade HCH was administered through a diet to Wistar rats demonstrated numerous physiological changes such as depression, liver increase, fatty accumulation and kidney degeneration (USEPA. 1984h). Lindane intake affects stimulation of the central nervous system, causing violent convulsions and is generally followed by either death or slow recovery. Elevated body temperatures and pulmonary edema have been reported in children (USEPA. 1986b).

In a study where rats were administered lindane (99.85%) in a diet, lindane exposure related effects were not noted on mortality, hematology, clinical chemistry or urinanalysis (USEPA. 1986b). Rats receiving 20 and 100 ppm lindane were observed to have a higher incidence of livehypertrophy, interstitial nephritis and kidney tubular degeneration. Since these effects were mild and rare at a level of 4 ppm, this represents a No Observable Adverse Effect Level (NOAEL) (USEPA. 1986b). An oral reference dose value for a-HCH and g-HCH (lindane) has been determined to be 3×10^{-4} mg/kg/day.

The teratogenic and other fetotoxic effects on female rats treated with lindane for four months resulted in: (a) disturbed estrous cycles, (b) lowered embryonic viability, (c) reduced fertility and (d) delayed sexual maturation at the 0.5 mg/kg bw/day level (USEPA. 1984h). These effects were not observed at a 0.05 mg/kg bw/day level.

Lindane appears to fall between a Group B2 and Group C for its carcinogenic risk category (USEPA. 1986c) while alpha HCH is considered a Group B2 carcinogen. The carcinogenic potency factor for the alpha isomer is 6.3 $(mg/kg/day)^{-1}$ based on increased incidence of liver tumors in mice and rats, while it is 1.33 for the gamma isomer (USEPA. 1987f).

TOXICITY PROFILE FOR: POLYCHLORINATED BIPHENYLS (PCBs)

BACKGROUND INFORMATION

Polychlorinated Biphenyls are a family of the chlorinated aromatic compounds. The physical, chemical and biological characteristics of these chemicals vary widely, depending on the number of chlorine atoms substituted in the aromatic ring(s) (Klaassen. 1986). The Aroclors are characterized by an exclusive four digit number. The first two digits indicate whether the compound contains biphenyls (denoted by a 12) triphenyls (by a 54) or both compounds (25,44), while the last two digits state the weight percent of chlorine in the compound (Merck. 1983). The chlorine content ranges from 12 to 68 percent. In general, all PCBs have very low water solubilities (0.003-0.6 mg/l) and vapor pressures 10^{-3} to 10^{-5} mm Hg at 20° C (USEPA. 1984k).

Polychlorinated Biphenyls or PCBs were once widely used industrial chemicals. Their high stability contributed to both their commercial usefulness and their subsequent long-term environmental and health effects. PCBs have been commercially available since 1930. PCBs have been used primarily as insulating material in electrical capacitors and transformers, for the insulation of electrical cables and wires, fire retardants, and in heat transfer systems (Clayton. 1981). The manufacture and distribution of PCB-containing products has been banned since 1979 (Klaassen. 1986).

FATE & TRANSPORT

PCB's ubiquitous nature can be attributed to volatilization mechanism followed by adsorption onto dust and fallout (Klaassen. 1986). Lighter PCB species, with fewer chlorine atoms, tend to volatalize more easily.

PCBs are relatively inert and therefore persistent. Adsorption to organic material in sediment is probably a fate mechanism for at least the more heavily chlorinated PCBs. Slow desorption can provide continuous low-level contamination. These less heavy PCBs can be biodegraded by some soil microorganisms. The heavier PCBs are not measurably biodegraded, but can be photodegraded by ultraviolet light at a very slow rate. (Clement. 1985). PCBs are bioaccumulated and biomagnified in the aquatic environment.

HEALTH EFFECTS

In 1968, accidental ingestion of PCBs occurred in Yusho, Japan, as a result of rice bran oil contamination with Kanechlor-400, a PCB product used as a heat transfer agent (USEPA. 1984). This incident known as Yusho poisoning, affected approximately 1,000 persons, altering their dermal and respiratory systems. Palmar sweating and muscular weakness were also common complaints. By 1979, 31 Yusho patients had already died (USEPA. 1984k) from causes such as malignant neoplasms, stomach and liver cancers, and malignant lymphomas.

Cancer caused by Kanechlor-500 has been demonstrated in laboratory mice, while Aroclor 1260 has also been shown to be carcinogenic in rats (USEPA. 1984k). The reference dose of $1x10^{-4}$ mg/kg/day is based on a study of rhesus monkeys where exposures to Aroclor 1016 in the diet during mating and gestation resulted in smaller offsprings in the study animals than those of the control group (USEPA. 1987h). Studies have shown an increased number of different liver cancers such as adenocarcinomas, trabecular carcinomas and neoplastic nodules in rats fed PCBs. No significant teratogenic effects were recorded but fetotoxicity was evident (USEPA. 1984k).

PCBs have been classified as a group B2, Probable Human Carcinogen compound. A draft document is available from the U.S.

Public Health Services (Nov. 1987), which designates a carcinogenic potency factor of 7.7 $(mg/kg/day)^{-1}$ for PCBs based on the carcinogenicity of Aroclor 1260 (USPHS. 1987). Previous to this newly developed CPF the generally accepted value of 4.34 $(mg/kg/day)^{-1}$ was used. A congener specific analysis of Quincy Bay biota samples was conducted by USEPA Exposure Assessment Group where it was concluded that based on the thirteen congeners measured, the mixture of PCBs in the Quincy Bay seafood resembles Aroclor 1254 more closely than Aroclor 1260 or 1242 (USEPA. 1988b). Additional work by the US EPA Carcinogen Assessment Group indicates that the plausible upper bound cancer potency factor for Aroclor 1254 is 2.6 $(mg/kg/day)^{-1}$. It is based on a National Cancer Institute study in which statically significant dose related increases in liver modules, benign tumors, and malignant tumors, were seen in rats fed a diet containing Aroclor 1254 (USEPA. 1988c). The CPF of 2.6 $(mg/kg/day)^{-1}$ was used in the evaluation of risk for this study. This CPF and the two others mentioned previously are not considered substantially different due to the uncertainty associated with the experimental data from which the CPF value of 2.6 $(mg/kg/day)^{-1}$ was derived (USEPA. 1988c).

TOXICITY PROFILE FOR: POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

BACKGROUND INFORMATION

PAHs are chemicals which consist of two or more fused benzene rings and occur in a variety of commercial products such as soot, coal, tar, tobacco smoke, cutting oils and petroleum (Klaassen. 1986). These compounds form as a result of breakdown of hydrocarbon compounds when exposed to ultraviolet radiation or by incomplete combustion of organic compounds with insufficient oxygen availability.

TRANSPORT & FATE

Little information is available on the range of compounds that are classified as PAHs, however, much is inferred from the more researched benzo(a) pyrene. Atmospheric fallout, surface runoff are likely existing sources to aquatic environments and adsorption on to sediments is a probable transport mechanism. (Clement. 1985).

PAHs are relatively insoluble in water, but the dissolved portion is believed to undergo direct photolysis. Some may also be oxidized by chlorine and ozone. (Clement. 1985).

PAHs are bioaccumulated, although rapidly metabolized and eliminated by most organisms (not shellfish). Biodegradation is believed to occur more slowly in water than in soil, but to more significant in systems chronically affected by PAH contamination. (Clement. 1985).

HEALTH EFFECTS

Due to the high lipophylic nature of PAHs, they are readily absorbed in the gastrointestinal tract of animals. In a study

where rats were administered B[a]P contained in a starch solution, 50% of the compound was absorbed. There is often no sign of toxicity until the dose is high enough to produce a high tumor incidence thus carcinogenicity dominates health effect considerations (Clement. 1985). When benzo [a] pyrene is administered to the skin of mice quick carcinoma formation results. Subcutaneous injection produces sarcomas in rats and mice. Oral administration of some PAHs to rhesus monkeys and other primates has so far not yielded tumors (Klaassen. 1986).

Benzo [a] pyrene was administered to study mice through diet at concentrations ranging from 1 to 250 ppm and stomach tumors (papillomas and carcinomas) were reported. Control mice did not have similar tumors (USEPA. 1984j). At increased concentrations ranging from 250 to 1000 ppm, B[a]P produced a higher incidence of stomach tumors, as well as lung adenoma and leukemia in the studied mice (USEPA. 1984j).

The US EPA used incidences of stomach tumors in B[a]P exposed mice in a 1957 study to derive a carcinogenic potency factor of 11.53 $(mg/kg/day)^{-1}$ for oral intake. This CPF is issued for all PAHs using the conservative default assumption that with the absence of sufficent data to the contrary all PAHs are carcinogenic and equal in potency to B[a]P.

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d)	Copper.	PB86-134368
e)	DDT.	PB86-134376
f)	Hexachlorobenzene.	PB86-134285
g)	Lead.	PB86-134665
h)	Lindane.	PB86-134673
i)	Mercury.	PB86-134533
j)	Polycyclic Aromatic Hydrocarbons.	PB86-134244
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d)	Chromium VI.	CAS No.: 7440-47-3
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Appendix B

Risk Calculations

TABLE B-1. RISK CHARACTERIZATION FOR A MAXIMALLY EXPOSED INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER IN COMBINATION WITH OTHER SEAFOOD (a) (b)
(using wet weight data)

CHEMICAL IDENTIFIED	FDA LIMITS (ppm)	CPF Carcinogenic Potency Factor (mg/kg/day)-1 Oral	CPF REF	RfD Reference Dose (mg/kg/day)	RfD REF	1987 Data FLOUNDER (Flesh) (ug/g) max	1987 Data FLOUNDER (Flesh) (ug/g) mean	FLOUNDER DOSE (c) (ug/kg/day) max	FLOUNDER DOSE (c) (ug/kg/day) mean	HAZARD RATIO (d) max	HAZARD RATIO (d) mean	UPPER BOUND INCREASED CANCER RISK (e) max	UPPER BOUND INCREASED CANCER RISK (e) mean
ELEMENTS/METALS													
Cadmium		NA		2.90E-04	1	9.00E-03	1.00E-03	1.45E-02	1.61E-03	0.05	0.01	-	-
Chromium		NA		5.00E-03	3	3.77E-01	2.90E-02	6.09E-01	4.68E-02	0.12	0.01	-	-
Copper		NA		3.70E-02	3	2.15E-01	1.09E-01	3.47E-01	1.76E-01	0.01	0.00	-	-
Lead *		NA		1.40E-03	3	4.30E-02	1.50E-02	6.94E-02	2.42E-02	0.05	0.02	-	-
Mercury *		NA		2.00E-03	3	8.60E-02	3.00E-02	1.39E-01	4.84E-02	0.07	0.02	-	-
ORGANIC COMPOUNDS													
Chlordane (total)	0.3	1.3	1	5.00E-05	1	3.00E-02	3.15E-03	4.84E-02	5.09E-03	0.97	0.10	6.3E-05	6.6E-06
PP-DDD	5.0	0.34	3	5.00E-04	3	1.33E-02	1.58E-03	2.14E-02	2.55E-03	0.04	0.01	7.3E-06	8.7E-07
PP-DDE	5.0	0.34	3	5.00E-04	3	1.59E-02	5.19E-03	2.57E-02	8.38E-03	0.05	0.02	8.7E-06	2.8E-06
PP-DDT	5.0	0.34	3	5.00E-04	3	4.97E-03	8.55E-04	8.03E-03	1.38E-03	0.02	0.00	2.7E-06	4.7E-07
Hexachlorobenzene (HCB)		1.69	3	8.00E-04	4	2.52E-04	1.27E-04	4.07E-04	2.05E-04	0.00	0.00	6.9E-07	3.5E-07
Hexachlorocyclohexane (HCH)													
a-HCH		6.3	1	3.00E-04	3	8.93E-04	1.82E-04	1.44E-03	2.94E-04	0.00	0.00	9.1E-06	1.9E-06
g-HCH (Lindane)		1.33	2	3.00E-04	3	1.70E-04	1.56E-04	2.74E-04	2.52E-04	0.00	0.00	3.6E-07	3.3E-07
PAH (f)		11.53	2	NA		2.61E-04	2.45E-04	4.21E-04	3.95E-04	-	-	4.9E-06	4.6E-06
PCBs (g)	2.0	2.6	2	1.00E-04	5	7.43E-01	2.73E-01	1.20E+00	4.41E-01	11.99	4.41	3.1E-03	1.1E-03
												3.2E-03	1.2E-03

FOOTNOTES:

* = Data correspond to inorganic compounds.

NA: Not available

(a): Mean calculated using detection limits for undetected observations. See Appendix Table B-5 for calculations using zero instead of detection limits.

(b): See Table 2 for weight of evidence classification for carcinogens.

(c): Calculated dose= contaminant concentration (ug/g) x 16 grams of fish ingested /day/70 kilogram body weight.

(d): Hazard Ratio= Calculatated dose(ug/kg/day)/[Reference Dose(mg/kg/day)*1000ug/mg].

(e): Increased Upper Bound Cancer Risk= [Calculated dose (ug/kg/day)*0.001mg/ug] * Carcinogenic Potency Factor (mg/kg/day)-1.

(f): Total Polycyclic Aromatic Hydrocarbons.

(g): Total Polychlorinated Biphenyls.

REFERENCES

(1) Integrated Risk Information System Chemical Files.

(2) Health Effects Assessment Documents.

(3) Superfund Public Health Evaluation Manual.

(4) Health Advisories for 25 Organics.

(5) USEPA. 1988a, b and c.

TABLE B-2. RISK CHARACTERIZATION FOR THE MAXIMALLY EXPOSED INDIVIDUAL FROM INGESTION OF QUINCY BAY SOFT SHELL CLAMS IN COMBINATION WITH OTHER SEAFOOD (a) (b)
(using wet weight data)

CHEMICAL IDENTIFIED	FDA LIMITS (ppm)	CPF Carcinogenic Potency Factor (mg/kg/day)-1 Oral	REF	RfD Reference Dose (mg/kg/day)	REF	1987 Data CLAMS (Soft-shell) (ug/g) max	mean	CLAM DOSE (c) (ug/kg/day) max	mean	HAZARD RATIO (d) max	mean	UPPER BOUND INCREASED CANCER RISK (e) max	mean
ELEMENTS/METALS													
Cadmium		NA		2.90E-04	1	2.50E-02	2.10E-02	5.71E-03	4.80E-03	0.02	0.02	-	-
Chromium		NA		5.00E-03	3	2.45E-01	2.06E-01	5.59E-02	4.71E-02	0.01	0.01	-	-
Copper		NA		3.70E-02	3	1.95E+00	1.85E+00	4.46E-01	4.23E-01	0.01	0.01	-	-
Lead *		NA		1.40E-03	3	4.60E-01	4.50E-01	1.05E-01	1.03E-01	0.08	0.07	-	-
Mercury *		NA		2.00E-03	3	2.00E-03	----	4.57E-04	-	0.00	-	-	-
ORGANIC COMPOUNDS													
Chlordane (total)	0.3	1.3	1	5.00E-05	1	3.48E-03	2.88E-03	7.95E-04	6.58E-04	0.02	0.01	1.0E-06	8.6E-07
PP-DDD	5.0	0.34	3	5.00E-04	3	1.42E-03	1.23E-03	3.25E-04	2.81E-04	0.00	0.00	1.1E-07	9.6E-08
PP-DDE	5.0	0.34	3	5.00E-04	3	4.76E-03	4.26E-03	1.09E-03	9.74E-04	0.00	0.00	3.7E-07	3.3E-07
PP-DDT	5.0	0.34	3	5.00E-04	3	3.37E-04	3.03E-04	7.70E-05	6.93E-05	0.00	0.00	2.6E-08	2.4E-08
Hexachlorobenzene (HCB)		1.69	3	8.00E-04	4	1.03E-04	1.02E-04	2.35E-05	2.33E-05	0.00	0.00	4.0E-08	3.9E-08
Hexachlorocyclohexane(HCH)													
a-HCH		6.3	1	3.00E-04	3	1.28E-04	1.19E-04	2.93E-05	2.73E-05	0.00	0.00	1.8E-07	1.7E-07
g-HCH (Lindane)		1.33	2	3.00E-04	3	1.18E-04	1.17E-04	2.70E-05	2.67E-05	0.00	0.00	3.6E-08	3.6E-08
PAH (f)		11.53	2	NA		4.51E-02	4.35E-02	1.03E-02	9.94E-03	-	-	1.2E-04	1.1E-04
PCB (g)	2.0	2.6	2	1.00E-04	5	1.53E-01	1.51E-01	3.49E-02	3.44E-02	0.35	0.34	9.1E-05	8.9E-05
												2.1E-04	2.1E-04

FOOTNOTES:

* = Data correspond to inorganic compounds.

NA: Not available

(a): Mean calculated using detection limits for undetected observations. See Appendix Table B-5 for calculations using zero instead of detection limits.

(b): See Table 2 for weight of evidence classification for carcinogens.

(c): Calculated dose= contaminant concentration (ug/g) x 16 grams of fish ingested /day/70 kilogram body weight.

(d): Hazard Ratio= Calculatated dose(ug/kg/day)/[Reference Dose(mg/kg/day)*1000ug/mg].

(e): Increased Upper Bound Cancer Risk= [Calculated dose (ug/kg/day)*0.001mg/ug] * Carcinogenic Potency Factor (mg/kg/day)-1.

(f): Total Polycyclic Aromatic Hydrocarbons.

(g): Total Polychlorinated Biphenyls.

- : Only one value was available.

REFERENCES

(1) Integrated Risk Information System Chemical Files

(2) Health Effects Assessment Documents

(3) Superfund Public Health Manual.

(4) Health Advisories for 25 Organics.

(5) USEPA. 1988a, b and c.

TABLE B-3. RISK CHARACTERIZATION FOR THE MAXIMALLY EXPOSED INDIVIDUAL FROM INGESTION OF QUINCY BAY LOBSTER TISSUE IN COMBINATION WITH OTHER SEAFOOD (a) (b)
(using wet weight data)

CHEMICAL IDENTIFIED	FDA LIMITS (ppm)	CPF Carcinogenic Potency Factor (mg/kg/day)-1 Oral	REF	RfD Reference Dose (mg/kg/day)	REF	1987 Data LOBSTER (Tissue) (ug/g) max	mean	LOBSTER DOSE (c) (ug/kg/day) max	mean	HAZARD RATIO (d) max	mean	UPPER BOUND INCREASED CANCER RISK (e) max	mean
METALS													
Cadmium		NA		2.90E-04	1	5.00E-03	2.00E-03	2.14E-03	8.57E-04	0.01	0.00	-	-
Chromium		NA		5.00E-03	3	2.60E-01	2.40E-02	1.11E-01	1.03E-02	0.02	0.00	-	-
Copper		NA		3.70E-02	3	6.22E+00	4.06E+00	2.67E+00	1.74E+00	0.07	0.05	-	-
Lead *		NA		1.40E-03	3	2.07E-01	1.69E-01	8.87E-02	7.24E-02	0.06	0.05	-	-
Mercury *		NA		2.00E-03	3	1.68E-01	8.50E-02	7.19E-02	3.64E-02	0.04	0.02	-	-
ORGANIC COMPOUNDS													
Chlordane (total)	0.3	1.3	1	5.00E-05	1	6.07E-04	3.76E-04	2.60E-04	1.61E-04	0.01	0.00	3.4E-07	2.1E-07
PP-DDD	5.0	0.34	3	5.00E-04	3	6.64E-04	5.28E-04	2.85E-04	2.26E-04	0.00	0.00	9.7E-08	7.7E-08
PP-DDE	5.0	0.34	3	5.00E-04	3	7.46E-03	5.03E-03	3.20E-03	2.16E-03	0.01	0.00	1.1E-06	7.3E-07
PP-DDT	5.0	0.34	3	5.00E-04	3	6.12E-04	5.45E-04	2.62E-04	2.34E-04	0.00	0.00	8.9E-08	7.9E-08
Hexachlorobenzene (HCB)		1.69	3	8.00E-04	4	2.19E-04	1.33E-04	9.40E-05	5.70E-05	0.00	0.00	1.6E-07	9.6E-08
Hexachlorocyclohexane (HCH)													
a-HCH		6.3	1	3.00E-04	3	1.83E-04	1.63E-04	7.84E-05	6.99E-05	0.00	0.00	4.9E-07	4.4E-07
g-HCH (lindane)		1.33	2	3.00E-04	3	1.81E-04	1.22E-04	7.76E-05	5.23E-05	0.00	0.00	1.0E-07	7.0E-08
PAH (f)		11.53	2	NA		7.43E-02	5.19E-02	3.18E-02	2.22E-02	-	-	3.7E-04	2.6E-04
PCBs (g)	2.0	2.6	2	1.00E-04	5	3.82E-01	2.37E-01	1.64E-01	1.02E-01	1.64	1.02	4.3E-04	2.6E-04
												8.0E-04	5.2E-04

FOOTNOTES:

* = Data correspond to inorganic compounds.

NA: Not available

(a): Mean calculated using detection limits for undetected observations. See Appendix Table B-5 for calculations using zero instead of detection limits.

(b): See Table 2 for weight of evidence classification for carcinogens.

(c): Calculated dose= contaminant concentration (ug/g) x 16 grams of fish ingested /day/70 kilogram body weight.

(d): Hazard Ratio= Calculatated dose(ug/kg/day)/[Reference Dose(mg/kg/day)*1000ug/mg].

(e): Increased Upper Bound Cancer Risk= [Calculated dose (ug/kg/day)*0.001mg/ug] * Carcinogenic Potency Factor (mg/kg/day)-1.

(f): Total Polycyclic Aromatic Hydrocarbons.

(g): Total Polychlorinated Biphenyls.

REFERENCES

(1) Integrated Risk Information System Chemical Files.

(2) Health Effects Assessment Documents.

(3) Superfund Public Health Manual.

(4) Health Advisories for 25 Organics.

(5) USEPA. 1988a, b and c.

TABLE B-4. RISK CHARACTERIZATION FROM MAXIMALLY EXPOSED INDIVIDUAL INGESTION OF QUINCY BAY LOBSTER HEPATOPANCREAS (a) (b)
(using wet weight data)

CHEMICAL IDENTIFIED	FDA LIMITS (ppm)	CPF Carcinogenic Potency Factor (mg/kg/day)-1 Oral	REF	RfD Reference Dose (mg/kg/day)	REF	1987 Data HEPATOPANCREAS (ug/g) max	mean	HEPATOPANCREAS DOSE (c) (ug/kg/day) max	mean	HAZARD RATIO (d) max	mean	UPPER BOUND INCREASED CANCER RISK (e) max	mean
ELEMENTS/METALS													
Cadmium		NA		2.90E-04	1	2.23E+00	1.31E+00	1.92E-01	1.12E-01	0.66	0.39	-	-
Chromium		NA		5.00E-03	3	2.38E+00	7.20E-01	2.04E-01	6.17E-02	0.04	0.01	-	-
Copper		NA		3.70E-02	3	2.79E+02	1.37E+02	2.39E+01	1.17E+01	0.65	0.32	-	-
Lead *		NA		1.40E-03	3	7.00E-01	3.35E-01	6.00E-02	2.87E-02	0.04	0.02	-	-
Mercury *		NA		2.00E-03	3	1.12E-01	6.50E-02	9.60E-03	5.57E-03	0.00	0.00	-	-
ORGANIC COMPOUNDS													
Chlordane (total)	0.3	1.3	1	5.00E-05	1	2.40E-01	9.75E-02	2.06E-02	8.36E-03	0.41	0.17	2.7E-05	1.1E-05
PP-DDD	5.0	0.34	3	5.00E-04	3	3.12E-01	1.00E-01	2.67E-02	8.57E-03	0.05	0.02	9.1E-06	2.9E-06
PP-DDE	5.0	0.34	3	5.00E-04	3	1.89E+00	1.30E+00	1.62E-01	1.11E-01	0.32	0.22	5.5E-05	3.8E-05
PP-DDT	5.0	0.34	3	5.00E-04	3	7.34E-02	2.95E-02	6.29E-03	2.53E-03	0.01	0.01	2.1E-06	8.6E-07
Hexachlorobenzene (HCB)		1.69	3	8.00E-04	4	1.92E-02	1.37E-02	1.65E-03	1.17E-03	0.00	0.00	2.8E-06	2.0E-06
Hexachlorocyclohexane (HCH)													
a-HCH		6.3	1	3.00E-04	3	3.37E-02	1.84E-02	2.89E-03	1.58E-03	0.01	0.01	1.8E-05	9.9E-06
g-HCH (lindane)		1.33	2	3.00E-04	3	3.18E-03	1.78E-03	2.73E-04	1.53E-04	0.00	0.00	3.6E-07	2.0E-07
PAH (f)		11.53	2	NA		4.78E+00	3.37E+00	4.10E-01	2.89E-01	-	-	4.7E-03	3.3E-03
PCBs (g)	2.0	2.6	2	1.00E-04	5	6.18E+01	4.39E+01	5.30E+00	3.76E+00	52.98	37.63	1.4E-02	9.8E-03
												1.9E-02	1.3E-02

FOOTNOTES:

* = Data correspond to inorganic compounds.

NA: Not available

(a): Mean calculated using detection limits for undetected observations. See Appendix Table B-5 for calculations using zero instead of detection limits.

(b): See Table 2 for weight of evidence classification for carcinogens.

(c): Calculated dose= contaminant concentration (ug/g) x 16 grams of fish ingested /day/70 kilogram body weight.

(d): Hazard Ratio= Calculatated dose(ug/kg/day)/[Reference Dose(mg/kg/day)*1000ug/mg].

(e): Increased Upper Bound Cancer Risk= [Calculated dose (ug/kg/day)*0.001mg/ug] * Carcinogenic Potency Factor (mg/kg/day)-1.

(f): Total Polycyclic Aromatic Hydrocarbons.

(g): Total Polychlorinated Biphenyls.

REFERENCES

(1) Integrated Risk Information System Chemical Files

(2) Health Effects Assessment Documents

(3) Superfund Public Health Manual.

(4) Health Advisories for 25 Organics.

(5) USEPA. 1988a, b and c.

TABLE B-5. RISK CHARACTERIZATION FOR A MAXIMUMLY EXPOSED INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER, CLAMS, LOBSTER AND HEPATOPANCREAS (a)(b)

CHEMICAL IDENTIFIED	FLOUNDER HAZARD RATIO		CLAMS HAZARD RATIO		LOBSTER HAZARD RATIO		HEPATOPANCREAS HAZARD RATIO		TOTAL HAZARD RATIO		FLOUNDER UPPER BOUND INCREASED CANCER RISK		CLAMS UPPER BOUND INCREASED CANCER RISK		LOBSTER UPPER BOUND INCREASED CANCER RISK		HEPATOPANCREAS UPPER BOUND INCREASED CANCER RISK		TOTAL UPPER BOUND INCREASED CANCER RISK	
	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean
ELEMENTS/METALS																				
Cadmium	0.05	0.00	0.02	0.02	0.01	0.00	0.66	0.39	0.74	0.41	-	-	-	-	-	-	-	-	-	-
Chromium	0.12	0.01	0.01	0.01	0.02	0.00	0.04	0.01	0.20	0.03	-	-	-	-	-	-	-	-	-	-
Copper	0.01	0.00	0.01	0.01	0.07	0.05	0.65	0.32	0.74	0.38	-	-	-	-	-	-	-	-	-	-
Lead	0.01	0.00	0.08	0.07	0.06	0.05	0.01	0.00	0.16	0.13	-	-	-	-	-	-	-	-	-	-
Mercury	0.07	0.02	0.00	-	0.03	0.02	0.00	0.00	0.11	0.04	-	-	-	-	-	-	-	-	-	-
ORGANIC COMPOUNDS																				
Chlordane (total)	0.97	0.10	0.02	0.00	0.01	0.00	0.41	0.17	1.40	0.27	6.3E-05	6.6E-06	1.0E-06	8.6E-08	3.4E-07	1.2E-07	2.7E-05	1.1E-05	9.1E-05	1.8E-05
PP DDD	0.04	0.00	0.00	0.00	0.00	0.00	0.05	0.02	0.10	0.02	7.3E-06	7.8E-07	1.1E-07	9.6E-08	2.2E-08	2.4E-09	9.1E-06	2.9E-06	1.7E-05	3.8E-06
PP-DDE	0.05	0.02	0.00	0.00	0.01	0.00	0.32	0.22	0.38	0.25	8.7E-06	2.8E-06	3.7E-07	3.3E-07	1.1E-06	7.3E-07	5.5E-05	3.8E-05	6.5E-05	4.2E-05
PP-DDT	0.02	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.03	0.01	2.7E-06	2.9E-07	2.6E-08	2.4E-08	0.0E+00	0.0E+00	2.1E-06	8.6E-07	4.9E-06	1.2E-06
Hexachlorobenzene (HCB)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6.9E-07	3.2E-07	4.0E-08	3.9E-08	1.6E-07	9.6E-08	2.8E-06	2.0E-06	3.7E-06	2.4E-06
Hexachlorocyclohexane (HCH)																				
α HCH	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	9.1E-06	8.1E-07	1.8E-07	1.7E-07	0.0E+00	0.0E+00	1.8E-05	9.9E-06	2.7E-05	1.1E-05
g HCH (lindane)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	5.3E-08	1.7E-08	3.6E-07	2.0E-07	4.2E-07	2.2E-07
PAH (total)	-	-	-	-	-	-	-	-	-	-	0.0E+00	0.0E+00	1.2E-04	1.1E-04	3.7E-04	2.6E-04	4.7E-03	3.3E-03	5.2E-03	3.7E-03
PCBs (total)	11.99	4.41	0.35	0.34	1.64	1.02	52.98	37.63	66.96	43.40	3.1E-03	1.1E-03	9.1E-05	8.9E-05	4.3E-04	2.6E-04	1.4E-02	9.8E-03	1.7E-02	1.1E-02
											3.2E-03	1.2E-03	2.1E-04	2.0E-04	7.9E-04	5.2E-04	1.9E-02	1.3E-02	2.3E-02	1.5E-02

Ingestion rates:	grams/day
Flounder	113
Clams	16
Lobster	30
Hepatopancreas	6
TOTAL	165

(a) - Mean calculated using zero for undetected observations.

(b) - See Table 2 for weight of evidence classification for carcinogens.

TABLE B-6. RISK CHARACTERIZATION FOR A MAXIMUM EXPOSED INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER ONLY(a)(b)
(using wet weight data)

CHEMICAL IDENTIFIED	FDA LIMITS (ppm)	CPF Carcinogenic Potency facto (mg/kg/day)-1 Oral	REF	RfD Reference Dose (mg/kg/day)	REF	1987 Data FLOUNDER (Flesh) (ug/g) max	mean	FLOUNDER DOSE(c) (ug/kg/day) max	mean	HAZARD RATIO(d) max	mean	INCREASED UPPER BOUND CANCER RISK(e) max	mean
ELEMENTS/METALS													
Cadmium		NA		2.90E-04	1	9.00E-03	3.60E-04	2.12E-02	8.49E-04	0.07	0.00	-	-
Chromium		NA		5.00E-03	3	3.77E-01	1.90E-02	8.89E-01	4.48E-02	0.18	0.01	-	-
Copper		NA		3.70E-02	3	2.15E-01	1.09E-01	5.07E-01	2.57E-01	0.01	0.01	-	-
Lead *		NA		1.40E-03	3	8.00E-03	1.00E-03	1.89E-02	2.36E-03	0.01	0.00	-	-
Mercury *		NA		2.00E-03	3	8.60E-02	3.00E-02	2.03E-01	7.07E-02	0.10	0.04	-	-
ORGANIC COMPOUNDS													
Chlordane (total)	0.3	1.3	1	5.00E-05	1	3.00E-02	3.14E-03	7.07E-02	7.40E-03	1.41	0.15	9.2E-05	9.6E-06
PP-DDD	5.0	0.34	3	5.00E-04	3	1.33E-02	1.43E-03	3.12E-02	3.37E-03	0.06	0.01	1.1E-05	1.1E-06
PP-DDE	5.0	0.34	3	5.00E-04	3	1.59E-02	5.19E-03	3.75E-02	1.22E-02	0.08	0.02	1.3E-05	4.2E-06
PP-DDT	5.0	0.34	3	5.00E-04	3	4.97E-03	5.34E-04	1.17E-02	1.26E-03	0.02	0.00	4.0E-06	4.3E-07
Hexachlorobenzene (HCB)		1.69	3	8.00E-04	4	2.52E-04	1.16E-04	5.94E-04	2.73E-04	0.00	0.00	1.0E-06	4.6E-07
Hexachlorocyclohexane (HCH)													
a-HCH		6.3	1	3.00E-04	3	8.93E-04	7.99E-05	2.11E-03	1.88E-04	0.01	0.00	1.3E-05	1.2E-06
g-HCH (lindane)		1.33	2	3.00E-04	3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00	0.00	0.0E+00	0.0E+00
PAH (f)		11.53	2	NA		0.00E+00	0.00E+00	0.00E+00	0.00E+00	-	-	0.0E+00	0.0E+00
PCBs (g)	2.0	2.6	2	1.00E-04	5	7.43E-01	2.73E-01	1.75E+00	6.44E-01	17.51	6.44	4.6E-03	1.7E-03
												4.7E-03	1.7E-03

FOOTNOTES:

* = Data correspond to inorganic compounds.

NA: Not available

(a): Mean calculated using zero for undetected observations.

(b): See Table 2 for weight of evidence classification for carcinogens.

(c): Calculated dose= contaminant concentration (ug/g) x 165 grams of fish ingested /day/70 kilograms body weight.

(d): Hazard Ratio = Calculated Dose(ug/kg/day)/[Reference Dose(mg/kg/day)*1000ug/mg].

(e): Increased Upper Bound Cancer Risk= [Calculated Dose(ug/kg/day) * 0.001mg/ug] * Carcinogenic Potency Factor (mg/kg/day)-1.

(f): Total Polycyclic Aromatic Hydrocarbons

(g): Total Polychlorinated Biphenyls

REFERENCES

(1) Integrated Risk Information System Chemical Files

(2) Health Effects Assessment Documents

(3) Superfund Public Health Evaluation Manual

(4) Health Advisories for 25 Organics

(5) USEPA. 1988a, b and c.

TABLE B-9. WET WEIGHT CONCENTRATIONS OF ORGANIC COMPOUNDS AND METALS IN AQUATIC ORGANISMS FROM QUINCY BAY 1
(DETECTION LIMITS INCLUDED IN AVERAGES)
(ug/g wet)

SAMPLE NO.	STATION	SAMPLE TYPE	g DRY/g WET RATIO	AROCLOR 1242	AROCLOR 1254	TOTAL PCBs	HCB	A-HCH	G-HCH	A-CHLOR	G-CHLOR	PP-DDE	PP-DDD	PP-DDT	TOTAL PAHs	Cu	Cr	Pd	Cd	Hg
75259	QBMHD	Soft Shell Clam	0.149	8.94E-03	(1.40E-01)	1.49E-01	1.01E-04	1.10E-04	1.16E-04*	9.61E-04	1.32E-03	3.77E-03	1.04E-03	2.70E-04	4.19E-02	--	--	--	--	--
75260	QBMIS	Soft Shell Clam	0.151	(1.99E-02)	1.33E-01	(1.53E-01)	(1.03E-04)	(1.28E-04)	(1.18E-04)*	(1.56E-03)	(1.92E-03)	(4.76E-03)	(1.42E-03)	(3.37E-04)	(4.51E-02)	--	--	--	--	--
		AVERAGE		1.44E-02	1.36E-01	1.51E-01	1.02E-04	1.19E-04	1.17E-04	1.26E-03	1.62E-03	4.26E-03	1.23E-03	3.03E-04	4.35E-02	--	--	--	--	--
75237	QBC	Lobster - Tissue	0.220	1.98E-03*	2.03E-01	2.03E-01	1.06E-04	1.74E-04*	6.60E-05	1.82E-04*	1.39E-04	4.11E-03	6.29E-04*	5.81E-04*	4.55E-02	--	--	--	--	--
75239	QBC	Lobster - Tissue	0.202	1.82E-03*	2.30E-01	2.30E-01	1.33E-04	1.60E-04*	1.58E-04*	1.67E-04*	6.26E-05	4.95E-03	5.78E-05*	5.33E-04*	5.03E-02	--	--	--	--	--
75241	QBC	Lobster - Tissue	0.179	1.61E-03*	1.88E-01	1.88E-01	8.41E-05	1.41E-04*	2.68E-05	1.48E-04*	1.24E-04*	4.87E-03	5.12E-04*	4.73E-04*	3.69E-02	--	--	--	--	--
75219	QBD	Lobster - Tissue	0.227	2.04E-03*	2.61E-01	2.61E-01	1.68E-04	1.79E-04*	8.17E-05	1.09E-04	3.04E-04	6.42E-03	1.52E-04	5.99E-04*	5.86E-02	--	--	--	--	--
75220	QBD	Lobster - Tissue	0.225	2.02E-03*	2.77E-01	2.77E-01	1.73E-04	1.78E-04*	1.76E-04*	1.86E-04*	3.04E-04	5.60E-03	6.43E-04*	5.94E-04*	6.28E-02	--	--	--	--	--
75244	QBE	Lobster - Tissue	0.151	1.36E-03*	2.20E-01	2.20E-01	1.18E-04	1.19E-04*	1.18E-04*	1.89E-04	(4.15E-04)	5.16E-03	4.32E-04*	3.99E-04*	5.75E-02	--	--	--	--	--
75245	QBE	Lobster - Tissue	0.206	1.85E-03*	2.25E-01	2.25E-01	1.17E-04	1.63E-04*	9.27E-05	1.09E-04	2.47E-04	3.93E-03	5.89E-04*	5.44E-04*	5.29E-02	--	--	--	--	--
75212	QBF	Lobster - Tissue	0.220	1.98E-03*	2.08E-01	2.08E-01	1.47E-04	1.74E-04*	1.72E-04*	1.82E-04*	3.52E-04	3.32E-03	6.29E-04*	5.81E-04*	5.90E-02	--	--	--	--	--
75214	QBF	Lobster - Tissue	0.207	1.86E-03*	2.50E-01	2.50E-01	(2.19E-04)	1.64E-04*	1.61E-04*	1.71E-04*	3.02E-04	5.32E-03	5.92E-04*	5.46E-04*	(7.43E-02)	--	--	--	--	--
75223	QBG	Lobster - Tissue	0.209	1.88E-03*	2.72E-01	2.72E-01	1.17E-04	1.65E-04*	1.63E-04*	1.73E-04*	3.01E-04	(7.46E-03)	5.98E-04*	5.52E-04*	5.73E-02	--	--	--	--	--
75225	QBG	Lobster - Tissue	0.232	2.09E-03*	2.88E-01	2.88E-01	1.21E-04	(1.83E-04)*	1.81E-04*	(1.92E-04)*	3.04E-04	7.08E-03	(6.64E-04)*	(6.12E-04)*	4.27E-02	--	--	--	--	--
75228	QBG	Lobster - Tissue	0.188	1.69E-03*	2.14E-01	2.14E-01	1.26E-04	1.49E-04*	8.65E-05	1.56E-04*	1.37E-04	4.19E-03	5.38E-04*	4.96E-04*	4.08E-02	--	--	--	--	--
75249	QBI	Lobster - Tissue	0.211	1.90E-03*	2.22E-01	2.22E-01	1.03E-04	1.67E-04*	6.33E-05	1.75E-04*	8.23E-05	4.26E-03	1.14E-04	5.57E-04*	5.51E-02	--	--	--	--	--
75250	QBI	Lobster - Tissue	0.223	2.01E-03*	1.43E-01	1.43E-01	1.05E-04	1.76E-04*	1.74E-04*	1.85E-04*	8.03E-05	2.83E-03	6.38E-04*	5.89E-04*	3.92E-02	--	--	--	--	--
75230	QBJ	Lobster - Tissue	0.199	1.79E-03*	2.15E-01	2.15E-01	1.21E-04	1.57E-04*	6.96E-05	1.65E-04*	6.37E-05	3.44E-03	5.69E-04*	5.25E-04*	3.58E-02	--	--	--	--	--
75234	QBJ	Lobster - Tissue	0.201	(3.68E-03)*	(3.80E-01)	(3.82E-01)	1.63E-04	1.59E-04*	1.57E-04*	1.66E-04*	1.39E-04*	7.44E-03	5.75E-04*	5.31E-04*	6.23E-02	--	--	--	--	--
		AVERAGE		1.97E-03	2.37E-01	2.37E-01	1.33E-04	1.63E-04	1.22E-04	1.66E-04	2.10E-04	5.03E-03	5.28E-04	5.45E-04	5.19E-02	--	--	--	--	--
75237	QBC	Lobster - Hepato	0.525	1.25E+00	4.11E+01	4.23E+01	1.20E-02	7.04E-03	8.30E-04	1.49E-02	3.45E-02	1.11E+00	5.09E-02	8.09E-03	2.79E+00	--	--	--	--	--
75219	QBD	Lobster - Hepato	0.592	(2.27E+00)	5.78E+01	6.04E+01	(1.92E-02)	(3.37E-02)	(3.18E-03)	(8.76E-02)	(1.52E-01)	(1.89E+00)	(3.12E-01)	(7.34E-02)	4.69E+00	--	--	--	--	--
75244	QBE	Lobster - Hepato	0.436	1.08E+00	3.14E+01	3.25E+01	1.01E-02	4.09E-03		4.18E-02	9.81E-02	1.16E+00	6.32E-02	2.13E-02	3.66E+00	--	--	--	--	--
75212	QBF	Lobster - Hepato	0.570	1.85E+00	4.41E+01	4.60E+01	1.78E-02	3.25E-02	2.78E-03	3.16E-02	6.44E-02	1.41E+00	1.03E-01	6.84E-02	(4.78E+00)	--	--	--	--	--
75223	QBG	Lobster - Hepato	0.412	1.13E+00	3.77E+01	3.89E+01	9.56E-03	1.47E-02	1.91E-03	1.73E-02	3.36E-02	1.10E+00	4.03E-02	1.26E-02	1.96E+00	--	--	--	--	--
75228	QBG	Lobster - Hepato	0.481	1.74E+00	4.51E+01	4.68E+01	1.74E-02	3.29E-02	3.17E-03	4.12E-02	9.57E-02	1.52E+00	1.14E-01	3.42E-02	3.55E+00	--	--	--	--	--
75249	QBI	Lobster - Hepato	0.547	2.03E+00	(5.96E+01)	(6.18E+01)	1.53E-02	1.17E-02	1.12E-03	1.30E-02	3.25E-02	1.52E+00	1.01E-01	1.36E-02	4.28E+00	--	--	--	--	--
75230	QBJ	Lobster - Hepato	0.480	6.53E-01	2.22E+01	2.28E+01	8.64E-03	1.08E-02	1.26E-03	7.15E-03	1.50E-02	6.58E-01	1.82E-02	4.34E-03	2.26E+00	--	--	--	--	--
		AVERAGE		1.50E+00	4.24E+01	4.39E+01	1.37E-02	1.84E-02	1.78E-03	3.18E-02	6.57E-02	1.30E+00	1.00E-01	2.95E-02	3.37E+00	--	--	--	--	--
75259	QBMHD	Soft Shell Clam	0.143	--	--	--	--	--	--	--	--	--	--	--	--	1.76	(0.245)	0.440	0.017	0.002
75260	QBMIS	Soft Shell Clam	0.137	--	--	--	--	--	--	--	--	--	--	--	--	(1.95)	0.167	(0.460)	(0.025)	--
		AVERAGE		--	--	--	--	--	--	--	--	--	--	--	--	1.85	0.206	0.450	0.021	0.002
75237	QBC	Lobster - Hepato	0.573	--	--	--	--	--	--	--	--	--	--	--	--	135	0.103*	0.195*	0.693	0.070
75237	QBC	Lobster - Hepato	0.488	--	--	--	--	--	--	--	--	--	--	--	--	137	0.420*	0.498*	0.756	--
75219	QBD	Lobster - Hepato	0.525	--	--	--	--	--	--	--	--	--	--	--	--	215	0.242*	0.215	1.64	0.082*
75219	QBD	Lobster - Hepato	0.585	--	--	--	--	--	--	--	--	--	--	--	--	219	0.187*	0.140*	1.61	--
75244	QBE	Lobster - Hepato	0.303	--	--	--	--	--	--	--	--	--	--	--	--	181	(2.35)	0.685*	1.58	0.062*
75244	QBF	Lobster - Hepato	0.337	--	--	--	--	--	--	--	--	--	--	--	--	217	1.72	0.600*	(2.23)	--
75212	QBF	Lobster - Hepato	0.533	--	--	--	--	--	--	--	--	--	--	--	--	176	0.634	0.181*	1.88	(0.112)*
75212	QBF	Lobster - Hepato	0.500	--	--	--	--	--	--	--	--	--	--	--	--	188	0.960	0.460*	1.90	--
75223	QBG	Lobster - Hepato	0.479	--	--	--	--	--	--	--	--	--	--	--	--	17.8	0.168	0.326*	1.11	0.053*
75223	QBG	Lobster - Hepato	0.434	--	--	--	--	--	--	--	--	--	--	--	--	17.7	0.417*	0.347*	1.06	--

TABLE B-8. RISK CHARACTERIZATION FOR A TYPICAL QUINCY AREA INDIVIDUAL FROM INGESTION OF QUINCY BAY FLOUNDER, LOBSTER AND HEPATOPANCREAS (a)(b)

CHEMICAL IDENTIFIED	FLOUNDER HAZARD RATIO		LOBSTER HAZARD RATIO		HEPATOPANCREAS HAZARD RATIO		TOTAL HAZARD RATIO		FLOUNDER UPPER BOUND INCREASED CANCER RISK		LOBSTER UPPER BOUND INCREASED CANCER RISK		HEPATOPANCREAS UPPER BOUND INCREASED CANCER RISK		TOTAL UPPER BOUND INCREASED CANCER RISK	
	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean	max	mean
ELEMENTS/METALS																
Cadmium	0.00	0.00	0.00	0.00	0.04	0.03	0.04	0.03	-	-	-	-	-	-	-	-
Chromium	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	-	-	-	-	-	-	-	-
Copper	0.00	0.00	0.00	0.00	0.04	0.02	0.05	0.02	-	-	-	-	-	-	-	-
Lead	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-	-	-	-	-	-	-	-
Mercury	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-	-	-	-	-	-	-	-
ORGANIC COMPOUNDS																
Chlordane (total)	0.01	0.00	0.00	0.00	0.03	0.01	0.04	0.01	5.6E-07	5.8E-08	1.9E-08	6.9E-09	1.8E-06	7.2E-07	2.4E-06	7.9E-07
PP-DDD	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6.4E-08	6.9E-09	1.3E-09	1.4E-10	6.1E-07	1.9E-07	6.7E-07	2.0E-07
PP-DDE	0.00	0.00	0.00	0.00	0.02	0.01	0.02	0.02	7.7E-08	2.5E-08	6.2E-08	4.2E-08	3.7E-06	2.5E-06	3.8E-06	2.6E-06
PP-DDT	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	2.4E-08	2.6E-09	0.0E+00	0.0E+00	1.4E-07	5.7E-08	1.7E-07	6.0E-08
Hexachlorobenzene (HCB)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6.0E-09	2.8E-09	8.9E-09	5.4E-09	1.8E-07	1.3E-07	2.0E-07	1.4E-07
Hexachlorocyclohexane (HCH)																
a-HCH	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	8.0E-08	7.2E-09	0.0E+00	0.0E+00	1.2E-06	6.6E-07	1.3E-06	6.7E-07
g-HCH (lindane)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.0E+00	0.0E+00	3.0E-09	9.8E-10	2.4E-08	1.4E-08	2.7E-08	1.5E-08
PAH (total)	-	-	-	-	-	-	-	-	0.0E+00	0.0E+00	2.1E-05	1.5E-05	3.1E-04	2.2E-04	3.4E-04	2.4E-04
PCBs (total)	0.11	0.04	0.09	0.06	3.53	2.51	3.73	2.61	2.8E-05	1.0E-05	2.4E-05	1.5E-05	9.2E-04	6.5E-04	9.7E-04	6.8E-04
									2.8E-05	1.0E-05	4.5E-05	3.0E-05	1.2E-03	8.8E-04	1.3E-03	9.2E-04

Ingestion rates:	grams/day
Flounder	1
Clams	0
Lobster	1.7
Hepatopancreas	0.4
TOTAL	3.1

(a) - Mean calculated using zero for undetected values.

(b) - See Table 2 for weight of evidence classification for carcinogens.

TABLE B-9. WET WEIGHT CONCENTRATIONS OF ORGANIC COMPOUNDS AND METALS IN AQUATIC ORGANISMS FROM QUINCY BAY 1
(DETECTION LIMITS INCLUDED IN AVERAGES)
(ug/g wet)

SAMPLE NO.	STATION	SAMPLE TYPE	g DRY/g WET RATIO	AROCLOR 1242	AROCLOR 1254	TOTAL PCBs	HCB	A-HCH	G-HCH	A-CHLOR	G-CHLOR	PP-DDE	PP-DDD	PP-DDT	TOTAL PAHs	Cu	Cr	Pd	Cd	Hg
75259	QBMMD	Soft Shell Clam	0.149	8.94E-03	(1.40E-01)	1.49E-01	1.01E-04	1.10E-04	1.16E-04*	9.61E-04	1.32E-03	3.77E-03	1.04E-03	2.70E-04	4.19E-02	--	--	--	--	--
75260	QBMIS	Soft Shell Clam	0.151	(1.99E-02)	1.33E-01	(1.53E-01)	(1.03E-04)	(1.28E-04)	(1.18E-04)*	(1.56E-03)	(1.92E-03)	(4.76E-03)	(1.42E-03)	(3.37E-04)	(4.51E-02)	--	--	--	--	--
		AVERAGE		1.44E-02	1.36E-01	1.51E-01	1.02E-04	1.19E-04	1.17E-04	1.26E-03	1.62E-03	4.26E-03	1.23E-03	3.03E-04	4.35E-02	--	--	--	--	--
75237	QBC	Lobster - Tissue	0.220	1.98E-03*	2.03E-01	2.03E-01	1.06E-04	1.74E-04*	6.60E-05	1.82E-04*	1.39E-04	4.11E-03	6.29E-04*	5.81E-04*	4.55E-02	--	--	--	--	--
75239	QBC	Lobster - Tissue	0.202	1.82E-03*	2.30E-01	2.30E-01	1.33E-04	1.60E-04*	1.58E-04*	1.67E-04*	6.26E-05	4.95E-03	5.78E-05*	5.33E-04*	5.03E-02	--	--	--	--	--
75241	QBC	Lobster - Tissue	0.179	1.61E-03*	1.88E-01	1.88E-01	8.41E-05	1.41E-04*	2.68E-05	1.48E-04*	1.24E-04*	4.87E-03	5.12E-04*	4.73E-04*	3.69E-02	--	--	--	--	--
75219	QBD	Lobster - Tissue	0.227	2.04E-03*	2.61E-01	2.61E-01	1.68E-04	1.79E-04*	8.17E-05	1.09E-04	3.04E-04	6.42E-03	1.52E-04	5.99E-04*	5.86E-02	--	--	--	--	--
75220	QBD	Lobster - Tissue	0.225	2.02E-03*	2.77E-01	2.77E-01	1.73E-04	1.78E-04*	1.76E-04*	1.86E-04*	3.04E-04	5.60E-03	6.43E-04*	5.94E-04*	6.28E-02	--	--	--	--	--
75244	QBE	Lobster - Tissue	0.151	1.36E-03*	2.20E-01	2.20E-01	1.18E-04	1.19E-04*	1.18E-04*	1.89E-04	(4.15E-04)	5.16E-03	4.32E-04*	3.99E-04*	5.75E-02	--	--	--	--	--
75245	QBE	Lobster - Tissue	0.206	1.85E-03*	2.25E-01	2.25E-01	1.17E-04	1.63E-04*	9.27E-05	1.09E-04	2.47E-04	3.93E-03	5.89E-04*	5.44E-04*	5.29E-02	--	--	--	--	--
75212	QBF	Lobster - Tissue	0.220	1.98E-03*	2.08E-01	2.08E-01	1.47E-04	1.74E-04*	1.72E-04*	1.82E-04*	3.52E-04	3.32E-03	6.29E-04*	5.81E-04*	5.90E-02	--	--	--	--	--
75214	QBF	Lobster - Tissue	0.207	1.86E-03*	2.50E-01	2.50E-01	(2.19E-04)	1.64E-04*	1.61E-04*	1.71E-04*	3.02E-04	5.32E-03	5.92E-04*	5.46E-04*	(7.43E-02)	--	--	--	--	--
75223	QBG	Lobster - Tissue	0.209	1.88E-03*	2.72E-01	2.72E-01	1.17E-04	1.65E-04*	1.63E-04*	1.73E-04*	3.01E-04	(7.46E-03)	5.98E-04*	5.52E-04*	5.73E-02	--	--	--	--	--
75225	QBG	Lobster - Tissue	0.232	2.09E-03*	2.88E-01	2.88E-01	1.21E-04	(1.83E-04)*	1.81E-04*	(1.92E-04)*	3.04E-04	7.08E-03	(6.64E-04)*	(6.12E-04)*	4.27E-02	--	--	--	--	--
75228	QBG	Lobster - Tissue	0.188	1.69E-03*	2.14E-01	2.14E-01	1.26E-04	1.49E-04*	8.65E-05	1.56E-04*	1.37E-04	4.19E-03	5.38E-04*	4.96E-04*	4.08E-02	--	--	--	--	--
75249	QBI	Lobster - Tissue	0.211	1.90E-03*	2.22E-01	2.22E-01	1.03E-04	1.67E-04*	6.33E-05	1.75E-04*	8.23E-05	4.26E-03	1.14E-04	5.57E-04*	5.51E-02	--	--	--	--	--
75250	QBI	Lobster - Tissue	0.223	2.01E-03*	1.43E-01	1.43E-01	1.05E-04	1.76E-04*	1.74E-04*	1.85E-04*	8.03E-05	2.83E-03	6.38E-04*	5.89E-04*	3.92E-02	--	--	--	--	--
75230	QBJ	Lobster - Tissue	0.199	1.79E-03*	2.15E-01	2.15E-01	1.21E-04	1.57E-04*	6.96E-05	1.65E-04*	6.37E-05	3.44E-03	5.69E-04*	5.25E-04*	3.58E-02	--	--	--	--	--
75234	QBJ	Lobster - Tissue	0.201	(3.68E-03)*	(3.80E-01)	(3.82E-01)	1.63E-04	1.59E-04*	1.57E-04*	1.66E-04*	1.39E-04*	7.44E-03	5.75E-04*	5.31E-04*	6.23E-02	--	--	--	--	--
		AVERAGE		1.97E-03	2.37E-01	2.37E-01	1.33E-04	1.63E-04	1.22E-04	1.66E-04	2.10E-04	5.03E-03	5.28E-04	5.45E-04	5.19E-02	--	--	--	--	--
75237	QBC	Lobster - Hepato	0.525	1.25E+00	4.11E+01	4.23E+01	1.20E-02	7.04E-03	8.30E-04	1.49E-02	3.45E-02	1.11E+00	5.09E-02	8.09E-03	2.79E+00	--	--	--	--	--
75219	QBD	Lobster - Hepato	0.592	(2.27E+00)	5.78E+01	6.04E+01	(1.92E-02)	(3.37E-02)	(3.18E-03)	(8.76E-02)	(1.52E-01)	(1.89E+00)	(3.12E-01)	(7.34E-02)	6.69E+00	--	--	--	--	--
75244	QBE	Lobster - Hepato	0.436	1.08E+00	3.14E+01	3.25E+01	1.01E-02	4.09E-03		4.18E-02	9.81E-02	1.16E+00	6.32E-02	2.13E-02	3.66E+00	--	--	--	--	--
75212	QBF	Lobster - Hepato	0.570	1.85E+00	4.41E+01	4.60E+01	1.78E-02	3.25E-02	2.78E-03	3.16E-02	6.44E-02	1.41E+00	1.03E-01	6.84E-02	(4.78E+00)	--	--	--	--	--
75223	QBG	Lobster - Hepato	0.412	1.13E+00	3.77E+01	3.89E+01	9.56E-03	1.47E-02	1.91E-03	1.73E-02	3.36E-02	1.10E+00	4.03E-02	1.26E-02	1.96E+00	--	--	--	--	--
75228	QBG	Lobster - Hepato	0.481	1.74E+00	4.51E+01	4.68E+01	1.74E-02	3.29E-02	3.17E-03	4.12E-02	9.57E-02	1.52E+00	1.14E-01	3.42E-02	3.55E+00	--	--	--	--	--
75249	QBI	Lobster - Hepato	0.547	2.03E+00	(5.96E+01)	(6.18E+01)	1.53E-02	1.17E-02	1.12E-03	1.30E-02	3.25E-02	1.52E+00	1.01E-01	1.36E-02	4.28E+00	--	--	--	--	--
75230	QBJ	Lobster - Hepato	0.480	6.53E-01	2.22E+01	2.28E+01	8.64E-03	1.08E-02	1.26E-03	7.15E-03	1.50E-02	6.58E-01	1.82E-02	4.34E-03	2.26E+00	--	--	--	--	--
		AVERAGE		1.50E+00	4.24E+01	4.39E+01	1.37E-02	1.84E-02	1.78E-03	3.18E-02	6.57E-02	1.30E+00	1.00E-01	2.95E-02	3.37E+00	--	--	--	--	--
75259	QBMMD	Soft Shell Clam	0.143	--	--	--	--	--	--	--	--	--	--	--	--	1.76	(0.245)	0.440	0.017	0.002
75260	QBMIS	Soft Shell Clam	0.137	--	--	--	--	--	--	--	--	--	--	--	--	(1.95)	0.167	(0.460)	(0.025)	--
		AVERAGE		--	--	--	--	--	--	--	--	--	--	--	--	1.85	0.206	0.450	0.021	0.002
75237	QBC	Lobster - Hepato	0.573	--	--	--	--	--	--	--	--	--	--	--	--	135	0.103*	0.195*	0.693	0.070
75237	QBC	Lobster - Hepato	0.488	--	--	--	--	--	--	--	--	--	--	--	--	137	0.420*	0.498*	0.756	--
75219	QBD	Lobster - Hepato	0.525	--	--	--	--	--	--	--	--	--	--	--	--	215	0.242*	0.215	1.64	0.082*
75219	QBD	Lobster - Hepato	0.585	--	--	--	--	--	--	--	--	--	--	--	--	219	0.187*	0.140*	1.61	--
75244	QBE	Lobster - Hepato	0.303	--	--	--	--	--	--	--	--	--	--	--	--	181	(2.35)	0.685*	1.58	0.062*
75244	QBF	Lobster - Hepato	0.337	--	--	--	--	--	--	--	--	--	--	--	--	217	1.72	0.600*	(2.23)	--
75212	QBF	Lobster - Hepato	0.533	--	--	--	--	--	--	--	--	--	--	--	--	176	0.634	0.181*	1.88	(0.112)*
75212	QBF	Lobster - Hepato	0.500	--	--	--	--	--	--	--	--	--	--	--	--	188	0.960	0.460*	1.90	--
75223	QBG	Lobster - Hepato	0.479	--	--	--	--	--	--	--	--	--	--	--	--	17.8	0.168	0.326*	1.11	0.053*
75223	QBG	Lobster - Hepato	0.434	--	--	--	--	--	--	--	--	--	--	--	--	17.7	0.417*	0.347*	1.06	--

TABLE B-9. WET WEIGHT CONCENTRATIONS OF ORGANIC COMPOUNDS AND METALS IN AQUATIC ORGANISMS FROM QUINCY BAY 1
(DETECTION LIMITS INCLUDED IN AVERAGES)
(ug/g wet)

SAMPLE NO.	STATION	SAMPLE TYPE	g DRY/g WET RATIO	AROCLOR 1242	AROCLOR 1254	TOTAL PCBs	HCB	A-HCH	G-HCH	A-CHLOR	G-CHLOR	PP-DDE	PP-DDD	PP-DDT	TOTAL PAHs	Cu	Cr	Pd	Cd	Hg
75228	QBG	Lobster - Hepato	0.500	--	--	--	--	--	--	--	--	--	--	--	--	(279)	1.41	0.120*	1.25	0.046
75228	QBG	Lobster - Hepato	0.494	--	--	--	--	--	--	--	--	--	--	--	--	252	2.01	0.217*	1.16	--
75249	QBI	Lobster - Hepato	0.557	--	--	--	--	--	--	--	--	--	--	--	--	28.5	0.201*	0.201*	0.830	0.028
75249	QBI	Lobster - Hepato	0.473	--	--	--	--	--	--	--	--	--	--	--	--	24.5	0.284*	(0.700)*	0.733	--
75230	QBJ	Lobster - Hepato	0.551	--	--	--	--	--	--	--	--	--	--	--	--	54.3	0.220*	0.287*	1.29	0.064
75230	QBJ	Lobster - Hepato	0.547	--	--	--	--	--	--	--	--	--	--	--	--	51.5	0.170	0.186*	1.27	--
		AVERAGE		--	--	--	--	--	--	--	--	--	--	--	--	137	0.720	0.335	1.31	0.065
75237	QBC	Lobster - Tissue	0.237	--	--	--	--	--	--	--	--	--	--	--	--	3.77	0.005*	0.168	0.002	0.073
75237	QBC	Lobster - Tissue	0.237	--	--	--	--	--	--	--	--	--	--	--	--	4.43	0.005*	0.166	0.002	--
75239	QBC	Lobster - Tissue	0.255	--	--	--	--	--	--	--	--	--	--	--	--	(6.22)	0.033	(0.207)	0.002	0.079
75239	QBC	Lobster - Tissue	0.255	--	--	--	--	--	--	--	--	--	--	--	--	5.79	0.036	0.171	0.001	--
75241	QBC	Lobster - Tissue	0.232	--	--	--	--	--	--	--	--	--	--	--	--	2.67	0.190	0.176	0.001	0.050
75241	QBC	Lobster - Tissue	0.230	--	--	--	--	--	--	--	--	--	--	--	--	2.69	(0.260)	0.152	0.001	--
75219	QBD	Lobster - Tissue	0.249	--	--	--	--	--	--	--	--	--	--	--	--	5.25	0.005*	0.179	0.001	0.022*
75219	QBD	Lobster - Tissue	0.241	--	--	--	--	--	--	--	--	--	--	--	--	5.93	0.005*	0.188	0.001	--
75220	QBD	Lobster - Tissue	0.236	--	--	--	--	--	--	--	--	--	--	--	--	4.01	0.009*	0.182	0.001	0.065
75220	QBD	Lobster - Tissue	0.235	--	--	--	--	--	--	--	--	--	--	--	--	4.61	0.009*	0.197	0.001	--
75244	QBE	Lobster - Tissue	0.187	--	--	--	--	--	--	--	--	--	--	--	--	4.30	0.062	0.159	(0.005)	0.028
75244	QBE	Lobster - Tissue	0.291	--	--	--	--	--	--	--	--	--	--	--	--	3.58	0.049	0.166	0.001	--
75245	QBE	Lobster - Tissue	0.236	--	--	--	--	--	--	--	--	--	--	--	--	3.94	0.005*	0.170	0.002	0.159
75245	QBE	Lobster - Tissue	0.230	--	--	--	--	--	--	--	--	--	--	--	--	4.55	0.005*	0.122	0.001	--
75212	QBF	Lobster - Tissue	0.232	--	--	--	--	--	--	--	--	--	--	--	--	4.59	0.005*	0.179	0.001	0.104
75212	QBF	Lobster - Tissue	0.228	--	--	--	--	--	--	--	--	--	--	--	--	3.85	0.005*	0.176	0.002	--
75214	QBF	Lobster - Tissue	0.225	--	--	--	--	--	--	--	--	--	--	--	--	3.65	0.005*	0.149	0.003	(0.168)
75214	QBF	Lobster - Tissue	0.222	--	--	--	--	--	--	--	--	--	--	--	--	4.13	0.013	0.173	(0.003)	--
75223	QBG	Lobster - Tissue	0.237	--	--	--	--	--	--	--	--	--	--	--	--	3.32	0.005*	0.171	0.001	0.104
75223	QBG	Lobster - Tissue	0.253	--	--	--	--	--	--	--	--	--	--	--	--	3.26	0.005*	0.147	0.001	--
75225	QBG	Lobster - Tissue	0.235	--	--	--	--	--	--	--	--	--	--	--	--	3.95	0.005*	0.190	0.001	0.118
75225	QBG	Lobster - Tissue	0.240	--	--	--	--	--	--	--	--	--	--	--	--	4.15	0.005*	0.197	0.001	--
75228	QBG	Lobster - Tissue	0.213	--	--	--	--	--	--	--	--	--	--	--	--	2.96	0.009	0.143	0.001	0.065
75228	QBG	Lobster - Tissue	0.285	--	--	--	--	--	--	--	--	--	--	--	--	2.88	0.003	0.185	0.001	--
75249	QBI	Lobster - Tissue	0.240	--	--	--	--	--	--	--	--	--	--	--	--	4.08	0.005*	0.187	0.002	0.087
75249	QBI	Lobster - Tissue	0.237	--	--	--	--	--	--	--	--	--	--	--	--	5.26	0.005*	0.121	0.003	--
75250	QBI	Lobster - Tissue	0.246	--	--	--	--	--	--	--	--	--	--	--	--	3.37	0.002	0.167	0.001	0.053
75250	QBI	Lobster - Tissue	0.239	--	--	--	--	--	--	--	--	--	--	--	--	3.73	0.002	0.177	0.001	--
75230	QBJ	Lobster - Tissue	0.239	--	--	--	--	--	--	--	--	--	--	--	--	3.92	0.005*	0.163	0.002	0.087
75230	QBJ	Lobster - Tissue	0.241	--	--	--	--	--	--	--	--	--	--	--	--	3.76	0.007	0.154	0.001	--
75234	QBJ	Lobster - Tissue	0.234	--	--	--	--	--	--	--	--	--	--	--	--	4.07	0.005	0.178	0.002	0.091
75234	QBJ	Lobster - Tissue	0.230	--	--	--	--	--	--	--	--	--	--	--	--	3.11	0.005*	0.147	0.001	--
		AVERAGE		--	--	--	--	--	--	--	--	--	--	--	--	4.06	0.024	0.169	0.002	0.085
75124	QBT1	Flounder(W)-Fresh	0.185	4.79E-03	5.68E-01	5.72E-01	1.28E-04	1.46E-04*	1.44E-04*	5.07E-04	2.28E-03	1.07E-02	1.87E-04	4.88E-04*	--	--	--	--	--	--
75168	QBT1	Flounder(W)-Fresh	0.210	1.39E-03	3.74E-01	3.76E-01	1.01E-04	1.66E-04*	1.64E-04*	3.23E-04	1.60E-03	5.77E-03	6.01E-04*	5.54E-04*	--	--	--	--	--	--
75185	QBT1	Flounder(W)-Fresh	0.197	1.77E-03	1.59E-01	1.59E-01	8.47E-05	1.56E-04*	1.54E-04*	1.99E-04	7.09E-04	1.99E-03	2.94E-04	5.20E-04*	2.46E-04*	--	--	--	--	--
75190	QBT1	Flounder(W)-Fresh	0.210	2.86E-03	1.28E-01	1.31E-01	8.61E-05	1.66E-04*	1.64E-04*	3.76E-04	9.60E-04	2.10E-03	2.56E-04	5.54E-04*	--	--	--	--	--	--
75191	QBT1	Flounder(W)-Fresh	0.205	1.84E-03	1.63E-01	1.63E-01	1.00E-04	1.62E-04*	1.60E-04*	1.64E-04	4.88E-04	2.42E-03	5.86E-04*	5.41E-04*	--	--	--	--	--	--
75194	QBT1	Flounder(W)-Fresh	0.197	1.77E-03	4.45E-01	4.45E-01	9.85E-05	1.56E-04*	1.54E-04*	5.46E-04	3.01E-03	8.33E-03	2.68E-04	5.20E-04*	--	--	--	--	--	--

TABLE B-9. WET WEIGHT CONCENTRATIONS OF ORGANIC COMPOUNDS AND METALS IN AQUATIC ORGANISMS FROM QUINCY BAY 1
(DETECTION LIMITS INCLUDED IN AVERAGES)
(ug/g wet)

SAMPLE NO.	STATION	SAMPLE TYPE	g DRY/g WET RATIO	AROCLOR 1242	AROCLOR 1254	TOTAL PCBs	HCB	A-HCH[2]	G-HCH[2]	A-CHLOR	G-CHLOR	PP-DDE	PP-DDD	PP-DDT	TOTAL PAHs	Cu	Cr	Pd	Cd	Hg
75195	08T1	Flounder(W)-Fresh	0.191	1.72E-03	2.01E-01	2.01E-01	8.60E-05	1.51E-04*	1.49E-04*	5.86E-04	1.40E-03	3.82E-03	4.72E-04	5.04E-04*	--	--	--	--	--	--
75198	08T1	Flounder(W)-Fresh	0.204	1.84E-03	2.41E-01	2.41E-01	1.86E-04	1.61E-04*	1.59E-04*	2.35E-04	7.04E-04	3.16E-03	3.73E-04	4.61E-04*	--	--	--	--	--	--
75101	08T2	Flounder(W)-Fresh	0.200	1.80E-03*	1.81E-01	1.81E-01	1.26E-04	1.58E-04*	1.56E-04*	2.86E-04	7.84E-04	1.79E-03*	5.72E-04	5.28E-04*	2.50E-04*	--	--	--	--	--
75113	08T2	Flounder(W)-Fresh	0.190	1.71E-03*	2.20E-01	2.20E-01	1.35E-04	1.50E-04*	1.48E-04*	2.85E-04	9.14E-04	2.36E-03*	5.43E-04	5.02E-04*	2.38E-04*	--	--	--	--	--
75114	08T2	Flounder(W)-Fresh	0.206	3.91E-03	3.96E-01	4.00E-01	2.18E-04	1.63E-04*	1.61E-04*	5.81E-04	1.63E-03	5.87E-03	8.94E-04	5.44E-04*	--	--	--	--	--	--
75115	08T2	Flounder(W)-Fresh	0.214	1.93E-03*	1.09E-01	1.09E-01	4.90E-05*	1.69E-04*	1.67E-04*	4.02E-04	7.64E-04	2.42E-03	1.03E-03	5.65R-04*	--	--	--	--	--	--
75179	08T2	Flounder(W)-Fresh	0.206	1.56E-03	1.64E-01	1.65E-01	7.38E-05*	1.63E-04*	1.61E-04*	5.71E-04	1.05E-03	2.99E-03	1.19E-03	5.44E-04*	--	--	--	--	--	--
75160	08T2	Flounder(W)-Fresh	0.218	1.96E-03*	2.02E-01	2.02E-01	1.33E-04	1.72E-04*(1.70E-04)*		2.31E-04	6.21E-04	2.38E-03*	6.23E-04	5.76E-04*	--	--	--	--	--	--
75164	08T3	Flounder(W)-Fresh	0.203	1.04E-02	3.15E-01	3.25E-01	1.66E-04	(8.93E-04)	1.58E-04*(7.67E-03)		(2.23E-02)	7.35E-03	(1.33E-02)	(4.97E-03)	2.54E-04*	--	--	--	--	--
75167	08T3	Flounder(W)-Fresh	0.200	1.10E-02*	4.88E-01	4.98E-01	1.60E-04	1.12E-04	1.56E-04*	1.49E-03	5.20E-03	1.22E-02	1.84E-03	1.16E-03	--	--	--	--	--	--
75170	08T3	Flounder(W)-Fresh	0.195	1.76E-03	1.85E-01	1.85E-01	8.35E-05*	7.41E-05	1.52E-04*	4.41E-04	1.35E-04*	3.84E-03	1.51E-03	4.49E-04	--	--	--	--	--	--
75172	08T3	Flounder(W)-Fresh	0.190	(1.77E-02)	(7.26E-01)	(7.43E-01)	1.60E-04	1.56E-04	1.48E-04*	1.26E-03	1.60E-04	1.37E-02	3.15E-03	1.96E-03	--	--	--	--	--	--
75180	08T3	Flounder(W)-Fresh	0.206	1.85E-03*	1.17E-01	1.17E-01	8.24E-05	2.88E-04	1.61E-04*	1.36E-03	8.24E-05	2.82E-03	3.69E-03	1.32E-03	--	--	--	--	--	--
75182	08T3	Flounder(W)-Fresh	0.209	1.88E-03*	1.24E-01	1.24E-01	5.56E-05*	1.65E-04*	1.63E-04*	1.09E-03	1.44E-04*	1.76E-03	3.96E-03	5.52E-04*	(2.61E-04)	--	--	--	--	--
75128	08T1T3	Flounder(W)-Fresh	0.181	1.63E-03*	3.33E-01	3.33E-01	1.57E-04	6.70E-05	1.41E-04*	3.48E-04	1.30E-03	6.64E-03	6.21E-04	4.16E-04	2.26E-04*	--	--	--	--	--
75133	08T1T3	Flounder(W)-Fresh	0.184	1.66E-03*	1.71E-01	1.71E-01	1.58E-04	8.65E-05	1.44E-04*	7.14E-04	1.69E-03	3.90E-03	4.49E-04	6.44E-04	--	--	--	--	--	--
75145	08T1T3	Flounder(W)-Fresh	0.195	1.76E-03*	1.60E-01	1.60E-01	1.44E-04	1.93E-04	1.52E-04*	8.48E-04	2.01E-03	4.08E-03	1.19E-03	7.88E-04	--	--	--	--	--	--
75148	08T1T3	Flounder(W)-Fresh	0.191	8.00E-03	5.41E-01	5.48E-01	(2.52E-04)	1.28E-04	1.49E-04*	2.16E-03	6.04E-03	(1.59E-02)	1.20E-03	1.18E-03	--	--	--	--	--	--
75149	08T1T3	Flounder(W)-Fresh	0.200	1.80E-03*	6.12E-02	6.12E-02	1.42E-04	1.58E-04*	1.56E-04*	1.66E-04*	5.40E-05	1.51E-03*	5.72E-04	5.28E-04*	--	--	--	--	--	--
75124	08T1	Flounder(W)-Fresh	0.203	--	--	--	--	--	--	--	--	--	--	--	--	0.093	0.012*	0.012*	0.002*	0.080
75168	08T1	Flounder(W)-Fresh	0.207	--	--	--	--	--	--	--	--	--	--	--	--	0.122	0.019*	0.006*	0.001*	0.035
75185	08T1	Flounder(W)-Fresh	0.209	--	--	--	--	--	--	--	--	--	--	--	--	0.144	0.008	0.006*	0.001*	0.045
75190	08T1	Flounder(W)-Fresh	0.223	--	--	--	--	--	--	--	--	--	--	--	--	0.087	(0.377)	0.033*	0.001*	0.014
75191	08T1	Flounder(W)-Fresh	0.205	--	--	--	--	--	--	--	--	--	--	--	--	0.105	0.025*	0.018*	0.001*	0.012
75194	08T1	Flounder(W)-Fresh	0.205	--	--	--	--	--	--	--	--	--	--	--	--	0.096	0.043*	(0.043)*	0.001*	0.036
75195	08T1	Flounder(W)-Fresh	0.200	--	--	--	--	--	--	--	--	--	--	--	--	0.078	*0.010*	*0.012**	*0.001**	*0.016*
75198	08T1	Flounder(W)-Fresh	0.217	--	--	--	--	--	--	--	--	--	--	--	--	0.126	0.009	0.013*	0.001*	0.036
75101	08T2	Flounder(W)-Fresh	0.207	--	--	--	--	--	--	--	--	--	--	--	--	0.048	0.000*	0.008	0.001*	0.047
75113	08T2	Flounder(W)-Fresh	0.189	--	--	--	--	--	--	--	--	--	--	--	--	0.096	0.006*	0.028*	0.003[1]	(0.086)
75114	08T2	Flounder(W)-Fresh	0.197	--	--	--	--	--	--	--	--	--	--	--	--	0.110	0.016	0.008	(0.009)	0.010
75115	08T2	Flounder(W)-Fresh	0.221	--	--	--	--	--	--	--	--	--	--	--	--	0.159	0.053*	0.002	0.004*	0.011
75179	08T2	Flounder(W)-Fresh	0.206	--	--	--	--	--	--	--	--	--	--	--	--	0.122	0.016	0.012*	0.001*	0.008
75160	08T2	Flounder(W)-Fresh	0.198	--	--	--	--	--	--	--	--	--	--	--	--	0.042	0.024*	0.042*	0.001*	0.021
75164	08T3	Flounder(W)-Fresh	0.208	--	--	--	--	--	--	--	--	--	--	--	--	0.079	0.019*	0.019*	0.001*	0.035
75167	08T3	Flounder(W)-Fresh	0.206	--	--	--	--	--	--	--	--	--	--	--	--	0.142	0.004	0.012*	0.001*	0.041
75170	08T3	Flounder(W)-Fresh	0.192	--	--	--	--	--	--	--	--	--	--	--	--	(0.215)	0.025	0.012*	0.001*	0.026
75172	08T3	Flounder(W)-Fresh	0.193	--	--	--	--	--	--	--	--	--	--	--	--	0.197	0.016*	0.000*	0.001*	0.047
75180	08T3	Flounder(W)-Fresh	0.210	--	--	--	--	--	--	--	--	--	--	--	--	0.036	0.002	0.002	0.001*	0.006
75182	08T3	Flounder(W)-Fresh	0.212	--	--	--	--	--	--	--	--	--	--	--	--	0.102	0.008	0.000*	0.001*	0.010
75128	08T1T3	Flounder(W)-Fresh	0.183	--	--	--	--	--	--	--	--	--	--	--	--	0.099	0.005*	0.016*	0.001*	0.024
75133	08T1T3	Flounder(W)-Fresh	0.202	--	--	--	--	--	--	--	--	--	--	--	--	0.147	0.018*	0.018*	0.001*	0.016
75133	08T1T3	Flounder(W)-Fresh	0.195	--	--	--	--	--	--	--	--	--	--	--	2.43E-04*	--	--	--	--	--
75145	08T1T3	Flounder(W)-Fresh	0.203	--	--	--	--	--	--	--	--	--	--	--	--	0.071	0.006*	0.012*	0.001*	0.019
75148	08T1T3	Flounder(W)-Fresh	0.201	--	--	--	--	--	--	--	--	--	--	--	--	0.127	0.012*	0.024*	0.001*	0.053
75149	08T1T3	Flounder(W)-Fresh	0.211	--	--	--	--	--	--	--	--	--	--	--	--	0.089	0.006*	0.006*	0.001*	0.017
		AVERAGE		3.61E-03	2.71E-01	2.73E-01	1.27E-04	1.82E-04	1.56E-04	9.14E-04	2.24E-03	5.19E-03	1.58E-03	8.55E-04	2.45E-04	0.109	0.029	0.015	0.001	0.030

1. Adapted from U.S. EPA, December 2, 1987.
2. Also A BHC and G BHC, respectively.
* = Detection limit.
() = Maximum Concentration

TABLE B-10. WET WEIGHT CONCENTRATIONS OF ORGANIC COMPOUNDS AND METALS IN AQUATIC ORGANISMS FROM QUINCY BAY 1
(ug/g wet)

SAMPLE NO.	STATION	SAMPLE TYPE	g DRY/g WET RATIO	AROCLOR 1242	AROCLOR 1254	TOTAL PCBs	HCB	A-HCH	G-HCH	A-CHLOR	G-CHLOR	PP-DDE	PP-DDD	PP-DDT	TOTAL PAHs	Cu	Cr	Pd	Cd	Hg
75259	08MHD	Soft Shell Clam	0.149	8.94E-03	(1.40E-01)	1.49E-01	1.01E-04	1.10E-04	0.00E+00	9.61E-04	1.32E-03	3.77E-03	1.04E-03	2.70E-04	4.19E-02	--	--	--	--	--
75260	08M1S	Soft Shell Clam	0.151	(1.99E-02)	1.33E-01	(1.53E-01)	(1.03E-04)	(1.28E-04)	0.00E+00	(1.56E-03)	(1.92E-03)	(4.76E-03)	(1.42E-03)	(3.37E-04)	(4.51E-02)	--	--	--	--	--
		AVERAGE		1.44E-02	1.36E-01	1.51E-01	1.02E-04	1.19E-04	0.00E+00	1.26E-03	1.62E-03	4.26E-03	1.23E-03	3.03E-04	4.35E-02	--	--	--	--	--
75237	08C	Lobster - Tissue	0.220	0.00E+00	2.03E-01	2.03E-01	1.06E-04	0.00E+00	6.60E-05	0.00E+00	1.39E-04	4.11E-03	0.00E+00	0.00E+00	4.55E-02	--	--	--	--	--
75239	08C	Lobster - Tissue	0.202	0.00E+00	2.30E-01	2.30E-01	1.33E-04	0.00E+00	0.00E+00	0.00E+00	6.26E-05	4.95E-03	0.00E+00	0.00E+00	5.03E-02	--	--	--	--	--
75241	08C	Lobster - Tissue	0.179	0.00E+00	1.88E-01	1.88E-01	8.41E-05	0.00E+00	2.68E-05	0.00E+00	0.00E+00	4.87E-03	0.00E+00	0.00E+00	3.69E-02	--	--	--	--	--
75219	08D	Lobster - Tissue	0.227	0.00E+00	2.61E-01	2.61E-01	1.68E-04	0.00E+00	8.17E-05	1.09E-04	3.04E-04	6.42E-03	(1.52E-04)	0.00E+00	5.86E-02	--	--	--	--	--
75220	08D	Lobster - Tissue	0.225	0.00E+00	2.77E-01	2.77E-01	1.73E-04	0.00E+00	0.00E+00	0.00E+00	3.04E-04	5.60E-03	0.00E+00	0.00E+00	6.28E-02	--	--	--	--	--
75244	08E	Lobster - Tissue	0.151	0.00E+00	2.20E-01	2.20E-01	1.18E-04	0.00E+00	0.00E+00	(1.89E-04)	(4.15E-04)	5.16E-03	0.00E+00	0.00E+00	5.75E-02	--	--	--	--	--
75245	08E	Lobster - Tissue	0.206	0.00E+00	2.25E-01	2.25E-01	1.17E-04	0.00E+00	(9.27E-05)	1.09E-04	2.47E-04	3.93E-03	0.00E+00	0.00E+00	5.29E-02	--	--	--	--	--
75212	08F	Lobster - Tissue	0.220	0.00E+00	2.08E-01	2.08E-01	1.47E-04	0.00E+00	0.00E+00	0.00E+00	3.52E-04	3.32E-03	0.00E+00	0.00E+00	5.90E-02	--	--	--	--	--
75214	08F	Lobster - Tissue	0.207	0.00E+00	2.50E-01	2.50E-01	(2.19E-04)	0.00E+00	0.00E+00	0.00E+00	3.02E-04	5.32E-03	0.00E+00	0.00E+00	(7.43E-02)	--	--	--	--	--
75223	08G	Lobster - Tissue	0.209	0.00E+00	2.72E-01	2.72E-01	1.17E-04	0.00E+00	0.00E+00	0.00E+00	3.01E-04	(7.46E-03)	0.00E+00	0.00E+00	5.73E-02	--	--	--	--	--
75225	08G	Lobster - Tissue	0.232	0.00E+00	2.88E-01	2.88E-01	1.21E-04	0.00E+00	0.00E+00	0.00E+00	3.04E-04	7.08E-03	0.00E+00	0.00E+00	4.27E-02	--	--	--	--	--
75228	08G	Lobster - Tissue	0.188	0.00E+00	2.14E-01	2.14E-01	1.26E-04	0.00E+00	8.65E-05	0.00E+00	1.37E-04	4.19E-03	0.00E+00	0.00E+00	4.08E-02	--	--	--	--	--
75249	08I	Lobster - Tissue	0.211	0.00E+00	2.22E-01	2.22E-01	1.03E-04	0.00E+00	6.33E-05	0.00E+00	8.23E-05	4.26E-03	1.14E-04	0.00E+00	5.51E-02	--	--	--	--	--
75250	08I	Lobster - Tissue	0.223	0.00E+00	1.43E-01	1.43E-01	1.05E-04	0.00E+00	0.00E+00	0.00E+00	8.03E-05	2.83E-03	0.00E+00	0.00E+00	3.92E-02	--	--	--	--	--
75230	08J	Lobster - Tissue	0.199	0.00E+00	2.15E-01	2.15E-01	1.21E-04	0.00E+00	6.96E-05	0.00E+00	6.37E-05	3.44E-03	0.00E+00	0.00E+00	3.58E-02	--	--	--	--	--
75234	08J	Lobster - Tissue	0.201	(3.68E-03)	(3.80E-01)	(3.82E-01)	1.63E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-03	0.00E+00	0.00E+00	6.23E-02	--	--	--	--	--
		AVERAGE		2.30E-04	2.37E-01	2.37E-01	1.33E-04	0.00E+00	3.04E-05	2.54E-05	1.93E-04	5.03E-03	1.66E-05	0.00E+00	5.19E-02	--	--	--	--	--
75237	08C	Lobster - Hepato	0.525	1.25E+00	4.11E+01	4.23E+01	1.20E-02	7.04E-03	8.30E-04	1.49E-02	3.45E-02	1.11E+00	5.09E-02	8.09E-03	2.79E+00	--	--	--	--	--
75219	08D	Lobster - Hepato	0.592	(2.27E+00)	5.78E+01	6.04E+01	(1.92E-02)	(3.37E-02)	(3.18E-03)	(8.76E-02)	(1.52E-01)	(1.89E+00)	(3.12E-01)	(7.34E-02)	4.69E+00	--	--	--	--	--
75244	08E	Lobster - Hepato	0.436	1.08E+00	3.14E+01	3.25E+01	1.01E-02	4.09E-03	0.00E+00	4.18E-02	9.81E-02	1.16E+00	6.32E-02	2.13E-02	3.66E+00	--	--	--	--	--
75212	08F	Lobster - Hepato	0.570	1.85E+00	4.41E+01	4.60E+01	1.78E-02	3.25E-02	2.78E-03	3.16E-02	6.44E-02	1.41E+00	1.03E-01	6.84E-02	(4.78E+00)	--	--	--	--	--
75223	08G	Lobster - Hepato	0.412	1.13E+00	3.77E+01	3.89E+01	9.56E-03	1.47E-02	1.91E-03	1.73E-02	3.38E-02	1.10E+00	4.03E-02	1.26E-02	1.96E+00	--	--	--	--	--
75228	08G	Lobster - Hepato	0.481	1.74E+00	4.51E+01	4.68E+01	1.74E-02	3.29E-02	3.17E-03	4.12E-02	9.57E-02	1.52E+00	1.14E-01	3.42E-02	2.55E+00	--	--	--	--	--
75249	08I	Lobster - Hepato	0.547	2.03E+00	(5.96E+01)	(6.18E+01)	1.53E-02	1.17E-02	1.12E-03	1.30E-02	3.25E-02	1.52E+00	1.01E-01	1.36E-02	4.28E+00	--	--	--	--	--
75230	08J	Lobster - Hepato	0.480	6.53E-01	2.22E+01	2.28E+01	8.64E-03	1.08E-02	1.26E-03	7.15E-03	1.50E-02	6.58E-01	1.82E-02	4.34E-03	2.26E+00	--	--	--	--	--
		AVERAGE		1.50E+00	4.24E+01	4.39E+01	1.37E-02	1.84E-02	1.78E-03	3.18E-02	6.57E-02	1.30E+00	1.00E-01	2.95E-02	3.37E+00	--	--	--	--	--
75259	08MHD	Soft Shell Clam	0.143	--	--	--	--	--	--	--	--	--	--	--	--	1.76	(0.245)	0.440	0.017	0.002
75260	08M1S	Soft Shell Clam	0.137	--	--	--	--	--	--	--	--	--	--	--	--	(1.95)	0.167	(0.460)	(0.025)	--
		AVERAGE		--	--	--	--	--	--	--	--	--	--	--	--	1.85	0.206	0.450	0.021	0.002
75237	08C	Lobster - Hepato	0.573	--	--	--	--	--	--	--	--	--	--	--	--	135	0.000*	0.000*	0.693	(0.070)
75237	08C	Lobster - Hepato	0.488	--	--	--	--	--	--	--	--	--	--	--	--	137	0.000*	0.000*	0.756	--
75219	08D	Lobster - Hepato	0.525	--	--	--	--	--	--	--	--	--	--	--	--	215	0.000*	(0.215)	1.64	0.000*
75219	08D	Lobster - Hepato	0.585	--	--	--	--	--	--	--	--	--	--	--	--	219	0.000*	0.000*	1.61	--
75244	08E	Lobster - Hepato	0.303	--	--	--	--	--	--	--	--	--	--	--	--	181	(2.38)	0.000*	1.58	0.000*
75244	08E	Lobster - Hepato	0.337	--	--	--	--	--	--	--	--	--	--	--	--	217	1.72	0.000*	(2.23)	--
75212	08F	Lobster - Hepato	0.533	--	--	--	--	--	--	--	--	--	--	--	--	176	0.634	0.000*	1.88	0.000*
75212	08F	Lobster - Hepato	0.500	--	--	--	--	--	--	--	--	--	--	--	--	188	0.960	0.000*	1.90	--
75223	08G	Lobster - Hepato	0.479	--	--	--	--	--	--	--	--	--	--	--	--	17.8	0.168	0.000*	1.11	0.000*
75223	08G	Lobster - Hepato	0.434	--	--	--	--	--	--	--	--	--	--	--	--	17.7	0.000*	0.000*	1.06	--
75228	08G	Lobster - Hepato	0.500	--	--	--	--	--	--	--	--	--	--	--	--	(279)	1.41	0.000*	1.25	0.046

TABLE B-10. WET WEIGHT CONCENTRATIONS OF ORGANIC COMPOUNDS AND METALS IN AQUATIC ORGANISMS FROM QUINCY BAY 1
(ug/g wet)

SAMPLE NO.	STATION	SAMPLE TYPE	g DRY/g WET RATIO	AROCLOR 1242	AROCLOR 1254	TOTAL PCBs	HCB	A-HCH	G-HCH	A-CHLOR	G-CHLOR	PP-DDE	PP-DDD	PP-DDT	TOTAL PAHs	Cu	Cr	Pd	Cd	Hg
75228	08G	Lobster - Hepato	0.494	--	--	--	--	--	--	--	--	--	--	--	--	252	2.01	0.000*	1.16	--
75249	08I	Lobster - Hepato	0.557	--	--	--	--	--	--	--	--	--	--	--	--	28.5	0.000*	0.000*	0.830	0.028
75249	08I	Lobster - Hepato	0.473	--	--	--	--	--	--	--	--	--	--	--	--	24.5	0.000*	0.000*	0.733	--
75230	08J	Lobster - Hepato	0.551	--	--	--	--	--	--	--	--	--	--	--	--	54.3	0.000*	0.000*	1.29	0.064
75230	08J	Lobster - Hepato	0.547	--	--	--	--	--	--	--	--	--	--	--	--	51.5	0.170	0.000*	1.27	--
		AVERAGE		--	--	--	--	--	--	--	--	--	--	--	--	137	0.590	0.013	1.31	0.026
75237	08C	Lobster - Tissue	0.237	--	--	--	--	--	--	--	--	--	--	--	--	3.77	0.000*	0.168	0.002	0.073
75237	08C	Lobster - Tissue	0.237	--	--	--	--	--	--	--	--	--	--	--	--	4.43	0.000*	0.166	0.002	--
75239	08C	Lobster - Tissue	0.255	--	--	--	--	--	--	--	--	--	--	--	--	(6.22)	0.033	(0.207)	0.002	0.079
75239	08C	Lobster - Tissue	0.255	--	--	--	--	--	--	--	--	--	--	--	--	5.79	0.036	0.171	0.001	--
75241	08C	Lobster - Tissue	0.232	--	--	--	--	--	--	--	--	--	--	--	--	2.67	0.190	0.176	0.001	0.050
75241	08C	Lobster - Tissue	0.230	--	--	--	--	--	--	--	--	--	--	--	--	2.69	(0.260)	0.152	0.001	--
75219	08D	Lobster - Tissue	0.249	--	--	--	--	--	--	--	--	--	--	--	--	5.25	0.000*	0.179	0.001	0.000*
75219	08D	Lobster - Tissue	0.241	--	--	--	--	--	--	--	--	--	--	--	--	5.93	0.000*	0.188	0.001	--
75220	08D	Lobster - Tissue	0.236	--	--	--	--	--	--	--	--	--	--	--	--	4.01	0.000*	0.182	0.001	0.065
75220	08D	Lobster - Tissue	0.235	--	--	--	--	--	--	--	--	--	--	--	--	4.61	0.000*	0.197	0.001	--
75244	08E	Lobster - Tissue	0.187	--	--	--	--	--	--	--	--	--	--	--	--	4.30	0.062	0.159	(0.005)	0.028
75244	08E	Lobster - Tissue	0.291	--	--	--	--	--	--	--	--	--	--	--	--	3.58	0.069	0.166	0.001	--
75245	08E	Lobster - Tissue	0.236	--	--	--	--	--	--	--	--	--	--	--	--	3.94	0.000*	0.170	0.002	(0.159)
75245	08E	Lobster - Tissue	0.230	--	--	--	--	--	--	--	--	--	--	--	--	4.55	0.000*	0.122	0.001	--
75212	08F	Lobster - Tissue	0.232	--	--	--	--	--	--	--	--	--	--	--	--	4.59	0.000*	0.179	0.001	0.104
75212	08F	Lobster - Tissue	0.228	--	--	--	--	--	--	--	--	--	--	--	--	3.85	0.000*	0.176	0.002	--
75214	08F	Lobster - Tissue	0.225	--	--	--	--	--	--	--	--	--	--	--	--	3.65	0.000*	0.149	0.003	0.168
75214	08F	Lobster - Tissue	0.222	--	--	--	--	--	--	--	--	--	--	--	--	4.13	0.013	0.173	0.003	--
75223	08G	Lobster - Tissue	0.237	--	--	--	--	--	--	--	--	--	--	--	--	3.32	0.000*	0.171	0.001	0.104
75223	08G	Lobster - Tissue	0.253	--	--	--	--	--	--	--	--	--	--	--	--	3.26	0.000*	0.147	0.001	--
75225	08G	Lobster - Tissue	0.235	--	--	--	--	--	--	--	--	--	--	--	--	3.95	0.000*	0.190	0.001	0.118
75225	08G	Lobster - Tissue	0.240	--	--	--	--	--	--	--	--	--	--	--	--	4.15	0.000*	0.197	0.001	--
75228	08G	Lobster - Tissue	0.213	--	--	--	--	--	--	--	--	--	--	--	--	2.96	0.009	0.143	0.001	0.065
75228	08G	Lobster - Tissue	0.285	--	--	--	--	--	--	--	--	--	--	--	--	2.88	0.003	0.185	0.001	--
75249	08I	Lobster - Tissue	0.240	--	--	--	--	--	--	--	--	--	--	--	--	4.08	0.000*	0.187	0.002	0.087
75249	08I	Lobster - Tissue	0.237	--	--	--	--	--	--	--	--	--	--	--	--	5.26	0.000*	0.121	0.003	--
75250	08I	Lobster - Tissue	0.246	--	--	--	--	--	--	--	--	--	--	--	--	3.37	0.002	0.167	0.001	0.053
75250	08I	Lobster - Tissue	0.239	--	--	--	--	--	--	--	--	--	--	--	--	3.73	0.002	0.177	0.001	--
75230	08J	Lobster - Tissue	0.239	--	--	--	--	--	--	--	--	--	--	--	--	3.92	0.000*	0.163	0.002	0.087
75230	08J	Lobster - Tissue	0.241	--	--	--	--	--	--	--	--	--	--	--	--	3.76	0.007	0.154	0.001	--
75234	08J	Lobster - Tissue	0.234	--	--	--	--	--	--	--	--	--	--	--	--	4.07	0.000*	0.178	0.002	0.091
75234	08J	Lobster - Tissue	0.230	--	--	--	--	--	--	--	--	--	--	--	--	3.11	0.000*	0.147	0.001	--
		AVERAGE		--	--	--	--	--	--	--	--	--	--	--	--	4.06	0.021	0.169	0.002	0.083
75124	08T1	Flounder(W)-Fresh	0.185	4.79E-03	5.68E-01	5.72E-01	1.28E-04	0.00E+00	0.00E+00	5.07E-04	2.28E-03	1.07E-02	1.87E-04	0.00E+00	--	--	--	--	--	--
75168	08I1	Flounder(W)-Fresh	0.210	1.39E-03	3.74E-01	3.76E-01	1.01E-04	0.00E+00	0.00E+00	3.23E-04	1.60E-03	5.77E-03	0.00E+00	0.00E+00	--	--	--	--	--	--
75185	08I1	Flounder(W)-Fresh	0.197	0.00E+00	1.59E-01	1.59E-01	8.47E-05	0.00E+00	0.00E+00	1.99E-04	7.09E-04	1.99E-03	2.94E-04	0.00E+00	0.00E+00	--	--	--	--	--
75190	09I1	Flounder(W)-Fresh	0.210	2.86E-03	1.28E-01	1.31E-01	8.61E-05	0.00E+00	0.00E+00	3.76E-04	9.60E-04	2.10E-03	2.56E-04	0.00E+00	--	--	--	--	--	--
75191	08I1	Flounder(W)-Fresh	0.205	0.00E+00	1.63E-01	1.63E-01	1.00E-04	0.00E+00	0.00E+00	1.64E-04	4.88E-04	2.42E-03	0.00E+00	0.00E+00	--	--	--	--	--	--
75194	08I1	Flounder(W)-Fresh	0.197	0.00E+00	4.45E-01	4.45E-01	9.85E-05	0.00E+00	0.00E+00	5.46E-04	3.01E-03	8.33E-03	2.68E-04	0.00E+00	--	--	--	--	--	--
75195	08I1	Flounder(W)-Fresh	0.191	0.00E+00	2.01E-01	2.01E-01	8.60E-05	0.00E+00	0.00E+00	5.86E-04	1.40E-03	3.82E-03	4.72E-04	0.00E+00	--	--	--	--	--	--
75198	08I1	Flounder(W)-Fresh	0.204	0.00E+00	2.41E-01	2.41E-01	1.86E-04	0.00E+00	0.00E+00	2.35E-04	7.04E-04	3.16E-03	3.73E-04	4.61E-04	--	--	--	--	--	--

TABLE B-10. WET WEIGHT CONCENTRATIONS OF ORGANIC COMPOUNDS AND METALS IN AQUATIC ORGANISMS FROM QUINCY BAY 1
(ug/g wet)

SAMPLE NO.	STATION	SAMPLE TYPE	g DRY/g WET RATIO	AROCLOR 1242	AROCLOR 1254	TOTAL PCBs	HCB	A-HCH[2]	G-HCH[2]	A-CHLOR	G-CHLOR	PP-DDE	PP-DDD	PP-DDT	TOTAL PAHs	Cu	Cr	Pd	Cd	Hg
75101	QBT2	Flounder(W)-Fresh	0.200	0.00E+00	1.81E-01	1.81E-01	1.26E-04	0.00E+00	0.00E+00	2.86E-04	7.84E-04	1.79E-03	0.00E+00	0.00E+00	0.00E+00	--	--	--	--	--
75113	QBT2	Flounder(W)-Fresh	0.190	0.00E+00	2.20E-01	2.20E-01	1.35E-04	0.00E+00	0.00E+00	2.85E-04	9.14E-04	2.36E-03	0.00E+00	0.00E+00	0.00E+00	--	--	--	--	--
75114	QBT2	Flounder(W)-Fresh	0.206	3.91E-03	3.96E-01	4.00E-01	2.18E-04	0.00E+00	0.00E+00	5.81E-04	1.63E-03	5.87E-03	8.94E-04	0.00E+00	--	--	--	--	--	--
75115	QBT2	Flounder(W)-Fresh	0.214	0.00E+00	1.09E-01	1.09E-01	0.00E+00	0.00E+00	0.00E+00	4.02E-04	7.64E-04	2.42E-03	1.03E-03	0.00E+00	--	--	--	--	--	--
75179	QBT2	Flounder(W)-Fresh	0.206	1.54E-03	1.64E-01	1.65E-01	0.00E+00	0.00E+00	0.00E+00	5.71E-04	1.05E-03	2.99E-03	1.19E-03	0.00E+00	--	--	--	--	--	--
75160	QBT2	Flounder(W)-Fresh	0.218	0.00E+00	2.02E-01	2.02E-01	1.33E-04	0.00E+00	0.00E+00	2.31E-04	6.21E-04	2.38E-03	0.00E+00	0.00E+00	--	--	--	--	--	--
75164	QBT3	Flounder(W)-Fresh	0.203	1.04E-02	3.15E-01	3.25E-01	1.66E-04	(8.93E-04)	0.00E+00	(7.67E-03)	(2.23E-02)	7.35E-03	(1.33E-02)	(4.97E-03)	0.00E+00	--	--	--	--	--
75167	QBT3	Flounder(W)-Fresh	0.200	1.10E-02	4.88E-01	4.98E-01	1.60E-04	1.12E-04	0.00E+00	1.49E-03	5.20E-03	1.22E-02	1.84E-03	1.16E-03	--	--	--	--	--	--
75170	QBT3	Flounder(W)-Fresh	0.195	0.00E+00	1.85E-01	1.85E-01	0.00E+00	7.41E-05	0.00E+00	4.41E-04	0.00E+00	3.84E-03	1.51E-03	4.49E-04	--	--	--	--	--	--
75172	QBT3	Flounder(W)-Fresh	0.190	(1.77E-02)	(7.26E-01)	(7.43E-01)	1.60E-04	1.56E-04	0.00E+00	1.26E-03	1.60E-04	1.37E-02	3.15E-03	1.96E-03	--	--	--	--	--	--
75180	QBT3	Flounder(W)-Fresh	0.206	0.00E+00	1.17E-01	1.17E-01	8.24E-05	2.88E-04	0.00E+00	1.36E-03	8.24E-05	2.82E-03	3.69E-03	1.32E-03	--	--	--	--	--	--
75182	QBT3	Flounder(W)-Fresh	0.209	0.00E+00	1.24E-01	1.24E-01	0.00E+00	0.00E+00	0.00E+00	1.09E-03	0.00E+00	1.76E-03	3.97E-03	0.00E+00	0.00E+00	--	--	--	--	--
75128	QBT1T3	Flounder(W)-Fresh	0.181	0.00E+00	3.33E-01	3.33E-01	1.57E-04	6.70E-05	0.00E+00	3.48E-04	1.30E-03	6.64E-03	6.21E-04	4.16E-04	0.00E+00	--	--	--	--	--
75133	QBT1T3	Flounder(W)-Fresh	0.184	0.00E+00	1.71E-01	1.71E-01	1.58E-04	8.65E-05	0.00E+00	7.14E-04	1.69E-03	3.90E-03	4.49E-04	6.46E-04	--	--	--	--	--	--
75145	QBT1T3	Flounder(W)-Fresh	0.195	0.00E+00	1.60E-01	1.60E-01	1.44E-04	1.93E-04	0.00E+00	8.48E-04	2.01E-03	4.08E-03	1.19E-03	7.88E-04	--	--	--	--	--	--
75148	QBT1T3	Flounder(W)-Fresh	0.191	8.00E-03	5.41E-01	5.48E-01	(2.52E-04)	1.28E-04	0.00E+00	2.16E-03	6.04E-03	(1.59E-02)	1.20E-03	1.18E-03	--	--	--	--	--	--
75149	QBT1T3	Flounder(W)-Fresh	0.200	0.00E+00	6.12E-02	6.12E-02	1.42E-04	0.00E+00	0.00E+00	0.00E+00	5.40E-05	1.51E-03	0.00E+00	0.00E+00	--	--	--	--	--	--
75124	QBT1	Flounder(W)-Fresh	0.203	--	--	--	--	--	--	--	--	--	--	--	--	0.093	0.000*	0.000*	0.000*	0.080
75168	QBT1	Flounder(W)-Fresh	0.207	--	--	--	--	--	--	--	--	--	--	--	--	0.122	0.000*	0.000*	0.000*	0.035
75185	QBT1	Flounder(W)-Fresh	0.209	--	--	--	--	--	--	--	--	--	--	--	--	0.144	0.008	0.000*	0.000*	0.045
75190	QBT1	Flounder(W)-Fresh	0.223	--	--	--	--	--	--	--	--	--	--	--	--	0.087	(0.377)	0.000*	0.000*	0.014
75191	QBT1	Flounder(W)-Fresh	0.205	--	--	--	--	--	--	--	--	--	--	--	--	0.105	0.000*	0.000*	0.000*	0.012
75194	QBT1	Flounder(W)-Fresh	0.205	--	--	--	--	--	--	--	--	--	--	--	--	0.096	0.000*	0.000*	0.000*	0.036
75195	QBT1	Flounder(W)-Fresh	0.200	--	--	--	--	--	--	--	--	--	--	--	--	0.078	0.010	0.000*	0.000*	0.016
75198	QBT1	Flounder(W)-Fresh	0.217	--	--	--	--	--	--	--	--	--	--	--	--	0.126	0.009	0.000*	0.000*	0.036
75101	QBT2	Flounder(W)-Fresh	0.207	--	--	--	--	--	--	--	--	--	--	--	--	0.048	0.000*	(0.008)	0.000*	0.047
75113	QBT2	Flounder(W)-Fresh	0.189	--	--	--	--	--	--	--	--	--	--	--	--	0.096	0.000*	0.000*	0.000*	(0.086)
75114	QBT2	Flounder(W)-Fresh	0.197	--	--	--	--	--	--	--	--	--	--	--	--	0.110	0.016	(0.008)	(0.009)	0.010
75115	QBT2	Flounder(W)-Fresh	0.221	--	--	--	--	--	--	--	--	--	--	--	--	0.159	0.000*	0.002	0.000*	0.011
75179	QBT2	Flounder(W)-Fresh	0.206	--	--	--	--	--	--	--	--	--	--	--	--	0.122	0.014	0.000*	0.000*	0.008
75160	QBT2	Flounder(W)-Fresh	0.198	--	--	--	--	--	--	--	--	--	--	--	--	0.042	0.000*	0.000*	0.000*	0.021
75164	QBT3	Flounder(W)-Fresh	0.208	--	--	--	--	--	--	--	--	--	--	--	--	0.079	0.000*	0.000*	0.000*	0.035
75167	QBT3	Flounder(W)-Fresh	0.206	--	--	--	--	--	--	--	--	--	--	--	--	0.142	0.004	0.000*	0.000*	0.041
75170	QBT3	Flounder(W)-Fresh	0.192	--	--	--	--	--	--	--	--	--	--	--	--	(0.215)	0.025	0.000*	0.000*	0.026
75172	QBT3	Flounder(W)-Fresh	0.193	--	--	--	--	--	--	--	--	--	--	--	--	0.197	0.000*	0.000*	0.000*	0.047
75180	QBT3	Flounder(W)-Fresh	0.210	--	--	--	--	--	--	--	--	--	--	--	--	0.036	0.002	0.002	0.000*	0.006
75182	QBT3	Flounder(W)-Fresh	0.212	--	--	--	--	--	--	--	--	--	--	--	--	0.102	0.008	0.000*	0.000*	0.010
75128	QBT1T3	Flounder(W)-Fresh	0.183	--	--	--	--	--	--	--	--	--	--	--	--	0.099	0.000*	0.000*	0.000*	0.024
75133	QBT1T3	Flounder(W)-Fresh	0.202	--	--	--	--	--	--	--	--	--	--	--	--	0.147	0.000*	0.000*	0.000*	0.016
75145	QBT1T3	Flounder(W)-Fresh	0.203	--	--	--	--	--	--	--	--	--	--	--	--	0.071	0.000*	0.000*	0.000*	0.019
75145	QBT1T3	Flounder(W)-Fresh	0.195	--	--	--	--	--	--	--	--	--	--	--	0.00E+00	--	--	--	--	--
75148	QBT1T3	Flounder(W)-Fresh	0.201	--	--	--	--	--	--	--	--	--	--	--	--	0.127	0.000*	0.000*	0.000*	0.053
75149	QBT1T3	Flounder(W)-Fresh	0.211	--	--	--	--	--	--	--	--	--	--	--	--	0.089	0.000*	0.000*	0.000*	0.017
		AVERAGE		2.46E-03	2.71E-01	2.73E-01	1.16E-04	7.99E-05	0.00E+00	9.07E-04	2.23E-03	5.19E-03	1.43E-03	5.34E-04	--	0.109	0.019	0.001	0.000	0.030

1. Adapted from U.S. EPA, December 2, 1987.
2. Also A BHC and G BHC, respectively.
* = Detection limit set equal to zero.
() = Maximum Concentration

Table B-11. ORGANICS AND METALS INCLUDED IN ANALYTICAL RESULTS PROVIDED BY US ENVIRONMENTAL PROTECTION AGENCY(a)

ELEMENTS/METALS

Silver
Arsenic
Beryllium
Cadmium
Cobalt
Chromium
Copper
Iron
Mercury
Magnesium
Manganese
Nickel
Lead
Antimony
Selenium
Thallium
Vanadium
Zinc

ORGANIC COMPOUNDS

Bis(2-Ethyl-Hexyl)Phthalate
Chlordane (total)
  a-Chlordane
  g-Chlordane
Coprostanol (Coprosterol)
PP-DDD
PP-DDE
PP-DDT
Hexachlorobenzene (HCB)
Hexachlorocyclohexane (HCH)
  a-HCH
  g-HCH (lindane)
Heptachlor
Methylene chloride
Methyl Chloride
Endrin
Toxaphene (chlorocamphene)
Polyarom. Hydrocarbons(PAH)
  Fluorene
  Phenanthrene
  Anthracene

ORGANIC COMPOUNDS(Continued)

C1PA(homologs/Phen-Anthr)
C2PA(homologs/Phen-Anthr)
C3PA(homologs/Phen-Anthr)
C4PA(homologs/Phen-Anthr)
Fluoranthene
Pyrene
Benzo [a] anthracene
Chrysene
Benzofluoranthenes (sum)
Benzo [e] pyrene
Benzo [a] pyrene
Perylene
Indeno [1,2,3-cd] pyrene
Benzo [ghi] perylene
PAHs (Sum of mol. weight 276)
PAHs (Sum of mol. weight 278)
Corene
PAHs (Sum of mol. weight 302)
Total of measured PAHs
PCBs (total) (a)
Aroclor 1242
Aroclor 1254
CB052 (2,2',5,5'-PCB)
CB047 (2,2',4,4'-PCB)
CB101 (2,2',4,5,5'-PCB)
CB151 (2,2',3,5,5',6-PCB)
CB118 (2,3',4,4',5-PCB)
CB153 (2,2,4,4',5,5'-PCB)
CB138 (2,2',3,4,4',5',-PCB)
CB128 (2,2',3,3',4,4',-PCB)
CB180 (2,2',3,4,4,5,5'-PCB)
CB195 (2,2',3,3',4,4',5,6-PCB)
CB194 (2,2',3,3',4,4',5,5'-PCB)
CB206 (2,2',3,3',4,4',5,5',6-PCB)
CB209 (CL10-PCB)

(a): Gardner & Pruell. 1987.

Appendix C

Development of Carcinogenic Potency Factor for PCBs

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

March 15, 1988

OFFICE OF
RESEARCH AND DEVELOPMENT

MEMORANDUM

SUBJECT: Cancer potency for Aroclor^{R} 1254

FROM: Jim Cogliano
Carcinogen Assessment Group (RD-689)

TO: Kevin Garrahan
Exposure Assessment Group (RD-689)

In response to your inquiry about a separate cancer potency for Aroclor^{R} 1254, I have prepared the following analysis.

My preliminary calculations indicate a cancer potency of 2.6 per mg/kg/d continuous lifetime exposure to Aroclor^{R} 1254. This is a plausible upper bound, meaning that the true potency is not likely to exceed this estimate and may be lower. It is based on the 1978 National Cancer Institute (NCI) study of Aroclor^{R} 1254, in which statistically significant, dose-related increases in liver nodules, benign tumors, and malignant tumors combined were seen in Fischer 344 rats fed a diet containing Aroclor^{R} 1254.

Several uncertainties deserve your attention:

1. NCI used only 24 rats per group (50 is considered standard today), so the potency estimate is rather imprecise.

2. The NCI study lasted 24 months. Although this is today's standard, a recent, longer study by Norback and Weltman indicates that PCB-fed rats develop many tumors after 24 months. CAG considers the Norback and Weltman study superior for estimating the potency. The NCI study is analogous to the study that was superseded by the Norback and Weltman study.

3. NCI's female rats developed only benign liver tumors and nodules, so some may argue that there was no cancer. Norback and Weltman, however, demonstrated that nodules progress to benign tumors, which in turn progress to malignant tumors. Under EPA's cancer guidelines it is, therefore, appropriate to consider benign tumors and nodules. Furthermore, some male rats did develop malignant liver tumors.

CAG's current cancer potency for Aroclor^{R} 1260, which is presumed to apply to other PCB mixtures as well, is 7.7 per mg/kg/d continuous lifetime exposure. CAG's previous estimate was 4.3 per mg/kg/d. In light of the uncertainties cited above, these figures are not substantially different from the new figure for Aroclor^{R} 1254. Larger differences are commonly seen between different sexes and animal strains. For example, a comparison of the NCI and Norback and Weltman studies suggests that Aroclor^{R} 1254 may be more potent in male Fischer 344 rats than Aroclor^{R} 1260 is in male Sprague-Dawley rats.

Further investigation, perhaps taking into consideration potency differences between PCB mixtures for other toxic effects, is needed before there can be separate cancer potencies for each PCB mixture. Until then, it appears that the cancer potency of Aroclor^{R} 1254 is either similar to, or slightly less than, that of Aroclor^{R} 1260.

Attached is a summary of the new potency calculation. If you have any questions, or if I can be of further assistance, please call me at 382-2575.

Attachment: Summary of potency calculation for Aroclor^{R} 1254

cc: Charles Ris

SUBSTANCE	Aroclor(R) 1254
REFERENCE	NCI, 1978
SEX, STRAIN, SPECIES	Female Fischer 344 rats
EXPOSURE ROUTE, VEHICLE	Oral, diet
TUMOR SITE, TYPE	Liver nodular hyperplasia and adenomas

NOMINAL DOSE	0	25	50	100	ppm
	0	1.25	2.50	5.00	mg/kg/d (5% food factor)
AVERAGE DAILY DOSE	0	1.16	2.32	4.65	mg/kg/d (105/113 weeks)
EQUIVALENT HUMAN DOSE	0	0.17	0.33	0.64	mg/kg/d (surf-area adj)
TUMOR INCIDENCE	0/24	6/24	10/22	19/24	
TUMOR PERCENTAGE	0%	25%	45%	79%	
STATISTICAL SIGNIFICANCE	--	1E-02	2E-04	1E-08	
TREND SIGNIFICANCE	<0.001, linearity OK				
ANIMAL WEIGHT	250	220	200	180	g (at end of study)
EXPOSURE PERIOD	105 wk				
STUDY LENGTH	113 wk				
ANIMAL LIFESPAN	113 wk (assumed)				
POTENCY (q1*)	2.6 per mg/kg/d				

J. Cogliano 16:14 09-Mar-88

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 16 1988

OFFICE OF
RESEARCH AND DEVELOPMENT

MEMORANDUM

SUBJECT: Congener-Specific Analysis of Quincy Bay Biota Samples

FROM: Susan Braen Norton, Environmental Scientist
Exposure Assessment Applications Branch
Exposure Assessment Group (RD-689)

TO: William H. Farland, Ph.D.
Acting Director
Office of Health and Environmental Assessment (RD-689)

THRU: Michael A. Callahan, Director
Exposure Assessment Group (RD-689)

One of the concerns expressed in the March 1 meeting of the Fish Contamination Committee was that the mixture of PCBs measured in seafood from Quincy Bay may be more like Aroclor 1254 than Aroclor 1260. This concern was raised because the cancer potency factor for Aroclor 1260 was used to assess risks associated with the ingestion of seafood from Quincy Bay.

To address this issue, I conducted a simple analysis using the thirteen congeners that were measured in Quincy Bay seafood (U.S. EPA 1987). The conclusions of this analysis are that, based on the 13 congeners measured, the mixture of PCBs in the seafood resembles Aroclor 1254 more closely than Aroclor 1260 or Aroclor 1242.

Bar graphs of the 13 congeners measured in flounder, clams, oysters, lobster flesh, and lobster hepatopancreas are attached. The congener concentrations in these graphs have been normalized relative to congener 138 (2,2',3,4,4',5-PCB) in order to more easily distinguish patterns. Also attached are bar graphs of the normalized congener concentrations present in the commercial mixtures Aroclors 1254, 1242, and 1260 (as per Rapaport and

Eisenreich 1984; and Capel et al. 1985). On the basis of these graphs, Aroclors 1254 and 1260 were selected for further analysis.

To more quantitatively compare the PCBs in seafood with the commercial PCB mixtures, I summed the squares of the differences between each of the normalized congener concentrations in the seafood and the commercial mixture. The results of the sums of squares analysis are also attached. As can be seen, the mixture of PCBs measured in oyster tissue most resembles the commercial mixture Aroclor 1254 as quantified by Rapaport and Eisenreich, (1984). Residues measured in flounder, clams, and lobster flesh and hepatopancreas most closely resemble Aroclor 1254 as reported by Capel et al. (1985).

There are several important uncertainties in using the results of this analysis in risk assessment:

1. The analysis was based on only 13 of 209 possible PCB congeners. However, the 13 congeners vary greatly with chlorination; for the purposes of this analysis, they were considered to sufficiently represent the large range of possible congeners.

2. No congener-specific data were available on the actual PCB mixture that was fed to the test animals in the cancer bioassays. Because the congener concentrations can vary greatly with batch, the congener concentrations reported in the literature may differ from those used in the bioassays.

3. Congener-specific toxicity data are not yet available. Because it is not known whether the most toxic PCB congeners were used to compare seafood residues to the commercial mixtures, the PCB mixture in the seafood may actually be more or less toxic than Aroclor 1254.

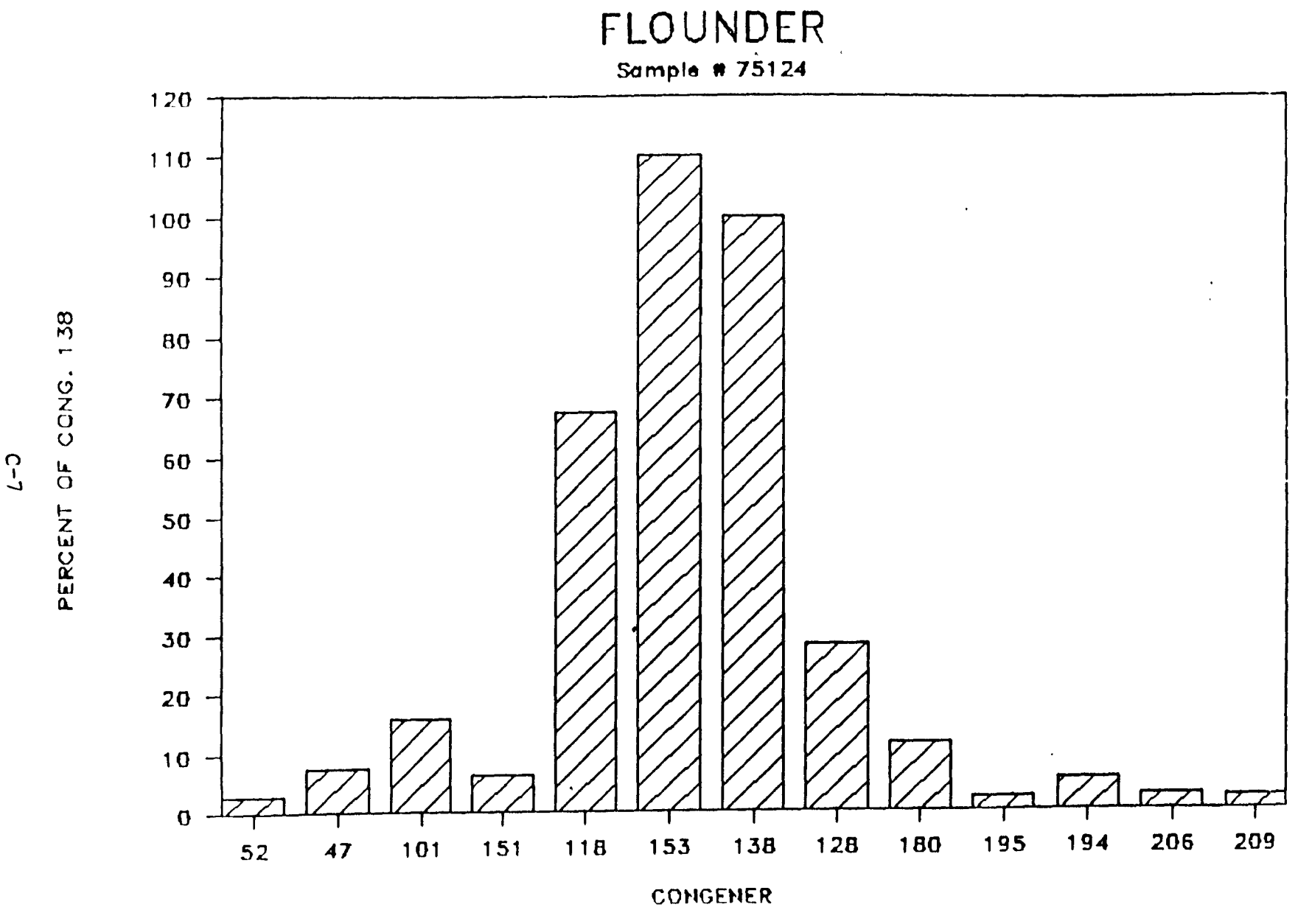
Attachments

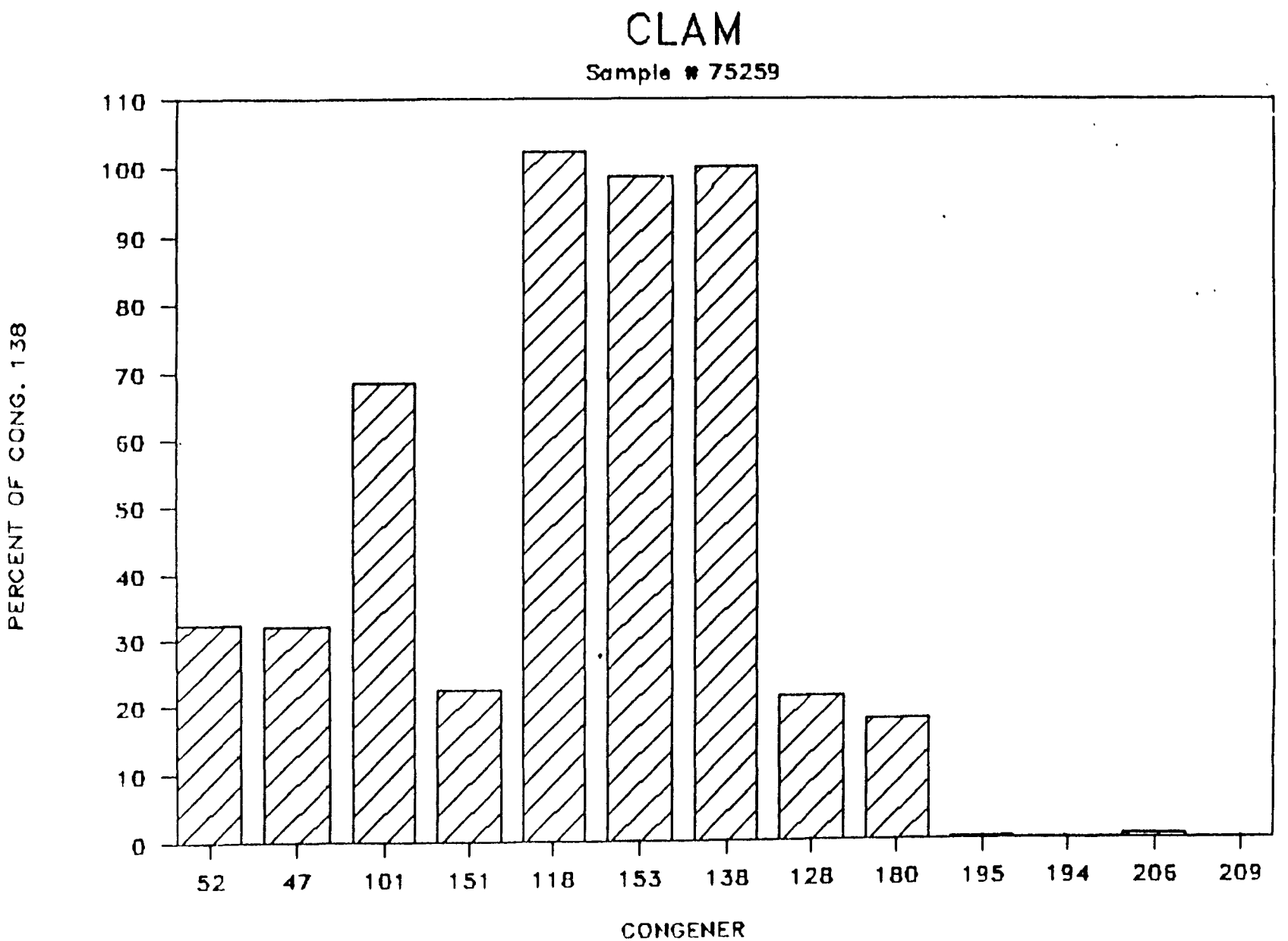
REFERENCES

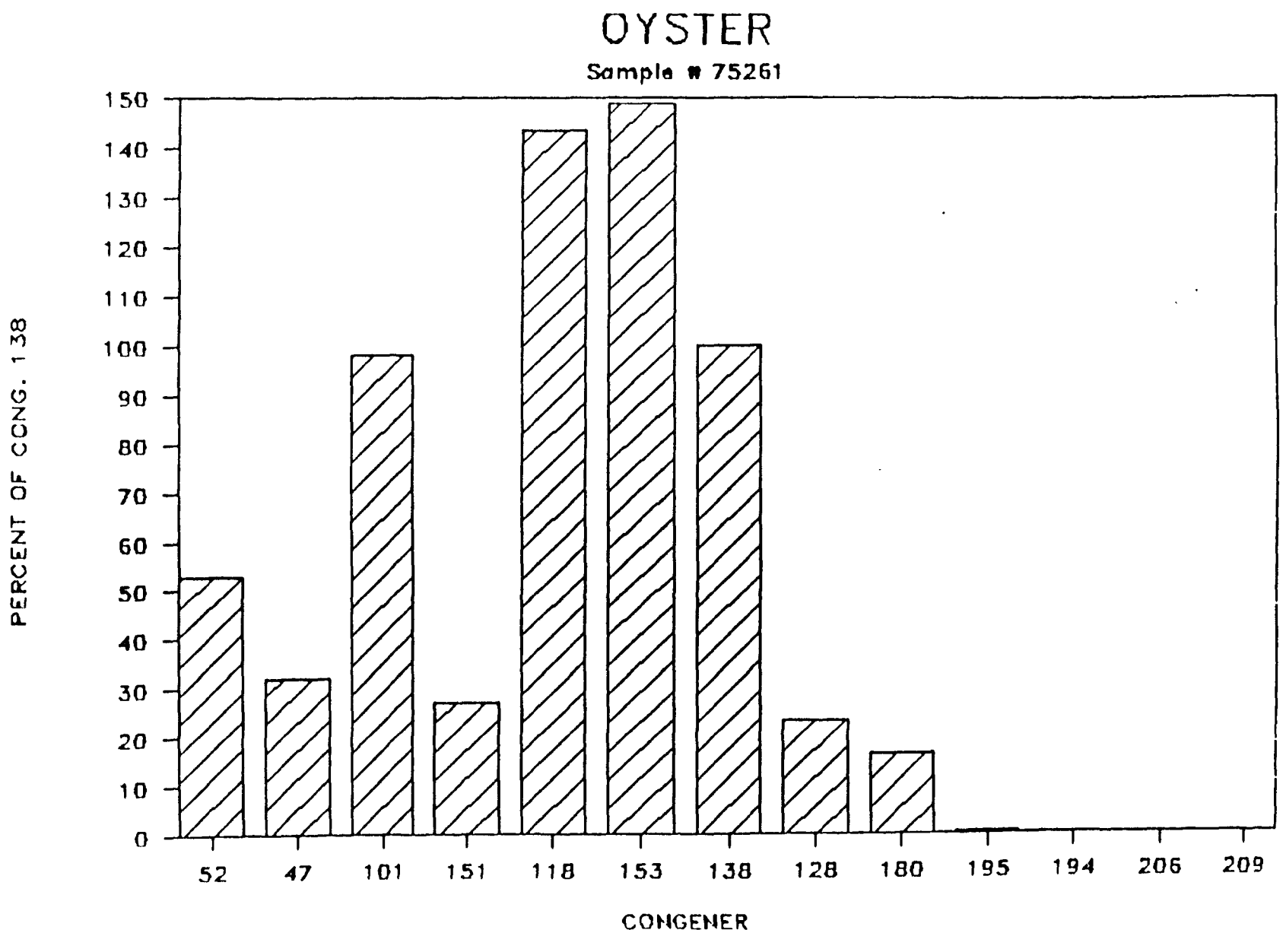
Capel, P.D., Rapaport, R.A., Eisenreich, S.J., and Looney, B.B. 1985. PCBQ: Computerized Quantification of Total PCB and Congeners in Environmental Samples. Chemosphere 14: 439-450

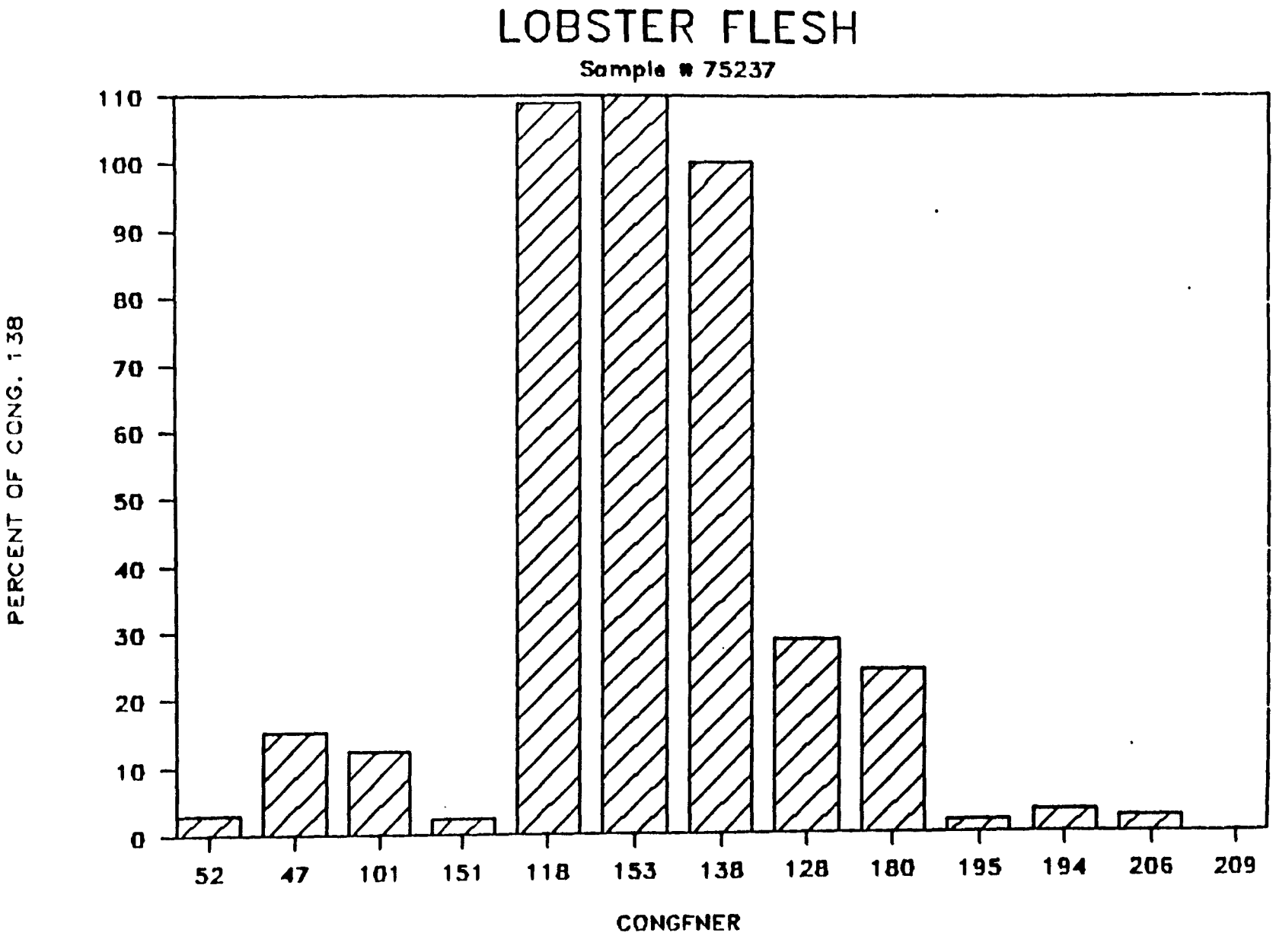
Rapaport, R.A. and Eisenreich, S.J. 1984. Chromatographic determination of octanol-water partition coefficients (Kow's) for 58 polychlorinated biphenyl congeners. Environ. Sci. Technol. 18: 163-170

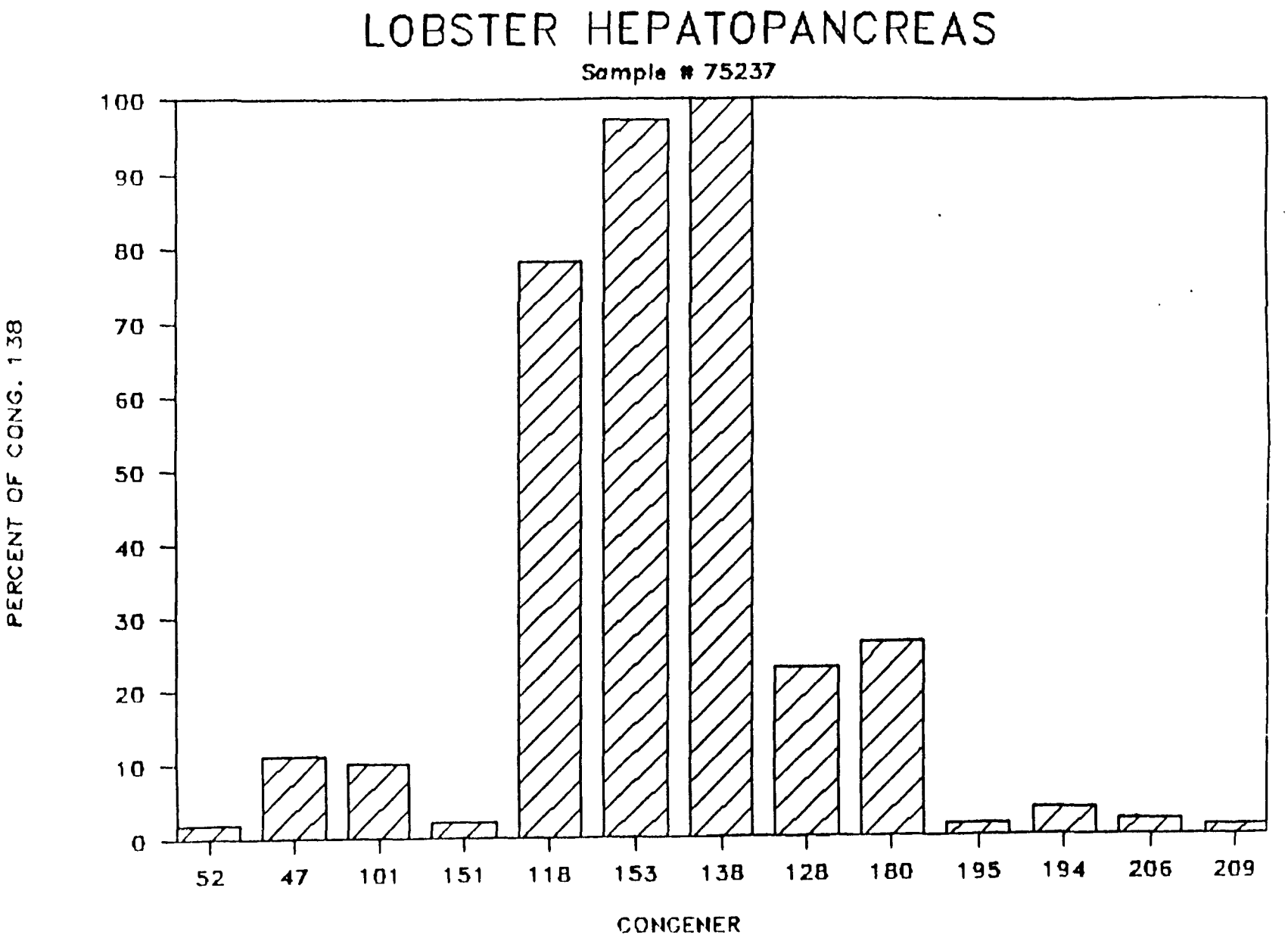
U.S. Environmental Protection Agency (USEPA). 1987. A Histopathological and Chemical Assessment of Winter Flounder, Lobster, and Soft-Shelled Clam Indigenous to Quincy Bay, Boston Harbor and an *in situ* Evaluation of Oysters Including Sediment (Surface and Cores) Chemistry. Environmental Research Laboratory Narragansett, Rhode Island. December 1, 1987

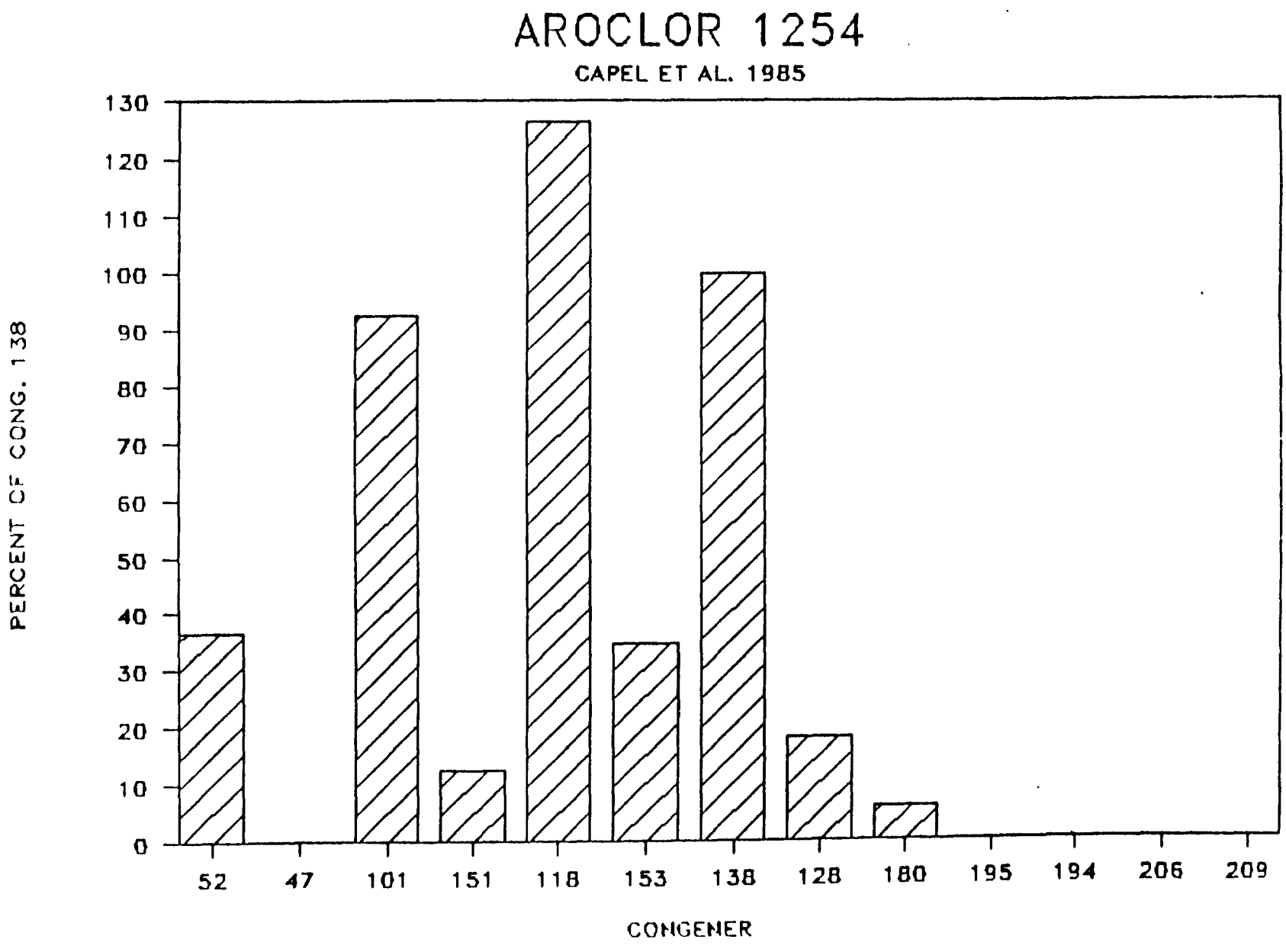

FLOUNDER
Sample # 75124
PERCENT OF CONG. 138
120
110
100
90
80
70
60
50
40
30
20
10
0
52
47
101
151
118
153
138
128
180
195
194
206
209
CONGENER


CLAM
Sample # 75259
PERCENT OF CONG. 138
110
100
90
80
70
60
50
40
30
20
10
0
52
47
101
151
118
153
138
128
180
195
194
206
209
CONGENER


OYSTER
Sample # 75261
PERCENT OF CONG. 138
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
52
47
101
151
118
153
138
128
180
195
194
206
209
CONGENER

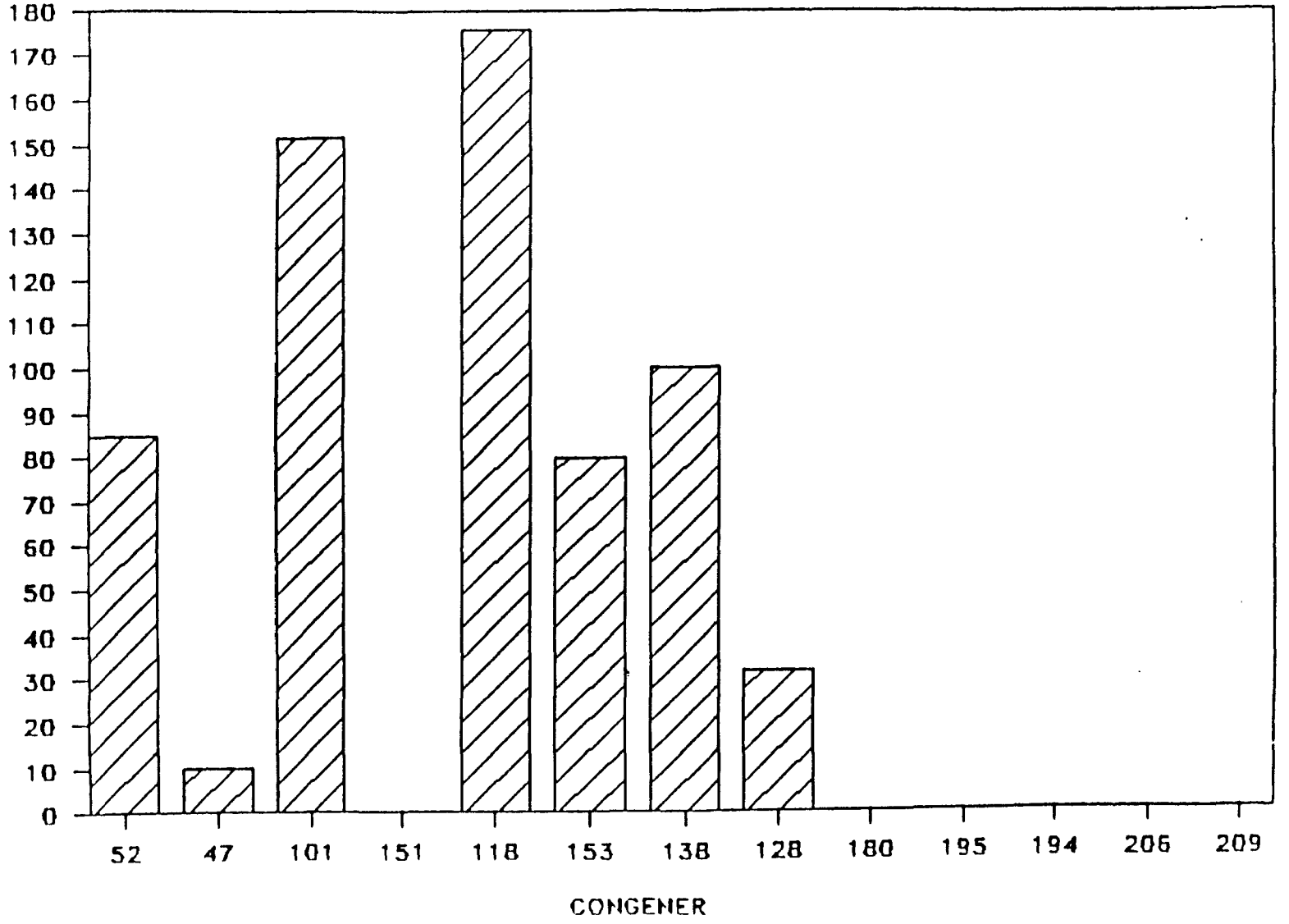

LOBSTER FLESH
Sample # 75237
PERCENT OF CONG. 138
110
100
90
80
70
60
50
40
30
20
10
0
52
47
101
151
118
153
138
128
180
195
194
206
209
CONGENER

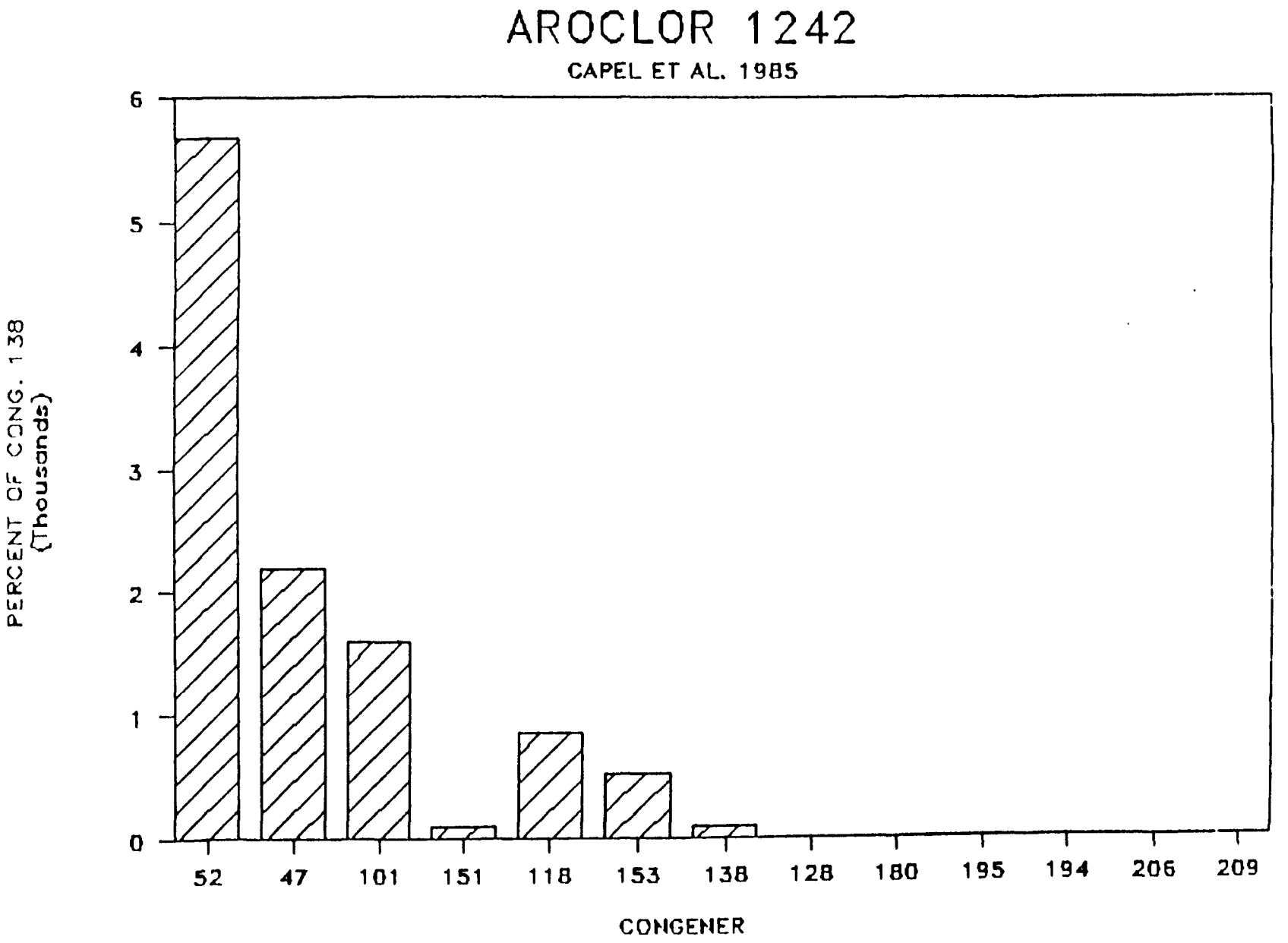

LOBSTER HEPATOPANCREAS
Sample # 75237
PERCENT OF CONG. 138
100
90
80
70
60
50
40
30
20
10
0
52
47
101
151
118
153
138
128
180
195
194
206
209
CONGENER

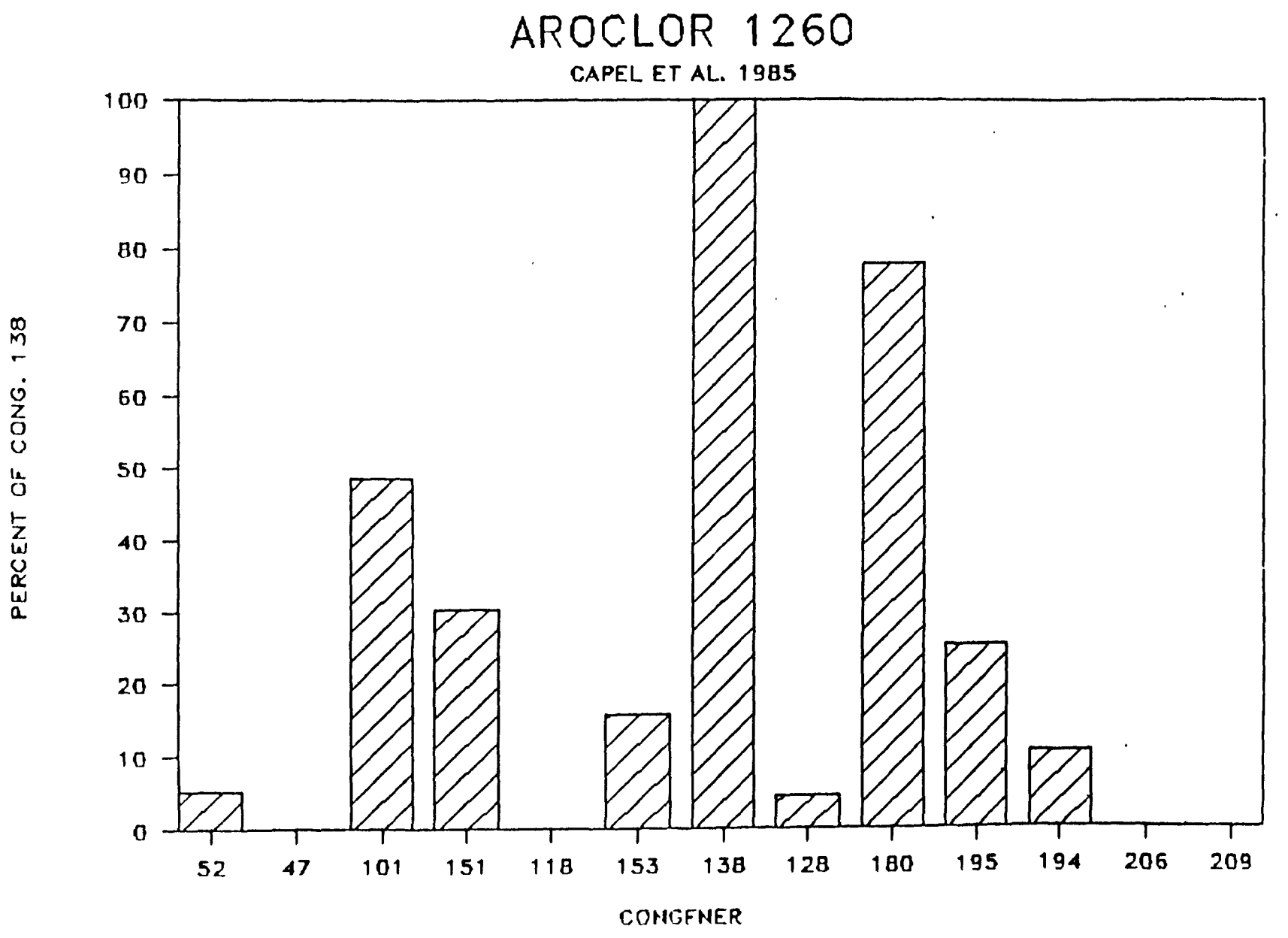

AROCLOR 1254
CAPEL ET AL. 1985
PERCENT OF CONG. 138
130
120
110
100
90
80
70
60
50
40
30
20
10
0
52
47
101
151
118
153
138
128
180
195
194
206
209
CONGENER

AROCLOR 1254

RAPAPORT AND EISENREICH 1984




AROCLOR 1242
CAPEL ET AL. 1985
PERCENT OF CONG. 138
(Thousands)
6
5
4
3
2
1
0
52
47
101
151
118
153
138
128
180
195
194
206
209
CONGENER


AROCLOR 1260
CAPEL ET AL. 1985
PERCENT OF CONG. 138
100
90
80
70
60
50
40
30
20
10
0
52
47
101
151
118
153
138
128
180
195
194
206
209
CONGENER

SUMS OF SQUARES COMPARISON BETWEEN QUINCY BAY SAMPLES AND LITERATURE VALUES

Sample Type	Sample Number	1254 (b)	1254 (a)	1260 (a)
Oyster	75261	11227 *	15053	48971
	75253	7578 *	18044	60767
	75255	7114 *	13699	54969
	75254	7715 *	9603	44954
	75256	6952 *	17593	63189
	75257	6539 *	16249	60065
Clam	75259	16850	6563 *	24173
	75260	11577	6537 *	31195
Lobster Flesh	75237	32169	14359 *	27108
	75239	31808	14633 *	28282
	75241	31489	17918 *	36550
	75219	27380	11104 *	25924
	75220	30404	12880 *	26322
	75244	28888	12859 *	28772
	75245	29832	13163 *	25922
	75212	31933	14221 *	25696
	75214	30014	12324 *	24942
	75223	32710	13691 *	24811
	75225	28051	10356 *	23140
	75228	32237	14601 *	27604
	75249	29021	12647 *	30535
	75250	31601	14819 *	29737
	75230	33365	16071 *	30424
	75234	34883	15928 *	26394
Lobster Hepato-pancreas	75237	37540	14927 *	18762
	75219	33769	11642 *	16306
	75244	34388	14272 *	20574
	75212	37119	14517 *	17962
	75223	37460	14677 *	18258
	75228	61796	39223 *	43196
	75249	36212	13482 *	18369
	75230	37267	16343 *	22266

* The sample most resembles the denoted mixture.

(a) Capel, P.D., Rapaport, R.A., Eisenreich, S.J., and Looney, B.B. 1985. PCBQ: Computerized Quantification of Total PCB and Congeners in Environmental Samples. Chemosphere 14: 439-450

(b) Rapaport, R.A., and Eisenreich, S.J. 1984. Chromatographic determination of octanol-water partition coefficients (Kow's) for 58 polychlorinated biphenyl congeners. Environ. Sci. Technol. 18: 163-170

SUMS OF SQUARES COMPARISON BETWEEN QUINCY BAY SAMPLES AND LITERATURE VALUES (continued)

Sample Type	Sample Number	1254 (b)	1254 (a)	1260 (a)
Flounder	75124	38091	16492 *	20642
	75168	39240	19739 *	23926
	75185	38799	20954 *	25383
	75190	35293	16045 *	19456
	75191	36659	17618 *	19812
	75194	40640	17993 *	19216
	75195	34944	15859 *	18789
	75198	43187	23745 *	24368
	75101	39044	18299 *	19839
	75113	43059	23844	23843 *
	75114	39194	19690 *	21606
	75115	24979	10781 *	21346
	75179	31190	17733 *	28029
	75160	34156	17567 *	24312
	75164	29918	12239 *	20876
	75167	33996	15024 *	22622
	75170	31627	18337 *	27089
	75172	38724	19059 *	22791
	75180	24708	12261 *	26087
	75182	35733	18046 *	23313
	75128	39303	18148 *	18788
	75133	35544	15479 *	20236
	75145	36571	18824 *	22508
	75148	36325	15417 *	19947
	75149	27244	14093 *	23345

* The sample most resembles the denoted mixture.

(a) Capel, P.D., Rapaport, R.A., Eisenreich, S.J., and Looney, B.B. 1985. PCBQ: Computerized Quantification of Total PCB and Congeners in Environmental Samples. Chemosphere 14: 439-450

(b) Rapaport, R.A., and Eisenreich, S.J. 1984. Chromatographic determination of octanol-water partition coefficients (Kow's) for 58 polychlorinated biphenyl congeners. Environ. Sci. Technol. 18: 163-170