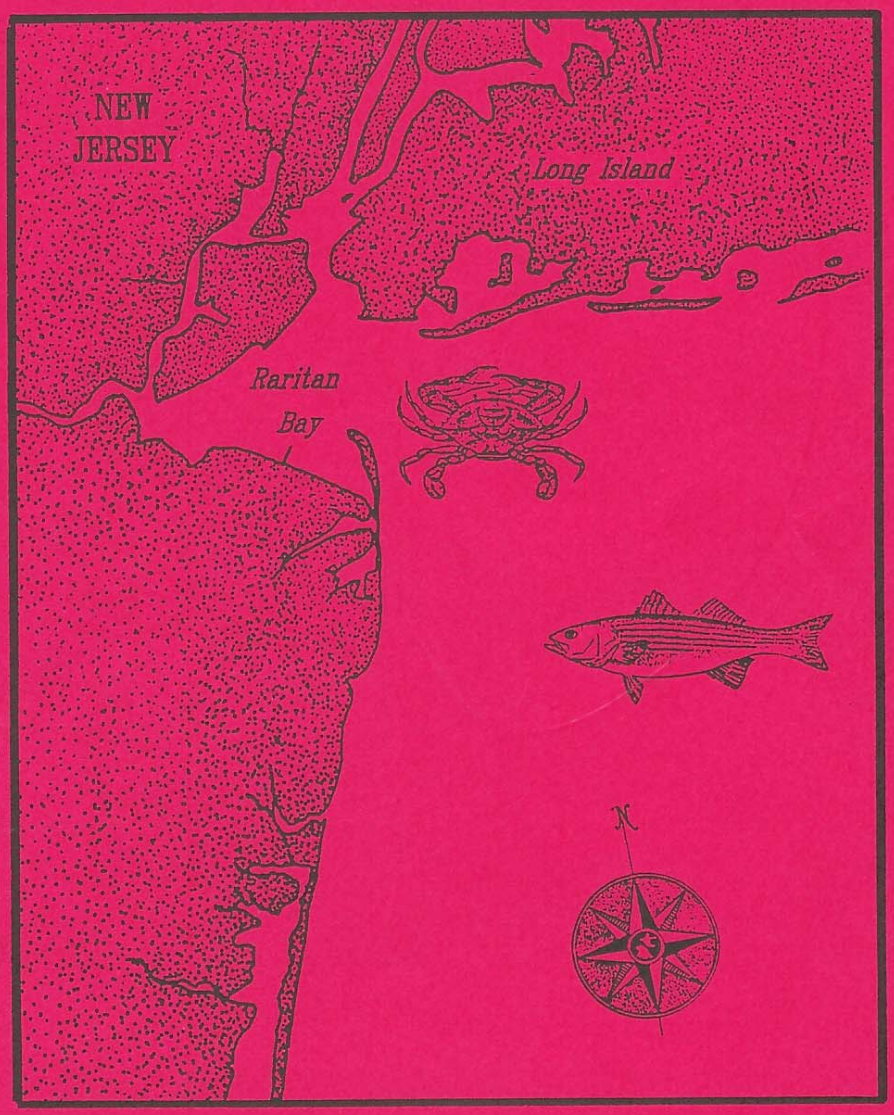
NJ Department of Environmental Protection

A Study of Dioxin in Aquatic Animals and Sediments



Office of Science and Research

205856

A Study of Dioxin (2,3,7,8-Tetrachlorodibenzo-p-Dioxin) Contamination in Select Finfish, Crustaceans and Sediments of New Jersey Waterways

by

Thomas J. Belton, M.A.
Robert Hazen, Ph.D.
Bruce E. Ruppel
Keith Lockwood
Robert Mueller, M.S.
Edward Stevenson
JoAnn J. Post, M.A.

Office of Science and Research
New Jersey Department of Environmental Protection

CN 409

Trenton, New Jersey 08625

October 30, 1985

Thomas H. Kean
Governor

Robert E. Hughey
Commissioner,Department of Environmental Protection

Thomas A. Burke, Ph.D., M.P.H.
Director, Office of Science and Research

Thomas J. Belton
Manager, Water and Biota Unit

Acknowledgements/Disclaimer

The office of Science and Research, NJDEP would like to thank the U.S. Environmental Protection Agency's Region II Emergency Response Group for their financial and scientific support during the earlier phase of this work and to thank the N.J. Spill Research Fund for similar support during phase two. The contents of this report, however does not necessarily reflect the views and policies of the USEPA, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

Table of Contents

Section	Title		Page
	Executive Summary		1-3
1.	Introduction		4-6
2.	Methods		7-9
3.	Sediments		
	3.1 Results	Phase I	10
		Phase II	10
	3.2 Discussion		11-13
4.	Finfish and Crustaceans		
	4.1 Results	Phase I	14
		Phase 11	15-17
	4.2 Discussion		18-27
5.	Risk Assessment		28
6.	N.J. Regulatory Actions		29-31
7.	Conclusions		32-33
8.	References		34-40
9.	Bibliography (Texts Available at OSR Library)		41-50
10.	Additional Sources of Information (Telephones)		51
11.	Appendices		
	A. EPA Classification Scheme for Potential Dioxin Sites		52-53
	B. FDA Levels of Concern		54-58
	C. Maps		59-68
	D. Figures		69-73
	E. Tables		74-97
	F. Methodology for Analysis of 2,3,7,8-TCDD in Fish		98-102

List of Maps Fires and Tables

Appendix C		Page
Map 1	Potential Dioxin Contamination Sites	60
Map 2	Phase I Passaic River Sediment Sampling Location and Database	61
Map 3	Phase I Biota Sampling Locations and Database	62
Map 4	Phase II, 2,3,7,8-TCDD Biota Sampling Locations	63
Map 5	Phase II, 2,3,7,8-TCDD Biota Database Newark Bay Complex (Positive Results)	64
Map 6	Phase II, 2,3,7,8-TCDD Biota Database Northern Coastal Waters (Positive Results)	65
Map 7	Lobster Sampling Locations in New York Bight Waters	66
Map 8	Commercial Fisheries: Lobster and Red Crab	67
Map 9	Revised Prohibition Area	68
Appendix D		
Figure 1	Newark Bay Area Photo	70
Figure 2	Signs Posted on Waterways	71
Figure 3	Blue Crab Anatomy	72
Figure 4	American Lobster Anatomy	73
Appendix E		
Table I	2,3,7,8-TCDD Concentrations in Sediments and Biota from Raccoon Creek	75
Table II	Phase II Complete Dioxin Fish Tissue Data (2,3,7,8-TCDD)	76-88
Table III	Positive Results for 2,3,7,8-TCDD In Biota (Phase II)	89-90
Table IV	Comparison of 2,3,7,8-TCDD In Passaic River Finfish and Crustaceans (Phase I and II)	91
Table V	Blue Crabs and American Lobster: 2,3,7,8-TCDD Concentrations by Tissue Type for all Locations (Phase II)	92
Table VI	2,3,7,8-TCDD Results for Striped Bass Caught at Confluence of Passaic River and Newark Bay and Their Relationship to Two FDA "Levels of Concern"	93
Table VII	Volumetric Sedimentation Rates in Channel and Off-Channel Areas of (Newark Bay) Study Areas	94
Table VIII	PCB and Pesticide Analysis of American Lobsters (Homarus americanus) Caught in New Jersey Waters	95
Table IX	Carcinogen Risk Assessment	96
Table X	Mean 2,3,7,8-TCDD Results for all Phase II Samples and Percentage of Samples Exceeding FDA "Level of Concern"	97

Executive Summary

Extensive contamination of soils by the chemical toxin 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) was discovered by the New Jersey Department of Environmental Protection (NJDEP) at the former Diamond-Alkali site, Newark, N.J., in the summer of 1983 (Range: 0.06-50 ppm). This prompted the Office of Science and Research (OSR) within NJDEP to immediately undertake an aquatic study of sediments and biota (i.e. finfish and crustaceans) in the tidal Passaic River which flows past the property. After this initial investigation (Phase I) uncovered 2,3,7,8-TCDD contamination in the sediments and biota of this tidal section a second study was undertaken (Phase II) targeted at investigating the potential extent of dioxin contamination both upstream and downstream of this site as well as other potential dioxin contaminated areas in other waterways of the State.

Sediment concentrations of 2,3,7,8-TCDD were discovered at three locations during this survey. The highest levels were found adjacent to the Diamond-Alkali site in spite of the fact that the plant has not been operating for twelve years (Range: non-detectable to 6.9 ppb). This indicates that continuous release of the contaminant has probably occurred over time due to either the weathering o£ soils, surface runoff, groundwater discharge or a combination of all three. A second set of TCDD contaminated sediments (0.11 ppb and 0.18 ppb) were found upstream at the confluence of the Passaic and Third Rivers adjacent to the Givaudan Chemical Company whose property was shown to contain dioxin contaminated storm drains and soils. Finally, a sediment sample from Raccoon Creek; Gloucester County was shown to have TCDD contaminated sediments (Mean = 0.03 ppb) as well as biota (white perch = 42 ppt). This last site was the only site to show dioxin contamination of sediments and biota not directly related to the Passaic River - Newark Bay System.

The data developed from this study during both Phase I and II sampling indicate that certain faunal species within the Passaic River, Newark Bay and its tributaries as well as the oceanic waters of the New York Bight are contaminated with 2,3,7,8-TCDD in excess of the two Food and Drug Administration (FDA) "Levels of Concern". The two FDA "Levels of Concern" are 25 and 50 parts per trillion (ppt), respectively. If exceeded these levels provide consumption recommendations to fish eaters in order to reduce human health risks for fish contaminated at these levels. For results in excess of 50 ppt the FDA recommends no consumption. For results between 25 and 50 ppt they recommend no more than one meal a week for infrequent consumers and no more than 1-2 times a month for frequent consumers of the fish. For results less than 25 ppt they place no limited on consumption.

Analysis of finfish and crustaceans for 2,3,7,8-TCDD in Phase I (1983) was restricted to the tidal Passaic River and showed that both resident and migratory species has elevated levels of dioxin in their edible tissue: carp (Mean = 110 ppt); catfish (Mean = 62 ppt); goldfish (66 ppt); American eel (Mean = 38 ppt); striped bass (Mean -± 45 ppt); and blue crab (Mean muscle tissue = 21 ppt and Mean hepatopancreas = 476 ppt). The Phase II (1984) tidal Passaic results from TCDD in biota replicated the results from Phase I although samples taken above the head of tide (Dundee Dam) showed no detectable levels of TCDD indicating that dioxin contamination is probably occurring only in the tidal section from the known point sources. Phase II collections of the same migratory species that were found contaminated on the tidal Passaic River (i.e. striped bass, blue crab and American eel) were then undertaken the contiguous waters of the Newark Bay-Hudson River-New York Bight Complex. The resulting data showed that these migratory species were found contaminated with TCDD in these adjacent waters as well.

Blue crabs revealed the highest tissue levels of TCDD identified in the study (Mean = 134 ppt for muscle and hepatopancreas mixture) whereas blue crabs from control sites (Great Egg Harbor and Delaware Bay) showed no detectable levels of TCDD. Phase II striped bass TCDD levels (Mean = 40 ppt) were consistent with Phase I levels from the Passaic River as well as New York of Environmental Conservation striped bass samples collected from the Hudson River. In Phase II a limited number of organisms from the contiguous ocean waters of the New York Bight were also analyzed including bluefish and American lobsters. We found that a small number of the bluefish fillets had elevated levels of TCDD (Mean = 45 ppt), and that the American lobster showed consistently high levels of TCDD in their hepatopancreas (Mean 77 ppt) (an edible organ) and combined muscle and hepatopancreas (Mean = 44 ppt) very similar to the results obtained for blue crabs. The latter finding held true for lobsters caught both at the mouth of the New York Harbor and twenty miles offshore indicating either a migratory movement of the animals or else possible exposure to TCDD from offshore sources.

The presence of TCDD contamination greater than the FDA "Levels of Concern" and the fact that there are commercial fishing closures and consumption advisories already in affect on these drainages due to PCB/pesticide contamination prompted DEP to further analyze the potential health effects of eating these finfish and shellfish species. To do this quantitative risk assessment methods were applied to the data and confirmed the unacceptable risk associated with using this fishery as a food source. As a result of this study the Commissioners of the New Jersey Department of Environmental Protection and Health then ordered in August of 1984 a prohibition on the sale and consumption of all fish and shellfish taken from the tidal Passaic River and also extended that ban to include striped bass and blue crabs taken from Newark Bay, the tidal Hackensack River, the Arthur Kill and the Kill Van Kull. Due to the limited information on lobsters, however, it was decided to gather more data prior to coming to any conclusions regarding these species.

The importance of these findings has stimulated OSR to continue TCDD related studies for 1985 in the areas of sediment transport and storage of dioxin within the affected drainages and a further study of lobster and blue crab contamination. Also because of the possibility of widespread contamination of this estuary due to 2,3,7,8-TCDD it is proposed that a long term research endeavor with requisite funding be established to finance future dioxin-related research issues. The lessons of New York State and the transport of thousands of kilograms of PCB laden sediments from the Ft. Edward section of the Hudson River down to New York Harbor and the inner New York Bight make it imperative that NJDEP continue to explore the ramifications of this TCDD contamination as it applies to such issues as disposal options for contaminated dredge spoils, the implications of migratory fish bioaccumulation, and possible detoxification technologies for dioxin contaminated sediments and soils. Only through long term monitoring of the problem, applied research and risk assessment can the State of New Jersey attempt to understand and manage this important contamination event.

1. Introduction

2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is one form of a group of seventy five tricyclic aromatic compounds containing from one to eight chlorine atoms. This particular TCDD isomer has four chlorine atoms at the 2,3,7 and 8 positions. There are no reports of dioxins being formed biosynthetically by living organisms nor do they have any desirable industrial properties (EPA 1980). They are inevitable, unwanted impurities produced mainly during the manufacture of other chlorinated chemicals such as the chlorophenols, especially 2,4,5-trichlorophenol (Firestone 1972). Chlorophenols are widely used throughout the world as pesticides and feedstocks for many other products. The most notable use of 2,4,5-trichlorophenol is in the manufacture of the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4.5-T) (EPA 1980a).

2,3,7,8-TCDD is considered by many toxicologists to be perhaps the most toxic synthetic chemical ever developed (Poland and Kende 1976). In animals it has been shown to be teratogenic, embryotoxic, carcinogenic and cocarcinogenic (Neubert and Dillman 1972, Courtney 1976, Kociba et al 1978, and Kouri et al 1978). The United States Environmental Protection Agency (EPA) has stated that because of the remarkable stability of this substance in biological systems and its extreme toxicity the cumulative effects of ever. extremely small doses are a major concern (EPA 1980).

The Office of Science and Research (OSR) within the New Jersey Department of Environmental Protection (NIDEP) has an institutional role heavily weighted toward public health concerns with the identification of new environmental issues and analytical needs as important corollary functions. In line with these objectives the discovery of polychlorinated dioxin (PCDD) contamination in soils (Missouri) and fish (New York and Michigan) which surfaced in 1982-1983 stimulated OSR to investigate the ramifications of such findings for New Jersey. An EPA document entitled "Dioxins" (EPA-600/2-80-197) listed several sites in New Jersey where dioxin contamination might be expected.

The Industrial Investigations Unit within OSR researched and compiled an inventory of potential dioxin contamination sites focusing on Class I and II organic chemical and pesticide producers as defined in the EPA report (see Appendix A). Research included: 1) a review of the New Jersey Industrial Survey records which are a data base developed from a mandatory survey of New Jersey industries concerning the manufacture, use, storage, processing, formation, release, disposal and repackaging of a group of chemical substances selected on the basis of their carcinogenicity or toxicity; 2) a review of state industrial directories and state library archives; 3) a review of all DEP/EPA inspection reports for active industries, and 4) some on-site investigations of former pesticide manufacturing locations. The EPA listings were checked against existing DEP information to confirm which New Jersey companies made or used the compounds most likely to have dioxin as a by-product. The result of this effort was an inventory of possible dioxin contaminated sites (see Map 1).

The former Diamond-Alkali plant located on the Lower Passaic River in Newark, New Jersey was determined to be a primary candidate for investigation of potential dioxin contamination (see Figure 1). This was due to the large quantities of the herbicide Agent Orange produced at this facility over a short period of time and the high incidence of chloracne reported for its workers in various health journals (Bleiberg et al 1964; Poland et al 1971). Dioxins are known to be a common by-product in the commercial production of Agent Orange (EPA 1980).

Agent Orange was used extensively by the U.S. military as a defoliant during the Vietnam War. It is a 50:50 combination of the two herbicides 2,4-dichlorophenoxy acetic acid (2,4-D) and 2,4,5-trichlorophenoxy acetic acid (2,4,5-T). When reports identifying its severe toxicity were released the U.S. government suspended all further military uses of Agent Orange, and in 1970 USEPA stopped many registered domestic uses including application to lakes, ponds, ditch banks, homesites, recreational areas, and most food crops (World Health Organization 1977). Chloracne, an often disfiguring and persistent skin disorder which is characterized by comedones, keration cysts, pustules, papules, and abscesses, is a classical sign of 2,3,7,8-TCDD exposure in humans (U.S. NIEHS IARC 1978). Chloracne can be caused by ingestion, inhalation, or skin contact with chlorodibenzo-dioxins, and while the disease may clear in a few months after exposure it may also persist for as long as fifteen years (Crow 1978).

In the summer of 1983 a soil sample taken by OSR staff from the former Diamond-Alkali plant, now called the 80 Lister Avenue site, was shown to contain 1.2parts per million (ppm) of 2,3,7,8-TCDD. The Center for Disease Control (CDC) had previously stated that exposure to soils with substantially lower: concentrations of TCDD (i.e. 1 part per billion) should be considered a potential health risk (Kimbrough et al 1983). Also, due to the close proximity of the Passaic River to the site it was felt that the water provided a potential route for offsite transport of TCDD. Therefore, a NJDEP and USEPA cooperative investigation into possible dioxin contamination of waters adjacent to the site was begun and OSR supplied EPA with samples of fish collected from the tidal section of the nearby Passaic River for 2,3,7,8-TCDD analysis. In addition, EPA hired a contract consultant to collect soils, sediments and biota from the vicinity of the site. Biota collections included blue crabs from the Passaic River within the immediate vicinity of the site.

The following results are presented in two sections: Phase I, the initial DEP/EPA study from the Summer of 1983 and Phase II, the results of the present study (1984). The initial Phase I survey indicated widespread dioxin contamination of aquatic biota within the tidal Passaic River drainage. Tissue samples revealed elevated amounts (>50 ppt) of 2,3,7,8-TCDD in a majority of finfish and crabs collected. As a result of this data the Commissioners of NJDEP and the N.J. Department of Health (DOH) jointly declared a prohibition on the sale or consumption of all fish and crabs taken from the tidal Passaic River (Administrative Order No. 80-70-17) and had the waterways posted (See Figure 2).

In Phase II an effort was made to further characterize the extent of 2,3,7,8-TCDD contamination within this region and other waterways in New Jersey. OSR applied for two research grants and was funded by the New Jersey Oil Spill Research Fund to do this work (Grants No. P-20768 and P-19666). The results of these research studies are the basis for the Phase II section of this report. The studies incorporate finfish and crustacean collections from major connecting drainages and waterways in the Newark Bay system and provide a

more comprehensive database that includes additional samples from other sites in the State of similar geographic or industrial characteristics or where potential sources of contamination exist (see Map 2). The objectives were: 1) to develop the laboratory capabilities to detect 2,3,7,8-TCDD concentrations in fish tissue; 2) to examine the potential extent of 2,3,7,8-TCDD contamination in fish collected from the Newark Bay Complex and; 3) to identify concentrations of 2,3,7,4-TCDD in, fish collected from other waterways in New Jersey.

2. Methods

2.1 Sample Collection

Finfish from freshwater locations were collected by electro-fishing and gill netting. Estuarine and marine finfish were captured by the use of gill nets, otter trawls, seines, hook and line, and baited traps. Blue claw crabs samples were collected by use of dip-nets, and commercial style crab pots. Lobster samples were collected in commercial style lobster pots and by the use of an otter trawl. All samples (both finfish and crustaceans) were transported to the laboratory in contaminant free ice chests containing sealed ice packs. All storage containers, packaging, work surfaces, and utensils were thoroughly scrubbed, rinsed with acetone or hexane and finally rinsed with distilled water. Each species collected from a particular site was processed based upon its ascending order of lipid content. Before actual processing all were weighed, measured and the species determined. Sediment samples were collected by a single grab ponar sampler. The sediment samples were placed in clean glass jars then sealed and stored frozen until analyzed.

2.2 Sample Processing

Samples of edible finfish consist of a standard fillet portion of muscle tissue. This standard fillet can be defined as that portion of the fish bounded anteriorly by the pectoral fin, posteriorly by the caudal fin, and from the mid-dorsal line to the mid-ventral line, including the rib cage and belly flap with skin attached**.** All catfish and American eel samples were skinned prior to processing. These standard fillets were either used as an individual sample from a single fish or combined with fillets from other individuals of the same species and size to form a composite sample consisting of five fish. The tissue was then thoroughly homogenized in a blender. Single samples were 100 grams in weight, while composites of five fish were 500 grams. Non-edible fish such as mummichogs that are too small to fillet were ground up whole in a blender, and used as whole fish samples. Composites made up of homogenized whole fish contained equal portions from all members in that composite. When the lobster and crab samples were ready to be processed they were removed from the freezer and thawed. When partially thawed, the samples were weighed on a triple beam balance and measured. Lobsters were measured from the rear of the eye socket to the rear edge of the carapace. Blue claw crabs were measured across the shell from point to point. Each individual specimen was sexed, and a notation was made regarding missing body parts, and shell condition (i.e. soft, hard etc). The thoracic body cavity was opened from the ventral surface and the hepatopancreas completely removed using a small lab spoon. All the edible meat was then removed. This included the thoracic, claw, leg and tail meat. All other body parts and organs were discarded. The meat was then either combined as muscle tissue alone (i.e. back lump, claw meat, etc.), or as hepatopancreas tissue alone, or finally as a combination of muscle and hepatopancreas tissue together utilizing all of the tissue from each animal before homogenizing thoroughly in a blender. Portions of this homogenized mixture were placed in clean, labeled wide-mouth 8 oz. glass jars and stored at -20°F until analysis. Between samples all processing equipment was rinsed with pesticide-grade hexane and distilled water. Processed samples were packaged in contaminant-free aluminum foil, labelled, and stored frozen until analysis.

2.3 Sample Analysis

Sediments: Phase I & II

Chemical analysis of sediments was performed by the same contract laboratory under EPA in Phase I and DEP in Phase II. Their method (EPA 1983) utilized a 10 gram sediment sample which was spiked with internal and surrogate standards of isotopically labelled 2,3,7,8-TCDD. Several cleanup procedures were utilized to aid in the elimination of any interferences that were encountered. Quantitation was based on the response of native TCDD relative to the isotopically labelled TCDD internal standard (via capillary gas chromatography in conjunction with low resolution mass spectometry). Performance was assessed based on the surrogate standard results.

Finfish and Crustaceans:

In Phase I samples of finfish and crustaceans were collected and processed by QSR staff and then given to the EPA Region II Emergency Response Group for analysis of 2,3,7,8-TCDD through a contract vendor. Analysis was performed by both high resolution GC/low resolution MS and high resolution GC/high resolution MS (Wright State, 1983). Following EPA quality assurance analysis of the two sets of results it was decided that the low resolution GC/MS data for these samples. was the most reliable and is reported here.

Phase 11 tissue analysis was performed by a contract laboratory for NJDEP using high resolution gas chromatography, low resolution mass spectrometry. It involved a modification of the EPA method using a saponification of the tissue prior to the initial extraction (Appendix F). The sample extracts were then analyzed using an electron impact GC/MS instrument with a direct capillary interface, and a 60 meter isomer specific fused silica capillary column (EPA, 1983).

A small set of the fish tissue samples were also analyzed for 2,3,7,8-tetrachlorodibenzo-p-furans, (2,3,7,8-TCDF). The $^{13}C_{12}$ labelled 2,3,7,8-TCDF was used as the internal standard. The ions m/z 304, 306, and 241 were monitored for 2,3,7,8-TCDF in the fish extract. If TCDF was not detected, a detection limit was calculated based on 2.5 tiffs signal to noise ratio at the retention time of 2,3,7,8-TCDF and the $^{13}C_{12}$ labelled internal standard.

It was also known from the beginning of this project that PCBs and chlorinated pesticides may interfere with 2,3,7,8-TCDD and 2,3,7,8-TCDF analysis in some samples. In those instances where detection limits were high due to chemical interferences some extracts were re-run on a 30 meter DB-5 column. In a few analyses this shorter column provided adequate separation of PCBs, pesticides and the target compounds and although the DB-5 may not be isomer specific we felt the usage of this column was appropriate as a confirming analysis provided that the primary column (ie. CP-Sil-88) indicated the possible presence of 2,3,7,8-TCDD.

Quality assurance/Quality Control procedures followed EPA recommended guidelines (EPA 1979, 1980b, 1983) and included spiking muscle tissue of each species with appropriate standards, analyzing replicate and blind control samples, and demonstrating the proper isomer specificity and ion ratios (Appendix F). The mean percent recovery for spiked samples with internal standards was 96.8° with a ± 1°% error for the full range of representative analyses.

3. Sediments
3.1 Results

Phase I

The EPA contractor collected sediment samples from the Passaic River on June 29, 1983 at locations adjacent to, upstream, and downstream of the Newark site (see Map 2). A total of five cross-river transects and one along-river transect on the south shore were sampled to determine the concentration levels and distribution of any TCDD contaminated sediments. Thirty-five samples were taken, of which 60% had some detectable level of 2,3,7,8-TCDD present; 43% had detectable levels below 1.0 ppb and 17% had levels greater than 1.0 ppb. The highest level identified was 6.9 ppb from a sample collected adjacent to the Diamond Alkali facility. In general, TCDD levels decreased across the river and downstream from the site except in areas of low flow. The highest levels were detected in sediments upstream of the site.

Seven sediment samples were also collected 11.5 miles upstream of the Newark site at the confluence of the Passaic and Third givers on October 31, 1983, near the Givaudan Chemical Company in Clifton, New Jersey. This Class I company uses 2,4,5-trichlorophenol as a raw material in the manufacture of the bactericide hexachlorophene. 2,3,7,8-TCDD levels measured in soils at the plant ranged from 0.09 to 9.7 ppb and an on-site stormwater drain sample showed a level of 2.5 ppb. The facility formerly discharged surface runoff directly to the Third River but is now tied into the Passaic Valley Sewerage Commission. Two sediment samples out of the seven taken from the third River showed positive results for TCDD. A sample taken. from Yantacaw Pond, directly across the street from the chemical company, to which a storm drain discharged showed a level of 0.18 ppb whereas a level of 0.11 ppb was found downstream at the confluence of the Third River and the mainstem of the Passaic River.

Phase II

In the Spring of 1984 twelve sediment grab samples were taken by OSR from the Passaic River, the Third River, the Hackensack River, Newark Bay and the Hudson River. All of these samples were not detected for TCDD at low detection limits (<0.01 ppb). Unfortunately, the Yantacaw Pond samples (Third River) had relatively high detection limits (i.e. none less than 0.5 ppb) in relation to the TCDD levels quantified in Phase I sampling (i.e. 0.18 ppb and 0.11 ppb) making it difficult to compare these results.

Sediment samples were also collected on Raccoon Creek, Gloucester County (Table I). The results revealed that two out of four sediment samples from Raccoon Creek had detectable yet low levels of TCDD. Both positive samples showed 0.03 ppb 2,3,7,8-TCDD, locations were both upstream and downstream of a waste treatment plant located on this drainage.

3.2 Discussion

Sediment transport and hydraulics can affect the dispersal of 2,3,7,8-TCDD in the environment. It is an extremely lipophilic molecule, only sparingly soluble in water (EPA 1980a) and has a high degree of adsorptivity to soil (Modell et al 1978). These properties enhance 2,3,7,8-TCDD ability to adsorb to sediment and organic matter in the water column whose movements and dispositions will then govern the fate of the dioxin molecules. This phenomena will also affect the availability of the contaminant for bioaccumulation in fish. Several studies have reported the transport of dioxin contaminated soils into surface waters. Findings by Bartleson, Harrison and Morgan (1975) indicate that horizontal translocation of 2,3,7,8-TCDD can occur through water runoff as well as wind and water erosion. Other studies have shown that TCDD's can migrate to nearby water bodies from industrial chlorophenol wastes buried in landfills and then contaminate the sediments at parts per billion levels (Chemical Week, 1979, Wright State University, 1979).

Another possible transportation route for dioxin to surface waters could be through atmospheric depositions of combustion emissions. Several reports describe the occurrence of dioxin in fly ash and flue gases from municipal incinerators and industrial heating facilities although 2,3,7,8-TCDD is often reported as a minor constituent of the total dioxins present (Olie, Vermeulen and Hutzinger 1977; Buser, Bosshardt and Rappe 1978).

The highly contaminated nature of the soils at the 80 Lister Avenue site including many industrial solvents could have facilitated continuous TCDD leaching over the years via the hydraulic head (pressure) caused by a twice daily (diurnal) tide of five feet experienced at this location or by normal precipitation events resulting in both surface runoff and subterranean discharge to the River. The distribution of TCDD in sediments from the Third River may be explained by the deposition of material contaminated by the storm drain discharge at Yantacaw Pond and its subsequent transport to a shoal or bar near the confluence of the two rivers.

Comparing the analytical results from Phase I and Phase II sediments shows a decrease in the number of sediments with detectable levels of TCDD for the latter. This may be due to the differing hydrologic conditions during sampling or the fewer samples collected in Phase II. For example, the Phase I sediments were collected in the summer and fall months after the spring floods had decreased and salt-water wedge intrusion was maximal. These relatively quiescent flows might have been more conducive to the net accumulation of TCDD in bottom sediments from the water column resulting in higher TCDD levels during the Phase I sampling. Conversely, the absence of positive TCDD values for most Phase II sediment samples, including those areas previously identified as contaminated, may be due to the transport and deposition of recent uncontaminated sediment from upstream during spring flooding and/or the transport out into the Newark Bay of contaminated sediments. Another explanation for the lack of positive results may be the limited number of grab samples taken in Phase II (one per site). In comparison the Phase I Newark investigation took 35 sediment grabs of which 40% had non-detectable levels even though parts per billion levels were found only a few yards away.

The distribution of TCDD in the river sediments adjacent to the Newark site might also be explained by the flow hydrology experienced at this river mile. At this point in the river the thalweg or channel is closer to the north bank of the river than the south. On the south side, closest to the contamination site, flow is slower and accumulation of sediment more likely than on the north bank. This may also reduce the ability for contaminated sediment on the south shore to be eroded and rapidly transported out of the system.

Transport of sediments away from the site may also be controlled by the flood dominated flow dynamics in the estuary. For example, a study of Newark Lay by Suszkowski (1978) identified a distinct "salt-water wedge" in the lower reaches of the tidal Passaic River. Under normal conditions there is nee: downstream transport in the upper water column while there is a dominating net upstream flow in the heavier saline-rich bottom waters. At full flood and subsequent slack tide we might expect then that the net effect would be to transport sediments up the river and deposit it in low energy, flood dominated areas where temporary storage take place. It is also possible that this upstream bottom flow could transport contaminated sediments miles upstream from the point source at the plant and store TCDD in upstream low energy tidal reaches where erosion is impeded (e.g. point bars, behind bridge abutments,etc.).

However, it is important when analyzing the river sediment results to stress that the samples taken were surficial grabs, therefore due to the strong flow (41 m^3/sec) and suspended sediment discharge from the Passaic River this material is only indicative of recent depositional contamination and not long term storage within the system. In spite of this limitation however studies of sediment deposition in Newark Myerson 1951) may indicate where downstream low environments are located and coincidently where Suszkowski (1978) showed, based on dredging records, that there are three areas within the bay complex that have high sedimentation rates; 1. the channel north of Shooters Island, 2. the lower Passaic River and 3. Port Newark (Table VII). The study by Myerson (1981) assumed correlations between suspended sediments and heavy metal concentrations showing that the east and west shores at both ends of Newark Bay had high metal levels in bottom sediments whereas the central portion of the bay and the two ends at Kearney Point and Shooters Island where scouring occurred had low metal concentrations.

Suszkowski (1978) also reports that net sediment transport occurs from Newark Bay into the Hackensack River. The implication of this finding is that the contaminated suspended sediments from the Passaic River and upper Newark Bay may be pushed up into the Hackensack Meadowlands and stored there within its marshes and mudflats. This might partially explain the high TCDD levels detected in blue crabs from the lower Hackensack River although their omnivorous nature and wide ranging seasonal movements are probably more important.

It should also be noted that fish normally wait in areas of lower flow in order to seek protection from the current and to catch smaller bait fish. Since these low energy areas have higher concentrations of fine-grained sediments and associated TCDD contamination it could increase the exposure time of fish to contaminated sediments possibly facilitating their uptake of TCDD. Studies have shown that regardless of the source once in the sediments TCDD can show strong resistence to biodegradation and may leach into the water column or be transported by suspended sediments where it is then made available to the biota for bioaccumulation in their tissues and biomagnification up through the food chain (Isensee and Jone 1975). Model ecosystem studies have demonstrated bioconcentration factors for 2,3,7,8-TCDD of 3,600 and 26,000 over a period of 3 to 31 days (Isensee and Jones 1975). Also in the Dow study (Dow Chemical 1978) no levels of TCDD were found in the river sediments although the fish downstream of the Dow Complex had elevated levels of TCDD in their flesh (90-230 ppt).

These facts make the low level contamination (<lppb) in sediments near the Third River and Raccoon Creek significant when considering the bioconcentrating power of the aquatic biota. For example, Raccoon Creek was the only non Newark Bay drainage to show TCDD levels in biota (i.e. white perch) although the sediment levels of the contaminant

were barely detectable (0.03 ppb). The fact that the positive sediment results were both upstream and downstream of a waste treatment facility may also be related to the fact that it is a tidal creek with flow reversals capable of moving sediments upstream.

Finally, the distribution and transport of sediments on the Newark Bay system will be affected by the routine dredging of ship channels and berths within the rivers, kills and heavily trafficked ports of Newark and Elizabeth. In the Newark Bay Complex the Army Corps of Engineers (ALOE) maintains approximately 35 km of navigation channels. It is estimated that combined federal and private dredging of Newark Bay removes approximately $961.9 \times 10^3 m^3$ of sediments annually (Suszkowski 1978) and since 1969 over 75% of this material has been dumped in the Atlantic Ocean. Therefore, the TCDD contaminated sediments from the Passaic River, due to the unusual tidal influences within Newark Bay, will probably be continuously reworked in semi-permanent storage within the system until they are either dredged out and disposed of in the ocean or secondarily transported out in the Kill plumes from the bottom of the bay. A more complete sampling design for future sedimentology work should probably include sediment cores, grain size analysis, and focused sampling for surficial grabs.

4. Finfish and Crustaceans

4.1 Results

Phase I

Samples collected represented aquatic organisms from several ecological compartments with species of resident and migratory nature and of major recreational and/or commercial importance. Resident species of carp Cyprinus carpio; goldfish, Cyprinus sps.; brown bullhead, Ictalurus nebulosus; channel catfish, Ictalurus punctatus; mummichog; Fundulus heteroclitus and white perch Morone americana, were collected at various locations along the tidal Passaic River. Migratory species of American eel, Anguilla rostrata; striped bass, Morone saxatilis; and blue crab, Callinectes sapidus, were also collected from this tidal region down to the confluence with Newark Bay.

Values for resident benthic species (carp and catfish) produced overall concentrations of TCDD consistently above 50 ppt (parts per trillion) in both single and composited samples (See Map 3). Results for carp ranged from 108 ppt to 155 ppt with mean values at 110 ppt. Catfish results were 50 ppt and 73 ppt with a mean of 62 ppt. One of these was collected at the extreme head of tide (Dundee Dam). In addition a composited sample of mummichogs, an abundant- forage fish, collected at the lower tidal section revealed significant levels of TCDD at 114 ppt.

Dioxin values for migratory species (American eel, striped bass, blue crab) also exceeded 50 ppt. Levels of TCDD for composited American eel samples ranged from 22 ppt to 61 ppt with a mean of 38 ppt. These samples were collected at the same site within the lower tidal portion of the Passaic River. Results for striped bass collected at the confluence of the Passaic River and Newark Bay are reported at 31 ppt and 58 ppt, with single and composite samples identified, respectively.

With respect to the blue crab results differential analysis of the muscle tissue and hepatopancreas (See figure 3), which are both edible (Davidson, 1978; Ross, 1978; Sarvis, 1968) showed starkly different levels of TCDD contamination between the tissue types. Previous research has demonstrated that dioxin shows a strong affinity for accumulation within fatty (i.e. lipid) internal organs of several aquatic species (Tucker et al 1983). In line with this observation the lipid-rich hepatopancreas of blue crabs collected in the vicinity of 80 Lister Avenue revealed the highest TCDD concentrations (450 ppt and 485 ppt) of any sample examined (See Figure 3). Muscle tissue values from these same animals however were greatly reduced (i.e. 27 ppt and 16 ppt) but still ranged within the FDA guidelines for reduced consumption.

Only one non-detectable value for Phase I tissue data was observed. This result was for a composited sample of white perch collected at the head of tide on the Passaic River below the Dundee Dam, Garfield, New Jersey.

Phase II

The first objective of the Phase Li_ study was to choose an analytical method for 2,3,7,8-TCDD analysis of tissue that was accurate, had a low detection limit and was cost effective. The last element was important in as much as the future of any routine chemical monitoring is usually predicated upon the fiscal accessibility of that technique. In line with this goal OSR worked with the contract chemists to help choose and develop an appropriate method. The Phase II tissue database reflects that "developmental approach" since the earlier runs had high detection limits in an arena where FDA "levels of concern" were being set at 10 through 25 ppt or at the limits of todays analytical technology We therefore added clean-up step that successfully removed most interferences for the latter runs giving us better detection limits with higher standard recoveries. The complete list of all the Phase II Biota analyses are shown in Table II.

It was then decided that all of the non-detectable analyses with detection limits in excess of 25 ppt (i.e. the FDA baseline level of concern) were non-representative and therefore not to be included in any summary statistics. Samples that showed non-detectable levels with a quantifiable signal-to-noise ratio or detectable limit below this 25 ppt were considered as being at. a zero contaminant level for statistical purposes unless otherwise noted (e.g. calculations of summary statistics to quantifiable levels only). It should be noted however that 2,3,7,8-TCDD may be present in these non representative samples but that it was impossible to measure because of the matrix interferences resulting in a high level of detection.

The second objective of the phase II study was to further characterize the extent of TCDD contamination within edible species from the Newark Bay system. We incorporated collections from major connecting drainages to Newark Bay, and in order to develop a more comprehensive database we also included additional samples from other sites for populations of representative resident and/or migratory target species (See Map 4). Overall, eleven species of fish and two crustacean species including both resident and migratory organisms were collected. They included blue crab, American lobster, striped bass, American eel, white perch, carp, white catfish, channel catfish, brown bullhead, largemouth bass, weakfish, bluefish tuna and skipjack tuna. Positive results for 2,3,7,8-TCDD were found for only seven of these species however (Table III).

Passaic River Collections

The results of the fish and crustacean samples collected from the tidal Passaic River consistently showed elevated levels of TCDD for most organisms and locations. This replicated and substantiated the findings from the Phase I study (See Table IV). Species with elevated TCDD concentrations included blue crab, brown-bullhead catfish and carp. The only variations in Phase I and II data were for the American eel which did not show detectable levels of TCDD in Phase II although they did so in Phase I. This apparently was due to matrix interference resulting in high detection limits for these samples as part of the analytical methodology development. Analyses of fish taken from above the head of tide or above Dundee Dam showed no detectable levels of TCDD in any of the representative species (i.e. American eel, largemouth bass and carp).

Newark Bay Complex Collections

Collections of fish and crustaceans were made throughout the Newark Bay system and in the tributaries of Newark Bay including the Hackensack River, Arthur Kill and the Kill Van Kull (Map 4). The data show that the migratory species identified with elevated TCDD concentrations on the tidal Passaic River in both Phases I and II were also contaminated throughout the Newark Bay Complex (Table III). Blue crabs, striped bass, and American eels were chosen for analysis since they represent the major target species for recreational and commercial fishermen in this area and as migratory animals posed the most serious threat for transport of TCDD out of the bay and to .he food distribution centers of the northeast populace. Quantitative concentrations of TCDD were identified in blue crabs and striped bass throughout the Newark Bay complex. (See Map 5)

American Eel Data

Due to problems encountered during the early methods development section it is difficult to make a comparison of the Phase I and the entire Phase II American eel datasets. In Phase I, TCDD was shown to be present in all of the eel samples analyzed from the tidal Passaic Unfortunately the Phase II eel samples from the same areas were among the first analyzed as part of the developing analytical methodology. This resulted in non-detectable values with extremely high detection limits for the initial Phase II eel samples (Table I). However, after establishing an acceptable method the later analyses on American eels from upstream of Dundee Dam, and downstream in Newark Bay and the Arthur Kill produced non-detectable results at low detection limits.

Blue Crab Data

Blue crab samples from Newark Bay, the Hackensack River, and the Passaic River revealed the highest TCDD levels identified (Map 5). While TCDD results for muscle meat were all non-detectable, TCDD levels in the crab hepatopancreas ranged from 10 to 1063 ppt with a mean value of 496 ppt (Table V).

On the Hudson River only one crab sample showed a low level of TCDD (10 ppt) in the hepatopancreas whereas all the other crab samples from that drainage showed non-detectable results with high detection limits. Like the Passaic River eels these Hudson River crabs were analyzed as part of the method development section and suffered the same loss of information due to the high detection limits. Two crab samples (i.e. combined muscle and hepatopancreas) from the mouth of the Raritan River showed elevated TCDD levels (i.e. 25 and 48 ppt) whereas no other samples from upstream in the Raritan River proper showed any dioxin concentrations. Control samples of blue crabs from Great Egg Harbor and Delaware Bay also showed no detectable levels of TCDD.

As noted, we processed some of the crabs as combined hepatopancreas-muscle tissue samples at the urging of the U.S. Food and Drug Administration (FDA) since they felt that this might be a better indicator of true consumer exposure. The resulting data ranged from 25 to 480 ppt with a mean of 184 ppt.

Striped Bass

The Phase II striped bass samples collected from the lower section of the Newark Bay exhibited levels similar to those found during Phase I (See Table VI). TCDD values ranged from 20 to 56 ppt with a mean of 40 ppt. Although no TCDD levels were found in stripers taken from the Hudson River as had been identified in 1982 by the New York Department of Conservation, (NYDEC) a sample taken near the Earle Navy Pier, Leonardo, N.J. on Raritan Bay did show 20 ppt of TCDD. Striped bass caught in Phase II were also supplied to NYDEC for inclusion in a study examining striped bass contamination on a coastwide basis (O'Keefe et al 1984). The results obtained by NYDEC for single fish analyses match quite well with composite samples reported here (Table VI). In addition to 2,3,7,8-TCDD the Newark Bay striped bass were analyzed for 2,3,7,8-tetrachlorodibenzofurans (TCDF) which have toxic properties similar to TCDD. This analysis was undertaken after NYDEC had reported finding furans in striped bass taken from the Hudson River. NJDEP levels ranged from 29 to 42 ppt with a mean of 26 ppt which matches quite well the samples OSR supplied to NYDEC (See Table VI).

New York Bight and Delaware Bay Samples

Samples of select species from the contiguous ocean of the inner New York Bight (Table III) revealed elevated levels of TCDD (21 and 37 ppt) in only two out of nineteen Bluefish (Pomatomus saltatrix) analyzed and in tissue samples from the American Lobster (Homarus americanus). (See Map 6)

We found that two composited samples (i.e. 15 organisms) of the lobster hepatopancreas (tomally) from animals caught in deep nearshore waters (i.e. the Mud Hole) were contaminated at 72 and 82 ppt, respectively (Map 6). Nine more samples of inshore lobsters collected from Raritan Bay and processed as single samples with hepatopancreas, claw and lump meat combined (as recommended by FDA for blue crabs) showed that four out of the nine samples had detectable levels of TCDD ranging from 25 to 62 ppt with a mean of 44 ppt. The five remaining samples had high detection limits initially and were re-analyzed. These second set of analyses also showed high detection limits due to matrix interferences that made it impossible to quantify TCDD in the parts per trillion range. Therefore TCDD may be present in these samples but it was impossible for us to quantify it.

Finally, one White Perch sample from Raccoon Creek on the Delaware River drainage showed 48 ppt of TCDD (Table I). This is the only positive TCDD quantification for biota that is not associated with the Hudson-Raritan-Newark Bay estuary and adjacent ocean waters.

4.2 Discussion

Fish and aquatic life are often better indicators of toxic contamination than sediments or water due to their propensity for biomagnifying chemicals to elevated concentrations even though the ambient levels in water and sediment may be non-detectable. There have been a number of reports showing 2,3,7,8-TCDD bioaccumulation in various finfish (Gaughwan and Meselson 1975, Dow Chemical 1978, Harless 1980, O'Keefe et al 1984, Kacymar, Zabia and D'Itri 1983) but only one study found TCDD in both finfish and crustaceans. Notably, it was found in fish and crustaceans collected in 1970 from South Vietnam in an effort to determine whether the spraying of the dioxin contaminated herbicide Agent Orange had led to the accumulation of TCDDs in the environment (Gaughwan and Meselson 1973). This report indicates carp and catfish were the most contaminated freshwater fish identified with TCDD levels of 320 and 1020 ppt respectively. Croaker fish and prawns (crustaceans) from the seacoast were also contaminated but at less elevated levels. More recently work done by New York DEC (1982) on TCDD contamination of striped bass taken from the Hudson River was an important stimulus for the present study.

Galston (1979) has established that under certain conditions 2,3,7,8-TCDD can enter the human body from a 2,4,5-T treated food chain and can accumulate in the fatty tissue and secretions, including milk. This is probably due to the fact that 2,3,7,8-TCDD is extremely lipophilic and only sparingly soluble in water and most organic liquids (WHO 1977).

The data developed from this study during both Phase I and II sampling indicate that the faunal species within the entire tidal section of the Passaic River are contaminated with 2,3,7,8-TCDD. This observation is supported by the fact that resident species such as carp, catfish and goldfish, which do not demonstrate a highly mobile lifestyle and are therefore indicative of local or upstream transport exhibited TCDD results in excess of the FDA "level of concern" along the entire tidal reach. (See Table IV, Maps 3 and 5).

The two FDA "Levels of Concern" are 25 and 50 parts per trillion (ppt), respectively. These guidelines provide consumption recommendations to fish eaters in order to reduce human health risks for fish contaminated at these levels. Overall 10 of the 16 tissue samples supplied exceeded 50 ppt (FDA Recommendation: No Consumption). Of the remaining 6 samples 3 ranged between 25 and 50 ppt (FDA Recommendation: No more than one meal per week for infrequent consumers and no more than 1-2 times a month for frequent consumers of the fish), 2 samples were below 25 ppt and only 1 showed no detectable levels of TCDD (FDA Recommendation: No limit on Consumption). *See* Appendix B for a further explanation of these "Levels of Concern".

Resident Fish in Passaic River

Carp and goldfish have fundamentally the same niche, feeding along the material and detritus. Their range requirements and to existing environmental conditions precludes the bottom on vegetative general adaptiveness likelihood that the organisms collected at upstream sites may have accumulated TCDD while in the vicinity of the Newark site, a distance of approximately 18 miles. It is more 'Likely that the Clifton area samples were contaminated either by TCDD being discharged from the Third River along with its contaminated sediments and/or from contaminated sediment transported from downstream by the strong bottom flow of the salt wedge.

The finding of 50 ppt (TCDD) in a channel catfish upstream of both these potential point sources is perplexing since it is unlikely that the salt wedge could transport sediment

that far up river. This species is routinely stocked by DEP, Division of Fish, Game and Wildlife (DFGW) in Dundee Lake above the spillway at the head of tide and was most likely washed over into the waters below the dam where because of its niche requirements (i.e. physical needs) it remained. The absence of TCDD results in either the sediments or fish from the lake above belies any easy explanation as to how this animal could have become contaminated. It may be speculated that in this instance the contamination may be in conjunction with movement of contaminated prey fish from downstream or movement of the channel catfish downriver caused by some environmental stress.

Samples collected below the confluence of the Second River at the 4th Street Bridge in Harrison, New Jersey incorporated species of more diverse lifestyles. Resident species include mummichog and brown bullhead catfish. These species exhibit omnivorous feeding characteristics although the brown bullhead catfish is a bottom. organism whereas the mummichog ranges throughout the water column but usually is closely associated with bottom or shore structures. Mummochog represent the major forage fish (i.e. prey for upper food chain predators) identified on this drainage and may provide one means for contaminant transport upward through the food chain. Direct uptake from the water via ingestion or adsorption at gill contacts during respiration cannot be precluded either. However, substantial contamination through food chain biomagnification could occur even if these higher order species are infrequent to the contaminated area (i.e. migratory). High concentrations within this food base may have implications for other animal species as well since shore birds and diving ducks can also feed upon this resource.

Analyses of fish taken from above the head of tide or above Dundee Dam showed no detectable levels of TCDD in any of the representative species (i.e. American eel and carp). This indicates that the dioxin contamination of fish and crustaceans within the Passaic River is probably occurring only in the tidal section. This observation may not be consistent for specific migratory species who may pick up contaminants elsewhere but it appears that for the majority of the resident species exposure to TODD on the Passaic River is contingent upon dwelling downstream of the Dundee Dam in Garfield.

Migratory Fish and Crustaceans

American eels, striped bass, and blue crabs will frequent various sections of the tidal Passaic River and will then migrate seasonally throughout the Newark Bay-Upper New York Bay-Inner New York Bight complex. The contamination of these species, therefore, could be instrumental in the transport of TCDD into the entire Hudson-Raritan estuary and its associated ocean waters. This raises serious health risk implications for the fish consuming public of the entire region since these organisms are some of the major recreational and commercial species sought in these waters.

American Eels

The American eel is a diadromous fish, meaning that it is capable of moving from fresh to salt waters and vice versa, usually for spawning reasons (Hardy, 1978). The eel is catadromous, spending most of its life in fresh or brackish water until it migrates to the ocean for spawning. The finding of elevated TCDD levels in Phase I and not in Phase II eels, both upstream and downstream of the potential point sources, is difficult to explain. There are similar findings however for eels taken from Lake Ontario (Ryan et al 1984). The study showed that American eel had the highest levels of 2,3,7,8-TCDD found (Range: 6.4-38.5 ppt) yet the levels and incidences were not uniform.

Confounding environmental factors related to this species' complex life style such as the ability to aestivate (i.e. hibernate) in contaminated muds or the tendency of only females to ascend rivers while the males remain behind in brackish waters, may account for this variability in results as well. In spite of this the potential for toxic transport out of this system by eels has been shown for PCBs and pesticides (Belton et al. 1982, Belton et al 1983). It is apparent that more samples at better detection limits will aid in interpeting the importance of TCDD contamination in this phenomenon.

Striped Bass

The striped bass is also a diadromous fish yet reciprocal in behavior to the eel. It is an anadromous species spending most of its life in the brackish estuarine and inshore salt waters of the ocean until it migrates into freshwater during the spawning season (Schaefer 1968). Therefore the young-of-the-year and juvenile striped bass utilizing Newark Bay as a nursery will spend their early lives feeding in this severely contaminated food chain and possibly absorbing dioxin directly from the water column where each tidal cycle will result in a resuspension of contaminated sediment (Califano et al. 1982; Suszkowski 1978).

The levels of TCDD for striped bass collected from Newark Bay are consistent with levels found in striped bass by O'Keefe (1984) in contiguous waters (see Table VI). The finding of TCDD in a striped bass taken from the south shore of Raritan Bay also demonstrates the potential for contaminant transport out of the estuary into the offshore fishery thereby creating a more widespread public health threat. And in fact the major objective of the NY state study (O'Keefe et al 1984) which NJDEP participated in, was to evaluate TCDD distribution in striped bass stocks from several locations along the Atlantic Coast. Rhode Island, New York, New Jersey and Maryland supplied fish for TCDD and TCDF analysis in this study and the results indicate that the Hudson River-Newark Bay area is the major source of 2,3,7,8-TCDD in striped bass. They also noted that similar concentrations in fish over a two year span suggests that there is a constant exposure level in the system. This observation is consistent with the data generated in this study.

NYDEC also noted that the concentrations of 2,3,7,8-tetrachlorodibenzofuran (TCDF) in striped bass appears to be elevated over a greater geographic range than 2,3,7,8-TCDD although the Hudson River-Newark Bay fish have the highest concentrations. The tetrachlorodibenzofurans have been associated with PCB mixtures which are much more ubiquitous in the environment than dioxin-contaminated compounds and this may provide an explanation for this observation of wider contamination for the furans.

Blue Crab

Crabs are bottom dwelling, macrophagous, scavengers and predators (with highly differential limbs) making them one of the most successful groups of predatory carnivores in the sea (Russel-Hunter 1969). In the higher crustacea (DECAPODA) such as the blue crab, the midgut of the alimentary canal may have diverticula (i.e. blind, branching sacs) such as the hepatopancreas which acts as the main site of lipid storage, enzyme secretion, and nutrient absorption (Johnson 1982) and where digestion is primarily extracellular (see Figure 3). Also, as with all animals possessing an exoskeleton for bodily support, the crab grows through a series of metamorphoses, or changes in form, until it reaches its adult form where it continues to grow via sucessional molts. During the molt stage the adult crab will lay down a new soft cuticle underneath its shell then shed its old cuticle or exoskeleton. The crab then swells by an uptake of water at which point the new, larger exoskeleton hardens and calcifys (Pyle and Cronin 1950).

Superimposed upon this complex developmental cycle is the fact that the blue crab, like the American eel and striped bass, has evolved a migratory behavior through the estuarine zone which allows the adults and juveniles to utilize different ecological niches at different stages of their life cycles and thereby prevent direct competition for resources (Williams & Duke 1979, Bliss 1982). Unlike the eel and the bass, however, the blue crab's life cycle is exceptionally short; about two or three years. During this time the adults will mate in brackish water within the estuary. The females then begin their migration to the high salinity ocean waters to spawn while the males stay behind in the brackish water. If the females do not reach the ocean by late autumn they burrow down into the mud and wait for the spring thaw before continuing oceanward (Map 6). This phenomenon results in a commercial winter dredge fishery for crabs in the lower New York and Raritan Bays. The females do not usually return to the estuary after spawning although multiple spawn years have been observed.

The finding of elevated 2,3,7,8-TCDD contamination in the hepatopancreas of blue crabs but reduced or non-existant levels in lump meat or other organs is consistent with similar blue crab studies of PCB's, heavy metals and organochloride pesticide bioaccumulation (Ruppel et al 1984, Belton et al 1982, Sheridan 1975). Sheridan (1975) detected DDT and its metabolites in five out of six major organs in blue crabs collected from the York River in Virginia. The hepatopancreas had the highest concentration while the edible claw and back fin muscles had the lowest. It was suggested by the data that DDT was transported from the gills to the hepatopancreas via the blood stream where DDT was rapidly dechlorinated to DDD.

Conversely, other studies on the mechanisms of organchloride bioaccumulation have shown that the food chain may be the primary route of exposure. For example, a study by Schimmel et al (1979) concerning uptake of the chlorinated pesticide Kepone in blue crabs compared direct uptake from seawater to the potential for dietary uptake via feeding on contaminated oyster meat. In that study the contaminant uptake was primarily through the food. They also noted that crabs fed oysters from the James River died in greater number or molted less frequently than control animals. They hypothesized that the foodchain contamination was possibly a factor in the present decline of the James River crab fishery.

It is also interesting to note that the concentration of lipids, organic food reserves and mineral salts in the hepatopancreas, and elsewhere in the animal, vary in a pattern according to their position in this molt cycle (Russel - Hunter 1969, Johnson 1982). Thus analytical results for contaminants may vary based on when in its molt cycle the crab is caught and processed. Only further studies will elucidate the size of this variability.

In the Newark Bay complex due to both the contamination of the food chain and the suspended sediments it is probable that both respiratory and digestive uptake are possible mechanisms for transport of TCDD into the crab hepatopancreas. The absence of the contaminant in the lump or muscle meat is probably due to a number of factors including the dechlorinating capacity of the hepatopancreas, the reduced lipid level in the muscle tissue and possibly the fact that the circulatory system's is generally open (i.e. no arteries or veins per se). Therefore, whither the contaminants enter through the gills or mouth the processing in the hepatopancreas appears to prevent migration into the muscle. The reports of various physiological dysfunctions in crabs due to organochloride contaminants, however, such as increased mortality, reduced fecundity and molting frequency all seem to indicate that the other parts of the animals anatomy, and especially the nervous system, are not as protected (Schimmel et al 1979, Cantelmo and Mantel 1979).

Concerning the geographical distribution of TCDD in crabs throughout the Hudson-Raritan estuary it is hard to interpret the results based on such a small sample size since the natural range and distribution of the crab stocks will vary based on seasonality and ecological limiting factors (i.e. salinity, temperature, D.O.) Also our data in no way circumscribes the actual range of the blue crab across the estuary nor does it take into account the possibility that Newark Bay, the Hudson River and the Raritan River crabs may not be related. In fact, it may be hypothesized that they are distinct sub-populations with little inter-mixing between groups. In support of this is a study that shows blue crab under some conditions actively avoiding low pH outflows from certain estuaries (Laughlin et al, 1978); Bliss (1983) postulates that this may allow crustaceans to discriminate chemicals signifying different water masses, as known for fish (Creutyberg 1961, Hasler and Scholz 1978). Only more data collections across all of these water segments as well as female samples from the overwintering grounds will elucidate these questions. This aspect of the blue crab study will be an important component of future research.

The more important observation however is the uniformity of TCDD-Crab contamination within the Newark Bay complex and the two rivers which discharge into it. Whether the contamination of Hackensack River crabs takes place within its waters or is just indicative of the already contaminated crabs moving up into the Meadowlands from Newark Bay to mate is open to conjecture.

In addition as part of this investigation we carried out the FDA's suggestion to combine the muscle meat and the hepatopancreas in order to better define the actual exposure of someone who eats the mustard and the lump meat. This exercise showed that 2,3,7,8-TCDD levels usually dropped an order of magnitude if the two tissue types were combined (Table V). This is not unusual considering that there is more tissue per unit contaminant using this method and that the hepatopancreas is less dense than the muscle tissue. In spite of this, however, the TCDD levels still remained elevated relative to the FDA's "Levels of Concern" and in fact one sample taken directly adjacent to the 80 Lister Avenue site remained in the hundreds of parts per trillion. This animal's hepatopancreas may have been engorged with food and sediment in the parts per billion range as indicated by the bottom sediment data from the same location. A second sample of combined tissue taken from the confluence of the Passaic with the Third River (River Mile 11.5) was also elevated and indicates the extent to which these animals may penetrate the river system (Map 5).

It is uncertain whether the two elevated (combined) samples from the mouth of the Raritan River can be solely associated with the Newark Bay crab population. Whether the Newark Bay crabs use the Arthur Kill or the Kill Van Kull or both for migratory purposes is unclear. The prevalence of higher salinity water for greater portions of the year (Suszkowski 1978) through the Kill Van Kull as opposed to the Arthur Kill may infer that the crabs may prefer this migratory route since salinity appears to be their major cut for migration behavior (Cantelmo, Personal Communication). If that is the case then the Raritan River Crabs remain an anomaly unless associated with other potential dioxin contaminated sites on that system (see Map 1) or the lower Arthur Kill which have so far failed to show current TCDD contamination or have not yet been visited by DEP (i.e. Class II Sites as defined in Appendix A).

Finally, we analyzed blue crabs from Delaware Bay, Great Bay and other coastal waters where the majority of New Jersey's commercial crabs are taken. None of the samples showed detectable levels of TCDD (See Table II) which emphasizes the predominantly regional nature of this contamination and its association with the Passaic River drainage.

The fact that blue crabs are caught throughout the Hudson-Raritan Estuary during their seasonal migration into the near ocean waters again underscores how bioaccumulation for organisms within the tidal Passaic River can become a toxic-exposure problem for someone crabbing miles away in better quality water. Studying the possible range of this crab/TCDD distribution will become a major objective in future OSR studies.

American Lobster

The American lobster like the blue crab is also a decapod crustacean that is bottom dwelling, and a macrophagous scavenger and predator with highly differentiated limbs. It feeds on bottom-living animals, such as fishes, starfishes, worms, clams, mussels, sea

urchins and crabs (Bliss 1982). Also, like the blue crab, the lobster has a midgut gland or heptaopancreas, (Figure 4) which is called the tomally, and is used for food storage, enzyme excretion and nutrient absorption (Bliss 1983). Accumulated food reserves make the tomally rich and flavorful when cooked and almost all cookbooks recommend utilizing the gland for some type of food preparation (Child 1961, Davidson 1978, Ross 1978, Jarvis 1968).

In contrast to the crab morphology, however, the general body plan of the lobster is more like that of a shrimp except that in the lobster the first pair of legs is modified as very large claws, used for seizing, cutting, and crushing (Bliss 1982). Unlike the blue crab, however, the American lobster is rarely found in salinity levels less than 25 ppt and prefers hard, rocky bottoms or those with a dense covering of seaweed or kelp (Doliber 1973). These conditions provide the animal with hiding places in which it is safe from predators and able to entrap its own prey. Mud bottoms are rarely attractive except in winter when lobsters burrow into the ocean floor (Doliber 1973).

Therefore, although the two crustacean species have many similarities including metamorphoses and molting their differing salinity tolerance effectively keeps them physically apart through much of their range until either the female crabs migrate into the ocean waters or low flow conditions increase salinity levels and allows the lobsters to enter the lower estuaries. The range of these two species does, however, overlap in the Hudson-Raritan estuary primarily along the Lower Bay, Raritan Bay and at the mouth of the Hudson River. Together, they represent two overlapping, benthic populations of crustaceans extending out of the, Hudson-Raritan estuary and onto the continental shelf and its submerged Hudson Canyon.

The geographical distribution and seasonal movements of the lobster population in the New York Bight and the presence or absence of true migratory behavior is not completely understood. Most commercial lobster fishermen believe all lobsters migrate with the seasons, going far offshore in the fall, to deeper, warmer waters and seeking the warmer inshore waters and rocks in early summer for protection during spawning and molting (Doliber 1973). Tagging studies however have shown that most inshore lobsters are non-migratory making annual movements of only 5 to 14 kilometers labeling this the "home territory" (Montreuil 1954, Cooper 1970, Bergeron 1967). The deep-water variety of lobsters, however, does show definite patterns of long-distance seasonal migration (Cooper and Uzmann, 1971 and 1980 Scud 1970, Wilder 1958) where offshore-onshore movement correlates with seasonal thermal conditions.

A recent lobster tagging study carried out by DEP (Andrews, 1980) helps to. clarify this controversy for New Jersey waters by indicating that the three geographic lobster fisheries in the New York Bight show only limited migratory behavior. For example the Ambrose, Alongshore and Offshore fisheries show only 24%, 27.3% and 10.9% of their recaptures as exhibiting migratory behavior defined as a track of greater than or equal to ten nautical miles and time at large less than or equal to 120 days or a track of greater than or equal to 40 nautical miles. The three groups also showed a mean distance traveled of only 13.2, 17.6 and 6.8 nautical miles respectively. This implies that if a contaminated lobster is caught offshore there is a 75% probability that it was exposed to the contaminant within the general vicinity of its capture.

Although TCDD contamination of lobsters has not been reported before in the literature there have been a number of reports concerning the contamination of lobsters by PCBs, pesticides and heavy metals (Ruppel et al 1984, NOAA 1982a, NOAA 1982b,

O'Connor 1982, Weaver 1984, Roberts 1982, Humason 1982, Mayer 1982). - A number of Atlantic coast studies have shown that the contamination of lobsters may be related to specific geographic locations such as New York Harbor (Roberts 1982), or the ocean waste disposal site for sewage sludge and dredge spoils (NOAA 1982, Mayer 1982, O'Connor 1982). Another geospecific association of contamination can be illustrated by the observation of severe PCB contamination within New Bedford harbor, Massachuetts. In 1979 the contamination of lobsters there resulted in a closure of some extremely productive lobstering grounds (Weaver 1984). The Massachusetts Department of Public Health processed composited samples as combined muscle-hepatopancreas mixtures. The results showed that between 1976 and 1980, a mean PCB concentration of 8.7 ppm (Range 0.1-84.0 ppm) was found for 183 lobster samples analyzed. New Bedford harbor is presently a Superfund site (CERCLA) and a two-year study of PCB contaminant distribution for sediment and biota is underway.

It is difficult to conclusively interpret the TCDD data for lobsters based upon a small sample size (Table V). The most compelling aspect of these findings, however, is that both individual tissue (hepatopancreas) and combined (muscle-hepatopancreas) samples showed elevated levels of 2,3,7,8-TCDD or else were so "dirty" from other chemical interferences that TCDD quantification was not possible. This latter observation was not entirely unexpected since other data collected by OSR has shown elevated levels of PCBs in lobster tissue (See Table VIII) which may mask the TCDD if it is present. This data also showed variable PCB-tissue concentrations for different tissue types (i.e. Higher PCB Levels in hepatopancreas). It was found that whereas muscle tissue had low to non-detectable levels of PCBs (Range: 0.1-0.31 ppm; Mean = 0.21 ppm) the hepatopancreas of the same animals had elevated levels (Range: 2.15-4.3 ppm; Mean = 3.15ppm). The analysis of combined muscle and hepatopancreas tissue also showed the same order of magnitude drop as seen for TCDD in crabs (Range: 0.65-0.79, ppm, mean = 0.72 ppm).

Data by O'Connor et al (1982) also demonstrated that American lobsters may have vastly elevated levels of PCBs and PAHs in the hepatopancreas despite the presence of low levels in the flesh. They note that this contaminant exposure may be dietary in origin and because of selective tissue accumulation, such (dietary) exposure may not be reflected by PCB concentration in the muscle.

However, in spite of the present data limitations it is useful to speculate on how this contamination could have occured. The obvious exposure routes are from contaminated water, suspended sediments, food and possibly aerosol deposition from the air. In relation to the inshore lobsters collected from the Chapel Hill Channel in Raritan Bay these animals were located directly in the path of the discharge plume from the Hudson River. The animals are also capable of moving into Upper New York Harbor when the conditions are optimal. The possibility then exists that these organisms could be subjected to significant concentrations of various toxic chemicals passing through the Hudson River plume and the New York Bight Apex. Therefore exposure via direct adsorption through gill contact of contaminants during respiration may contribute a portion of the total body burden. Of course the lobster's omnivorous feeding characteristics along with the identification of elevated concentrations within the hepatapancreas, which is part of the digestive tract, provides us with a stronger argument that a. contaminated food chain is responsible. In conjunction with this hypothesis several reports have indicated that the American lobster will prey upon various species of crab (Squires 1965, Bliss 1982, Evans and Mann. 1977). The geographic proximity of the Chapel Hill Channel collection site places these lobsters in an overwintering area that is utilized by the migrating female blue crab population. This can be illustrated by the presence of a winter crab dredge fishery in this area. Even if this potential food-chain activity does not entail active predation on the part of the lobster the female blue crabs do not return to the estuary after spawning but die in the ocean. Therefore, a substantial biomass of TCDD-contaminated crabs are annually deposited on the inner-coastal plain where the dying female blue crabs are then subject to scavenging by other fauna including lobsters.

The offshore lobster samples were collected on the eastern slope of the "Mud Hole" approximately 20 miles east of Long Branch, N.J. (Map 6). They were of the same size and weight as the inshore lobsters all of which were of a legal commercial size. Unfortunately, these were samples of opportunity collected after muscle tissue was removed and only combined hepatapancreas tissue was analyzed. The results, nonetheless, were unexpected. The hepatapancreas of the offshore lobsters demonstrated TCDD contamination in excess of the FDA "no consumption" 50 ppt advisory level (Mean: 77 ppt).

How these organisms became contaminated with TCDD is again open to conjecture. If there is a true migration for the offshore population of lobsters then these animals could have possibly moved into or through areas containing chemical contamination. Another possibility, although less likely, is the deposition of wind borne combustion products from shore based incinerator and industry stacks. Finally, it might be that these animals, whether migratory or not, accumulated the TCDD in the vicinity of the Mud Hole from prey food rather than from the water column since they are some distance from the Hudson River plume. The Ocean Waste Disposal site for sewage sludge, fly ash and dredge spoils is halfway between the Chapel Hill Channel and the offshores sampling locations. It is well within the forgoing zone of the home territories for both the Ambrose and alongshore lobster fisheries and offers another possible exposure source on (See Map 7). It has also been demonstrated (Mayer 1982) that dredge spoils and sludge dumped at the ocean disposal site may be dispersed through storm related activity thereby widening the zone of exposure. Subsequent contamination of the infauna (i.e. worms, clams, etc.) and certain benthic (bottom) species could then lead to food chain biomagnification with the highest TCDD contaminant concentrations residing in a top predator/scavenger such as the lobster. A DEP report (Long and Figley, 1982) on the offshore Commercial Lobster fishery in

northern N.J. waters (see Map 8) shows how lobsters on the coastal plain are primarily associated with the Hudson Canyon, including the Christiansen Basin and Mud Hole region.

Finally, regardless of the source of contamination, the finding of dioxin in such a commercially important species as the lobster, blue crab and striped bass underlines how contamination events in the estuaries may have long term and severe consequences for species that are far removed from these harsh environments. The implications of this data and the need for more information to be utilized in meaningful management decisions has therefore prompted the Office of Science and Research to continue research of this important species across its entire range within New Jersey waters as it relates to contamination by anthropogenic chemicals such as 2,3,7,8-TCDD.

5. Risk Assessment

Levels of TCDD found in striped bass and blue crabs from Newark Bay and American lobsters from the open ocean were used in estimating lifetime cancer risks. Risk assessment analysis requires the determination of a likely lifetime daily dose of the toxicant in question, in this case TCDD. The data from the analyses of striped bass blue crabs and lobsters were used to obtain average concentrations of $TCDD_5$ in edible portions. Risks were calculated using a potency of 1.56×10^5 $(mg/kg/day)^{-1}$ (EPA 1984). This factor is based on tumors in female Sprague-Dewley rats.

Table IX shows the mean tissue level of TCDD and the excess cancer risk from two lifetime ingestion scenarios. In one scenario there is an assumed average ingestion of 15.7 grams of fish or shellfish per day. This consumption rate was calculated from a study of fish consumption by 25,000 people living in the eight Great Lake states (Cordle, 1983). Data on their dietary intake of fish may be considered better representative of other populations having access to a fisheries resources (i.e. such as New Jerseyans) than an average fish consumption figure for the entire United States. The other scenario is based on the consumption of a given number of animals per year assuming on edible tissue mass of 675, 30 and 200 grams for Striped bass, blue crab and American lobster respectively (Ruppel and Lockwood 2985). This latter approach allows us to put the exposure in terms that the average consumer may understand rather than the hypothetical construct of everyday consumption. It can be seen in Table IX that the animal representing the highest risk varies depending on the ingestion scenario. This is most likely due to the difference in assumed tissue mass and number of organisms consumed however the important thing is that there is excess risk -for consumption of all these species at these levels regardless of approach.

6. N.J. Regulatory Actions

In 1983 the New Jersey Department of Environmental Protection (NJDEP), New Jersey Department of Health (NJDOH), and the United States Environmental Protection Agency (EPA) announced the results of their cooperative investigation into dioxin contamination of fish caught in the estuarine portion of the Passaic River, New Jersey (Phase I). The data received from this survey indicated widespread 2,3,7,8,-TCDD contamination within the aquatic biota in the tidal portion of the Passaic River. Tissue samples collected across this region exhibited concentrations of TCDD in excess of the FDA "Levels of Concern" recommending no consumption at this level (50 ppt). (See Appendix B). Therefore, in accordance with federal and State statutes New Jersey was required to take regulatory action based on this data, and after conferring with FDA's Bureau of Foods the NJDEP issued a fishing advisory for the tidal Passaic River and posted these waters in both English and Spanish with the assistance of municipal health officials (Figure 2) (N.J. Administrative Order No. EO-40-17) stating that: 1) until further notice by this Department, the sale or consumption of all fish and shellfish taken from that portion of the Passaic River from the Dundee Dam in Garfield/Clifton to the mouth of that River at Newark Bay is hereby prohibited, 2) pending the analysis of additional fish and shellfish samples, a presumptive fishing advisory for Newark Bay, the' Hackensack River up to the Oradell Dam, the Arthur Kill from Elizabeth to Perth Amboy, and the Kill Van Kull shall be in effect, and the Department strongly recommends that people not consume any fish or shellfish taken from these waters until further sampling is completed.

August 6th, 1984 the Commisioner of NJDEP, Robert Hughey, signed a second Administrative Order (No. EO-40-19) based an analysis of the data described in this Phase II study. It ordered that: 1) the Prohibition for the sale and consumption of fish and shellfish taken from that portion of the Passaic River from the Dundee Dam in Garfield/Clifton to the mouth of the Passaic River at Newark Bay shall be continued in full force and effect, 2) until further notice by NJDEP the sale or consumption of striped bass and blue crabs taken from Newark Bay, the tidal Hackensack River, the Arthur Kill and the Kill Van Kull is prohibited, and that 3) appropriate notices in English and Spanish concerning the prohibition of the consumption of striped bass and blue crabs taken from Newark Bay, the tidal Hackensack River, the Arthur Kill and the Kill Van Kull shall be posted in conspicuous locations along said waters (See Map 8 and Figure 2). In addition to the sign postings, telephone hotlines were also established for information dissemination through the State and municipal health departments.

The Commissioner also announced (NJDEP Press Release No. 85/11) that due to the likelihood of free movement of striped bass populations from the Passaic River-Newark Bay complex into the Hudson River and due to the identification of dioxin contaminated striped bass by New York DEC in their portion of the Hudson River that recreational fishermen on the Hudson be advised to adhere to the New York State advisory guidelines and to limit their consumption of striped bass caught in the New Jersey portion of the River to no more than one meal per month. Persons at extreme risk such as pregnant mothers, breastfeeding women, and small children were advised to restrict their input even further. The New Jersey and New York commercial striped bass fishery on these drainages is currently closed. The New Jersey 1983 PCB-based "prohibition of sale" regulation prohibited the sale of striped bass by recreational anglers for these waters and an advisory to limit consumption is in place for nearby oceanic waters. (15 N.J.R. 39).

The most immediate public health threat apparently is from the consumption of blue crabs taken throughout the tidal Newark Bay complex, including the two connecting rivers and kills. This area's fishery is primarily recreational however, although a limited amount of small-scale commercial crabbing does occur in the bay and its nearby waters. The majority of New Jersey commercial blue crab fishing takes place in Delaware Bay and as reported no detectable levels of TCDD were found in crabs from those waters.

However, even though the fishery in Newark Bay is predominantly a recreational activity, on peak days a hundred or more individuals have been observed crabbing (Belton and Roundy 1985) If they consume either the hepatopancreas or the contaminated muscle tissue (i.e. cross-contaminated during processing) they may be exposed to a dose of this dangerous contaminant. Kneip et al (1982) identified that cadmium concentrations of cooking liquid from boiled crabs taken from the Foundry Cove section of the Hudson River can contain high contaminant concentrations leached out during cooking. If these liquids were then added to sauces or stews as flavorings they would thereby significantly increase the dietary intake of cadmium. This same process may also increase the dietary intake of other contaminants present such as TCDD. In addition, if skin contact with dioxin-laden hepatopancreas tissue occurs while cleaning out muscle meat for consumption they may also facilitate ingestion of TCDD since crabs from the shell is usually eaten with the fingers. Therefore, in light of the elevated dioxin concentrations found in the crab tissue and the propensity for exposure even if an attempt was made to discard the “mustard” it was felt best to assume a conservative approach and to prohibit the consumption and sale of this species from these waters.

It is also important to emphasize that the striped bass and the blue crabs which frequent these waters are migratory and will distribute themselves seasonally throughout the Newark Bay-Upper New York Bay-Inner New York Bight complex. The contamination of striped bass and blue crabs therefore may be instrumental in the transport of TCDD into the entire Hudson-Raritan estuary and its associated ocean waters. This possibility raises serious health risk implications for the fish-consuming public of the entire region since these organisms are some of the major recreational and commercial species sought in these waters. Studying the possible range of TCDD distributions in these organisms will be the target of ongoing research at OSR as well as investigations of TCDD associated sediment transport and storage of dioxin within the affected drainages. In fact, OSR's planned Newark Bay sediment study will be an in-depth analysis of the problem utilizing suspended sediment samples, bottom grabs and bottom cores which will be dated via geochronological techniques in order to more fully understand how contaminant storage take place in the Newark Bay system and how much is exported via the sediment plume and the migrating biota.

Finally, due to the extreme toxicity of 2,3,7,8-TCDD and the elevated levels in bottom sediments and biota near the Diamond Alkali plant in spite of the fact that the site has been inactive for over twelve years may indicate that, transport and storage of dioxin downstream as well as subsequent transport offshore during dredge spoil disposal from the Ports of Newark and Elizabeth may have occurred historically. Therefore the need for future research as to the levels and extent of 2,3,7,8-TCDD contamination and the possible detoxification schemes for soils and sediments as well as the implications for the management of public goods such as harbors and fisheries make it plausable to claim that an "Environmental Endowment" needs to be set up structured along the lines of the "Virginia Environmental Endowment" due to Kepone on the James River, Va. and the "Hudson River Settlement Panel", N.Y. due to PCBs on the Hudson River. This fund could then be used to address future dioxin related research issues for the Ports of New York and New Jersey, as they arise.

7. Conclusions

1. Extensive Contamination of soils by the chemical toxin 2,3,7,8-tetrochlorodibenzo-p-dioxin (2,3,7,8-TCDD) was discovered at the former Diamond-Alkali plant in Newark, N.J. (Range: 0.06-50 ppm).

2. Bottom sediments taken from the Passaic River which flows past the plant were shown to be contaminated with 2,3,7,8-TCDD at levels ranging from non-detectable to 6.9 parts per billion (ppb).

3. 2,3,7,8-TCDD was also found in bottom sediments 11.5 miles upstream of the Newark site at the confluence of the Passaic and Third Rivers (ie. 0.11 ppb and 0.18 ppb). Givaudan Chemical Company which is situated near these locations was found to have on site soils and storm drains contaminated by 2,3,7,8-TCDD.

4. The only other locations to show positive 2,3,7,8-TCDD levels in sediments (ie. 0.05 ppb) was outside of the Newark Bay System on Raccoon Creek, Gloucester County, N.J. It also possessed one white perch sample with elevated levels of 2,3,7,8-TCDD (42 ppt).

5. Analysis of edible finfish and crustaceans in Phase I sampling (1983) consistently showed elevated levels (>10 ppt) of 2,3,7,8-TCDD in both resident and migratory species collected on the tidal Passaic River. Resident species with elevated TCDD concentrations included: carp (Mean = 110 ppt); catfish (Mean = 62 ppt); and goldfish (66 ppt). Contaminated migratory species caught on the tidal Passaic River included: American eel (Mean = 38 ppt); striped bass (Mean = 45 ppt); and blue crab (Mean muscle tissue = 21 ppt and Mean hepatopancreas tissue= 467 ppt).

6. Subsequent biota collections in Phase II sampling (1984) showed positive TCDD results for seven species.

7. Phase II, tidal Passaic River results replicated the findings from Phase I showing elevated levels in blue crab, catfish and carp although analysis of fish taken from above the head of tide showed no detectable levels of TCDD in any of the representative species indicating that the sources of dioxin contamination are probably confined to the tidal section of the river.

8. Phase II collections of migratory fish from the Newark Bay Complex downstream of the point sources (i.e. Newark Bay, Hackensack River, Kill Van Kull and Arthur Kill) showed that those species identified with elevated TCDD concentration on the tidal Passaic River were also contaminated throughout the adjacent waterways as well.

9. Phase II blue crab hepatopancreas samples contained the highest TCDD levels identified within the study (Mean = 496 ppt) whereas muscle or lump meat showed no detectable levels (< 10 ppt). Combined muscle-hepatopancreas samples were analyzed as recommended by the U.S. Food and Drug Administration (FDA) and still showed elevated levels of TCDD (Mean = 184 ppt). Control samples of blue crabs from Great Egg Harbor and Delaware Bay showed no detectable levels of TCDD.

10. Phase II striped bass results (Mean = 40 ppt) showed 2,3,7,8-TCDD levels consistent with amounts found in Phase I and in studies by the New York Department of Environmental Conservation (NYDEC) on the Hudson River.

11. Phase II striped bass were also contaminated with 2,3,7,8-tetrochlorodibenzo- p-furan (Mean: 26 ppt) which has toxic properties similar to TCDD.

12. Phase II American eel results were either non-detectable for TCDD or else displayed high detection limits making it impossible to quantify them relative to the elevated levels found for eels on the tidal Passaic River in Phase I.

13. Phase II samples of select species from the contiguous ocean waters of the New York Bight revealed elevated levels of TCDD in two bluefish out of nineteen analyzed (Mean = 45 ppt).

14. Phase II American lobster like the blue crabs were analyzed for 2,3,7,8-TCDD as combined samples of muscle--hepatopancreas and as hepatopancreas tissue above. Combined tissue samples of lobster caught nearshore within Raritan Bay showed elevated levels of TCDD (Mean = 44) ppt). Samples of lobster hepatopancreas caught much further offshore in deep water were similarly contaminated with dioxin (Mean = 77 ppt).

15. Quantitative risk assessment was applied to Phase II striped bass, blue crabs and American lobsters to estimate excess human cancer risk. The risk from eating the contaminated striped bass is estimated to be 1300 cancers per million people, 3300 cancers per million people (for contaminated crab meat (muscle-hepatopancreas mix)) and 1500 cancers per million people (for contaminated lobster meat (muscle-hepato mix) although based on a smaller size. A second risk assessment based on the number of organisms eaten also showed excess risk of cancer.

16. The public health implications of the levels and geographic distribution for 2,3,7,8-TCDD found in the various finfish and crustaceans relative to FDA's "Levels of Concern" resulted in the New Jersey Departments of Environmental Protection and Health issuing prohibitions on the sale and consumption of all fish and shellfish taken from the tidal Passaic River, and on striped bass and blue crabs taken from Newark Bay, the tidal Hackensack River, the Arthur Kill and the Kill Van Kull.

8. References

Andrews, W.D., 1980, Management of the lobster, Homarus americanus, Resources of the Continental Shelf, Canyons and Slopes of the Northern Portion of Area IV and Southern Portion of Area III, New Jersey Dept. of Envir. Prot., Div. Fish, Game and Wildlife.

Bartleson, F.D., Jr., D.D. Harrison, and J.B. Morgan, 1975. Field Studies of Wildlife Exposed to TCDD-Contaminated Soils. Air Force Armament Lab. Eglin A.F. Base, Flordia.

Baughman, R.W. and M. Meselson, 1973. An Analytical Method for Detecting TCDD (Dioxins): Levels of TCDD in Samples from Vietnam. Environmental Health Perspectives, 5:27-35, September.

Belton, T.J. and R. Roundy, 1985, A study of Toxic Hazards for Urban Subsistence and Recreational Fishermen: 1983 to 1984, DEP technical report to Hudson River Foundation.

Belton, T.J., Ruppel, B., Lockwood, K., and M. Boriek, (1983) PCB in Selected finfish Caught within New Jersey Waters 1981-1982 (With Limited _hlo dane Data), New Jersey Department of Environmental Protection, Tec:tnical Report, 43 pp.

Belton, T,J., Ruppel, B., and K. Lockwood, (1982), Polychlorinated Biphenyls, (Aroclor 1254) in Fish Tissue Throughout: the State of New Jersey: A Comprehenxive Survey, New Jersey Department of Environmental Protection Technical Report, 43 pp.

Bergeron, J. (1967), Natur. Can. 94, 169.

Bleiberg et al. "Industrially Acquired Prophyria," Archives of Dermatology, 89:793-797, 1964.

Bliss, D., 1982, Shrimps, Lobsters and Crabs, New Century Publishers, Inc. pp 166-170.

Bliss, D.E., Vernberg, F.J. and W.B. Vernberg, 1983, the Biology of the Crustacea, Vol. 7, Behavior and Ecology, Academic Press 1983.

Buser, H.R., H.P. Bosshardt and C. Rappe, 1978. Identification of Polychlorinated Dibenzo-p-dioxin Isomers Formed in Fly Ash. Chemosphere, 7(2):165-172.

Califano, R.J., J.M. O'Connor and J.A. Hernondey, 1982. Polychlorinated Biphenyl Dynamics in Hudson River Striped Bass, I., Accumulations in early Life History Stages. Aquat. Toxicolo., 2, pp 187-204.

Cantelmo, A. and L. Mantel, 1979, the Effect of Petroleum Hydrocarbons on the Physiology of the Blue Crab (Callinectes Sapidus), Report #79-040 to the N.J. Oil Spill Research Fund.

Chemical Week, 1979. Hooker Dumpsites May Pose Dioxin Threat. 124(1):16, January 3.

Child, J., Bertholle, L. and S. Beck, 1961, "Mastering the Art of French Cooking: Vols. I and II, Alfred A. Knopf, Inc., N.Y.

Cooper, R.A., (1970) Trans. Amer. Fish. Soc. 99, 409 (1970): D. M. Harriman, Progr. Rep. on Lobster Tagging-Monhegan, 1951-1952, Maine Dep. Seashore Fish, No. 5 (1953).

Cooper, R., and Uzmann, J. (1971). Migrations and growth of deep-sea lobsters, Homarus americanus. Science 171,288-290.

Cooper, R., and Uzmann, J. (1980). Ecology of juvenile and adult Homarus. In "The Biology and Management of Lobsters" (S. Cobb and B. Phillips, ed.) Vol. II, pp. 97-142. Academic Press, New York.

Cordle, F. (1983) "Use of epidemiology in the regulation of dioxins" Chapter 16, pp. 245-256, Accidental Exposure to Dioxins, Academic Press.

Courtney, K.D., 1976. Mouse teratology studies with Chlordibenzo-pDioxins. Bulletin of Environmental Contamination and Toxicology, 16(6):674-681.

Creutzberg, F. (1961). On the orientation of migrating elvers (Anguilla vulgaris Turt.) in a tidal area. Neth. J. Sea Res. 1, 257-338.

Crow, K.D. 1_,8. Chloracne--fin Up-To-Date Assessment. Ann. Occur. Hyg. 21(3):297-8.

Davidson, A., 1979 "North Atlantic Seafood", Viking Press.

Doliber, E.L., 1973, "Lobstering: Inshore and Offshore", Associated Press, N.Y.

Dow Chemical Company, 1978. The Trace Chemistries of Fire-The Source of and Routes for the Entry of Chlorinated Dioxins into the Environment. The Chlorinated Dioxins Task Force, The Michigan Division, Dow Chemical, U.S.A.

EPA (1979). "Hankbook for Analytical Quality Control in Water and Wastewater Laboratories" EPA-600/4-79-019, March, 1979.

EPA, 1980a, Dioxins, by M.P. Esposito, T.O. Tiernan and Forrest E. Dryden. EPA-600/2-80-197.

EPA 1980b, "Manual of Analytical Methods for the Analysis of Pesticides in Humans and Environmental Samples," EPA 600/8-80-038, June 1980; and

EPA (1983). "Determination of 2,3,7,8-TCDD in Soil and Sediment" EPA, Region VII, Kansas City, September 1983.

EPA (1984) Health Assessment Document for Polychlorinated Dibenzo-pDioxins; Parts 1 and 2, EPA-600/8-84-014A, May 1984.

Evans, P.D. and Mann, K. H. (1977). Selection of prey by American lobsters (Homarus americans) when offered a choice between sea urchins and crabs. J. Fish. Res. Board Can. 34, 2203-2207.

Firestone, D., 1972. Determination of Polychlorodibenzo-p-dioxins and Related Compounds in Commercial Chlorophenols. Journal of the AOAC, 55(1):85-92.

FDA 1981, Food and Drug Administration Correspondence from FDA Commissioner Dr. Arthur Hull Hayes, Jr., to Governor Milliken of Michigan concerning advice on the Public Health Significance of TCDD Contaminant "Levels of Concern" for Finfish in Great Lakes (8/26/81).

FDA 1983, Food and Drug Administration Correspondence from John Taylor (Director, Div. Regulatory Guidance) to Thomas Belton (NJDEP) outlining on Advisory Opinion on N.J. Public Health Announcements Concerning TCDD Contamination on Finfish and Crabs (12/5/83).

Galston, A.W., 1979. Herbicides: A Mixed Blessing. Bioscience, 29(2):85-89, February.

Hardy Jr., J.D., 1978. Development of Fishes of the Mid-Atlantic Bight, Vol. II, Anguillidae through Synganathidae, U.S. Fish and Wildlife Publication, FWS/OBS-78/12 January 1978.

Harless, R.L. 1980. Direct Testimony before the Administrator, U.S. EPA, Re: FIFRA Docket Nos. 415 et al. EPA Exhibit No. 212. April 23.

Hasler, A., and Scholz, A. (1978). Olfactory imprinting in coho salmon (Onchorhynchus kisutch). In "Animal Migration, Navigation, and Homing" (K. Schmidt-Koenig and W. Keeton, eds), pp. 356-369. Springer-Verlag, Berlin and New York.

Humason, A.W. and D.F. Gadbois, 1982, Determination of Polynuclear Aromatic Hydrocarbons in the New York Bight Area, Bull. Environ. Contam. Toxicol. 29, 645-650, (1982).

Isensee, A.R., and G.E. Jones, 1975. Distribution of 2,3,7,8-Tetrachloro-dibenzo-p-Dioxin (TCDD) in Aquatic Model Ecosystems. Environmental Science and Technology 9(7):668-672.

Johnson, P.T., 1982, Histology of the Blue Crab, Callinectes sapidus, A Model for the Decapoda, U.S. Department of Commerce, NOAA, NMFS, Northeast Fisheries Ctr, Praeger Inc.

Kaczmar, S., M.J. Zabik, and F. D'Itri, 1983, "The Extent and Geographical Distribution of 2,3,7,8-TCDD Residues in Michigan", Report from Michigan State University, February 1983.

Kimbrough, R.D., et al. (1983) Health implications of 2,3,7,8-tetrachlorodibenzodioxin (TCDD) contamination of residential soil. Center for Environmental Health; Center for Disease Control; Atlanta, Georgia 30333.

Kneip, T.J. O'Connor, J., Wiedow, A., and R. Hazen, (1979) Cadmium in Foundry Cove Crabs: Health Hazard Assessment, New York University Medical Center Report to Health Research Council, State of New York, September 1979.

Kociba, R.J., et al. 1978. Results of a Two Year Chronic Toxicity and Oncogenicity Study of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin in Rats. Toxicol. Appl. Phacmacol., 46(2):279-304.

Kouri, R.E., et al. 1978. 2,3,7,8-Tetrachlorodibenzo-p-Dioxin a Cocarcinogen Causing 3-Methylcholanthrene-Initiated Substaneous Tumors in Mice Genetically "Nonresponsive" at Ah Locus. Cancer Research, 38:2777-2783, September.

Krewski, L. et al., (1981). "Dose response models for quantal response toxicity data, pp. 201-231. Statistics and Related Topics, North Holland, Amsterdam.

Laughlin, R., Cripe, R., and Livingston, R. (1978). Field and laboratory avoidance reactions by blue crabs (Callinectes sapidus) to storm water runoff. Trans. Am. Fish. Soc. 107, 78-86.

Long, D. and W. Figley, 1982, New Jersey Recreational and Commercial Ocean Fishery Grounds, Technical Report #82-1.

Longstreth, J.D. et al. (1983). "Risk assessment for 2,3,7,8-tetrachlorodibenzo-p-dioxin," pp. 639-664. Human and environmental risks of chlorinated dioxins and related compounds, Plenum, New York.

Mayer, G.F. ed., 1982, Ecological Stress and the New York Bight: Science and Management, Estuarine Research Federation Publication, Columbia, S.C.

Modell, J., R.P. deFilippi, and V. Krukonis. 1978. Regeneration of Activated Carbon with Supercritical Carbon Dioxide. Presented at the ACS Conference. Miami, Florida. September 14.

Montrevil, P. (1954), "Sta. Biol. Mar. Grande Riviere Que. Rapp. Annu. 1953 (1954), p. 75.

Myerson, A.L., Luther, G.W., Krajeswki, J. & Hires, R.I. (1981). Heavy Metal Distribution in Newark Bay Sediments. Mar. Pollut. Bull., 12, 244-250.

Neubert, D., and I. Dillman, 1972. Embryotoxic Effects in Mice Treated with 2,4,5-Trichloropehnoxyacetic Acid and 2,3,7,8-Tetrachlorodibenzo-p-Dioxin. Naunyn-Schmierdebergs's Ach. Exp. Path. Pharmacol, 272:243-264.

NOAA 1982a: "Gulf and Atlantic Survey for Selected Organic Pollutants in Finfish", by P.D., Boehm and P. Hirtzer (Energy Resource Co.), NOAA Tech. Memo. NMFS-F/NEC-13.

NOAA 1982b: "Contaminants in New York Bight and Long Island Sound Sediments and Demersal Species, and Contaminant Effects on Benthos, Summer 1980, eds., Reid, R.N., O'Reilly, J.E. and V. S. Zdanowicz, NOAA Tech. Memo NMFS-F/NEC-16.

O'Connor, J.M., Klotz, J.B. and T.J. Kneip, 1982, "Sources, Sinks, and Distribution of Organic Contaminants in the New York Bight Ecosystem" in "Ecological Stress and the New York Bight: Science and Management, Mayer G.F., ed., Estuarine Research Federation Publ., Columbia, S.C.

O'Keefe, P., Hilker, D., Meyer, C., Aldous, K., Shane, L., Donnelly, R., Smith, R.; Sloan, R., Skinner, L. and E. Horn, 1984, "Determination of Tetrachlorodibenzo-p-Dioxins and Tetrachlorodibenzofurans in Atlantic Coast Striped Bass and in selected Hudson River Aquatic Biota and Sediments", New York State Department of Health and Environmental Conservation (Unpublished Report).

O'Connor, J. (1984). Institute of Environmental Medicine, Tuxedo, New York. Personal communication.

Olie, Vermeulen, Hutzinger, 1977. Chlorodibenzo-p-Dioxins and chlorodibenzofuran are Trace Components of Fly Ash and Flue Gas. Some Municipal Incinerators in the Netherlands, Chemosphere, 8:455-459.

Poland, A. et. al. "A Health Survey of Workers in a 2,4-D and 2,4,5-T Plant, "Archives of Environmental Health, 22:316-327, 1971.

Poland, A. and A. Kende, 1976. 2,3,7,8-Tetrachlorodibenzo-p-Dioxin; Environmental Contaminant and Molecular Probe. Federal Proceedings, 35(2):2404-2411.

Pyle R. and E. Cronin, 1950, The General Anatomy of the Blue Crab (Callinectes sapidus Rathbun), Publ. No. 87, State of Maryland, Board of Nat. Res., Department of Research and Education.

Roberts, A.E., Hill, D.R., and E.C. Tifft, Jr., 1982, Evaluation of New York Bight Lobsters for PCBs, DDT, Petroleum Hydrocarbons, Mercury and Cadmium, Bull. Environm. Contam. Toxicol. 29, 711-718(1982).

Ross, J., 1978, "The Seafood Book", McGraw-Hill.

Ruppel, B., Belton, T. and K. Lockwood, 1984, The Occurrence of Cadmium, PCB's and Selected Pesticides in Blue Crabs, Callinectes sapidus from Estuarine Water of New Jersey, Proceedings: Conference on the Fate and Transport of Toxicants in the Hudson River, Northeast Science Foundation (abstract).

Ruppel, B. and K. Lockwood, NJDEP (1983) Personal Communication.

Russel-Hunter, W.D. 1969, "A Biology of Higher Invertebrates", Macmillian Public Co., N.Y.

Ryan, J.J.; Lau, P-Y, Pilan, J.C. Lewis, D., McLeod, H.A., and A. Gervals, 1984, Incidence and Levels of 2,3,7,8-TCDD in Lake Ontario Commercial Fish, Envir. Sci. Technol., Vol. 18, No. 9, pp 719-721.

Sarvis. J., 1968, "Crab and Abalone; West Coast Ways with Fish and Shellfish", The Bobbs-Merrill Co. Inc.

Schaefer, R. H. 1968. Size, Age Composition and Migration of Striped Bass from Surf Waters of Long Island. New York Fish and Game Journal, Vol. 15, (1), pp 1-51.

Schimmel, J.C., Patrick, J.M. Faas, L.F. Oglesby, J.L., and Wilson, A.J., Jr. 1979, Kestone: Toxicity to and bioaccumulation of blue crabs. Estuaries 2,9-15.
Scud, T., 1970, Inshore and Offshore Lobster Form Separate Populations, National Fisherman Yearbook, 1970, p. 44.

Sheridan, P.F. 1975, Uptake, metobolism and distribution of DDT in organs of the blue crab, Callinectes sapidus. Chesapeake Scio. 16,20-26.

Simmons, H.B. & Bobb, W.H. (1965). Hudson River Channel, New York and New Jersey. Plans to reduce shoaling in Hudson River channels adjacent pier slips. U.S. Army Engineer Waterways Experiment Station. Vicksburg, Miss. Tech. Report No. 2-694, 100p.

Squires, H.J. (1965). Decapod crustaceans of Newfoundland, Labrador and the Canadian eastern Arctic. Fish. Res. Board Can., Rep. Ser. (Biol.) No. 810, pp. 1-212.

Suszkowski, D. J. (1978) "Sedimentology of Newark Bay, New Jersey; An Urban Estuarine Bay", Ph.D. Dissentation, University of Delaware.

Tucker, R.E., Young, A.L. and A.P. Gray, eds., 1983, Human and Environmental Risks of Chlorinated Dioxins and Related Compounds, Plenum Press, N.Y.

U.S. Department of Interior, 1968, "Lobster Tagging Procedures Interesting Information", Commercial Fisheries Review, 31, p. 4.

U.S. NIEHS IARC, 1978, U.S. National Institute of Environmental Health Sciences/International Agency for Research on Cancer. 1978. Long-Term Hazards of Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans. Joint Working Group Report, IARC, Lyon, France.

Weaver, G. 1984, "PCB Contamination in and around New Bedford, Mass.", Environ. Sci. Technol, Vol. 18, No. 1, 1984.

Wilder, D.G. and R.C. Murray, 1958, Do Lobsters more offshore and onshore in the fall and spring? Atlantic Progress: Report No. 69. Atlantic: Fisheries Research Board of Canada. pp 12-15.

Williams, A.B. and T.W. Duke, 1979 Crabs (Arthropods: Crustacea: Decapoda: Brachyrua) pp 171-233, in "Pollution Ecology of Estuarine Invertebrates", eds. Hart, C.W. and S.L.H. Fuller, Academic Press, 1979.

World Health Organization, 1977. International Agency for Research on Cancer. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Some Fumigants, the Herbicides 2,4-D and 2,4,5-T, Chlorinated Dibenzodioxins and Miscellaneous Industrial Chemicals. Vol. 15, Lyon, France, August.

Wright State University, 1979. Report on Analyses of Love Canel Samples for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD). New York State Department of Health Purchase Order No. 5975. January 21.

9. BIBLIOGRAPHY

OFFICE OF SCIENCE AND RESEARCH Information Resource Center Bibliography:
DIOXINS

(Information Resource Center)
(609) 984-2249 or 984-7320
prepared by JoAnn J. Post

Call Numbers	Books
	American Chemical Society. Division of Environmental Chemistry. 182nd National Meeting, New York, New York, August 23-28, 1981. Vol. 21, No. 2 (Detoxification of dioxins). [Washington, D.C.: ACS], 1981.
	American Medical Association. Council on Scientific Affairs. The Health Effects of "Agent Orange" and Polychlorinated Dioxin Contaminants; Technical Report. Chicago, Ill.: AMA, 1981. (CRF)
	The American Society of Mechanical Engineers. Research Committee on Industrial and Municipal Wastes. Study on State-of-the Art of Dioxin from Combustion Sources. New York, N.Y.: The Society, 1981.
TP248 .P47P48	Bandal, S. Kris; Marco, Gino J.; Golberg, Leon; and Leng, Marguerite L. L., eds. The Pesticide Chemist and Modern Toxicology, "Based on a symposium sponsored by the ACS Division of Pesticide Chemistry at a Special Conference at Downington, PA, June 26, 1980, ACS Symposium Series 160. Washington, D.C.: American Chemical Society, 1981.
	Barnes, Donald G. "Assessing TCDD Emissions from Municipal Waste . Combustors," (paper presented at the 3rd International Symposium on Chlorinated Dioxins and Related Compounds, Salzburg, Austria, October 12-14, 1982).
SB951.4 .B66	Bovey, Rodney W. The Science of 2,4,5-T and Associated Phenoxy Herbicides. New York, N.Y.: Wiley, c1980.
	Canada. Ministry of Labour. Special Studies and Services Branch. Chlorinated Dioxins and Chlorinated Dibenzofurans: Ambient Air Guideline, by D.H. Harding. [Canada: Environment Canada, 1982].
RA1242 T44D56	Cattabeni, Flaminio; Cavallaro, Aldo; and Galli, Giovanni, eds. . Dioxin: Toxicological and Chemical Aspects. Jamaica, N.Y.: SP Medical and Scientific Books, 1978.
	Choudhary, Gangadhar, ed. Chemical Hazards in the Workplace: Measurement and Control. ACS Symposium Series 149. Washington, D.C.: American Chemical Society, 1981.
RA1242 T44A25	Coulston, Federick; and Pocchiari, Francesco, eds. Accidental . Exposure to Dioxins: Human Health Aspects. New York, N.Y.: Academic Press, 1983.

Electric Power Research Institute. The Chlorinated Phenoxy Acids and their Related Dioxins: An Overview of Issues and Evidence. Draft Report, by Anna M. Fan, W.S. Simmons, and A.V. Colucci. Palo Alto, CA: The Institute, 1979.

Esposito, M. Pat and Walkins, David R. "Airborne Dioxins: the Problem in Review," (paper presented at the 73rd Annual Meeting of the Air Pollution Control Association, Montreal, Quebec, June 22-27, 1980).

Exner, Jurgen H. "Detoxication of Chlorinated Dioxins," (paper presented before the Division of Environmental Chemistry, American Chemical Society, New York, N.Y., August 1983).

Gross, Michael L.; Weerasinghe, N.C.A.; Meehan, J.L.; and Harless, Robert. "The Analysis of Tetrachlorodibenzo-p-dioxins and tetra-chlorodibenzofurans in Chemical Waste and in the Emission from its Combustion," (paper presented before the Division of Environmental Chemistry, American Chemical Society, Washington, D.C., September, 1983).

Hay, Alastair. "The Mutagenic 'Properties of 2,3,7,8-Tetrachloro-dibenzo-p-dioxin," (paper presented before the Division of Environmental Chemistry, American. Chemical Society, Washington, D.C., August, 1983).

Hob.1, L.B.; Lee, L.E.; Gross, M.L.; and Young, A.L. "Dioxin in Body Fat and Health Status: A Feasibility Study," (paper presented before the Division of Environmental Chemistry, American Chemical Society, Washington, D.C., September, 1983).

Kainz, Robert and Simpson, Michael M. Dioxin: Environmental Impacts and Potential Human Health Effects. Issue Brief No. IB83079. Washington, D.C.: The Library of Congress, 1983.

Kleopfer, Robert D. and Kirchmer, Cliff J. "Quality Assurance Plan for 2,3,7,8-TCDD Monitoring Project," (paper presented before the Division of Environmental Chemistry, American Chemical Society, Washington, D.C., September, 1983).

National Institute of Environmental Health Sciences and International Agency for Research on Cancer Working Group. Long-term Hazards of Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans. Lyon: IARC, 1978.

RA591 National Research Council. Safe Drinking Water Committee. .N37 Drinking Water and Health. Washington, D.C.: National
Vol. 1C.3 Academy of Sciences, 1977

NTP80-31 National Toxicology Program. NTP Technical Report on the Carcino-genesis Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (CAS No. 1746-

01-6) in Osborne-Mendel Rats and B6C3F, Mice (Gavage Study). NIH 82-1765. Washington D.C.: U.S.G.P.O., 1982.

NTP80-32 NTP Technical Report on the Carcinogenesis Bioassay 2,3,7,8-Tetrachlorodibenzo-p-dioxin (CAS No. 1746-01-6) in Swiss- Webster Mice (Dermal Study). NIH 82-1757. Washington, D.C.: U.S.G.P.0, 1982.

New Jersey. Department of Environmental Protection. Office of Science and Research. Background Report on the Dioxin Study by Thomas Belton. [Trenton, N.J.: NJDEP], 1983.

_____. Combustion, Resource Recovery and Chlorinated Organic By-Products by Ronald Harkov. [Trenton, N.J.: NJDEP, 1983]. (Contact R. Harkov)

NJAOC-81 New Jersey Agent Orange Commission. New Jersey State Commission on Agent Orange; Legislative Report. Trenton, N.J., 1981.

NYTCDE 83-2 New York. Temporary Commission on Dioxin Exposure. What You Should Know About Dioxin. Albany, N.Y.: The Commission, 1983.

Saxena, Jitendra, ed. Hazard Assessment of Chemicals: Current Developments, Vol. 2. New York: Academic Press, 1983.

Taylor, Michael L.; Tiernan, Thomas 0.; Garrett, John H.; Van Ness Garrett F.; and Solch, Joseph G. "Assessments of Incineration Processes as Sources of Supertoxic Chlorinated Hydrocarbons: Concentrations of CDDs/CDFs and Possible Precursor Compounds in Incinerator Effluents," (paper presented at the Symposium on Chlorinated Dioxins and Dibenzofurans in the Total Environment, Division of Environmmental Chemistry, American Chemical Society National Meeting, Kansas City, MO., September 1982).

Tung, Ton-That. Primary Cancer of the Liver in Viet Nam. Translated from French by the Medical Documentation Service, College of Physicians, Philadelphia, PA. Chirurgie, Paris: In Press, 1973?

United States. Air Force. The Surgeon General. Project Ranch Hand II: An Epidemilogic Investigation of Health Effects in Air Force Personnel Following Exposure to Herbicides; Baseline Mortality Study Results. [Washington, D.C.: U.S.A.F.], 1983.

USAF/OEHL _____. The Toxicology, Environmental Fate, and Human Risk Herbicide Organe and its Associated Dioxin. Final Report. Washington D.C.: U.S.G.P.O 1979

U.S. Environmental Protection Agency. Environmental Monitoring and Support Laboratory. EPA-Test Method: 2,3,7,8-Tetrachlorodibenzo-p-Dioxin-Method 613, [Cincinnati, OH: U.S.E.P.A.], 1982.

EPA-600/ Environmental Research Laboratory. Transport of

3-78-056 Agricultural Chemicals from Small Upland Piedmont Watersheds, by C.N. Smith, et al. Athens, GA: U.S.E.P.A., 1978.

EPA-600/ 2-80-197 _____. Dioxins, by M.P. Esposito; T.O. Tiernan; and Forrest E. Dryden. Cincinnati, OH: U.S.E.P.A., 1980.

EPA-600/ 2-80-156 _____. Dioxins: Vol. I. Sources, Exposure, Transport and Controls, by T.O. Tiernan et al. Cincinnati, OH: U.S.E.P.A., 1980.

EPA-600/ 2-80-157 _____. Dioxins: Vol. II. Analytical Method for Industrial Wastes, by T.O. Tiernan et al. Cincinnati, OH: U.S.E.P.A., 1980.

_____. Office of Solid Waste and Emergency Response. Health and Environmental Effects Profile for: Tetra-, Penta - and Hexachlorodibenzo-p-dioxins. (Peer review draft) [Cincinnati, OH: U.S.E.P.A.], 1983.

EPA-600/ 6-81-003 _____. Office of the General Counsel. Risk Assessment on (2,4,5-Trichloropehnoxy) Acetic Acid (2,4,5-Trichlorophenoxy) Propionic Acid, 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), by the Carcinogen Assessment Group. [Washington, D.C.: U.S.G.P.0.], 1980.

_____. Office of Water Regulations and Standards. Ambient Water Quality Criteria for 2,3,7,8-Tetrachlordibenzo-p-dioxin. (peer review draft) [Cincinnati, OH: U.S.E.P.A.], 1983. (Contact D. Patel)

GAO/CED 78-158 U.S. General Accounting Office. Community and Economic Development Division. The Military Use of Herbicides in South Vietnam, by Henry Eschwege to The Honorable Ralph H. Metcalfe. Washington, D.C.: U.S.G.A.O., 1978.

GAO/CED 79-22 Comptroller Genera. Health Effects of Exposure to Herbicide Orange in South Vietnam Should be Resolved. Washington, D.C.: U.S.G.A.0, 1979.

GAO/FPCD 80-23 U.S. Ground Troops in South Vietnam were in Areas Sprayed with Herbicide Orange. Washington, D.C.: U.S.G.A.O., 1979.

V101(93) P-823 Veterans Administration. Department of Medicine and Surgery. Review of Literature on Herbicides, Including Phenoxy Herbicides and Associated Dioxins. Vols. I & 2. Washington, D.C.: V.A., 1982.

Whiteside, Thomas. The Pendulum and the Toxic Cloud: The Course of Dioxin Contamination. New Haven, CT: Yale University Press, 1979. (N.J. State Library)

Young, A.L. and Arnold, E.L. "Environmental Fate of TCDD - Conclusion from Three Long-Term Field Studies", (paper presented before the Division of Environmental Chemistry, American Chemical Society, Washington, D.C., September, 1983).

Periodical Articles

Ayres, D.C. "Destruction of Polychlorodibenzo-p-dioxins," Nature 290 (March 26, 1981) pp. 323-234.

Barnes, Donald G. "Dioxin Questions and Answers," The Environmental Forum 2(5):41-44, 1983.

Baughman, Robert and Meselson, Matthew. Vietnam," 1973. "An Analytical Method for Detecting TCDD (Dioxin): Levels of TCDD in Samples from Environmental Health Perspectives 5 (Sept.):27-35, 1973

Bumb, R.R.; Crummett, W.B.; Cutie, S.S.; Gledhill, J.R.; Hummel, R.H.; Kagel, R.O.; Lamparski, L.L; Luoma, E.V.; Miller, L; Nestrick, T.J.; Shadoff, L.A.; Stehl, R.H.; and Woods, J.S. "Trace Chemistries of Fire: A Source of Chlorinated Dioxins,' Science 210(4468): 385-390, 1980.

Buser, Hans R. and Bosshardt, Hans-Paul. "Identification of Polychlorinated Dibenzofuran Isomers in Fly Ash and PCB Pyrolyses," Chemosphere 5:419-429, 1978.

Carter, Coleman D.; Kimbrough, Renate D.; Liddle, John A.; Cline, Richard E.; Zack, Matthew M.; Barthel, William F.; Koehler, Robert E.; and Phillips, Patrick E. "Tetrachlorodibenzodioxin. An Accidental Poisoning Episode in Horse Arenas," Science 188 (May 16):738-740, 1975.

Cocucci, S.; DiGerolamo, F.; Verderio, A.; Cavallaro, A.; Colli, Gorn., A.; Invernizzi, G.; and Luciani, L. "Absorption and Translocation of Tetrachlorodibenzo-p-dioxin, by Plants from Polluted Soil," Experientia 35(4):482-484,1979.

Cook, Ralph R.; Townsend, Jean C.; Ott, M. Gerald; Silverstein, Lawrence G. "Mortality Experience of Employees Exposed to 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), "Journal of Occupational Medicine 22(8):530, 1980.

Council on Scientific Affairs. "Health Effects of Agent Orange Dioxin Contaminants," Journal of the American Medical Association 248(15):1895-1897, 1982.

Crosby, D.G.; Wong, A.S.; Plimmer, J.R.; and Woolson, E.A. "Photodecomposition of Chlorinated Dibenzo-p-dioxins," Science 173 (August 20):748-749, 1971.

Crosby, Donald G.; and Wong, Anthony S. "Environmental Degradation of 2,3,7,8-TCDD," Science 195 (March 25):1337-1338, 1977.

Crossland, Janice. "Dioxin: the lingering Controversy," The Ecologist 10(3):87-93, 1980.

Dagani, Ron. "Seveso: Five Years Later, Questions Remain," Chemical and Engineering News 59 (June 29, 1981) pp. 18-20. (See CRF)

Des Rosiers, Paul E. "Remedial Measures for Wastes Containing Polychlorinated Dibenzo-p-Dioxins (PCDDs) and Dibenzofurans

(PCDFs): Destruction, Containment or Process Modification," Annals of Occupational Hygiene 27(1):57, 1983.

DiDomenico, Alessandro; Silano, Vittorio; Viviano, Giuseppe; and Zapponi, Giovanni. "Accidental Release of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) at Seveso, Italy," Ecotoxicology and Environmental Safety 4:283-356, 1980.

Dioxin - A C&EN Special Issue. Chemical and Engineering News 61(25):(entire issue), 1983.

Environmental Health perspectives (NIH 74-218) Experimental Issue No. 5, September 1973.

Fanelli, R.; Bertoni, M.P., Bonfanti, M.; Castelli, M.G.; Chiabrando, C.; Martelli, G.P.;Noe, M.A.; Noseda, A.; Garrattini, S.; Binaghi, C.; Marazza, V.; Pezza, F.; Pozzoli, D.; and Cicognetti, G. "2,3,7,8-Tetrachlorodibenzo-p-dioxin Levels in Cow's Milk from the Contaminated Area of Seveso, Italy," Bulletin of Environmental Contamination and Toxicology 24:634-639, 1980.

Fox, Jeffrey L. "Dioxins' Health Effects Remain Puzzling," Science 221(4616):1161-1162, 1983.

Friedman, Morton R. and Weckdresser, John. "Dioxin and Resource Recovery," The Environmental Forum 2(5):44-48, 1983.

Giavini, E.; Prati, Mariangela; and Vismara, Claudio. "Effects of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Administered to Pregnant Rats during the Preimplantation Period," Environmental Research 29(1):185-189, 1982.

Hanify, Jennifer A.; Metcalf, Peter; Nobbs, Christopher, L.; and Worsley, Keith J. "Aerial Spraying of 2,4,5-T and Human Birth Malformations: An Epidemiological Investigation," Science 212 (April 17, 1983) pp. 349-351.

Hay, Alastair. "Disposing of Dioxins by Oxidation," Nature 290 (March 26, 1981) p. 294. (See CRF)

Helling, C.S.; Isensee, A.R.; Woolson, E.A.; Ensor, P.D.J.; Jones, G.E.; Plimmer, J.R., and Kearney, P.C. "Chlorodioxins in Pesticides, Soils, and Plants," Journal of Environmental Quality 2(2):171-177, 1973.

Henig, R.M. "TCDD - A Useful Tool in Basic Biology Laboratories," BioScience 30(2):129-130, 1980.

Huff, J.F.; Moore, J.A.; Saracci, R., and Tomatis, L. "Long-Term Hazards of Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans," Environmental Health Perspectives 36:221-N 1980.

Hutzinger, 0.; Olie, K.; Lustenhouwer, J.W.A.; Okey, A.B.; Bandii S., and Safe, S. "Polychlorinated Dibenzo-p-dioxins and Dibenzofurans: A Bioanalytical Approach," Chemosphere 10(1):19-25, 1981.

Isensee, A.R.; and Jones, G.E. "Distribution of 2,3,7,8-Tetrachloro-dibenzo-p-dioxin (TCDD) in Aquatic Model Ecosystem," Environmental Science and Technology 9(7):668-672, 1975.

Jensen, D.J., and Hummel, R.A. "Secretion of TCDD in Milk and Cream Following the Feeding of TCDD to Lactating Dairy Cows, Bulletin of Environmental Contamination and Toxicology 29:440-446, 1982.

Josephson, Julian. "Chlorinated Dioxins and Furans in the Environment," Environmental Science and Technology 17(3):121 128A, 1983.

Junk, G.A., and Richard, J.J. "Dioxins not Detected in Effluents from Coal/Refuse Combustion," Chemosphere 10(11/12):1237-124 1981.

Karasuk, F.W., and Onuska, F.I. "Trace Analysis of the Dioxins," :analytical Chemistry 54(2):309A-310A, 1982.

Kearny, Philip C.; Woolson, E.A.; Isensee, A.R.; and Helling, C,S "Tetrachlorodibenzodioxin in the Environment: Sources, Fate and Decontamination," Environmental Health Perspectives 5(5):273-277, 1973.

Kearny, Philip C.; Woolson, Edwin, A.; Ellington, Charles P., Jr, "Persistence and Metabolism of Chlorodioxins in Soils," Environmental Science and Technology 6(12):1017-1019, 1972.

Kilbane, J.J. et al. "Detoxification of 2,4,5-Trichlorophenoxyaa Acid from Contaminated Soil by Pseudomonas Cepacia," ARRlieE and Environmental Microbiology 45(5):1697-1700, 1983.

Kriebel, David. "The Dioxins: Toxic and Still Troublesome," Environment 23(1):6-13, 1981.

Lustenhouwer, J.W.A.; Olie, K.; and Hutziner, 0. "Chlorinated Dibenzo-p-dioxins and Related Compounds in Incinerator Effluents: A Review of Measurements and Mechanisms of Formation," Chemosphere 9:501-522, 1980.

Mahle, Nels H., and Whiting, Larry F. "The Formation of Chlorodibenzo-p-dioxins by Air Oxidation and Chlorination of Bituminous Coal," Chemosphere 9:693-699, 1980.

Miller, A.S. "2,4,5-T: Chemical Time Bomb: Vietnam Vets Exposure to Agent Orange," Environment 21:2-4, 1979.

Olie, K.; Berg, M.v.d.; and Hutzinger, 0. "Formation and Fate of PCDD and PCDF from Combustion Processes," Chemosphere 12(4/5):627-636, 1983.

Pazderova-Vejlupkova, Jana; Lukas, Edgar; Nemcova, Marcela; Pickova, Jane; and Jirasek, Lubor. "The Development and Prognosis of Chronic Intoxication by Tetrachlorodibenzo-p dioxin in Men," Archives of Environmental Health 36(1):5-11, 1981.

Pocchiari, F. "Human Health Effects from Accidental Release of TCDD at Seveso, Italy,"Annals of the New York Academy of Science 320: 311-320, 1979.

Poiger, H.; and Schlatter, Chi. "Influence of Solvents and Absorbents on Dermal and Intestinal Absorption of TCDD," Journal of Environmental Contamination and Toxicology 18:477-481, 1980.

Pol, 1, Alan P., and Knutson, Joyce C. "2,3,7,8-`letrachlorodivenzo-p-Dioxin and Related Halogenated Aromatic Hydrocarbons: Examination of the Mechanism of Toxicity," Annual Review of Pharmacology and Toxicology 22:517-54, 1982.

Poland, Alan P,; Smith, Donald; Metter, Gerald; and Possick, Paul. "A Health Survey of Workers in a 2,4-D and 2,4,5-T Plant," Archives of Environmental Health 22 (March):316-327, 1971.

Pontzer, Dave. "Dioxin: Unsafe at Every Level Tested," Exposure (Oct.-Nov. 1982) pp. 8-9.

Rawls, Rebecca L. "Dioxin's Human Toxicity is Most Difficult Problem," Chemical and Engineering News 61(23):37-48, 1983.

Ryan, J.J.; Pilon, J.C.: Conacher, H.B.S.; and Firestone, D. "Interlaboratory Study on Determination of 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Fish," Journal of the Association of Official Analytical Chemists 66(3):700, 1983.

Silkworth, Jay; McMartin, Donald; DeCaprio, Anthony; Rej, Robert; O'Keefe, Patrick; and Kaminsky, Lawrence. "Acute Toxicity in Guinea Pigs and Rabbits of Soot from a Polychlorinated Biphenyl-Containing Transformer Fire," Toxicology and Applied Pharmacology 65:425-439, 1982.

Smith, R.M. "2,3,7,8-Tetrachlorodibenzo-p-dioxin in Sediment Samples from Love Canal Storm Sewers and Creeks," Environmental Science and Technology 17(1):6-10, 1933.

Stevens, Kingsley M. "Agent Orange Toxicity: a Quantitative Perspective," Human Toxicology 1:31-39, 1981.

Sun, Majorie. "Dioxin's Uncertain Legacy," Science 219(4584): 468-469, 1983.

Sun, Marjorie. "Missouri's Costly Dioxin Lesson," Science 219(4583): 367-369, 1983.

"Symposium Updates Health Effects of Dioxins, Benzofurans," Chemical and Engineering News 61(37):26-30, 1983.

Tiernan, Thomas 0.; Taylor, Michael L.; Solch, Joseph G.; Va6 Garrett F., and Garrett, John H. "Characterization of Tor Components in the Effluents from a Refuse - Fired Inciner Resources and Conservation 9:343-354, 1982.

U.S. Environmental Protection Agency. "Hazardous Waste Manage System: Prohibition of Dispsoal of Tetrachlorodibenzo-p• Dioxin," (Proposed Rule and Request for Comments)Federal Register 48(65):14514-14529, 1983.

Van den Berg, M.; 01ie; -C.; and Hutzinger, 0. "Uptake and Selective Retention in Rats of Orally Administered Chlorv Dioxins and Dibenzofurans from Fly-Ash and Fly-Ash Extract Chemosphere 12(4/5):537-544, 1983.

Ward, Claudia T.; and Matsumura, F. "Fate of 2,3,7,8-Tetrachloi dibenzo-p-dioxin (TCDD) in a Model Aquatic Environment, Archives of Environmental Contamination and Toxicology 7:349-357, 1978.

Wilson, David C. "Lessons from Seveso," Chemistry in Britian, July 1982.

Woolson, Edwin A.; Thomas, Ronald F.; and Ensor, Peter D.J. "Survey of Polychlorinated-p-Dioxin Content in Selected Pesticides," Agricultural and Food Chemistry 20(2):351-351 1972.

"Worker's Suits Over Dioxin Pick up Speed," Chemical Week 133(14):86-88, 1983.

Zack, J.A. and Suskind, R.R. “the Mortaility Experience of Workers Exposed to Tetrachlorodibenzo-dioxin in Trichlorophenol Process Accident “Journal of Occupational Medicine 22:11, 1980

10. Additional Sources of Information

Refer to Newsclipping File and Vertical File under "Dioxins". See Chemical Reference File under CAS #1746-01-6 (TCDD), #39277-47-9 (Agent Orange).

N.J. Dept. of Environmental Protection
Toll-free numbers: Health effects - (800) 792-8831 Poison information - (800) 962-1253
Government Press Office
Office of Science & Research - Contact: (609) 984-6070

N.J. Dept. of Health, John Fitch Plaza, CN360, Trenton, NJ 08625
Contact: Dr. Kenneth Rosenman
(609) 984-1863

N.J. Agent Orange Commission, Dept. of Biochemistry & Microbiology, Lipman Hall, Cook College, Rutgers Univ., P.O. Box 231, New Brunswick, NJ 08903 Contact: Dr. Peter Kahn
(201) 932-9522

New York Dept. of Health, Dioxin Unit
Contact: Dr. 'Nancy Kim
(518) 473-7238

U.S. Center for Environmental Health, Centers for Disease Control, Atlanta, GA 30333 Contact: Dr. Renate Kimbrough
(404) 452-4111

U.S. Dept. of Health & Human Services, Agent Orange Working Group, Director of Veterans Affairs Contact: Dr. Peter E.M. Beach
(202) 245-2210

Agent Orange Projects Office, Washington, D.C.
Contact: Han K. Kang
(202) 389-5534

U.S. Environmental Protection Agency, Chlorinated Dioxins Working Group, Washington, D.C. 20460 Contact: Dr. Donald G. Barnes
Science Advisor to the
Asst. Admin. for Pesticides
& Toxic Substances
(202) 382-2897

University of Medicine & Dentistry of New Jersey, Rutgers, Medical School, Piscataway, NJ
Contact: Dr. Michael Gallo
(201) 461-4771

Veterans Administration. Office of Environmental Medicine (10A7A), 810 Vermont Avenue, N.W., Washington, D.C. 20420
Contact: Alvin Young
(202) 398-5534

II. APPENDIX A

EPA Classification Scheme for
Potential Dioxin Sites.[1]

The classification scheme developed by EPA was designed as a means of focusing attention to those organic chemicals and pesticides most likely to be associated with the formation of dioxins based upon molecular structure, process sequence, and commercial significance. The product lists are based on commercial production during the past ten (10) years, and the listing is limited to those produced in quantities in excess of 1,000 pounds per year and/or whose sales reach $1,000 per year.

Organic Chemicals

Class I Polyhalogenated phenols, primarily with a halogen ortho to the hydroxyl group, with a high probability of dioxin formation. Products with such compounds appearing as intermediates are also considered. Manufacture of these materials normally involves reaction conditions of elevated temperature plus either alkalinity or free halogen presence, either of which is conducive to formation of halogenated dioxins.

Class II Ortho-halophenols and ortho-halophenyl ethers where the substituted groups are a mixture of halogens and nonhalogens. Processing conditions are similar to those defined for Class I and produce mixed substituted dioxins. The distinction. between Classes I and II is arbitrary and does not indicate necessarily a difference in likelihood of dioxin formation.

Class III Other chemicals having the possibility, but less likelihood, of dioxin formation. These include 1) ortho substituted aromatic compounds requiring an unusual combination of reaction steps to produce dioxins, 2) aromatic compounds that might form dioxins because of their production under semicombustion conditions, and 3) products that might contain dioxins by way of contamination of their starting materials.

Pesticide Chemicals

Class I Highly likely to be associated with the presence of halogenated dibenzo-p-dioxins because of the presence of an ortho-halogenated phenol in the reaction sequence, with subjection to elevated temperature ($\geq$145°C) plus either alkalinity or the presence of free halogen.

Class II Reasonable but lesser probability of such dioxin association because of the presence of phenolic or aromatic structures related to dioxins; although not directly involving dioxin precursive conditions, such chemicals might form dioxins under irregular operating conditions.

[1] Classification descriptions taken from Dioxins, EPA-600/2-80-197. In order to avoid repetition producers are listed in one class only. Producers with compounds in multiple classes were listed in the lowest (most conservative) classification. For example, a producer classified by EPA as Class II for organic chemicals and Class I for pesticides is included in the accompanying tables as a Class I site.

APPENDIX B

I. FDA Report-Attachment to Letter from FDA Commissioner, Dr. Arthur Hull Hayes, Jr. to Governor Milliken of Michigan concerning advice on the Public Health Significance of TCDD contaminant "Levels of Concern" for Finfish in Great Lakes (dated 8/26/81) *exerpted*

A. INTRODUCTION

Chlorinated dibenzo-p-dioxins are formed during the production of certain compounds such as the herbicide 2,4 ,5-trichlorophenoxyacetic acid (2,4,5-T), the fungicide pentachlorphenol and the germicide hexachlorophenes. The manufacture and use of these compounds has resulted in the introduction of these toxic impurities into the environment. TCDD* is one of the chlorinated dibenzo-p-dioxins that may be present in the impurities and is possibly the most potent man-made toxic chemical presently known.

Residues of TCDD have been detected in a variety of species of fresh water fish in Lake Ontario and in Michigan 's Tittabawassee and Saginaw Rivers which empty into Saginaw Bay. As a result of the identification of TCDD residues in fish and the Canadian report of TCDD residues in Great Lakes herring gull eggs, Canadian and U.S. officials first met at the State Department of Washington on December 19, 1980 to discuss the problems and to develop a plan for responding to the problem. Three needs were identified in order to respond: (1) results of analyses of a variety of fish consumption in the Great Lakes area, and (3) information concerning the toxicology of TCDD. The response to these needs has been completed. With this information, Canadian officials, representing a variety of ministries, and U.S. officials, representing the Food and Drug and Administration and the Environmental Protection Agency, met in Ottawa, Canada on July 15, and 16, 1981 to evaluate jointly the problem of residues of TCDD in fish from various areas of the Great Lakes.

B. EVALUATION OF THE HEALTH SIGNIFICANCE OF DIOXIN CONTAMINATED FISH

Data developed from a nationwide study of fish consumption carried out by the U.S. National Marine Fisheries Service indicate that fish consumption of the selected species most likely to contain TCDD residues at the 99th percentile of consumption is 36.83 per day on an individual basis in the eight States which border the Great Lakes. Consumption of the selected species at the 90 percentile of consumption is 15.70 grams per day. Results of analysis of fish samples collected by Canada and the U.S. indicate that TCDD levels of up to 30 parts per trillion (ppt) and TCDD (mean value, 20-25 ppt) are present in the edible portion of salmonoid fish (salmon, trout) from Lake Ontario. Lower levels of TCDD were reported to be present in the edible portion of commercial species (bullhead, perch, catfish, sucker, etc.) from Lake Ontario, although up to 40 ppt of TCDD were found in eel and smelt from the lake. Less than 10 ppt of TCDD was seen in samples from Lake Erie, and limited data from fish from the other Great Lakes were similar to those obtained from Lake Erie. Higher levels of TCDD (up to 30 ppt) were present in fish from Saginaw Bay.

Toxicologically in the rat, the only species adequately tested, lifetime studies have shown that at dose levels of 100 ng TCDD/kg/bw/day, there is an increase in liver carcinomas. At 10 ng TCDD/kg/bw/day, hyperplasia of the lungs and liver was observed. No toxic affects were reported at 1 ng TCDD/kg/bw/day.

Deleterious effects on reproduction and general health have been reported in female Rhesus monkeys receiving 50 ppt of TCDD in their diet (equivalent to 2.5 ng/kg/bw/day). In man, toxicity due to TCDD has been reported after occupational exposure during industrial syntheses of PCP and 2 ,4 ,5-T or following industrial accidents in plants producing these substances, e.g., the July, 1976 incident in Seveso, Italy. In all cases, chloracne was the most common effect reported. In the large and intensive follow-up program in the Seveso area of Italy, there has been no indication to date of any reproductive effects due to TCDD exposure.

The Bureau of Foods assessment of the available data, including fish consumption, toxicity studies, TCDD residues in fish, and analytical variability which may approach 100 percent, leads to the conclusion that there does not appear to be cause for concern for fish distributed in interstate commerce. This conclusion is based on the fact that the species of fish caught for sale in interstate commerce from waters having a TCDD problem generally would contain less than 25 ppt TCDD. At the same time, it is recognized that there may be reason for concern related to patterns of fish consumption in localized areas in some of the Great Lakes States (primarily Lake Ontario and the Saginaw Bay area), in particular, the sport fishermen or residents who may consume unusual quantities of locally caught fish.

C. RECOMMENDATIONS

Based on evaluation of the available toxicity data and patterns of fish consumption, as well as the analytical variability, FDA's recommended advice - to the Great Lakes States having a problem concerning consumption of TCDD contaminated fish by sport fishermen and consumers is as follows: if the TCDD levels found in fish average less than 25 ppt, FDA believes that there is little cause for concern. On the other hand, if the average values exceed 50 ppt, the State should seriously consider more stringent methods to limit the taking of fish from these areas. For those values between 25 and 50 ppt, sport fishermen who generally consume fish only a few times a year, should restrict their intake to no more than one meal a week. Permanent residents of these areas who might consume the fish over the entire year, should restrict their intake to no more than 1-2 times a month.

II. Letter from FDA's Director, Division of Regulatory Guidance, John Taylor to DEP Research Scientist, Thomas Belton outlining an Advisory Opinion on New Jersey's Public Health Announcements concerning TCDD Contamination of Finfish and Crabs (dated 12/5/83) *exerpted*

December 5, 1983

Mr. Thomas Belton
Research Scientist
Office of Science and Research
Department of Environmental Protection
CN 402
Trenton, New Jersey 08625

Dear Mr. Belton:

This is in reply to your letter of November 3, 1983, in which you request an FDA review of your data and health advisories concerning dioxin (2,3,7,8-TCDD) contamination of fish and crabs.

The FDA advisory opinion to the State of Michigan, which we furnished you, continues to represent our best judgement regarding the consumption of fish contaminated with TCDD. This advisory applies only to fin fish. We have not- yet addressed the contamination of shellfish with TCDD. The policies described in your health advisories regarding TCDD in fin fish from the Passaic River appear to be consistent with our Great Lakes advisory. However, we have concerns about the wording used in paragraph four of Administrative Order No. EO-40-17.

The order states that "fish and shellfish from the Passaic River are contaminated with dioxin in excess of the safe level established by FDA". We have not established a "safe level" for TCDD in fish. The levels listed in our advisory opinion represent only levels of health concern. For this reason, we believe the order should be modified to state that fish (edible portion) from the Passaic River are contaminated with dioxin in excess of 50ppt, a level at which FDA recommends that fish products should not be consumed.

The health advisory fact sheet refers to 50ppt as an FDA guideline. This term is often used interchangeably with the term action level and thus the use of the term guideline in reference to the 50ppt dioxin level could be misleading. We believe that the wording we suggested for the administrative order would more accurately describe FDA's position.

Your health advisories concerning shellfish are based on the limited data you obtained for TCDD in blue claw crab. This data shows that the edible muscle of the crab contains low levels of dioxin (16, 27ppt) whereas the hepatopancreas contains higher levels (450, 485ppt), as we would expect. In evaluating this data, you have assumed that there is a potential exposure

to TCDD from the hepatopancreas d~e to migration during the cooking process. We know of no studies that have established that such migration might occur. However, we believe there is a more meaningful way of estimating the potential exposure to TCDD from the hepatopancreas of crustaceans without having to consider the possibility of migration.

The findings of TCDD in the edible muscle would provide a good estimate of the potential exposure for individuals who consume only the back fin and lump meat. However, there are also individuals who consume the hepatopancreas along with the meat. To obtain a more realistic estimate of the potential TCDD exposure in this situation, it would be more appropriate to use a sample composite that includes the hepatopancreas and the edible muscle rather than the hepatopancreas alone.

The consumption of the hepatopancreas could result in a rather significant potential exposure to TCDD, but we are unable to reach definitive conclusions from the limited data you submitted. We believe there is a need to develop more data on the TCDD levels in the edible muscle alone and in composites of hepatopancreas with meat. When this information becomes available, we would be in a better position to comment on your advisories regarding shellfish.

For your information, we are enclosing the article "Use of Epidemiology in the Regulation of Dioxin in the Food Supply" by Dr. Frank Cordle. This paper describes some of the data and information that were considered in developing the FDA public health advisory.

If we can be of further assistance, let us know.

Sincerely yours,

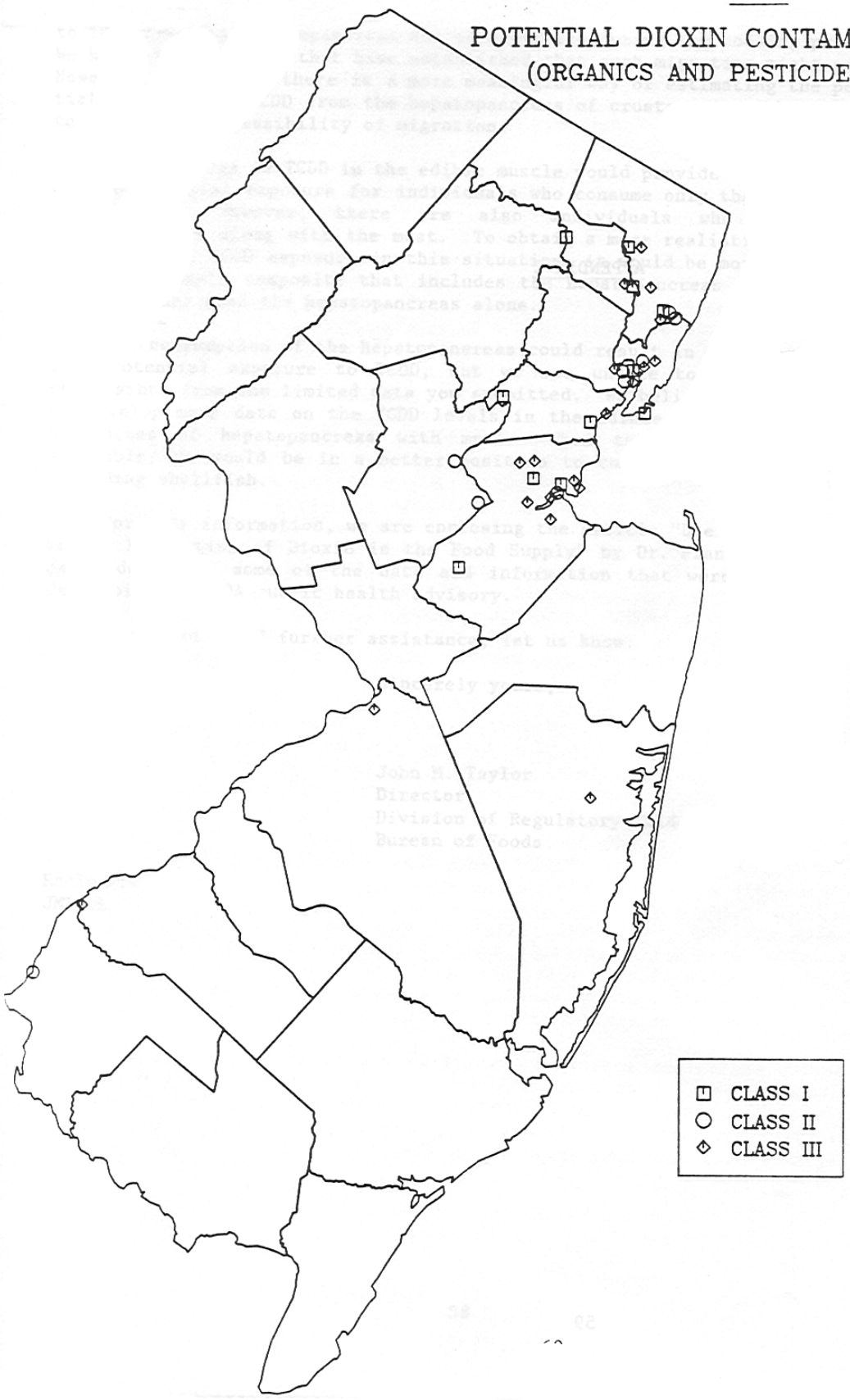
John M. Taylor
Director Division of Regulatory Guidance
Bureau of Foods

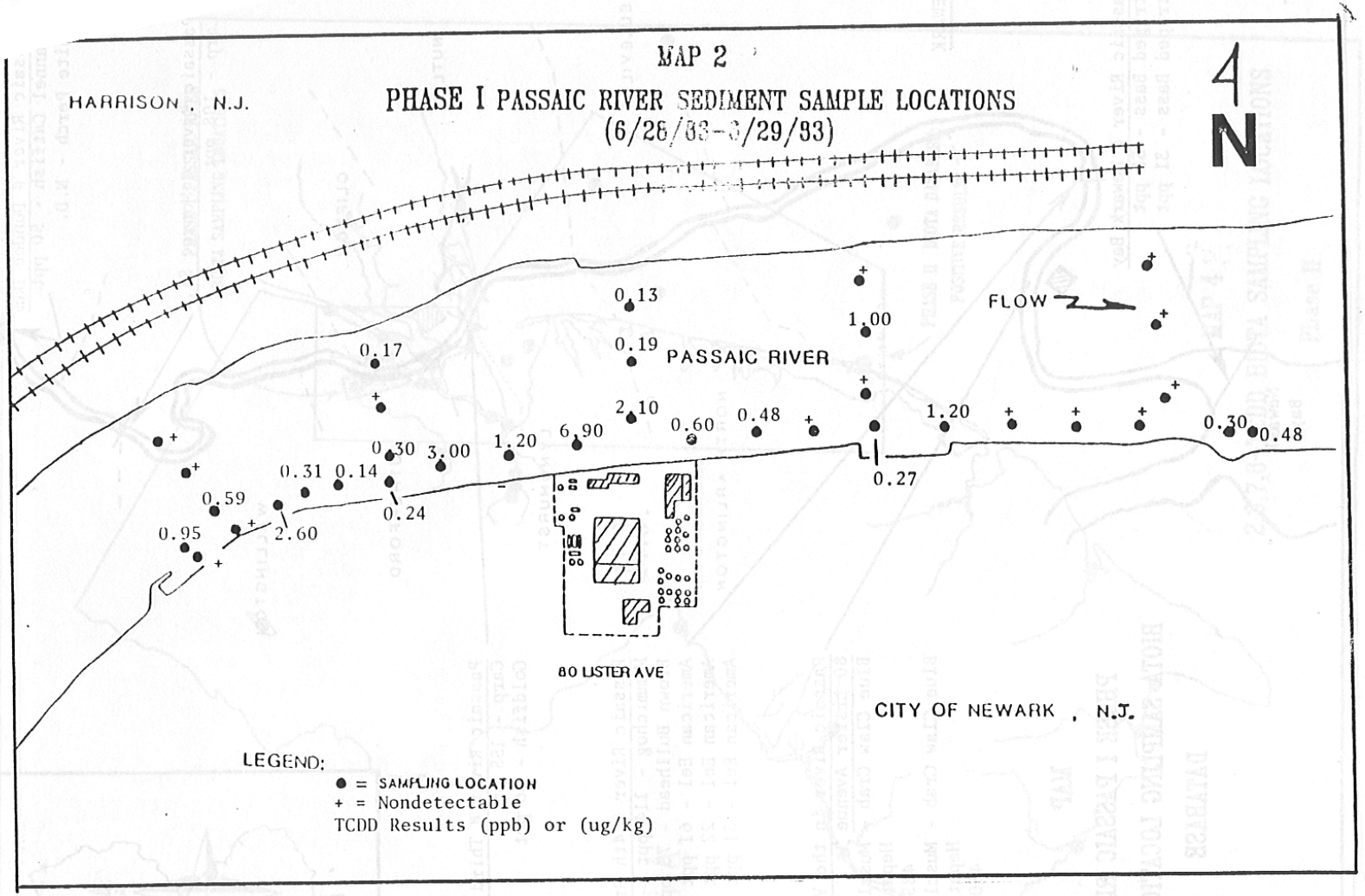
Enclosure
JMT/rk

APPENDIX C

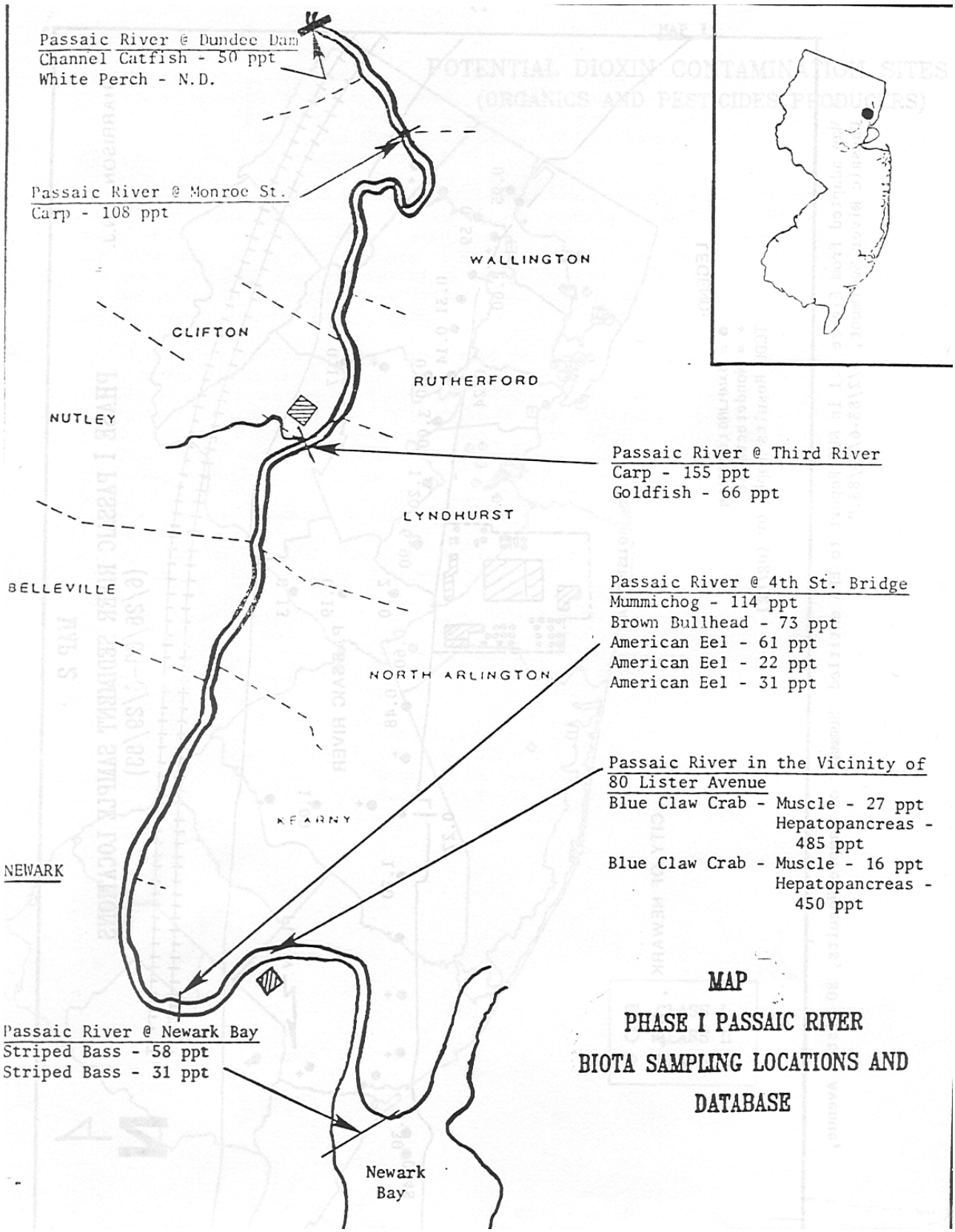
MAP I

POTENTIAL DIOXIN CONTAMINATION SITE
(ORGANICS AND PESTICIDES PRODUCERS)





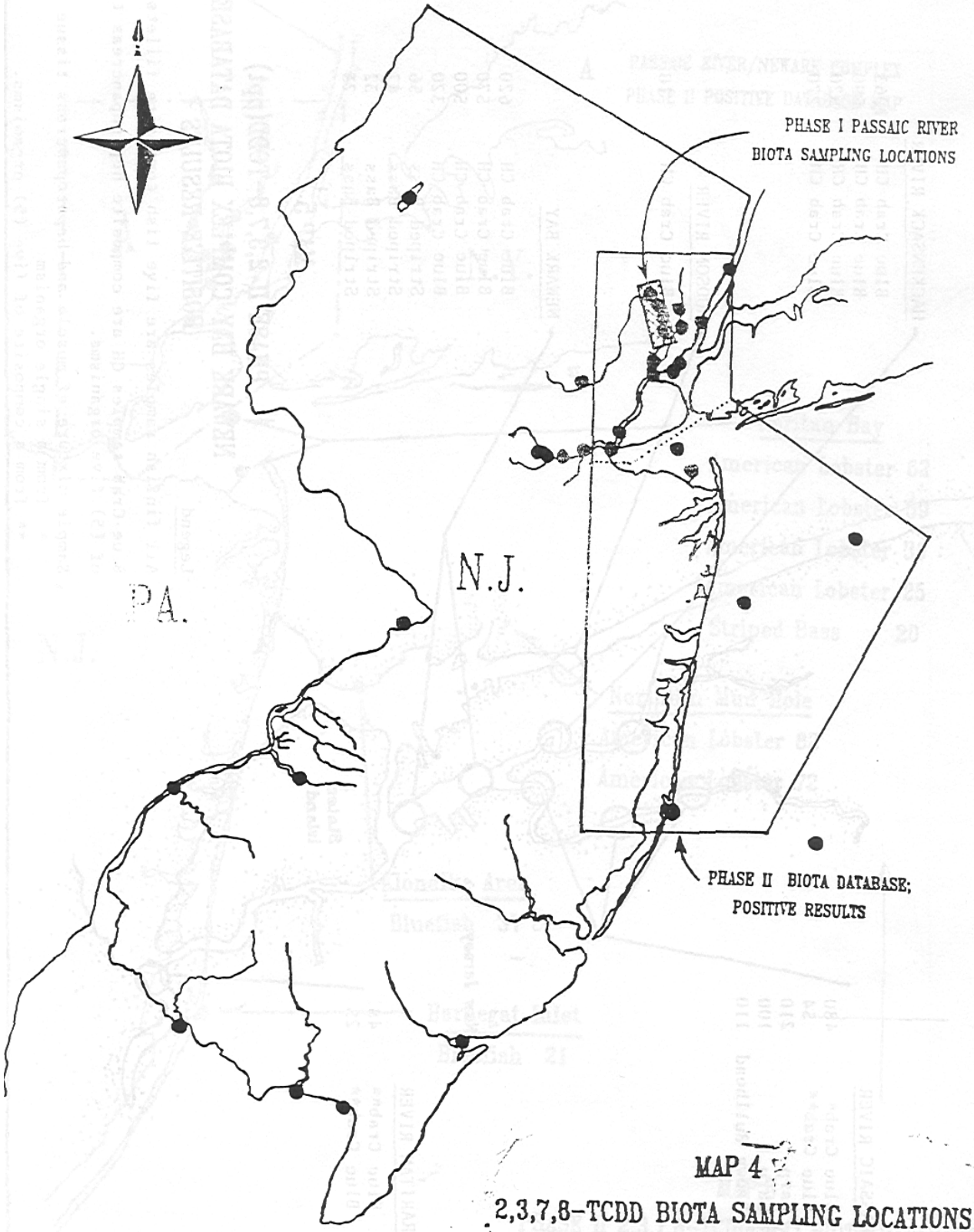
* Map adapted from Figure 9.1 in NUS Report to EPA entitled "Summary of Sample Results, 80 Lister Avenue, Passaic River Sediment, 6/2/83-6/29/83."



MAP

PHASE I PASSAIC RIVER

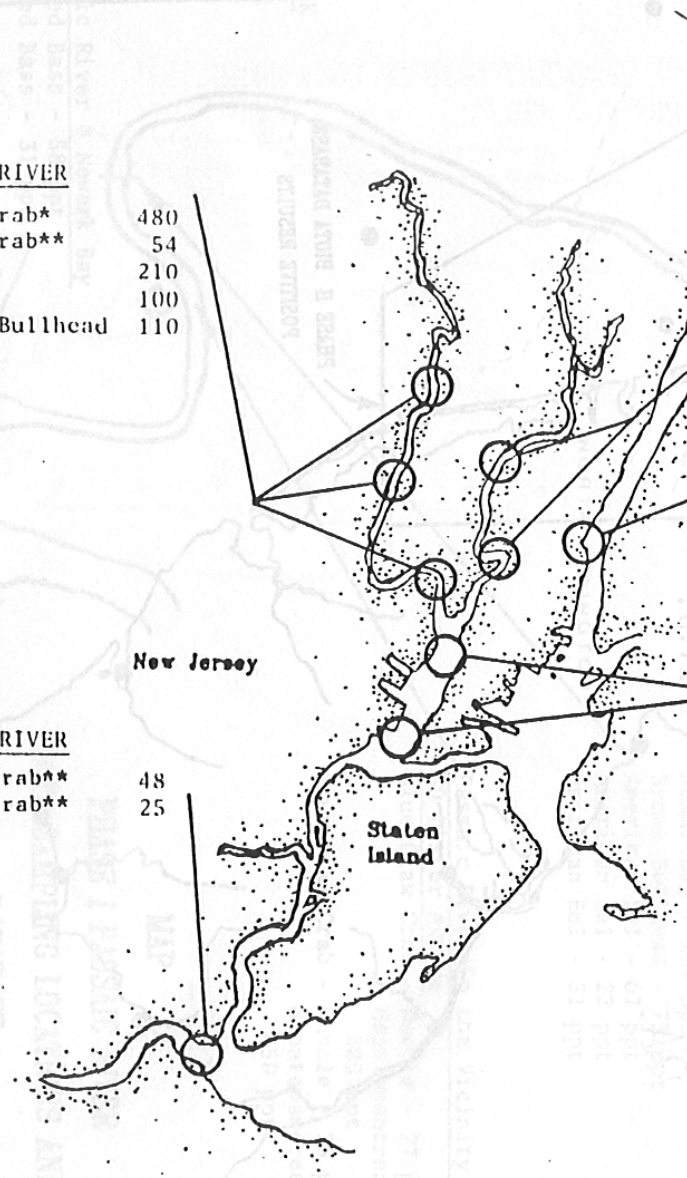
BIOTA SAMPLING LOCATIONS AND DATABASE



MAP 4

2,3,7,8-TCDD BIOTA SAMPLING LOCATIONS

Phase II



PASSAIC RIVER

Blue Crab*	480
Blue Crab**	54
Carp	210
Carp	100
Brown Bullhead	110

RARITAN RIVER

Blue Crab**	48
Blue Crab**	25

HACKENSACK RIVER

Blue Crab CH	1063
Blue Crab CH	590
Blue Crab CH	520
Blue Crab CH	270

HUDSON RIVER

Blue Crab CH	10

NEWARK BAY

Blue Crab CH	620
Blue Crab CH	570
Blue Crab CH	500
Blue Crab CH	320
Striped Bass	56
Striped Bass	47
Striped Bass	32
Striped Bass	23

MAP 5

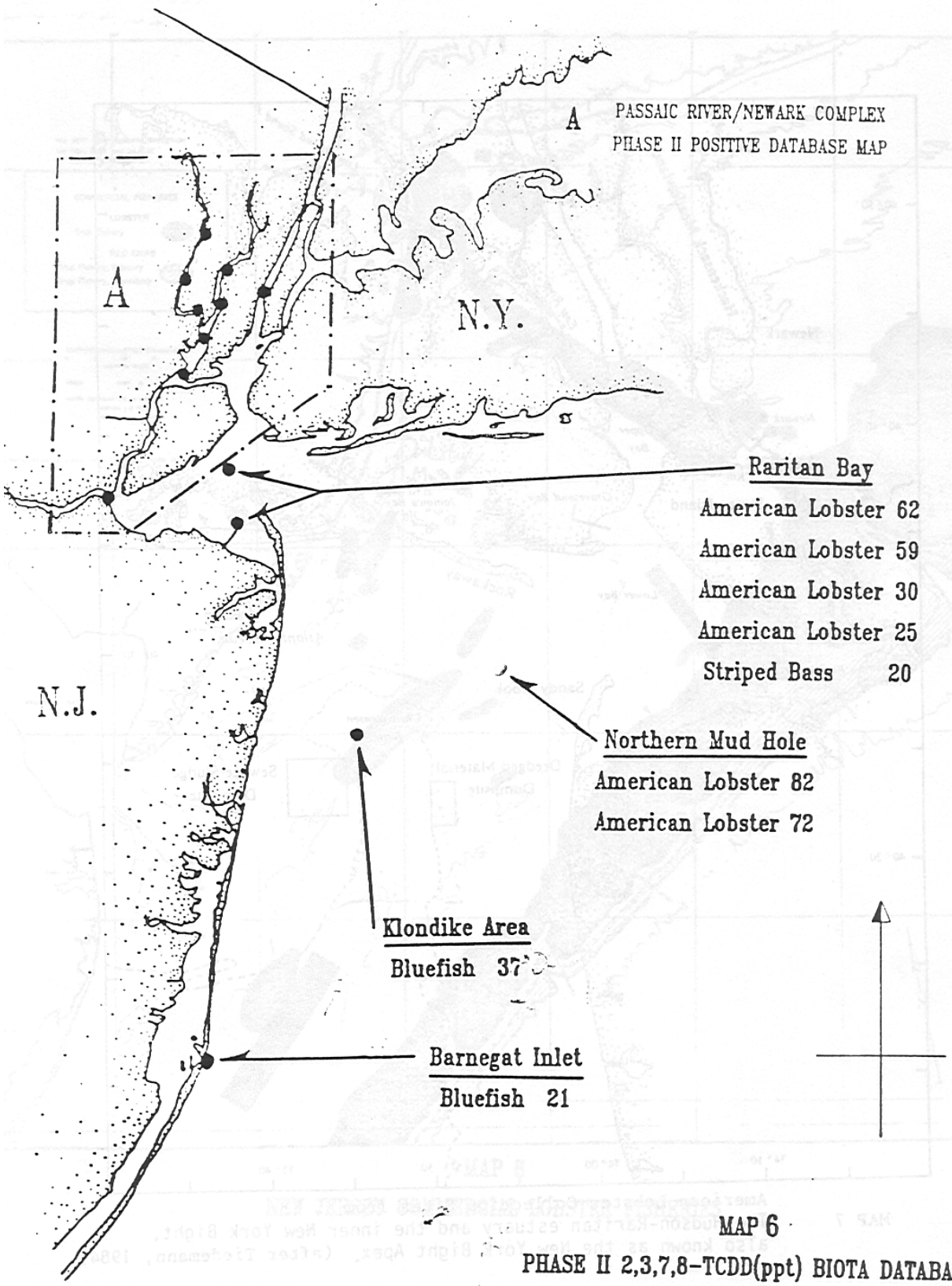
PHASE II 2,3,7,8-TCDD(ppt)
NEWARK BAY COMPLEX BIOTA DATABASE; POSITIVE RESULTS

Legend

All finfish samples are five fish composite fillets

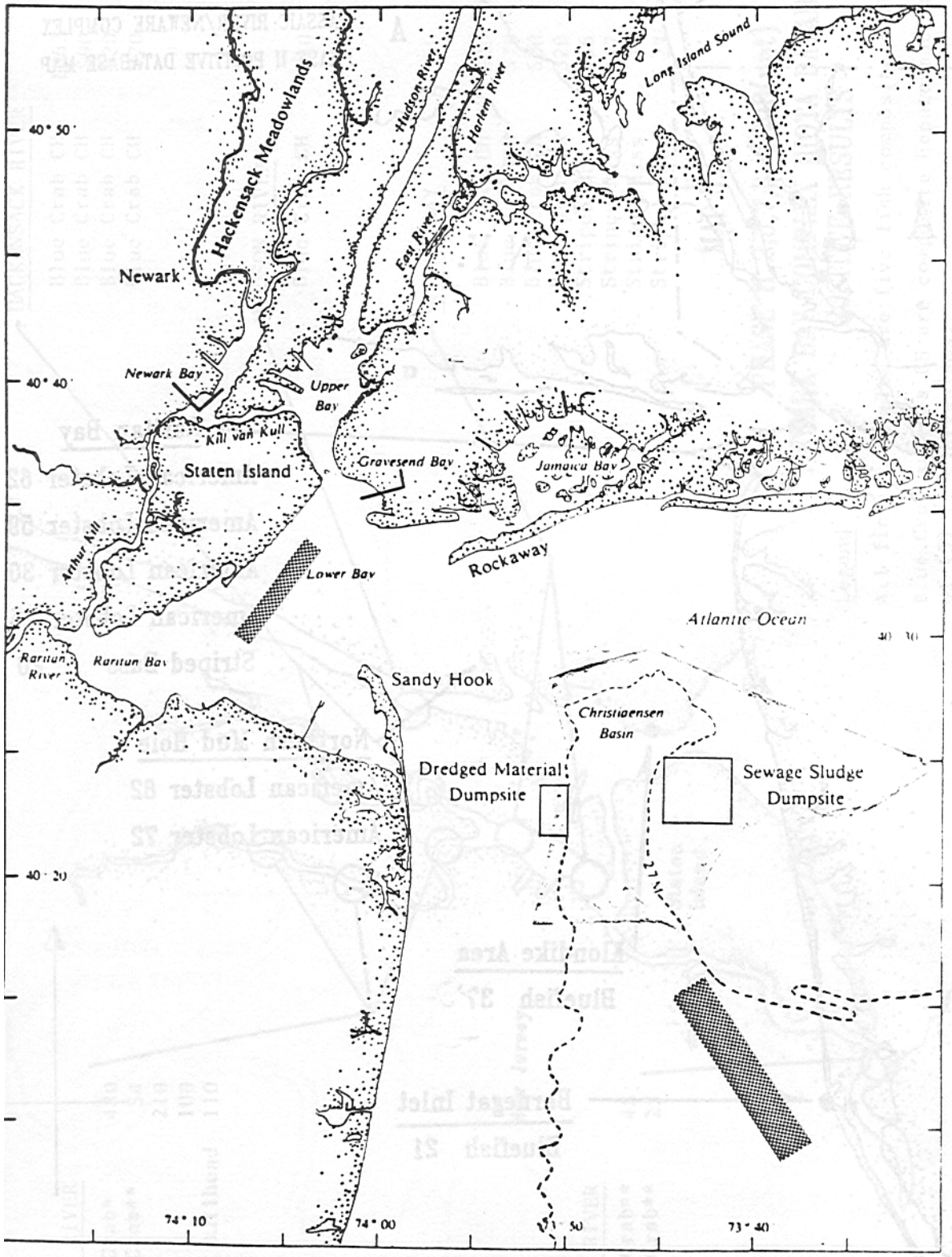
Blue Crab samples CH are composite hepatopancreas tissue of (5) five organisms.

Sample mixture of muscle and hepatopancreas tissue
- * from a single organism
- ** from a composite of five (5) organisms.

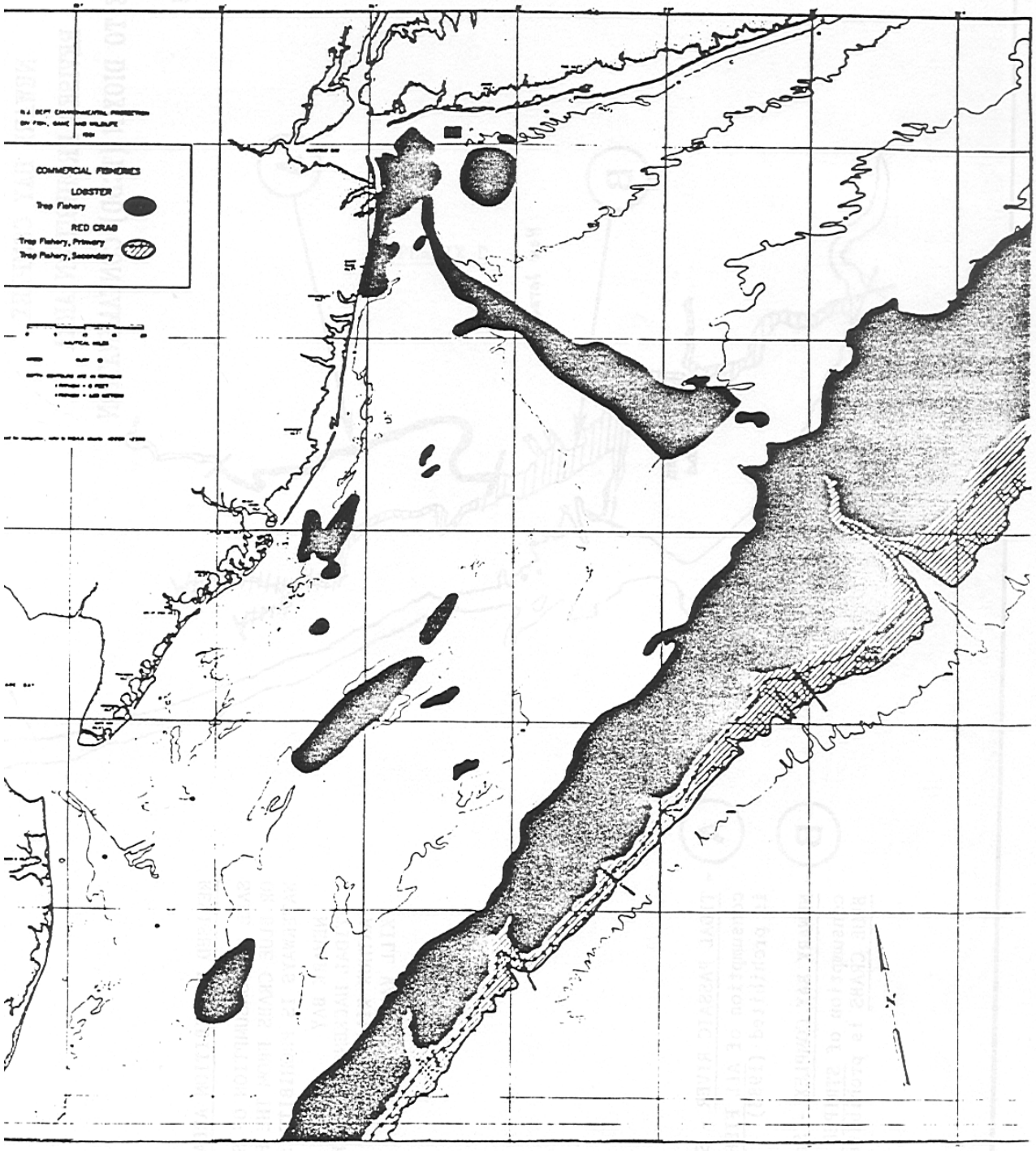


MAP 6

PHASE II 2,3,7,8-TCDD(ppt) BIOTA DATABASE;
NORTHERN COASTAL WATERS; POSITIVE RESULTS



MAP 7 American Lobster Collection Sites from The Hudson-Raritan estuary and the inner New York Bight, also known as the New York Bight Apex. (after Tiedemann, 1984)



MAP 8

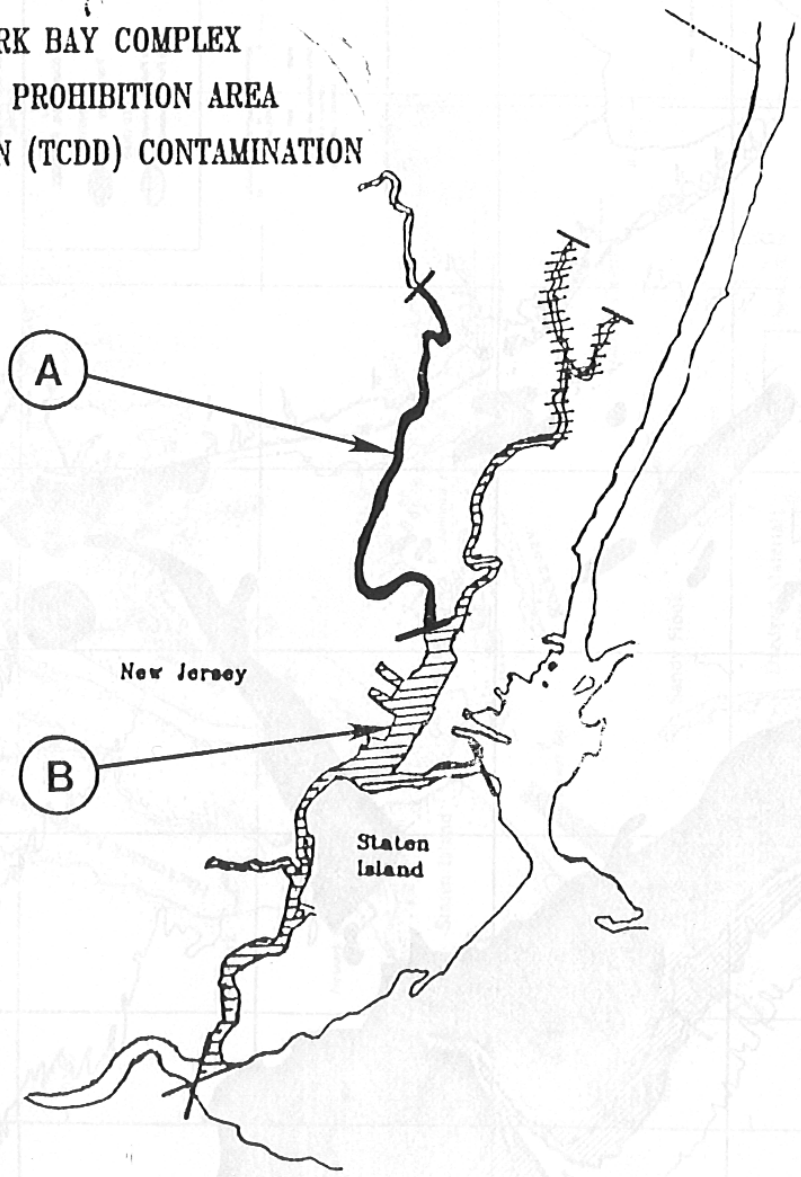
NEW JERSEY COMMERCIAL LOBSTER FISHERIES

MAP 9

NEWARK BAY COMPLEX

REVISED PROHIBITION AREA

DUE TO DIOXIN (TCDD) CONTAMINATION



REVISED PROHIBITION AREA

SALE OR CONSUMPTION OF STRIPED BASS OR BLUE CRABS FROM THE FOLLOWING WATERWAYS IS PROHIBITED:

NEWARK BAY
TIDAL HACKENSACK RIVER
ARTHUR KILL
KILL VAN KULL

A - TIDAL PASSAIC RIVER - Sale or consumption of ALL FISH AND CRABS is prohibited (1983)

B - NEWARK BAY COMPLEX - Sale or consumption of STRIPED BASS AND BLUE CRABS is prohibited.

APPENDIX D

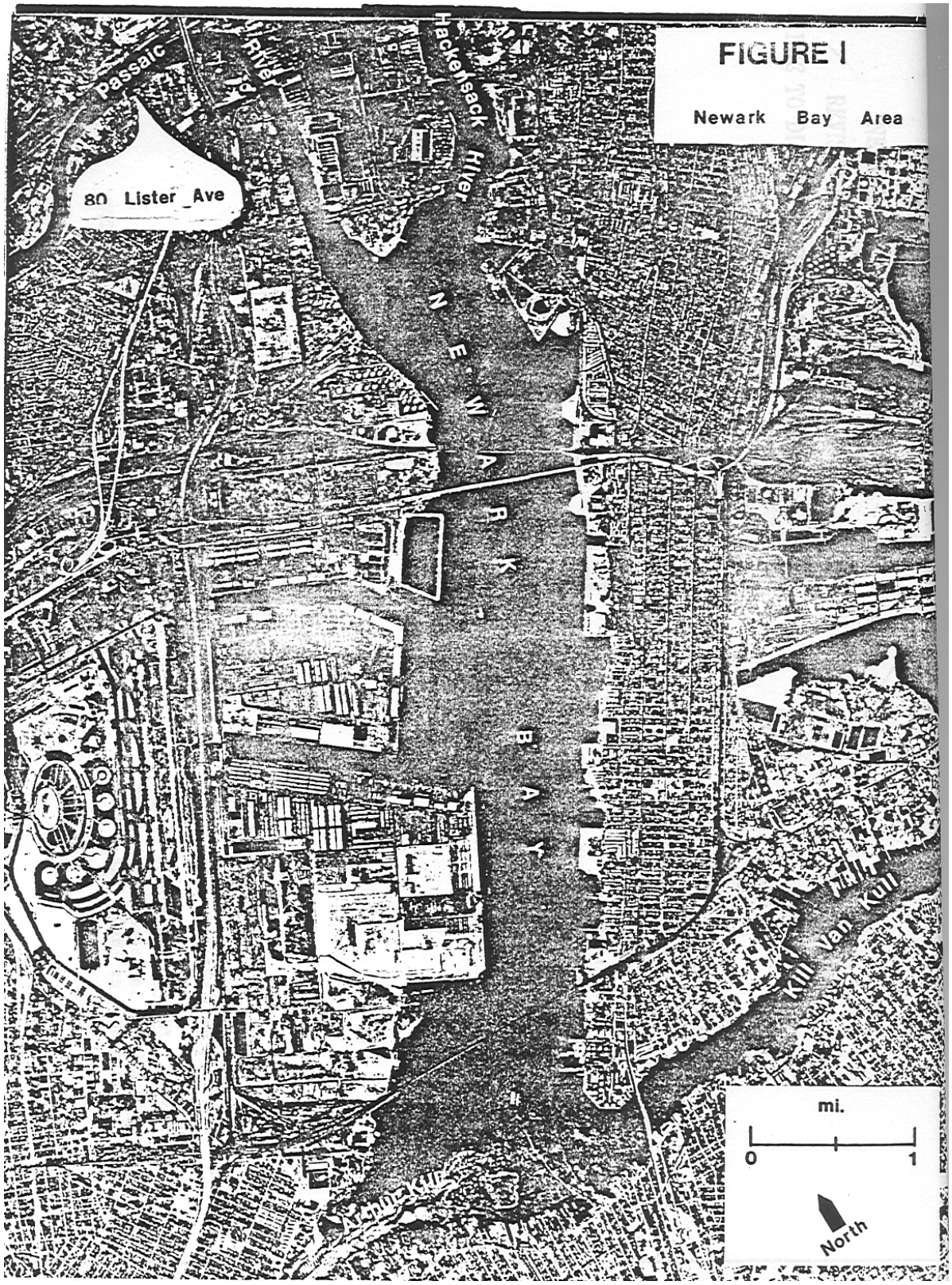


FIGURE I

Newark Bay Area

FIGURE 2

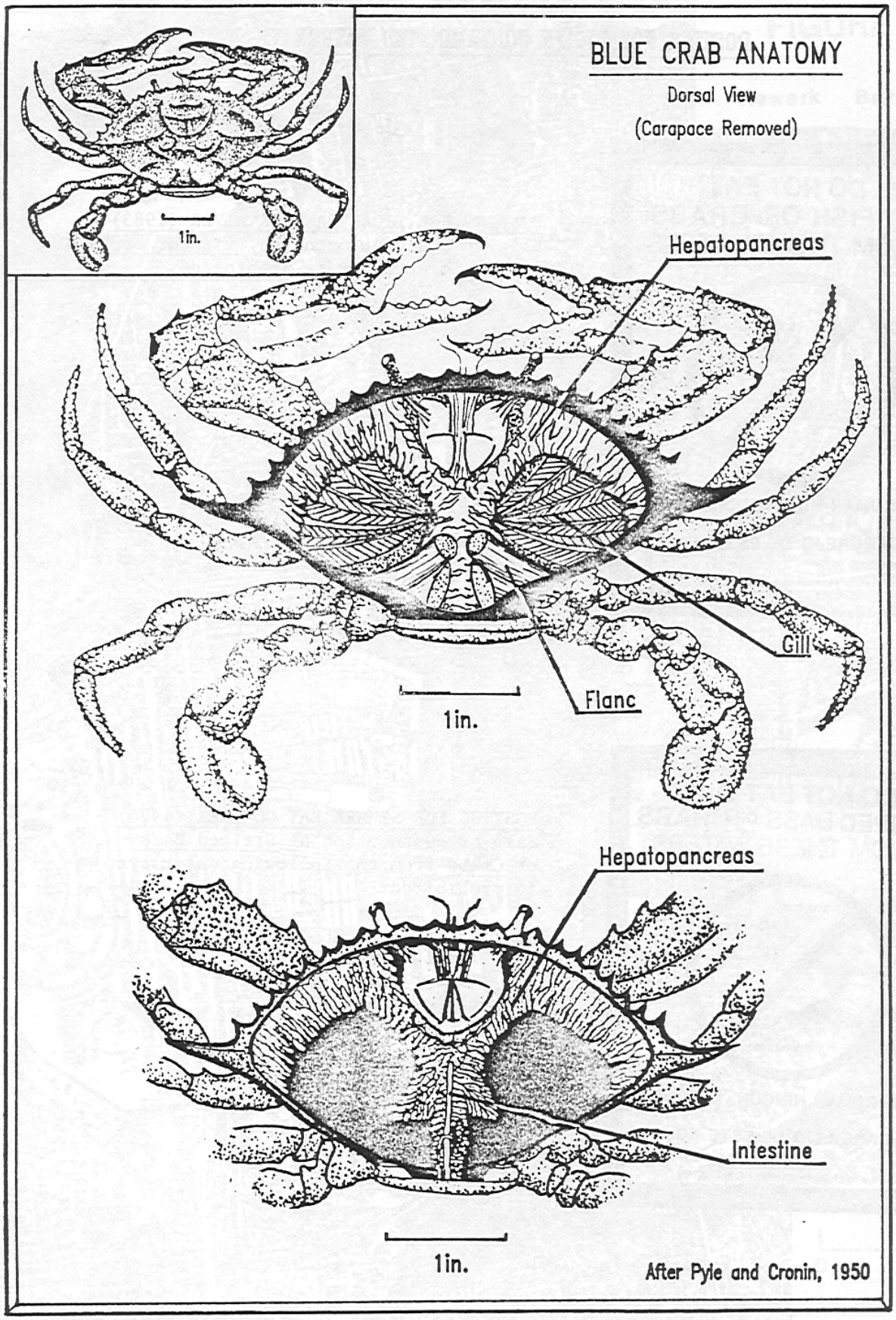
POSTING FOR DIOXIN CONTAMINATED WATERWAYS

POSTING FOR TIDAL PASSAIC RIVER (1983) - Sale or consumption of any fish or crabs from this waterway is prohibited.

POSTING FOR NEWARK BAY COMPLEX (1984) - Sale or consumption of Striped Bass or Crabs from the following waterways is prohibited:

Newark Bay
Tidal Hackensack River
Arthur Kill
Kill Van Kull

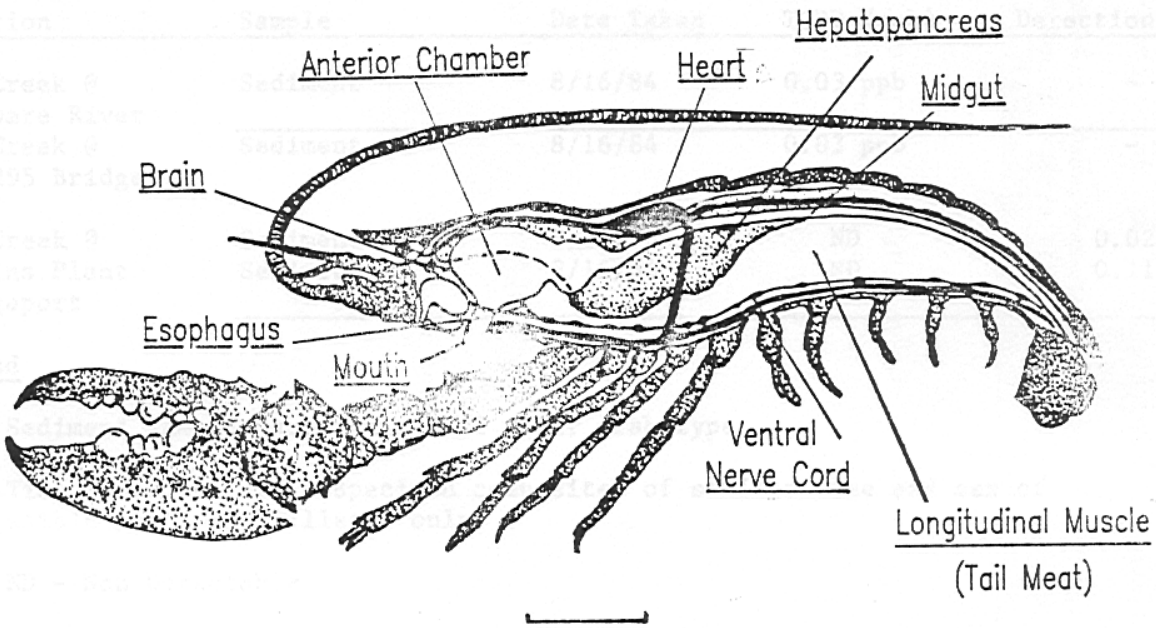
Figure 3


BLUE CRAB ANATOMY
Dorsal View
(Carapace Removed)
1in.
Hepatopancreas
Gill
Flanc
1in.
Hepatopancreas
Intestine
1in.
After Pyle and Cronin, 1950

AMERICAN LOBSTER

Internal Anatomy

(Sagital view, showing major organs)



After Bliss, 1982

APPENDIX E

Table I
2,3,7,8-TCDD Concentrations in Biota and Sediments
from Raccoon Creek

Biota Results

Location	Species	Date Taken	TCDD (ppt)	Detection Limit
Ra. Creek @ Confluence With Delaware River	Striped Bass	8/16/84	ND	26
	Striped Bass	8/16/84	ND	28
	Striped Bass	8/16/84	ND	21
	White Perch	6/9/82	42	-

Biota results are in ppt (parts per trillion)

Sediment Results

Location	Sample	Date Taken	TCDD (ppb)	Detection Limit
Ra. Creek @ Delaware River	Sediment	8/16/84	0.03 ppb	-
Ra. Creek @ Rt. 295 Bridge	Sediment	8/16/84	0.03 ppb	-
Ra. Creek @ Rollins Plant Bridgeport	Sediment	8/16/84	ND	0.02 ppb
	Sediment	8/16/84	ND	0.11 ppb

Legend

1. Sediment samples are individual ponar grab type

2. Tissue Samples are 5 specimen composites of similar size and sex of edible portions (Fillets) only

3. ND - Non Detectable

4. Sediment results are in parts per billion (ppb)

5. Tissue results are in parts per trillion (ppt)

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
I. PASSAIC RIVER							
Passaic River at the Turnpike Bridge, Near Diamond Alkali Site	8/24/83	Blue Crab*	M	189.0	17.0	480	-
Passaic River below South 4th St. Bridge, Harrison	6/1/82	American Eel		151.4	42.5	ND	40.0
Passaic River below South 4th St. Bridge, Harrison	6/1/82	American Eel		98.6	38.4	ND	62.0
Passaic River, Avondale Swing Bridge, Lyndhurst	8/23/83	White Perch		107.5	19.8	ND	76.0
Passaic River, Avondale Swing Bridge, Lyndhurst	8/23/83	American Eel		60.0	30.5	ND	113.0

Legend

All finfish samples are five fish composites unless otherwise noted.
+ Sample of (1) single organism.
ND = Non-detectable.
PPT = Parts per Trillion

Blue Crab and American Lobster samples are divided into two body tissues as indicated below:
Tissue samples designated CM are composite muscle tissue of (5) five organisms.
Tissue samples designated CH are composite hepatopancreas tissue of (5) five organisms for crabs and (15) fifteen for lobsters.
* Sample mixture of muscle and hepatopancreas tissue from a single organism.
** Sample mixture of muscle and hepatopancreas tissue from a composite of five (5) organisms.

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
I. PASSAIC RIVER (Continued)							
Passaic River, Avondale Swing Bridge, Lyndhurst	8/23/83	Blue Crab**	M	207.2	16.9	54	-
Passaic River, Avondale Swing Bridge	8/23/83	Carp		996.6	42.3	100	-
Passaic River, Rutherford Carlton Hills Area	8/24/83	Brown Bullhead		184.0	22.2	110	-
Passaic River, Rutherford Carlton Hills Area	8/24/83	American Eel+		312.0	57.1	ND	160.0
Passaic River, Rutherford Carlton Hills Area	8/24/83	Carp		1778.7	49.2	210	-
Passaic River @ Dundee Lake, Above Dam	5/18/84	Carp		1120.0	42.2	ND	4.0
Passaic River @ Dundee Lake, Above Dam	5/18/84	Carp				ND	4.0
Passaic River @ Dundee Lake, Above Dam	5/18/84	Carp+		4000.0	63.5	ND	29.0
Passaic River @ Elmwood Park	5/18/84	American Eel+		650.0	64.1	ND	5.0
Passaic River @ Elmwood Park	5/18/84	Carp		1167.2	43.4	ND	6.0

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
I. PASSAIC RIVER (Continued)							
Passaic River @ Elmwood Park	5/18/84	Carp		1687.2	48.1	ND	4.0
Passaic River at Berkeley Heights	10/11/83	Largemouth Bass+		406.0	30.0	ND	6.0
Passaic River at Berkeley Heights	10/11/83	American Eel+		829.0	74.4	ND	13.0
Passaic River at Berkeley Heights	10/11/83	Carp		1704.2	254.4	ND	30.0
Passaic River at Berkeley Heights	10/11/83	Carp		2200.4	54.3	ND	17.0
II. HACKENSACK RIVER							
Hackensack River at Laurel Hill	8/20/82	Blue Crab CM	F/M	156.2	16.3	ND 1063	19.0 -
Hackensack River at Laurel Hill	8/20/82	Blue Crab CM CH	F	136.9	15.6	ND 590	11.0 -
Hackensack River at Sawmill Creek	8/18/82	Blue Crab CM CH	M	143.0	13.6	ND 270	13.0 -
Hackensack River at Sawmill Creek	8/18/82	Blue Crab CM CH	F	105.0	14.6	ND 520	35.0 -

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
III. NEWARK BAY							
Newark Bay at Shooter's Island	5/26/82	American Eel		189.8	46.6	ND	8.6
Newark Bay at Shooter's Island	5/26/82	American Eel		160.0	43.9	ND	8.0
Newark Bay at Central Railroad at NJ Trestle	8/17/82	Blue Crab CM CH	M	174.4	14.8	ND 570	9.6 -
Newark Bay at Central Railroad at NJ Trestle	8/17/82	Blue Crab CM CH	M	169.4	14.2	ND 620	11.0 -
Newark Bay at Central Railroad at NJ Trestle	8/17/82	Blue Crab CM CH	M/F	141.4	14.4	ND 320	18.0 -
Newark Bay at Central Railroad at NJ Trestle	8/17/82	Blue Crab CM CH	M/F	197.4	15.5	ND 500	11.0 -
Newark Bay at Central Railroad at NJ Trestle	8/27/82	Blue Crab* (Soft Shell)	F	69.8	11.9	ND	36.0
Newark Bay at the Bayonne Bridge	6/14/83	Striped Bass	M	591.4	37.8	ND	39.0
Newark Bay at the Bayonne Bridge	6/14/83	Striped Bass	F	500.8	35.6	ND	51.0
Newark Bay @ the Passaic River Confluence	6/14/83	Striped Bass		321.8	31.1	23	-

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
III. NEWARK BAY (Continued)							
Newark Bay @ the Passaic River Confluence	6/14/83	Striped Bass		344.2	32.6	56	-
Newark Bay @ the Passaic River Confluence	6/14/83	Striped Bass		372.4	32.9	32	-
Newark Bay @ the Passaic River Confluence	6/14/83	Striped Bass		449.4	34.6	47	-
IV. ARTHUR KILL							
Arthur Kill at Boynton Beach, Sewaren	6/1/82	American Eel		233.6	49.5	ND	12.0
Arthur Kill at Boynton Beach, Sewaren	6/1/82	American Eel		159.0	44.7	ND	8.9
V. HUDSON RIVER							
Hudson River, Bayonne to George Washington Bridge	7/20/83	Blue Crab CM CH	M	249.4	15.9	ND 10	24.0 -
Hudson River, Bayonne to George Washington Bridge	7/20/83	Blue Crab CM	M	244.4	16.5	ND ND	14.0 42.0
Hudson River, below George Washington Bridge	8/2/83	Blue Crab CM CH	M	226.0	15.6	ND ND	12.0 58.0
Hudson River @ Weehawken	7/19/83	Striped Bass	F	615.8	39.9	ND	12.0

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
V. HUDSON RIVER (Continued)							
Upper New York Bay at Caven Point	10/19/83	Striped Bass		423.4	34.4	ND	6.0
Upper New York Bay at Robbins Reef	5/26/83	Bluefish		1996.0	62.0	ND	35.0
Upper New York Bay at Caven Point	8/17/82	American Eel		236.4	48.1	ND	12.0
Upper New York Bay at Caven Point	8/17/82	American Eel		314.0	54.0	ND	11.0
Upper New York Bay at Caven Point	8/17/82	American Eel+		598.0	61.5	ND	12.0
Upper New York Bay at Robbins Reef	8/27/82	Striped Bass		672.2	39.7	ND	17.0
Upper New York Bay at Robbins Reef	8/27/82	Striped Bass		571.8	37.3	ND	12.0
VI. RARITAN RIVER AND BAY							
Raritan Bay @ Victory Bridge	8/25/82	Bluefish		461.8	36.5	ND	5.0
Raritan River @ Victory Bridge	9/16/83	Blue Crab**		314.4	19.0	48	-

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
VI. RARITAN RIVER AND BAY (Continued)							
Raritan River @ Victory Bridge	9/16/83	Blue Crab**	M	284.0	17.8	25	-
Raritan River @ Kin Buc Landfill	9/16/83	Striped Bass		613.0	40.0	ND	7.6
Raritan River @ Kin Buc Landfill	9/16/83	White Perch		217.0	23.8	ND	21.0
Raritan River @ Rt. 1 Bridge	5/12/82	Carp		1816.0	49.7	ND	14.0
Raritan River @ Donaldson Park	9/20/82	Channel Catfish				ND	8.0
Raritan River @ Fieldville Dam	5/12/83	Carp		2640.0	54.4	ND	17.0
Raritan River @ Confluence with Raritan Bay	7/24/84	Weakfish+		-	-	ND	25
Raritan Bay @ Leonardo Earle Pier	8/23/83	Striped Bass+		9170	-	ND	14
Raritan Bay @ Leonardo Earle Pier	8/23/83	Striped Bass+		2758	62.5	20	-
Raritan Bay @ Leonardo Earle Pier	8/23/83	Striped Bass+		1690	53.5	ND	22

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
VI. RARITAN RIVER AND BAY (Continued)							
Raritan Bay @ Leonardo Earle Pier	8/23/83	Striped Bass+		1495	50.5	ND	12
Raritan Bay @ Leonardo Earle Pier	8/23/83	Striped Bass+		2550	61.0	ND	49
Raritan Bay @ Leonardo Earle Pier	7/24/84	Bluefish+		-	-	ND	8
Raritan Bay @ Leonardo Earle Pier	7/24/84	Bluefish+		-	-	ND	34.
Raritan Bay @ Leonardo Earle Pier	7/24/84	Bluefish		-	-	ND	34
Raritan Bay West Reach Channel	10/27/83	American Lobster**		106.4	5.3	ND	20
Raritan Bay West Reach Channel	10/27/83	American Lobster**		45.6	4.1	ND	110
Raritan Bay Chapel Hill Channel	7/20/84	American Lobster*	F	307.0	7.6	ND	41
Raritan Bay Chapel Hill Channel	7/20/84	American Lobster*	M	219.0	7.0	59	-
Raritan Bay Chapel Hill Channel	7/20/84	American Lobster*	F	406.5	7.5	62	-

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
VI. RARITAN RIVER AND BAY (Continued)							
Raritan Bay Chapel Hill Channel	7/20/84	American Lobster*	F	173.0	6.3	30	-
Raritan Bay Chapel Hill Channel	7/20/84	American Lobster*	F	201.0	6.8	ND	28
Raritan Bay Chapel Hill Channel	7/20/84	American Lobster*	F	176.0	6.7	ND	31
Raritan Bay Chapel Hill Channel	7/20/84	American Lobster*	F	174.0	6.4	25	-
VII. COASTAL REGION							
12-14 Miles East of Long Branch Northern Section of Mud Hole	11/29/83	American Lobster CH		-	6.6	72	-
12-14 Miles East of Long Branch Northern Section of Mud Hole	11/29/83	American Lobster CH		-	6.8	82	-
4.7 Miles East @ 103° Shark River Inlet-Klondike	8/20/82	Bluefish		2641.7	63.5	37	-
30 Miles East at 113° Shark River Inlet	9/16/82	Bluefish	F	1696.7	57.0	ND	4.5

TABLE II
PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
VII. COASTAL REGION (Continued)							
12-18 Miles East of Barnegat Inlet	9/16/82	Bluefish		799.0	42.3	ND	9.5
Barnegat Inlet @ Lighthouse	10/83	Bluefish		5945.0	84.6	ND	11.0
Barnegat Inlet @ Lighthouse	10/83	Bluefish		5000.0	79.9	21	-
6mi. East @ 110° Shark River Inlet	7/24/84	Bluefish		-	-	ND	19
6mi. East @ 110° Shark River Inlet	7/24/84	Bluefish		-	-	ND	16.
6mi. East @ 110° Shark River Inlet	7/24/84	Bluefish		-	-	ND	34.
65 Mi. East of Point Pleasant	7/24/84	Bluefish		-	-	ND	19
65 Mi. East of Point Pleasant	7/24/84	Bluefish		-	-	ND	25
65 Mi. East of Point Pleasant	7/24/84	Bluefish		-	-	ND	17
65 Mi. East of Point Pleasant	7/24/84	Bluefish		-	-	ND	24

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
VII. COASTAL REGION (Continued)							
65 Mi. East of Point Pleasant	7/24/84	Bluefish		-	-	ND	25
40 mi. East of Sandy Hook	7/24/84	Bluefish		-	-	ND	24
2 mi. East of Barnegat Inlet	7/24/84	Bluefish		-	-	ND	18
½ Mi. East of Seaside Heights	7/24/84	Bluefish		-	-	ND	9
Mud Hole 18-20 Mi. East of Belmar N.J.	8/19/84	Bluefin Tuna+		29770	-	ND	14
Mud Hole 18-20 Mi. East of Belmar N.J.	8/19/84	Skipjack Tuna		4.34	63.3	ND	10
Great Egg Harbor River at Beasley's Point	8/28/82	Blue Crab*	F	85.0	13.1	ND	20.0
VIII. DELAWARE BAY REGION							
Delaware Bay at the Maurice River Cove	10/19/82	Blue Crab**	M/F	123.4	13.5	ND	4.0

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
VIII. DELAWARE BAY REGION (Continued)							
Delaware Bay at the mouth of the Cohansey River	10/19/82	Blue Crab	M/F	100.8	13.5	ND	25.0
Delaware Bay at West Creek	10/18/83	American Eel		448.5	57.5	ND	3.0
IX. DELAWARE RIVER							
Delaware River @ Raccoon Creek	6/9/82	White Perch		181.0	23.4	42	-
Delaware River @ Duck Island	6/23/83	White Catfish				ND	8.0
Delaware River @ Duck Island	6/23/83	Brown Bullhead				ND	3.6
X. MISC. WATERWAYS							
Cooper River @ Downstream of Tidegate	5/11/84	Channel Catfish		1900.0	54.7	ND	24.0
Swartswood Lake, Sussex County	6/8/82	Carp		2076.0	52.5	ND	7.0
Swartswood Lake, Sussex County	6/8/82	Largemouth Bass		682.0	35.9	ND	13.0
Swartswood Lake, Sussex County	6/8/82	Brown Bullhead		308.6	20.6	ND	10.0

TABLE II

PHASE II COMPLETE - DIOXIN FISH TISSUE DATA (2,3,7,8-TCDD)

SITE	DATE COLLECTED	FISH	SEX	$\overline{X}$ WEIGHT (GM)	$\overline{X}$ LENGTH (CM)	TCDD (PPT)	DETECTION LIMIT (PPT)
X. MISC. WATERWAYS (Continued)							
Raccoon Creek at the Delaware River Confluence	8/16/84	Striped Bass	M	498.6	36.0	ND	26
Raccoon Creek at the Delaware River Confluence	8/16/84	Striped Bass	M	577.2	39.3	ND	28
Raccoon Creek at the Delaware River Confluence	8/16/84	Striped Bass		1067.0	46.8	ND	21
3rd River at Yantacaw Pond, Nutley	8/28/84	Carp		875.8	38.7	ND	17
3rd River at Yantacaw Pond, Nutley	8/28/84	Carp		1165.5	41.3	ND	10
3rd River at Yantacaw Pond, Nutley	8/28/84	American Eel		227.8	47.5	ND	18
3rd River at Yantacaw Pond, Nutley	8/28/84	American Eel		337.8	53.4	ND	13

88

Table III

POSITIVE RESULTS FOR 2,3,7,8-TCDD IN BIOTA (PHASE II)

SAMPLE NUMBER	LOCATION	DATE COLLECTED	SPECIES	TCDD (PPT)
I. PASSAIC RIVER				
6.13-1*	@ N.J. Turnpike Bridge Near Diamond-Alkali	8/24/83	Blue Crab	480
6.12-6**	@ Avondale Swing Bridge Lyndhurst	8/23/83	Blue Crab	54
6.12-8	@ Avondale Swing Bridge Lyndhurst	8/23/83	Carp	100
6.11-2	@ Rutherford, Carlton Hills	8/24/83	Brown Bullhead	110
6.11-5	@ Rutherford, Carlton Hills	8/24/83	Carp	210
II. HACKENSACK RIVER				
5.6-2CH	@ Laurel Hill	8/20/83	Blue Crab	1063
5.6-3CH	@ Laurel Hill	8/20/83	Blue Crab	590
5.0-1CH	@ Sawmill Creek	8/18/83	Blue Crab	270
5.0-2CH	@ Sawmill Creek	8/18/83	Blue Crab	520
III. NEWARK BAY				
15D-10CH	@ Central Railroad Trestle	8/17/82	Blue Crab	570
15D-12CH	@ Central Railroad Trestle	8/17/82	Blue Crab	620
15D-9CH	@ Central Railroad Trestle	8/17/82	Blue Crab	500
15D-4CH	@ Central Railroad Trestle	8/17/82	Blue Crab	320
7.2-15	@ Passaic River Confluence	6/14/83	Striped Bass	23
7.2-16	@ Passaic River Confluence	6/14/83	Striped Bass	56
7.2-17	@ Passaic River Confluence	6/14/83	Striped Bass	32
7.2-18	@ Passaic River Confluence	6/14/83	Striped Bass	47

Legend

Blue Crab and American Lobster samples designated CH are composite hepatopancreas tissue.

* Sample mixture of muscle and hepatopancreas tissue from a single organism.

** Sample mixture of muscle and hepatopancreas from a composite of five (5) organisms.

\+ Sample of (1) single organisms.

Table III

POSITIVE RESULTS FOR 2,3,7,8-TCDD IN BIOTA (PHASE II) (Continued)

SAMPLE NUMBER	LOCATION	DATE COLLECTED	SPECIES	TCDD (PPT)
IV. HUDSON RIVER				
2.2-17CH	Bayonne to George Washington Bridge	7/20/83	Blue Crab	10
V. RARITAN RIVER				
9.0-3**	@ Victory Bridge	9/16/83	Blue Crab	48
9.0-5**	@ Victory Bridge	9/16/83	Blue Crab	25
VI. COASTAL REGION				
90.6-10	18 Miles East @ 110° Shark River Inlet-Klondike	8/20/82	Bluefish	37
91.5-3	Barnegat Inlet @ Lighthouse	10/83	Bluefish	21
90.3-49CH	12-14 Miles East of Long Branch-Northern Mud Hole Section	11/29/83	Lobster	72
90.3-41CH	12-14 Miles East of Long Branch-Northern Mud Hole Section	11/29/83	Lobster	82
11.0-122*	Raritan Bay Chapel Hill Channel	7/20/84	Lobster	59
11.0-123*	Raritan Bay Chapel Hill Channel	7/20/84	Lobster	62
11.0-124*	Raritan Bay Chapel Hill Channel	7/20/84	Lobster	30
11.0-127*	Raritan Bay Chapel Hill Channel	7/20/84	Lobster	25
11.0-131	Raritan Bay at Earle Pier	8/23/84	Striped Bass	20
VI. MISC. WATERWAYS				
14.16-1	Delaware River @ Raccoon Creek	10/18/83	White Perch	42

Table IV

COMPARISON OF 2,3,7,8-TCDD (ppt) IN PASSAIC RIVER FINFISH AND CRUSTACEANS (PHASE I AND II)

Sample Locations	Species	Phase I Results (ppt)	Phase II Results (ppt)
A. ABOVE THE HEAD OF TIDE			
1. Berkeley Heights	Carp	-	ND (17)*
	American Eel	-	ND (13)
	Largemouth Bass	-	ND (6)
2. Elmwood Park	Carp	-	ND (6)
	Carp	-	ND (4)
	American Eel	-	ND (5)
3. Dundee Lake	Carp	-	ND (4)
	Carp	-	ND (4)
	Carp	-	ND (29)
B. BELOW THE HEAD OF TIDE			
1. Below Dundee Dam	Channel Catfish	50 ppt	-
	White Perch	ND (10)	-
2. At Monroe St. Bridge Garfield	Carp	108 ppt	-
3. Near Third River Confluence	Brown Bullhead	-	110ppt
	Carp	155 ppt	210 ppt
	Carp	-	100 ppt
	Goldfish	66 ppt	-
	Blue Crab**	-	54 ppt
	American Eel*	-	ND (160)
4. 4st Bridge Harrison	American Eel	31 ppt	-
	American Eel	22 ppt	-
	American Eel	61 ppt	-
	Brown Bullhead	73 ppt	-
	Mummichog	114 ppt	-
5. Newark 80 Lister Ave.	Blue Crabs M	27 ppt	-
	Blue Crabs H	485 ppt	-
	Blue Crab M	16 ppt	-
	Blue Crab H	450 ppt	-
	Blue Crab**+	-	480 ppt
6. Confluence with Newark Bay	Striped Bass	58 ppt	
	Striped Bass	31 ppt	
	Striped Bass	-	56 ppt
	Stripped Bass	-	47 ppt
			32 ppt
			23 ppt

* ND-Non Detectable with Detection Limit in Parenthesis (ppt)
\+ Single Sample Analysis
\- No Samples Analyzed
** Muscle and Hepatopancreas Tissue Combined
M Muscle Tissue Only
H Hepatopancreas Tissue Only

Table V

BLUE CRAB AND AMERICAN LOBSTER 2,3,7,8-TCDD CONCENTRATIONS (ppt)*
BY TISSUE TYPE FOR ALL LOCATIONS SAMPLED (Phase II)

	I. Lump (Muscle) Meat	II. Hepatopancreas Only	III. Combined*
A. Blue Crab Data			
Single Sample (Individual Organisms)	Not analyzed	Not analyzed	480
Composite Sample (5 Organisms Combined)	Not detected in 11 samples analyzed	10 1063 590 270 520 570 620 500 320	48 25 54
B. American Lobster			
Single Sample	Not analyzed	Not analyzed	39 62 30 25
Composite Sample (15 Organisms Combined)	Not analyzed	72 82	Not analyzed

* Combined Tissue - Muscle and hepatopancreas tissue mixture
Results expressed in parts-per-trillion (ppt)

Table VI

2,3,7,8-TCDD AND 2,3,7,8-TCDF (ppt) RESULTS FOR STRIPED BASS CAUGHT AT CONFLUENCE OF PASSAIC RIVER AND NEWARK BAY AND THEIR RELATIONSHIP TO TWO FDA "LEVELS OF CONCERN" FOR 2,3,7,8-TCDD

	2,3,7,8-TCDD (ppt)	2,3,7,8-TCDF (ppt)
A. NEW JERSEY DEP ANALYSES*		
Phase I Study	58	
	31	
	x=44.5 ppt	
Phase II Study	23	N.D. (16)++
	56	33
	32	29
	47	42
	x=39.5 ppt	x=26.0 ppt
B. NEW YORK DEC ANALYSES**		
	67, 53+	30, 34+
	24	28
	16	20
	29	26
	x=34.0 ppt	x=26.0 ppt
Grand Mean x =	39.3 ppt	26.0 ppt

C. COMPARISON TO FDA "LEVELS OF CONCERN" for 2,3,7,8-TCDD***

1. Percentage of all Fish Greater than 25 ppt = 70%

2. Percentage of all Fish Greater than 50 ppt = 30%

\+ Replicated Analyses
++ Detection Limit in Parenthesis and counted as zero for mean
* Composites of five fish analyzed
** Single fish analyzed (All fish caught and supplied by NJDEP)
From O'Keefe et al (1984)
*** See Appendix B

Table VII

VOLUMETRIC SEDIMENTATION RATES IN CHANNEL AND OFF-CHANNEL AREAS OF (NEWARK BAY) STUDY AREA

Location	Area[a]	Sedimentation Rates	
	($m^2 x 10^6$)	($m^3 x 10^3$/yr)	(mm/yr)
Off-Channel	14.21	49.7[b]	3.5
Channel[c]			
Main Channel-North	0.35	19.4	55.4
Main Channel-South	0.90	2.2	2.4
Port Newark	0.45	62.3	138.4
Port Elizabeth	0.69	8.9	12.9
Ch. N. of Shooter's Isl.	0.31	102.9	331.9
Ch. S. of Shooter's Isl.	0.23	19.0	82.6
Passaic R.	0.38	56.0	147.4
Hackensack R.	0.51	20.6	40.4
N.Y.-N.J. Channels (Kill Van Kull & Arthur Kill)	1.14	0	0
Channel Totals	4.96	291.3 x	58.7
Bay-wide Totals	19.17	341.0 x	17.7

a Channel areas include side slopes

b Calculated from isopachs in Figure 10

c Quantities calculated from U.S. Army Engineer District, New York (1977) unpublished dredging records and from the U.S. Army Corps of Engineers (1952-1976)

From Suszkowski (1978)

TABLE VIII

PCB and Pesticide Analysis
of American Lobsters (Homarus americanus)
Caught in New Jersey Waters

Location	Date of Capture	Number of organisms in Sample		Mean length (cm)	% Lipid	Total[3] chlordane (PPB)	Alpha BHC (PPB)	Total[4] DDT (PPB)	Total[5] PCB's (PPM)
Offshore (Mud[1] Hole)	11/16/82	5	M[2]	7.24	0.73	--	ND[6]	13.39	0.31
			H		19.06	74.40	8.14	291.40	4.30
	"	5	M	6.72	0.43	--	ND	5.58	ND
			H		14.78	48.89	8.77	237.79	2.40
	"	5	M	7.04	0.77	--	ND	16.18	0.18
			H		13.57	84.57	8.77	276.55	3.90
	"	5	M	6.34	0.62	--	ND	9.06	0.15
			H		14.38	71.80	ND	227.92	3.05
	"	5	M	7.90	0.92	--	ND	12.97	0.13
			H		20.86	87.84	10.34	277.34	3.87
	"	10	M	7.20	0.46	--	ND	8.51	ND
			H		12.26	31.89	ND	179.59	2.15
	11/29/82	15	H	6.6	10.28	45.61	ND	116.82	2.29
	"	15	H	6.8	20.43	44.15	ND	145.09	3.20
Raritan Bay (Chapel Hill Channel)	7/20/84	5	C	6.4	2.93	11.00	ND	25.57	0.65
	"	5	C	6.0	3.64	10.17	ND	27.80	0.79

1. Location of Sampling was 12 miles east of Long Branch, NJ on the eastern slope of the Mud Hole
2. Tissue Types
   M = Muscle Tissue
   H = Hepatopancreas
   C = Composite Muscle and Hepatopancreas
3. Includes alpha and gamma chlordane
4. Includes DDT, DDD and DDE
5. Includes Arochlor 1254 and 1248
6. (ND) None Detectable

TABLE IX

CARCINOGEN RISK ASSESSMENT

Animal	n*	Tissue Level of TCDD, Mean and SD pg/g (ppt)	Excess cancers per 10^6 people for consumption of 15.7 grams per day for 70 years	Excess cancers per 10^6 people for consumption of 10 bass or 50 crabs or 10 lobsters a year for 70 years
Striped Bass	(21)	38.3 ± 17.4	1300	1560
Crab**	(48)	94.6 ± 64.4	3300	870
Lobster	(4)	44	1500	530

* N = number of organisms showing positive TCDD values.

** The Blue Crab data was generated from two categories of analyses, Mix 1 and Mix 2. Mix 1 refers to TCDD concentrations found in hepatopancreas tissue alone. A factor of 0.226 was multiplied times the hepatopancreas concentration to obtain the potential dose of TCDD in all edible portions if the lump meat was included. This factor was obtained from tissue weight data on 13 blue crabs showing the hepatopancreas to be approximately one quarter the weight of the whole organism (O'Connor 1984). It was then assumed that all of the TCDD was contained in the hepatopancreas. Mix 2 refers to data from analyses on crabs where hepatopancreas tissue was combined with thoracic muscle and claw muscle tissue before analysis. After conversion of Mix 1 the two data sets were then pooled to get the mean tissue level for all 48 organisms.

Table X

MEAN 2,3,7,8-TCDD (ppt) RESULTS FOR ALL PHASE II SAMPLES AND PERCENTAGE OF SAMPLES EXCEEDING FDA "LEVELS OF CONCERN"+

Species	$\bar{X}$ TCDD (ppt) (N)*	**Percentage of All Samples Greater Than 25 ppt (N)	**Percentage of A Samples Greater Than 50 ppt (N)
Blue Crab Muscle	0.0 (13)	0% (13)	0% (13)
Blue Crab Hepatopancreas	495.8 (9)	100% (9)	100% (9)
Blue Crab Muscle and Hepatopancreas Mixture	151.75 (4)	100% (4)	50% (4)
Striped Bass	35.6 (5)	19 (16)	6% (16)
Carp	155.0 (2)	20% (10)	20% (10)
Catfish***	110.0 (1)	17% (6)	17% (6)
American Lobster Hepatopancreas	77.0 (2)	100% (2)	100% (2)
American Lobster Mixture Hepatopancreas and Muscle	44.0 (4)	44% (9)	22% (9)
Weakfish	0.0 (1)	0% (1)	0% (1)
Bluefin Tuna	0.0 (1)	0% (1)	0% (1)
Skipjack Tuna	0.0 (1)	0% (1)	0% (1)
Bluefish	29.0 (2)	4% (21)	0% (21)
White Perch	42.0 (1)	50% (2)	0% (2)
American Eels	0.0 (10)	0% (10)	0% (10)
Largemouth Bass	0.0 (2)	0% (2)	0% (2)

* Mean Value of all positive results only. N=number of analyses.

** Includes non-detectable values only if detection limit is less than 25 ppt and N.D. counted as zero for simple statistics.

*** Combined subspecies: Brown Bullhead, White and Channel Catfish.

\+ See Appendix E

APPENDIX F

A. Methodology for Analysis of 2,3,7,8-TCDD in Fish

The method employed in the analysis of fish samples was derived from three sources. Procedures utilized by the State of Michigan, the University of Nebraska and the U.S. EPA have been combined to yield extracts suitable for GC/MS Analysis. The methodology is described herein.

Approximately 20g of sample was accurately weighed and spiked with known amounts of surrogate and internal standard. It was then saponified in 15ml of ethanol and 30ml of 40% aqueous KOH in a reflux apparatus for 60 minutes with stirring. The sample was completely hydrolyzed before terminating the saponification.

The solution was transferred to a 250ml separatory funnel and diluted with 20ml of ethanol and 40ml of water extracted four times with nanograde hexane. The first extraction was done with 25ml of hexane, shaking vigorously for one minute. The lower aqueous layer was removed to a clean beaker, and the upper hexane layer was decanted to a 125ml separatory funnel. The aqueous layer was then extracted three times more with 15ml portions of hexane, each time adding the hexane to a 125ml separatory funnel. The combined hexane extracts were washed with 10ml of water to remove excess base.

The combined hexane extracts were washed 4 times with 10ml concentrated H_2SO_4, or until both layers were clear. As many as 4 extracts may be necessary, depending on the sample. Again the hexane was washed with 10ml of water. The hexane layer was decanted and concentrated under a stream or dry nitrogen to approximately 1ml.

The initial clean-up procedure involves passing the extract through a dual Macro-column system with effluent solvent evaporation using ultra-pure nitrogen. The top column contains two layers: 1g silica over 4g 44% H_2SO_4/Solica. The bottom column contains two layers: 2g 33% 1N NaOH/Silica over 1g silica. The total effluent and additional hexane column rinses are evaporated to dryness. The sample container and column hexane rinses use a nominal total volume of 35ml hexane.

A second clean-up procedure is performed by putting the residue on the following dual column system: The top column is a Macro: 1.5 g 10% $AgNO_3$/silica and the bottom is a high aspect: 5.0 g basic alumina. When drained to bed level, the vial is rinsed with 2-3 ml hexane and this is placed on the column system. Repeat the rinsing procedure two additional times. The system is now eluted with 30 ml hexane, when drained to bed level the top column is removed. Allow bottom column to drain to bed level, then elute with 50 ml 50% CCl_4/hexane. The hexane and 50% CCL_4/hexane effluents are discarded. Chlorinated dioxins are eluted with 60 ml of 50% CH_2Cl_2/hexane, and the solvent is evaporated under ultra-pure N_2.

The final Option D clean-up is performed by using the following procedure: Prepare 18% Carbopak C on Celite 545TM by thoroughly mixing 3.6 grams of Carbopak C (80/100 mesh) and 16.4 grams of Celite 545™ in a 40 ml vial. Activate at 130°C for six hours. Store in a desiccator. Prepare a column

using a standard size (5-3/4 inches long by 7.0 mm o.d.) disposable pipet fitted with a small plug of glass wool. Using a vacuum aspirator attached to the pointed end of the pipet, add the carbopak/celite mix until a 2cm column is obtained. Preelute the column with 2-ml of toluene followed by 1-ml of 75:20:5 methylene chloride/methanol/benzene, 1-ml of 1:1 cyclohexane in methylene chloride and 2-ml of hexane. While the column is still wet with hexane add the extract obtained from above. Elute the column sequentially with two 1-ml aliquots of hexane, 1-ml of 1:1 cycloehxane in methylene chloride, and 1-ml of 75:20:5 methylene chloride/methanol/benzene. Next collect the TCDD fraction by elution with 2-ml of toluene. The sample is stored at this point in a freezer until GC/MS analysis. Just before analysis begins, reconstitute the residue with 10ul of isooctane.

The GC/MS method employed for the analysis for TCDD is the September 1983 draft revision of "Determination of 2,3,7,8-TCDD in Soil and Sediment", U.S. Environmental Protection Agency, Region VII Laboratory, Kansas City, Kansas. The sample extracts are analyzed using an eletron impact GC/MS instrument with a direct capillary interface. A 60 meter isomer specific fused silica column was used for the analysis.

GC/MS ison measurement cycle time is 0.75 second/cycle.

The accuracy of the analysis is directly dependent on the accuracy of the native TCDD stock solution. We used the certified standard from the EPA as the primary standard from the EPA as the primary standard to calculate the values in the sample.

Native: 2,3,7,8-TCDD 99+% Standard - EPA Lot No. 20603-01/83

Labeled: $^{13}c12$ 98 +% Standard - KOR, Inc., Lot No. J-2-70

$^{37}c14$ 99% Standard - KOR, Inc., Lot No. SSY-G-123

B. Quality Assurance/Quality Control Procedures (QA/QC)

Quality assurance protocols were based on the following government guidelines:

- "Handbook for Analytical Quality Control in Water and Wastewater Laboratories", EPA-600/4-79-019, March 1979;
- National Enforcement Investigation Center Policies, and Procedures manual; EPA-330/9/79/001-R, October 1979;
- the recommended guidelines for EPA Methods 624 and 625. (Federal Register, December 3, 1979, pp. 69532-69559); and
- "Manual of Analytical Methods for the Analysis of Pesticides in Humans and Environmental Samples," EPA 600/8-80-038. June 1980.
- "Determination of 2,3,7,8-TCDD in Soil and Sediment" EPA, Region VII, Kansas City, September 1983.

However, protocols were modified to provide a higher level of QA/QC than the guidelines require. For example, a higher than required number of quality control samples were analyzed and especially careful attention was paid to the certification of the "reference standard" compounds used in analysis. Below are listed the key QA/QC elements for the methods used.

Analysis of 2,3,7,8-TCDD (Dioxin) by GC/MS (SIM)

- Each sample is dosed with a known quantity of $^{13}C_{12}$-2,3,7,8-TCDD as internal standard and $^{37}C_{14}$-TCDD as surogate standard. The action limits for surrogate standard results is +/-40% of the true value. Samples showing surrogate standard results outside of these limits are reextracted and reanalyzed.

- A laboratory "method blank" is run along with each set of 24 or fewer samples. The method blank is also dosed with the internal standard and surrogate standard.

- At least one per set of 24 samples is run in duplicate to determine intralaboratory precision.

- Qualitative Requirements. The following are met in order to confirm the presence of native 2,3,7,8-TCDD:

  a. Isomer specificity must be demonstrated intitally and verified once per 8-hour work shift. The verification consists of injecting a mixture containing TCDD isomers which elute close to 2,3,7,8-TCDD. The 2,3,7,8-TCDD must be separated from interfreeing isomers, with no more than 25% valley relative to the 2,3,7,8-TCDD peak.

  b. The 320/322 ratio is within the range of 0.67 to 0.87.

  c. Ions 320, 322, and 257 are all present and maximize together the signal to mean noise ratio must be 2.5 and 1 or better for all 3 ions.

  d. The retention time is equal (within 3 seconds) the retention time for the isotopically labeled 2,3,7,8-TCDD.

  e. At least one of the positives can be confirmed by obtaining partial scan spectra form mass 150 to mass 350. The partial scan guidelines are as follows:

     . the 320/324 ration should be 1.58+/-0.16

     . The 257/259 ratio should be 1.03+/-0.10

     . the 194/196 ratio should be 1.54+/0.15

- One sample is spiked with native 2,3,7,8-TCDD at a level of 1.0 PPB (for soil) for each set of 24 or fewer samples.