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DRAFT FINAL BASELINE PUBLIC HEALTH RISK ASSESSMENT; NEW BEDFORD HARBOR FEASIBILITY STUDY

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EXECUTIVE SUMMARY

New Bedford Harbor is an urban tidal estuary located between the City of New Bedford on the west and the Towns of Fairhaven and Acushnet on the east, at the head of Buzzards Bay, Massachusetts (Figure E-1). Between 1974 and July 1982, several environmental studies were conducted to assess the magnitude and extent of polychlorinated biphenyl (PCB) and heavy metal contamination in New Bedford Harbor. The studies revealed that sediments north of Hurricane Barrier contain elevated PCB and heavy metals levels. PCB concentrations range from a few parts per million (ppm) to over 100,000 ppm, and concentrations of metals range from a few ppm to over 5,000 ppm. PCB concentrations in surface water in excess of the Ambient Water Quality Criterion for PCBs were observed. Concentration of PCBs in locally caught fish were also detected in excess of the Food and Drug Administration PCB tolerance level of 2 ppm (previously 5 ppm). Data from these and more recent studies have been combined to form the central New Bedford Harbor Data Base.

The purpose of this risk assessment was to estimate potential risks to public health under baseline (i.e., current) conditions from exposure to PCBs and metals detected in the sediment, surface water biota and air within the New Bedford Harbor site. The baseline assessment is the first of a series of three risk assessments to provide the basis for evaluating the need for and the extent of remediation; it is based on existing conditions in the harbor and does not consider potential natural decreases in contaminant concentration due to transport and degradation through time.

Recent sampling data indicates that no appreciable changes in PCB concentrations have occurred over the past decade. Sustained elevated levels of PCBs (i.e., greater than 2 ppm) in lobster and several other species have been documented in fishing closure Area 3 (Kolek and Ceurvels, 1981; Massachusetts Divison of Marine Fisheries, unpublished data; Pruell et al., 1988), and elevated levels of PCBs (i.e., greater than 4,000 ppm) in sediment have been reported (USACE, 1988). While it is probable that natural processes such as biodegradation and photolysis will result in a decrease in PCB concentrations in sediment and biota, these changes are not expected to be significant over the next 10 years. The evaluation in this risk assessment indicates that an order-of-magnitude or more change in PCB concentrations would be necessary to reduce exposure concentrations to levels consistent with EPA and state public health guidance. Reduction of that magnitude is not expected to occur without remedial actions.

SOURCE: USGS QUADRANGLES;
NEW BEDFORD NORTH & SOUTH, MASS., 1977 & 1979

SCALE IN FEET

0 2000 4000

FIGURE E-1
SITE LOCATION MAP
NEW BEDFORD HARBOR

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To evaluate the effectiveness of various remedial alternatives, additional risk assessments will be conducted based on the results of the sediment contaminant transport and food-chain models. These risk assessments will allow an evaluation of the relative effectiveness of the various remedial alternatives against the baseline conditions.

The methodology and results of this baseline assessment is summarized in the following subsections.

PUBLIC HEALTH RISK SUMMARY

The purpose of the public health risk assessment was to accomplish the following:

- identify human receptors potentially at risk from contaminant exposure
- determine significant exposure routes
- characterize the intrinsic toxicity of PCBs, cadmium, copper, and lead
- estimate the potential carcinogenic and noncarcinogenic risks to public health from contaminant exposure.

Primary sources of information used in this report were the New Bedford Harbor Data Base, the Greater New Bedford Health Effects Study (GNBHES), various site investigation reports, and data from the pilot study recently conducted by the Army Corps of Engineers. The public health risk assessment consists of four sections. The first section, the Introduction, reviews the site history. The second section, the Exposure Assessment, identifies potential human receptors and describes mechanisms by which these receptors may be exposed to contaminants within the New Bedford Harbor area. The third section, the Toxicity Assessment, provides a description of the toxic properties of PCBs, cadmium, copper, and lead. The final section, the Risk Characterization, quantifies carcinogenic and noncarcinogenic risks to public health.

SUMMARY OF THE EXPOSURE ASSESSMENT

An analysis of demographic and land use information, and activity and behavior patterns, indicated that contaminant exposure in the New Bedford Harbor area could occur through dermal contact with sediments and water, ingestion of water and biota, and/or inhalation of airborne contaminants. A quantitative screening analysis of the exposure pathways was

performed to identify the principle pathways of exposure, which consist of the following:

- ingestion of aquatic biota
- direct contact with sediments
- ingestion of sediments
- inhalation of airborne contaminants

These exposure pathways accounted for over 99 percent of the potential exposures within the New Bedford Harbor area, and were the focus of the quantitative risk evaluation. Exposure to contaminants from direct contact with and/or ingestion of surface water was also evaluated. However, these exposure routes were not considered to present a public health risk. PCBs and metal concentrations in surface water were not at levels considered harmful to public health.

Exposure scenarios were developed to estimate the potential exposure dose contaminant and for each exposure pathway. These scenarios were based on a various exposure conditions, primarily focusing on areas where exposure was considered likely to occur.

The New Bedford Harbor site was divided into three areas (i.e., Areas I, II, and III) for purposes of assessing exposure to sediments. This division separates areas of high sediment contamination from areas of low sediment contamination. Area-specific contaminant concentrations provide a realistic estimate of the exposure point concentration. The areas were defined as follows:

- Area I - the area between the Wood Street and Coggeshall Street bridges
- Area II - the area between the Coggeshall Street Bridge and the Hurricane Barrier
- Area III - the area south of the Hurricane Barrier.

These areas are depicted in Figure E-2.

Exposure through the ingestion of biota was assessed separately for the following four areas:

- Area 1 - the area between the Wood Street Bridge and the Hurricane Barrier

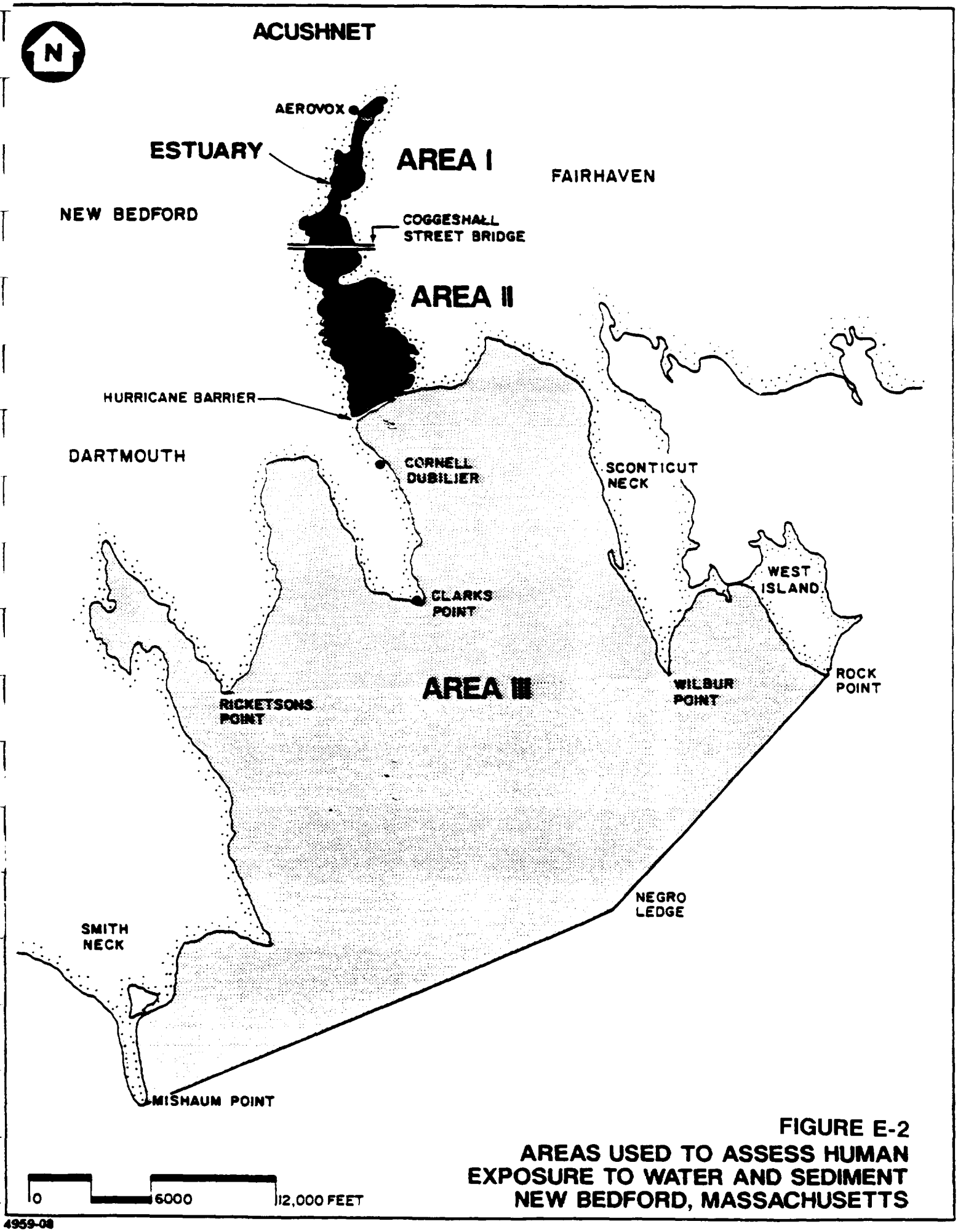


FIGURE E-2
AREAS USED TO ASSESS HUMAN EXPOSURE TO WATER AND SEDIMENT
NEW BEDFORD, MASSACHUSETTS

4959-08

- Area 2 - the area between the Hurricane Barrier and Wilbur and Ricketsons Points
- Area 3 - the area between Wilbur, Ricketsons, and Rock points, and Negro Ledge and Mishaum Point
- Area 4 - beyond Area 3 extending into Buzzards Bay

These areas are depicted in Figure E-3.

SUMMARY OF THE TOXICITY ASSESSMENT

This section provides appropriate toxicological information necessary to evaluate the potential public health risks from exposure to PCBs, cadmium, copper, and lead.

Toxicological evaluations, developed for each contaminant, describe the nature and severity of potential adverse effects associated with exposure to each compound. Information contained in these evaluations includes physiochemical data, pharmacokinetic and toxicity information, and descriptions of noncarcinogenic effects associated with acute, chronic, and lifetime exposures.

In addition, information about the potency of PCBs, cadmium, copper, and lead was presented as part of the dose-response assessment. The assessment included pertinent standards, criteria, advisories, and guidelines developed for protecting public health. These standards and criteria were used to evaluate potential noncarcinogenic and carcinogenic risks associated with contaminant exposure.

SUMMARY OF PUBLIC HEALTH RISKS

Estimates of carcinogenic and noncarcinogenic risks associated with PCB and metals exposure were developed for direct contact and ingestion of sediments, ingestion of biota, and inhalation of airborne contaminants.

Noncarcinogenic risk estimates were generated by comparing the exposure dose for each contaminant to the most applicable health-based standard or criteria value. Values used in this risk assessment represent contaminant concentrations that do not present a public health risk. The ratio of the estimated body dose levels to standard or criteria values is used to evaluate risk. In this risk assessment, the ratio is referred to as the risk ratio.

The risk ratio was evaluated against a value of 1. Generally, the Environmental Protection Agency (EPA) states that if the risk ratio is less than 1, the predicted body dose level is

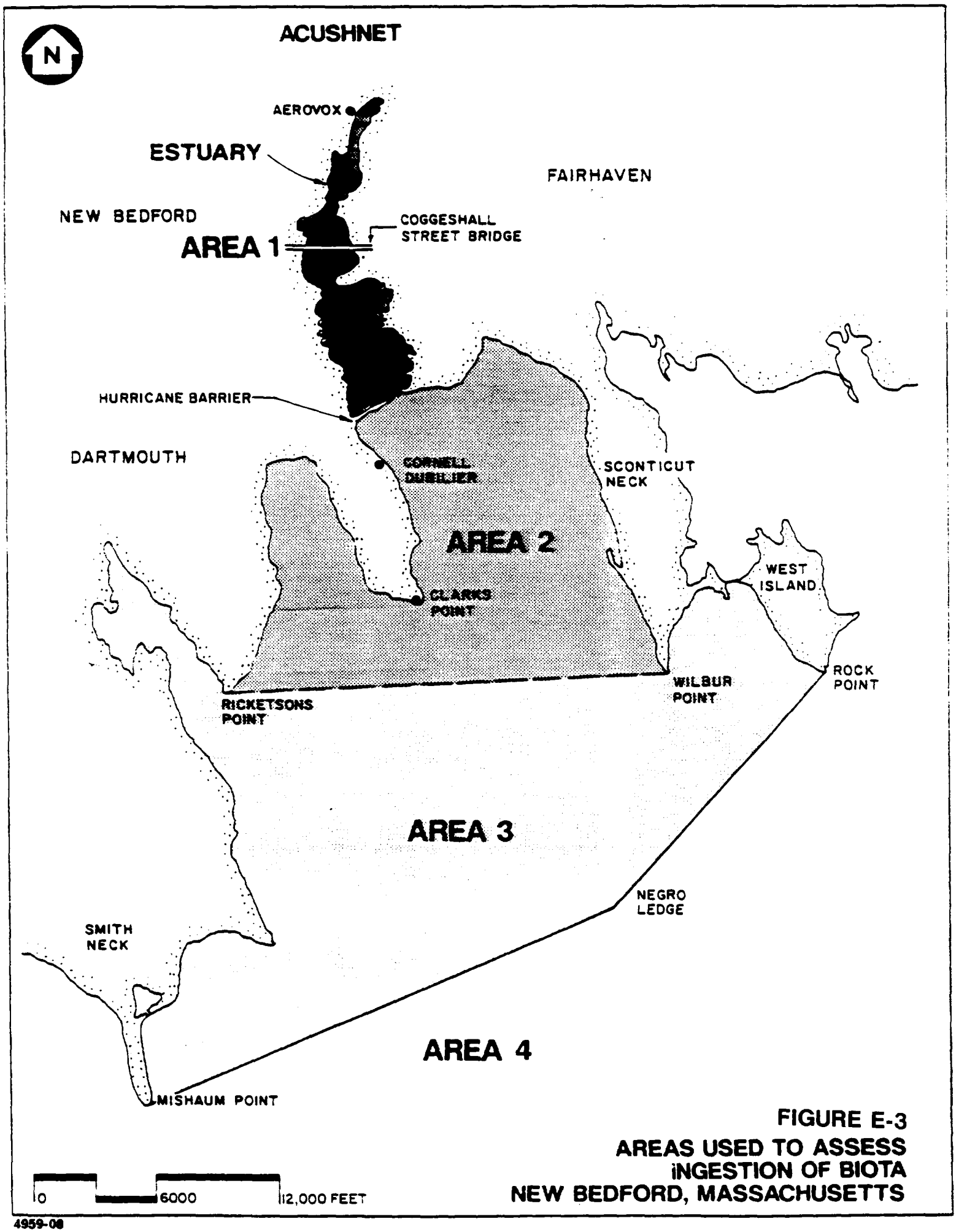


FIGURE E-3
AREAS USED TO ASSESS
INGESTION OF BIOTA
NEW BEDFORD, MASSACHUSETTS

4959-08

anticipated to be without lifetime risk to public health. The sum of these risk ratios, referred to as the Hazard Index (HI), represents the potential risk associated with concurrent exposure to multiple contaminants. As with the risk ratio, the HI is evaluated against a value of 1.

Carcinogenic risk estimates were calculated by multiplying the potency factor of the contaminant by the estimated body dose concentration. The product of the two values is an estimate of the incremental lifetime cancer risk, which is defined as the excess probability that an individual will develop cancer over a lifetime.

EPA guidance states that the target total carcinogenic risk for an individual resulting from exposure at a Superfund site may range from 10^{-4} to 10^{-7}. Therefore, response objectives and remedial alternatives are developed to reduce the total carcinogenic risks to levels within or below this range. Carcinogenic risk estimates developed in this report were evaluated using this target range.

In addition to the EPA target range, carcinogenic risk estimates were also evaluated against a total site cancer risk level of 10^{-5}. This risk level is stated in the portion of the Massachusetts Contingency Plan (MCP) relevant to risk assessment. The MCP requires that a permanent solution be implemented at all disposal sites that effectively eliminates significant or otherwise unacceptable risk to health, safety, public welfare, or the environment. As stated in the MCP, the total site cancer risk must be compared to a cancer risk of 10^{-5}.

The following subsections summarize risk estimates generated for each exposure route.

Direct Contact with Sediment

Noncarcinogenic and carcinogenic risks associated with direct contact exposure to PCB-, cadmium-, copper-, and lead-contaminated sediment were evaluated separately for Areas I, II, and III, and focused on locations within these areas where exposure was likely to occur. Contaminant concentrations detected in shoreline sediments were used when available.

Noncarcinogenic risk estimates for exposure to sediment in Area I exceeded 1 under the majority of scenarios evaluated, and ranged from 0.7 to 200. PCB exposure accounted for most of the risk. Individual risk ratios for cadmium, copper, and lead were all below 1. Noncarcinogenic risk ratios associated with PCB exposure in Area I indicate a potential public health risk.

Exposure to sediments from Areas II and III were associated with noncarcinogenic risk ratios ranging from less than 1 to 3. The only risk ratios to exceed 1 were based on conservative exposure assumptions, which were not considered representative of likely exposure conditions for these areas (including long-term repetitive exposure to the maximum detected contaminant concentration). Based on this evaluation, the noncarcinogenic risk for direct contact exposure in Areas II and III was not considered to pose a risk to public health.

Carcinogenic risks associated with direct contact exposure to sediments was greatest for Area I. Risk estimates based on exposure by a child, an older child, and an adult, ranged from $1x10^{-6}$ to $1x10^{-2}$, with most scenarios associated with risks in excess of the EPA target risk range of 10^{-4} to 10^{-7}. Based on this evaluation, methods to reduce these risks will be addressed in the Feasibility Study (FS).

Carcinogenic risks estimated for Area II assuming probable exposure conditions ranged from $2x10^{-7}$ to $8x10^{-6}$. The only risk estimates exceeding the target range were those associated with PCB exposure under conservative exposure conditions. Because these conditions assume repetitive, long-term exposure to the maximum PCB concentration, the associated risks were considered overly conservative. As stated, exposure under more realistic conditions were associated with risks in the lower end of the target range.

In Area III, carcinogenic risks ranged from $1x10^{-8}$ to $2x10^{-6}$ under probable exposure conditions, and from $2x10^{-7}$ to $1x10^{-4}$ under conservative exposure conditions. No risk estimates exceeded the EPA target risk range.

Ingestion of Sediment

Exposure through ingestion of sediment was considered an age-related activity and most significant for children less than six years old. Both noncarcinogenic and carcinogenic risks associated with this route of exposure were evaluated.

Noncarcinogenic risk associated with exposure to cadmium- and copper-contaminated sediments in Areas I, II, and III were below 1 for all scenarios evaluated. Risk ratios based on exposure to PCBs and lead-contaminated sediments exceeded 1 under certain scenarios. For Area I, risk ratios for PCBs and lead ranged from 11 to 175 and 26 to 33, respectively. The magnitude and extent to which the values exceed 1 indicates that ingestion of Area I sediment presents a potential health risk to children.

Risk ratios based on ingestion of PCB-contaminated sediment in Areas II and III ranged from below 1 to 17. However, the risk

ratios based on exposure at recreational locations and under probable exposure conditions within these areas were all below 1. Because these scenarios were considered to represent actual exposure conditions, ingestion of sediments from Areas II and III was not considered to present a noncarcinogenic health risk.

Incremental carcinogenic risks associated with exposure through the ingestion of sediment were greatest for Area I and ranged from $6x10^{-6}$ to $1x10^{-2}$. These risk estimates were based on exposure to sediments in areas where access by children is considered possible. These risks fell within and exceeded the EPA target range of 10^{-4} to 10^{-7}. As such, methods to reduce these risks will be addressed in the FS.

Risk estimates based on exposure in Area II ranged from $9x10^{-7}$ to $2x10^{-4}$, with most risk values falling between 10^{-5} and 10^{-6}. Risk estimates based on probable exposure conditions ranged from $9x10^{-7}$ to $2x10^{-5}$. The risks based on exposure in Area III fall within the lower end of the target range and are between $2x10^{-7}$ to $3x10^{-6}$.

Risks associated with exposure through direct contact and ingestion of contaminated shoreline sediment are greatest for Area I. Both the carcinogenic and noncarcinogenic risk estimates based on PCB exposure in this area exceeded the EPA-established criteria levels. Noncarcinogenic risks based on exposure to metals in this area were below levels considered to represent a public health risk. Methods to reduce carcinogenic risks from PCB exposure will be evaluated in the FS.

Risk estimates based on exposure to sediment from other New Bedford Harbor areas were less than those developed for Area I. Noncarcinogenic risks based on exposure to PCBs and metals were below levels considered to represent a public health concern. Carcinogenic risks associated with probable exposure condition through direct contact with and ingestion of sediments from Areas II and III ranged from less than 10^{-7} to $8x10^{-5}$. Most risks were between 10^{-6} and 10^{-5}. Young children were considered at greater risk from contaminant exposure than older children or adults.

Risk estimates based on acute exposure to sediments, representing intermittent or once-in-a-lifetime exposure were below EPA criteria levels . Therefore, these exposures were not considered to present a public health risk.

Ingestion of Aquatic Biota

Exposure to PCBs and metals through ingestion of biota was evaluated for potential noncarcinogenic and carcinogenic risks.

Three species were considered in this evaluation: winter flounder, clams, and lobster (both with and without tomalley). Separate scenarios were developed for each species and assumed that 100 percent of the seafood diet was comprised of said species. A standard 8-ounce fish meal (i.e., 227 grams) was assumed for older children and adults, and a 4-ounce fish meal (i.e., 115 grams) was assumed for younger children.

Risk ratios based on exposure to cadmium and copper by older children and adults ranged from below 1 to 7.9. Ratios in excess of 1 were based on daily ingestion frequencies and whole-body tissue concentrations. These conservative assumptions may overestimate the actual risks, suggesting that exposure to cadmium and copper may not present a public health concern.

However, exposure to cadmium and copper by children resulted in risk ratios ranging from below 1 to 15.8. Because young children are more sensitive to contaminant exposure than older children and adults, this exposure route was considered to present a greater risk to a child's health.

Risk ratios based on exposure to lead and PCBs via ingestion of biota for all age classes exceeded 1 for most scenarios evaluated. No particular area or species appeared to consistently present a greater risk from exposure to these compounds. Based on this evaluation, exposure to lead and PCBs through the ingestion of biota presents a public health risk.

Incremental carcinogenic risks associated with the ingestion of biota fall within or exceed the EPA target range. Many scenarios evaluated had associated risks in excess of 10^{-3}. The risk estimates range from $1x10^{-5}$ to $9x10^{-3}$; for Area 1; from $4x10^{-6}$ to $1x10^{-2}$ for Area 2; from $6x10^{-6}$ to $8x10^{-3}$ for Area 3 and from $1x10^{-6}$ to $2x10^{-3}$ for Area 4. The highest risks were associated with ingestion of lobster including the tomalley.

Methods to reduce the noncarcinogenic risks from exposure to cadmium, copper, lead, and PCBs, and carcinogenic risks from exposure to PCBs will be assessed in the FS.

Inhalation of Airborne Contaminants

Limited air data were available to assess risks associated with inhalation exposure to PCBs. Data available for risk evaluation were collected from sampling stations distant from receptor locations that were chosen to provide a measure of the maximum PCB concentrations in the air above the mud-flats in Area I. Using these concentrations to assess potential risk was considered overly conservative.

Lifetime exposure to the assumed background concentration of 10 nanograms per cubic meter for the New Bedford Harbor area was assessed and associated with incremental carcinogenic risks in the 10^{-6} range. These risk estimates were based on conservative exposure conditions suggesting that actual risks from this route of exposure are less than 10^{-6}.

SUMMARY OF TOTAL SITE RISKS

The total site risk associated with multimedia and multitoxic exposure was generated by summing the individual risk estimates developed for the ingestion and direct contact with sediments, ingestion of biota and inhalation of air. This scenario represents the risks associated with concurrent or sequential exposure to contaminants through multiple exposure pathways. Total site risk estimates were evaluated against the MCP criteria of $1x10^{-5}$ incremental carcinogenic risk level and of 0.2 noncarcinogenic HI.

The total site risks evaluated in this report were based on chronic exposure via ingestion of, direct contact with, and inhalation of PCBs, cadmium, copper, and lead under probable exposure conditions. The carcinogenic and noncarcinogenic risk estimates for each age class and areas assessed exceed 10^{-5} and 0.2 respectively. Based on this evaluation, methods to reduce the overall site risk will be addressed in the FS.

THE GREATER NEW BEDFORD HEALTH EFFECTS STUDY

In the fall of 1987, the Massachusetts Department of Public Health released the findings of the GNBHES, a three-year study to determine the prevalence of elevated serum PCB levels in a random sample of Greater New Bedford area residents and to test the relationship between serum PCB levels and various health effects. GNBHES was a collaborative effort of the MDPH, the Massachusetts Health Research Institute, and the U.S. Centers for Disease Control.

GNBHES provided retrospective exposure and demographic information for the greater New Bedford area, which was incorporated into this exposure assessment. Because GNBHES focused on seafood consumption and occupational exposure, no information for either inhalation or direct contact exposure to PCBs was presented. Additionally, GNBHES provided exposure and limited demographic information only for persons between 18 and 64 years of age.

The purpose of this risk assessment was to predict how people are or may be exposed to PCBs under various exposure conditions. Exposure scenarios were developed to describe the possible exposures received by a hypothetical individual.

GNBHES does not contradict this risk assessment. Measures recommended in the GNBHES can be viewed as ways to reduce many of the risks identified in this risk assessment.

1.0 INTRODUCTION

This report presents the baseline public health risk assessment for the New Bedford Harbor Superfund site. This work is a component of the New Bedford Harbor REM III Superfund Feasibility Study (FS) and was conducted under contract to Ebasco Services, Inc. (Ebasco) under U.S. Environmental Protection Agency (EPA) Contract Number 68-01-7250.

1.1 SITE DESCRIPTION AND HISTORY

New Bedford Harbor is an urban tidal estuary on the western shore of Buzzards Bay, Massachusetts, situated between the City of New Bedford on the west and the Towns of Fairhaven and Acushnet on the east. The area contains approximately six square miles of open water, tidal creeks, salt marshes, and wetlands, and provides habitats for a wide variety of aquatic organisms that use this area for spawning, foraging, and overwintering.

The Acushnet River runs through three communities: Fairhaven, New Bedford, and Acushnet, Massachusetts. The coastal town of Dartmouth, Massachusetts, is located south of and adjacent to New Bedford and borders Clark Cove and Buzzards Bay. These four towns comprise the Greater New Bedford Harbor area. The estimated population of this area is 145,600 (based on the 1987 town census for Acushnet, Fairhaven, and Dartmouth and the 1986 census for the City of New Bedford).

Between 1974 and 1982, a number of environmental studies were conducted to assess the magnitude and extent of PCB (polychlorinated biphenyl) contamination in New Bedford Harbor. Results of these studies revealed that sediment north of the Hurricane Barrier contained elevated levels of PCBs and heavy metals. Additional investigations revealed that PCBs had been discharged into the surface waters of New Bedford Harbor, causing elevated PCB concentrations in sediment, water, fish, and shellfish.

To reduce the potential for human exposure to PCBs, the Massachusetts Department of Public Health closed much of the New Bedford Harbor area to fishing. Three closure areas were established on September 25, 1979. Area 1 (New Bedford Harbor) is closed to the taking of all finfish, shellfish, and lobsters. Area 2 (Hurricane Barrier to a line extending from Ricketson Point to Wilbur Point) is closed to the taking of lobster and bottomfeeding fish (eel, scup, flounder, and tautog). Area 3 (from Area 2 out to a line from Mishaum Point, Negro Ledge, and Rock Point) is closed to the taking of lobster.

In July 1982, the U.S. Environmental Protection Agency (EPA) placed New Bedford Harbor on the Interim National Priority List (NPL). The final NPL was promulgated in September 1984. The

site, as listed, includes the Upper Estuary of the Acushnet River, New Bedford Harbor, and portions of Buzzards Bay. Following the NPL listing, EPA Region I initiated a comprehensive assessment of the PCB problem in the New Bedford area. This assessment included an area-wide ambient air monitoring program, a sediment profile for the Acushnet River and harbor, and a biota sampling program in the estuary and harbor.

As a result of these studies, a better understanding of the extent of PCB contamination has been gained. The entire harbor north of the Hurricane Barrier, an area of 985 acres, is underlain by sediment containing elevated levels of PCBs and heavy metals. PCB concentrations in this area range from a few parts per million (ppm) to over 100,000 ppm. Portions of western Buzzards Bay sediment are also contaminated, with PCB concentrations occasionally exceeding 50 ppm, primarily near locations of combined sewer outfalls. The water column in New Bedford has been measured to contain PCBs in excess of EPA's Ambient Water Quality Criterion (AWQC). Concentrations of PCBs in edible portions of locally caught fish have been measured in excess of the Food and Drug Administration (FDA) 2 ppm tolerance level for PCBs.

In 1984, EPA conducted an initial Feasibility Study (FS) of the highly contaminated mudflats and sediment in the upper estuary of the Acushnet River. Five clean-up options were presented in that report. EPA received extensive comments on these options from other federal, state, and local officials, potentially responsible parties, and the public. Many of the comments expressed concern regarding the proposed dredging techniques and potential impacts of dredging on the harbor, and potential leachate from the proposed unlined disposal sites.

In responding to these comments, EPA elected to conduct additional studies before choosing a clean-up alternative for the Upper Estuary. Concurrent with these studies, EPA is conducting additional surveys to better define the extent of PCB contamination throughout the overall Harbor and Bay. Through these efforts, clean-up options for this site are being developed.

PCBs are the primary contaminant of concern in the Hot Spot area and estuary. However, the Acushnet River Estuary is not a pristine estuarine environment, and has historically been polluted with industrial and sanitary waste discharges. Due to these other discharges, there are elevated levels of polycyclic aromatic hydrocarbons (PAHs) and heavy metals (i.e., copper, chromium, lead, and cadmium) in the estuary sediment. The presence of and potential risks from metal contamination are presented in the baseline risk assessment; risks from exposure

to PAHs in the Hot Spot area have been previously evaluated (E.C. Jordan/Ebasco, 1987).

PAH compounds were found to be collocated with PCBs; however, the range of PAH concentrations in sediment was significantly less than the range of PCB concentrations. Total PAH concentrations range from below detection limit to 930 ppm, with an average PAH sediment concentrtion of approximately 70 ppm. (The highest PAH concentration of 930 ppm was detected in the Hot Spot area of the upper estuary.) No discrete areas of elevated levels of PAH compounds were observed, suggesting that PAH contamination results from non-point sources such as urban runoff. PAH concentrations detected in New Bedford Harbor sediment are similar to PAH concentrations detected in other urban and industrialized areas (EPA, 1982).

The relative toxicity of PAH compounds with respect to PCBs indicates that the majority of risk from exposure to sediment can be attributed to PCBs. Since PAH compounds can be effectively treated by the technologies identified in the Hot Spot FS to treat PCB contamination, methods taken to reduce PCB contamination will effectively reduce PAH contamination (E.C. Jordan/Ebasco, 1989). However unlike PCBs, the discharge of PAH compounds is expected to continue after remediation into the upper estuary from non-point sources. Therefore, remedial actions may not permanently reduce levels of these contaminants.

1.2 OBJECTIVES OF THIS REPORT

EPA Region I is responsible for the cleanup of the New Bedford Harbor site under the authority of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) as amended by the Superfund Act Reauthorization Amendments (SARA) of 1985. Pursuant to this charter, Region I has direct responsibility for conducting the appropriate studies for this site to support the need for and extent of remediation. In accordance with the National Contingency Plan (NCP), these studies form the basis of the Remedial Investigation/Feasibility Study (RI/FS) for the site.

This risk assessment presents and quantifies risks to public health due to PCB, cadmium, copper, and lead exposure in the New Bedford Harbor area under baseline (existing) conditions. The baseline assessment is the first of a series of risk assessments that will provide the basis for evaluating the need for and extent of remediation. It is based on existing conditions in the harbor only and does not consider potential natural decrease in contaminant concentration in the harbor due to transport and degradation through time.

While it is probable that natural processes will result in a decrease in contaminant concentrations, these processes are not expected to show significant changes over the next decade. Recent sampling data indicates no appreciable change in PCB concentrations have occured over the past 10 years. Sustained elevated levels of PCBs (i.e., greater than 2 ppm) in lobster and other species have been documented (Kolek and Ceurvels, 1981; Massachusetts Division of Marine Fisheries, unpublished data; Pruell et al., 1988), and elevated levels of PCBs in sediment (i.e., greater than 4,000 ppm) have been recorded (USACE, 1988). Reduction of PCB concentrations to levels consistent with EPA and state public health guidance are not expected to occur without remedial actions.

Additional risk assessments will be conducted to determine the effectiveness of various remedial alternatives. Results of the sediment contaminant transport and food-chain models will be used to provide future potential exposure point concentrations under various conditions. Risk assessments conducted using these modeled results will allow an evaluation of the relative effectiveness of the various remedial alternatives against the baseline conditions.

1.3 REPORT STRUCTURE

This report consists of three sections. The first section is the Exposure Assessment, which identifies potential human receptors and describes the mechanism by which these receptors may be exposed to contaminants within the New Bedford Harbor area. The second section, Toxicity Assessment, provides a description of the toxic properties of PCBs, cadmium, copper, and lead. In addition, the existing standards and criteria for these compounds are presented and discussed. The final section, Risk Characterization, combines information presented in the first two sections to describe and quantify the potential risks to public health.

1.4 PROGRAM DATA BASE

Data on the distribution of PCBs in sediment and overlying waters of New Bedford Harbor and the Acushnet River Estuary were provided by Battelle Pacific Northwest Laboratories (PNL). For consistency with other aspects of the RI/FS process in New Bedford, the public health risk assessment was based primarily on a data set developed as the initial conditions for the physical/chemical transport model. The initial conditions were established by PNL using information on PCBs in the harbor obtained from three sources, each of which will be described briefly below: data collected by Battelle Ocean Sciences (BOS) (Duxbury, MA) specifically for the calibration and validation of the model, a data base compiled by GCA Corporation (now Alliance Technologies Corporation) from a variety of historical

sources, and a detailed survey of PCBs in the harbor developed by NUS. These three data sets were subsequently combined into the central New Bedford Harbor Data Base by BOS (Administrative Record).

1.4.1 BOS Calibration/Validation Data

From 1985 through 1986 BOS conducted four samplings of water, sediment, and biota in the Acushnet River Estuary, New Bedford Harbor, and adjacent areas of Buzzards Bay to provide data for calibration and validation of the physical/chemical transport model and food-chain model. Twenty-five stations were established and sampled on each of three surveys; the remaining survey was limited to eight stations and was conducted immediately following a storm event. Although the samples obtained during these surveys were collected and analyzed under rigorous quality control procedures, the data were intended for use primarily for model calibration/validation; their usefulness for determining patterns of PCB distribution in the harbor is limited by the relatively sparse spatial distribution.

1.4.2 Alliance Data Base

This previously compiled data base summarizing a number of diverse field investigations in the harbor represented an important source of data and was used extensively to set initial conditions for the model. The data base was originally constructed for EPA by Metcalf & Eddy, Inc. (1983) and was transferred to Alliance in 1986. Alliance began to expand the data base and converted it to run under dBase III, a personal computer data base management software package. This work was never completed, and the data base was subsequently provided to Jordan for their internal use, and to BOS for quality assurance checks and subsequent incorporation into the central New Bedford Harbor data base. The data base used to establish initial conditions for the model was provided to PNL by Jordan.

Several technical difficulties were encountered by PNL in using the Alliance data base in the dBASE III form. The most significant of these was that contaminant data were not indexed fully and consistently or, in some other cases, correctly. Data from the Alliance data base were eventually extracted from ASCII versions of the data base files using a combination of custom-written FORTRAN programs and hand editing at PNL.

1.4.3 NUS Data Base

The NUS data base was provided to PNL in digital form by BOS. The data base was apparently complete and contained data for PCBs expressed as the concentrations of various Aroclors for samples obtained on a regular grid. The GZA data proved to be

valuable because they provided concentration data for the entire study area.

Sediment Data. PCBs detected in sediment from New Bedford Harbor vary both in level and composition. The Aerovox facility and the Cornell Dubilier facility used blends of PCBs (marketed under the trade name "Aroclor") in the manufacture of electronic capacitors from the late 1940s to the late 1970s. Aroclor 1242 was used in substantial quantities in New Bedford until 1971 when Aroclor 1016 was introduced, replacing Aroclor 1242. Aroclors 1254 and 1252 were used in lesser quantities.

The data sets used to establish the initial condition for the modeling included PCB data in a variety of different forms. In some data sets, PCBs were reported as Aroclor 1242, Aroclor 1254, Aroclor 1242/1016, and non-specific PCB. Some samples included data on level-of-chlorination homologs. The desired final measure, total PCB, was obtained for each sample by summing the concentrations of all quantified Aroclors.

When quantitation in the Alliance data base had been performed on a wet-weight basis, a conversion to dry weight was performed using the group-average water content of 55 percent. Data obtained via this conversion were identified as "CDW" in the final data files. Only data with equivalent units of parts per million dry weight (ppm dw), milligrams per kilogram dry weight (mg/kg dw), or the same units in converted dry weight were used.

PCB concentrations in the NUS data base were reported as Aroclor 1242, Aroclor 1248, or Aroclor 1254 in units of micrograms per kilogram (ug/kg), and assumed to be dry weight. Typically, only one or two Aroclor concentrations were summed and converted to units of micrograms per gram (ug/g), equivalent to ppm dw. Some replicate samples occurred in the NUS data base; in these cases, the arithmetic average of the two reported concentrations was used.

The BOS data base reported PCB concentrations by level-of-chlorination homolog in units of ug/g dw. These concentrations were summed to produce an estimate of total PCB concentration.

Values below specified detection limits occurred in all data bases and were used in determining initial conditions; values reported as zero were not used. Data reported below detection limits were assigned a value equal to approximately 0.1 times the specified detection limit of the analytical procedure and were placed in a separate file. When detection limits were not reported, concentrations of zero were assigned values of approximately 0.1 times the lowest reported value. These arbitrary assignments were necessary because the data were later log-transformed and values of zero would have been unacceptable.

The selected and converted sediment PCB concentration data were combined into four files with common formats. Each record in the files contained information on the data source, location, total PCB concentration, units, and the number of samples summed to produce the total concentration. Original units were included in these files, but the units of ppm, ug/g, and mg/kg are numerically equivalent. The below-detection-limit values discussed in the preceding section were segregated to facilitate changes to the assigned values, if necessary.

Standard univariate statistics were calculated for the raw and log-transformed data. The log-transformed data produced near-normal distributions around the mean value for each data set.

Computerized contour plots of the PCB surface sediment concentrations were prepared at PNL using data contained in the New Bedford Harbor data base. These plots were used to estimate PCB exposure point concentrations at various locations within the study area. These concentrations are, therefore, based on both actual data and computerized interpolation of these data.

The metal concentrations used in this report were accessed directly from the New Bedford Harbor data base. These data were collected as part of the Battelle sampling programs and reported in wet weight concentrations. No conversion to equivalent dry weight concentrations were made. The mean metal value represents the mean concentration of only the detected (i.e., greater than the detection limit) samples. Using appropriate longitude and lattitude coordinates, area-specific metals data were obtained and used as exposure point concentrations.

Water Data. PCB concentrations in the water column for the risk assessment were also based on the values used for the physical/chemical transport model. Unlike sediment concentrations, however, the use of initial conditions, *per se*, is not appropriate because preliminary model runs indicated that concentrations in the water column are determined largely by the assigned sediment concentrations following a brief "spin up" period of approximately 90 days simulation. Accordingly, PNL did not determine initial conditions for the water column in a manner similar to that previously described for sediment, but assigned initial conditions that were generally consistent with the field data and then allowed the model to produce its own "starting conditions" based on the assigned sediment concentrations. These starting conditions in the water column were averaged vertically and provided to Jordan along with initial sediment conditions. As with the metals sediment data, metals water data were accessed directly from the Alliance data base.

1.4.4 Other Sources of Data

Additional information used in this risk assessment includes various site investigation reports, the Greater New Bedford Health Effects Study (GNBHES) (MDPH, 1986), the Pilot Study conducted by the U.S. Army Corps of Engineers, and the Damage Assessment Report prepared for National Oceanic and Atmospheric Administration (NOAA) (NOAA, 1986).

2.0 EXPOSURE ASSESSMENT

The purpose of this public health exposure assessment is to identify potential receptors (i.e., individuals or populations) and describe the mechanisms by which persons may be exposed to contaminants at the New Bedford Harbor site. This assessment is based on land-use and demographic information for this area and assumptions regarding the frequency and duration of activities likely to result in contaminant exposure. The demographic, land-use, and exposure information used to complete this section includes the GNBHES (MDPH, 1987), the federal census (U.S. Department of Commerce, 1980), the "Land-use and Point Source Inventory, New Bedford, Massachusetts" (EPA, 1982a), The Damage Assessment Report (NOAA, 1986), and the "New Bedford Harbor Site Visit; Summary Report" (GCA, 1986b).

Although it is not possible to identify specific individuals or determine the exact number of adults and/or children who may be exposed to contaminants in New Bedford Harbor, it is possible through interview, land-use and demographic information to estimate how and to what level of contamination individuals may be exposed. The following section describes possible contaminant exposure in qualitative terms which reflect behavioral patterns and physical and chemical conditions at the site.

2.1 DEMOGRAPHICS

PCB and heavy metal contamination in the Acushnet River is documented from the Wood Street Bridge throughout the harbor and into Buzzards Bay. The primary areas of concern for public health at this site include the Upper Estuary (from the Coggeshall Street Bridge to the Wood Street Bridge) where elevated levels of PCBs (i.e., greater than 4,000 ppm were documented in the sediment, and along the shoreline where access to the river is unrestricted.

The Acushnet River runs through three communities: Fairhaven, New Bedford, and Acushnet, Massachusetts (Figure 2-1). The coastal town of Dartmouth, Massachusetts, is located south of and adjacent to New Bedford, bordering Clarks Cove and Buzzards Bay. These four towns compose the Greater New Bedford Area, and are the focus of this exposure assessment. The inhabitants of these communities were considered most likely to be at potential risk to contaminant exposure due to their proximity to the river and harbor area. The total population of these four communities is 145,605 (Town Census for Acushnet, Fairhaven, and Dartmouth; 1986 Census for the City of New Bedford).

Although any individual within the defined population may potentially be exposed, four groups within the general population were considered more sensitive to environmental contaminant exposure:

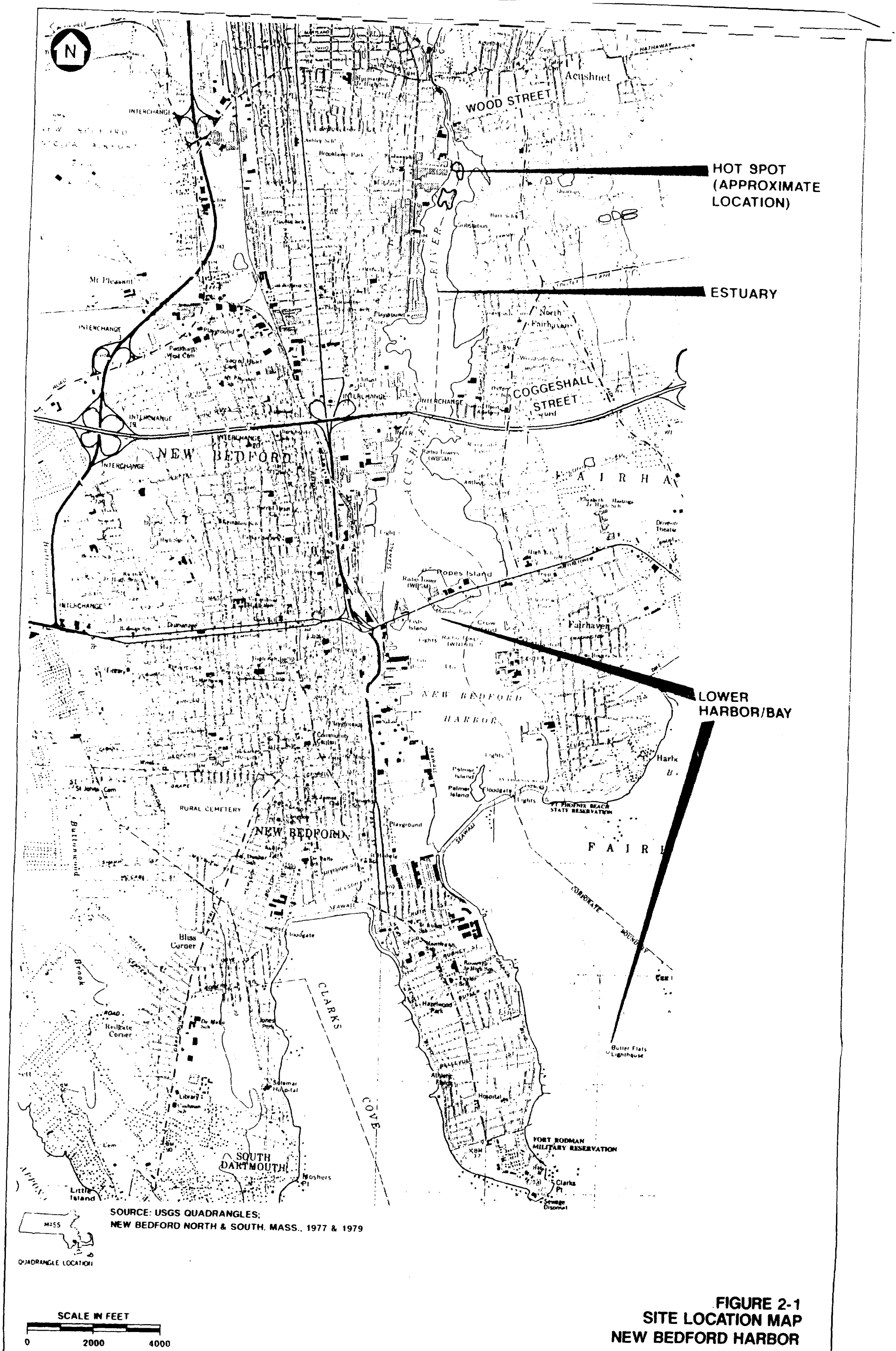


FIGURE 2-1
SITE LOCATION MAP
NEW BEDFORD HARBOR

- Infants and Young Children. Infants and children engage in more activities that could result in contaminant exposure. This subpopulation may be more sensitive to contaminant exposure because of their small body sizes, developing immune systems, and rapid development. These factors effectively reduce their ability to compensate for chemical insult.

- Developing Fetus. The fetus is often considered to be sensitive to chemical exposure because of rapid development, especially during the first trimester. Many environmental contaminants are capable of crossing the placental barrier and potentially interfering with fetal development. Because of its small body size, body weight, and rapid growth, the fetus is particularly sensitive to chemical insult.

- The Elderly. The elderly are considered a sensitive subpopulation because of potentially compromised immune systems and the frequent presence of disease and organ pathology. These conditions may reduce the functional ability to compensate for chemical injury through regeneration or repair of cells, or metabolic detoxification of chemicals.

- Chronically Ill. In addition to the groups discussed previously, there are also individuals in the mainstream population who may be hypersensitive to contaminant exposure because of their immunologic status, presence of disease or specific organ pathology, or medication status.

The 1980 Federal Census provides estimates of the number of infants/children (zero to 5 years), women of childbearing ages (14 to 44 years), and the elderly (older than 65 years) having permanent residence in the Greater New Bedford Area. These subpopulations are indicated by an asterisk in Table 2-1. Assuming that the age distribution within this population has not significantly changed since 1980, these high risk populations account for approximately 50 percent of the total population. Specifically, 7 percent of the people are less than 5 years old, 28 percent are women between 14 and 44 years, and 15 percent are over 65 years.

The group considered at highest risk of direct exposure to sediment within the New Bedford Harbor site area is children between the ages of 6 and 16, since individuals within this age group are most likely to wander and play in areas that may be contaminated, and are least likely to be aware of the potential dangers associated with contaminant exposure. Children younger than 5 years are at risk from contaminant exposure due to small

TABLE 2-1

POPULATION DISTRIBUTION BY AGE GROUP AND SEX FOR THE
GREATER NEW BEDFORD AREA
NEW BEDFORD, MASSACHUSETTS

Age	New Bedford	Acushnet	Dartmouth	Fairhaven	Total
Males					
0-5*	4,268	264	657	466	5,655
6-16*	8,007	873	2,256	1,412	12,548
17-44	17,452	1,708	4,832	2,985	26,977
45-64	10,257	1,014	2,644	1,793	15,708
>65*	5,889	401	1,237	937	8,464
Females					
0-5*	3,941	288	573	429	5,231
6-16*	7,959	780	2,087	1,303	12,129
17-44*	18,782	1,722	5,495	2,942	28,941
45-64	12,181	1,075	2,911	1,966	18,133
>65*	10,007	579	1,781	1,526	13,893
Total					
0-5	8,209	552	1,230	895	10,886
6-16	15,966	1,653	4,343	2,715	24,677
17-44	36,239	3,340	8,929	5,927	55,918
45-64	22,438	2,089	5,555	3,759	33,841
>65*	15,896	980	3,018	2,463	22,357

Source: U.S. Department of Commerce, 1980

Note:

* Indicates subpopulations considered to be more sensitive to contaminant exposure (see text).

body sizes, developing immune systems, and rapid growth and development. However, exposure to contaminants by this age class is expected to be limited, given that children under age 5 are generally supervised and have limited mobility. Therefore, they are unlikely to be playing in areas of high contamination.

Adults (including those older than 65 years) are also expected to have more limited exposure than older children. This age class is considered to be more aware of potential dangers associated with contaminant exposure and is likely to voluntarily restrict access to contaminated areas. However, it is considered likely that persons within this age class may fish or shellfish in contaminated areas.

According to the 1980 census, approximately 90 percent of New Bedford residents reported living in Bristol County during the previous five years, and approximately 60 percent of the population have not changed their residences. This indicates that chronic and/or lifetime contaminant exposure is possible for a large segment of the population.

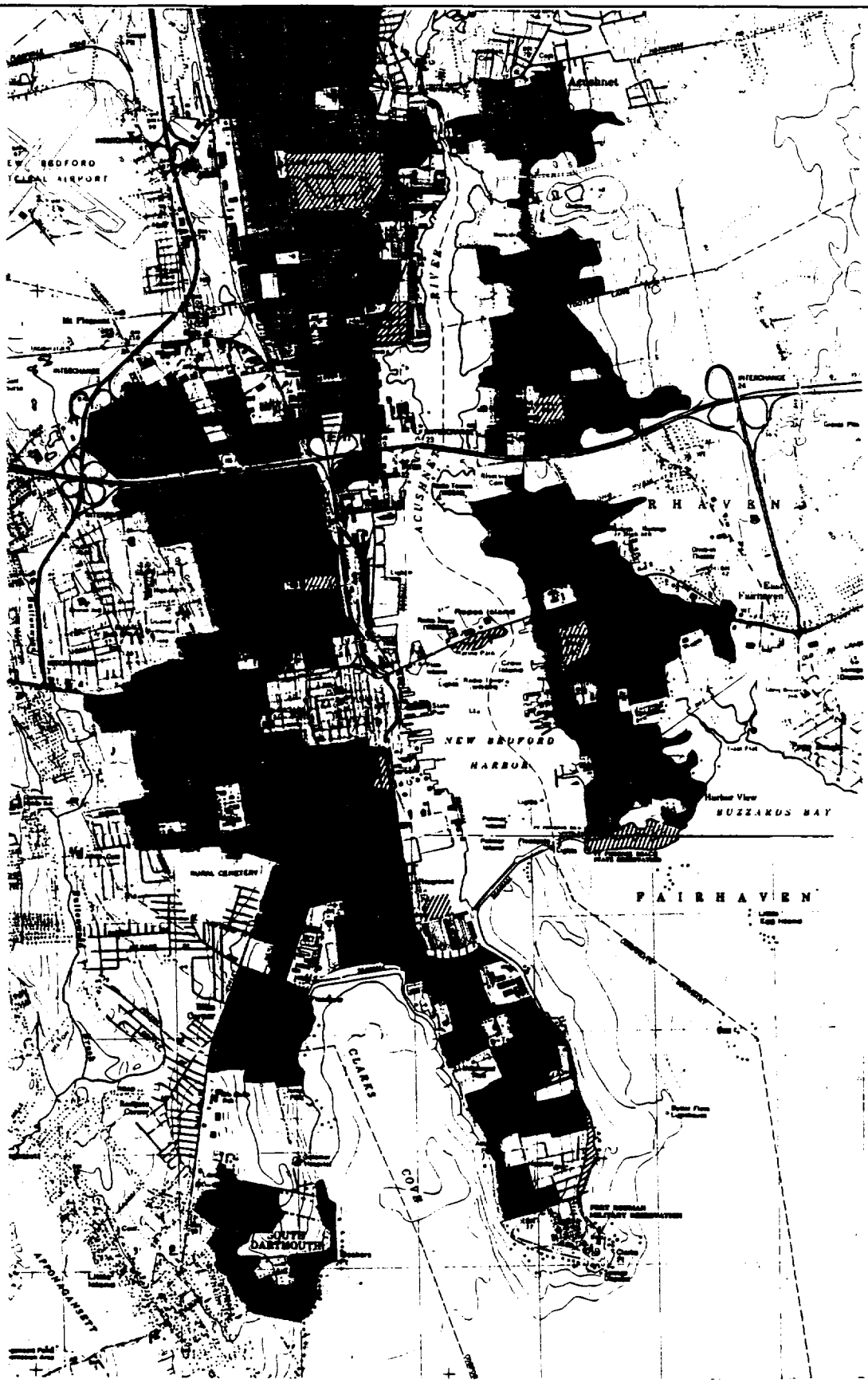
The Greater New Bedford Area experiences a seasonal fluctuation in population. Although this increase cannot be quantified, tourists and summer residents result in a temporary increase in population. Because of recreational activities associated with this area, summer residents and/or tourists have the potential for exposure to contaminants in the New Bedford Harbor Site Area while swimming, fishing, and shellfishing. However, given the temporary residence of this subpopulation, exposure is likely to be sporadic or short-term in duration.

Approximately 50 percent of the New Bedford population is of single Portuguese ancestry and 20 percent is from multiple ancestry (i.e., English, French, German, Irish, Italian, and Polish). Reportedly, 55 percent of the residents speak English, 35 percent Portugese, and 5 percent Spanish (U.S. Department of Commerce, 1980). Where possible, the REM III team has considered cultural differences that may affect exposure to contaminated media.

2.2 LAND-USE WITHIN THE NEW BEDFORD HARBOR SITE AREA

Land-use classifications for the Acushnet River/New Bedford Harbor Site Area include urban (residential and industrial), wetlands, beaches, and barren land; with the majority being classified as urban residential (Figure 2-2) (EPA, 1982a). The land-use information, combined with demographic data, can assist in determining how and where people may become exposed to contaminants.

Figure 2-2 identifies the residential and recreational areas located within an approximate 1-mile radius of the Acushnet



SOURCE: USGS (1990)

LEGEND

RESIDENTIAL AREAS

RECREATIONAL AREAS

0 2000 4000 FEET

FIGURE 2-2
RESIDENTIAL AND RECREATIONAL LAND-USES AROUND THE ACUSHNET RIVER NEW BEDFORD, MASSACHUSETTS

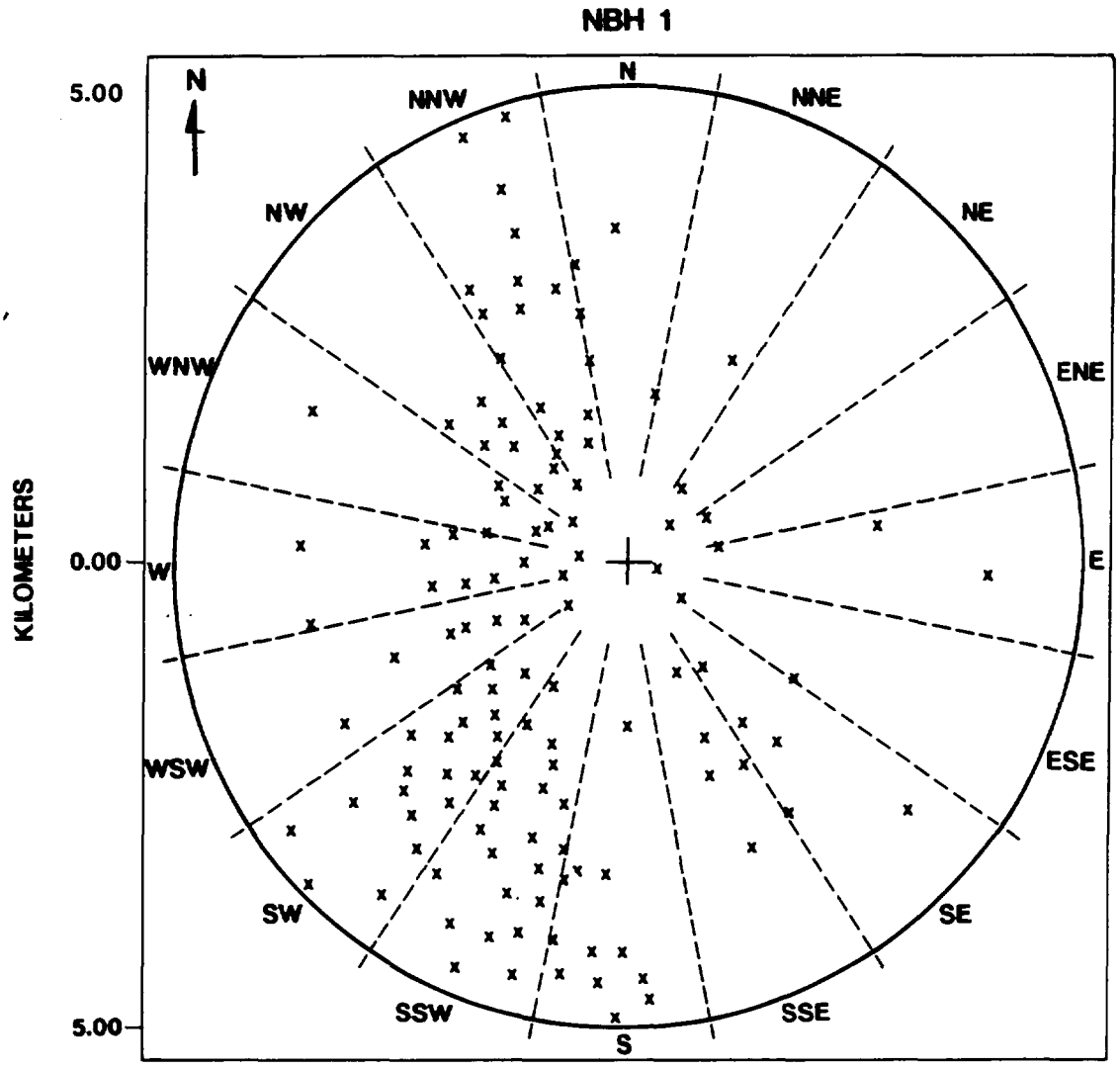
River. Most of the land within this area is used for residential purposes, with a much smaller portion set aside for recreational uses. The land directly adjacent to Acushnet River, on the New Bedford side, is primarily industrial. However, the amount of industrial land-use decreases southward from the harbor toward Buzzards Bay, where public beaches constitute most of the southern shoreline. The land-use on the Fairhaven side of the river is primarily residential. The population residing within a 3-mile radius of the Upper Estuary is estimated at 90,000 (Figure 2-3) (EPA, 1982b).

Recreational and land-use information obtained from NOAA (1986) and the GNBHES (MDPH, 1987) include data on beach use and recreational fishing in the Greater New Bedford area. Surveys conducted by NOAA show that 71 percent of the respondents reported visiting saltwater beaches in the Greater New Bedford area in 1985. Beaches located adjacent to the Acushnet River include the Fort Phoenix State Beach (Fairhaven, Massachusetts) and Fort Rodman/East Beach (New Bedford, Massachusetts). Twenty-three and 18 percent of respondents reported visiting these two locations, respectively (NOAA, 1986).

The NOAA study also reported that 19 percent of respondents fished in the New Bedford area in 1985. Eighty persons indicated having fished in the area north of Ricketson Point or Wilbur Point 14 times on average in 1985. The GNBHES reported that 12.9 percent of the Greater New Bedford population obtain fish by catching it themselves (MDPH, 1987). However, when looking at sources of seafood caught and consumed from contaminated areas, most people (61.5 percent) report they do not consume this seafood. The GNBHES concluded that the majority of the general public was not directly or knowingly catching and consuming fish from contaminated areas (MDPH, 1987). However, the GNBHES identified a small percentage of the population who did report catching and consuming locally caught fish (MDPH, 1986).

In addition to these data, qualitative information describing the Acushnet River and potential activities that may occur at various locations along the shoreline were made by GCA during a site visit to New Bedford Harbor (GCA, 1986b). These observations were limited to one season (late summer) and therefore cannot be considered representative of year-round conditions. However, these observations in conjunction with the GNBHES and NOAA reports, indicate that individuals access the river for various purposes. The major observations are summarized as follows:

Upper Estuary: Acushnet River Between Coggeshall and Wood Street Bridges



SOURCE: GRAPHICAL EXPOSURE MODELING SYSTEM (GEMS)

SECTOR	POPULATION
N	3090
NNE	1236
NE	1101
ENE	379
E	2947
ESE	1948
SE	2839
SSE	3790
S	11064
SSW	17767
SW	16680
WSW	7505
W	8245
WNW	4444
NW	7511
NNW	12906

X = RELATIVE POPULATION DENSITIES

+ = HOT SPOT LOCATION

FIGURE 2-3
POPULATION DISTRIBUTION
AROUND THE UPPER ESTUARY, ACUSHNET RIVER
NEW BEDFORD, MASSACHUSETTS

4959-08

- The New Bedford Harbor side of this section of the river is primarily industrial, while the Fairhaven side is much less commercially developed.

- Access to the river is unrestricted; however, warning signs are posted.

- Swimming is unlikely, although wading in the mudflat areas is possible.

- The Acushnet River is very "dirty" with brown and pungent water, oil stains, and trash.

- An approximate 10-foot width of bottom sediment is exposed at low tide.

- Children were observed in a playground located within 300 feet of the river bank (Cove Area).

Upper Harbor: Coggeshall Street to Fairhaven (Hutchinson Street) Bridge

- The Fairhaven side of this section of the river is less commercially developed than the New Bedford Harbor side.

- Access to the river is unrestricted, and no warning signs were observed.

- Wading and swimming in this section of the river are considered possible.

- The river shows visual signs of pollution (e.g., trash and oil stains).

- A pungent odor from the water was noted and the bottom sediment was exposed at low tide.

Lower Harbor: Fairhaven (Hutchinson Street) Bridge to Hurricane Barrier

- The Fairhaven side of this section of the river is primarily residential. The New Bedford Harbor side is less commercially developed than areas to the north.

- Access to the river is unrestricted along the Fairhaven side. Access along the New Bedford Harbor side is restricted by the presence of fenced private property (i.e., warehouses).

- Wading and swimming in this section of the river seem likely. Persons were observed fishing around the

Hurricane Barrier. Palmer Island can be accessed by foot at low tide.

Entrance to Buzzards Bay: Hurricane Barrier to Fort Rodman

- Fort Phoenix and Fort Rodman State Reservations are located in this section of the river.
- Children and adults were observed fishing, wading, and swimming in this area.
- Both sides of the river are primarily residential with some commercial development around the Hurricane Barrier.
- Fishing, wading, and swimming are likely activities in this area.
- Beaches run along the river bank for most of this area.

Access to the estuary and harbor is unrestricted in most areas, including locations of high contamination. Although warning signs are posted in the Upper Estuary, fishing, wading, and/or playing in this area was observed. However, activities along the shoreline were observed more frequently in the southern portion of Acushnet River near Buzzards Bay. This, in addition to the physical conditions of the Upper Estuary, suggests that exposure to sediment and water will be more common in the southern portion of the Lower Harbor/Bay Area. However, since access to the Upper Estuary is unrestricted, exposure to high levels of contaminated sediment is possible.

Summary. A culturally diverse population resides within the Greater New Bedford Area. A large percentage of residents report living in this area for at least five years. A seasonal influx of summer residents and tourists suggests that short-term or acute exposures to contaminated media may be occurring, in addition to possible chronic exposure experienced by permanent residents.

Activities observed or reported to occur include swimming, wading, fishing, and shellfishing (GCA, 1986b; NOAA, 1986c; MDPH, 1987). The areas of the Acushnet River where recreational activities are considered likely to occur include Palmer Island, Marsh Island, Popes Island, and Fort Rodman and Fort Phoenix State Beaches. These areas are either easily accessible or support organized recreational uses. However, because access to most portions of the Acushnet River is unrestricted, inadvertent contaminant exposure is considered possible for all areas of the river.

2.3 EXTENT OF CONTAMINATION

An extensive data base, containing contaminant concentrations for all media throughout the Acushnet River and Buzzards Bay, was developed and used in this risk assessment to provide exposure concentrations for various receptor locations within the New Bedford Harbor site area (New Bedford Harbor Data Base, 1987). The majority of sample analyses in this data base were obtained between 1981 and 1986 and, therefore, were considered to provide an accurate description of the current extent and level of PCB and metal contamination. This data base was also used to establish initial conditions for the physical/chemical transport model.

The Acushnet River/New Bedford Harbor Site Area was subdivided into three areas to assess sediment and water exposure in this risk assessment:

- Area I: the area between the Wood Street and Coggeshall Street bridges
- Area II: the area between the Hurricane Barrier and Coggeshall Street Bridge
- Area III: the area south of the Hurricane Barrier.

This subdivision, illustrated in Figure 2-4, separates areas of high contamination (i.e., hot spots) from areas of relatively low contamination (south of the Hurricane Barrier), thereby providing a more accurate estimate of exposure concentrations.

Another subdivision of the Acushnet River/New Bedford Harbor Site Area was used to assess exposure through the consumption of aquatic biota. The Acushnet River/Buzzards Bay Area was divided into four areas for purposes of modeling future contaminant concentrations in aquatic biota. Since these estimated concentrations will be used to evaluate future potential risks in this area, this subdivision was used to assess exposure via the ingestion of aquatic biota. These areas are shown in Figure 2-5.

In summary, exposure to sediment and water was assessed for the three areas, referred to by Roman numerals (i.e., I, II, and III), shown in Figure 2-4. Exposure through the ingestion of aquatic biota was assessed for the four areas established by HydroQual, referred to by Arabic numerals (i.e., 1, 2, 3, and 4), and shown in Figure 2-5.

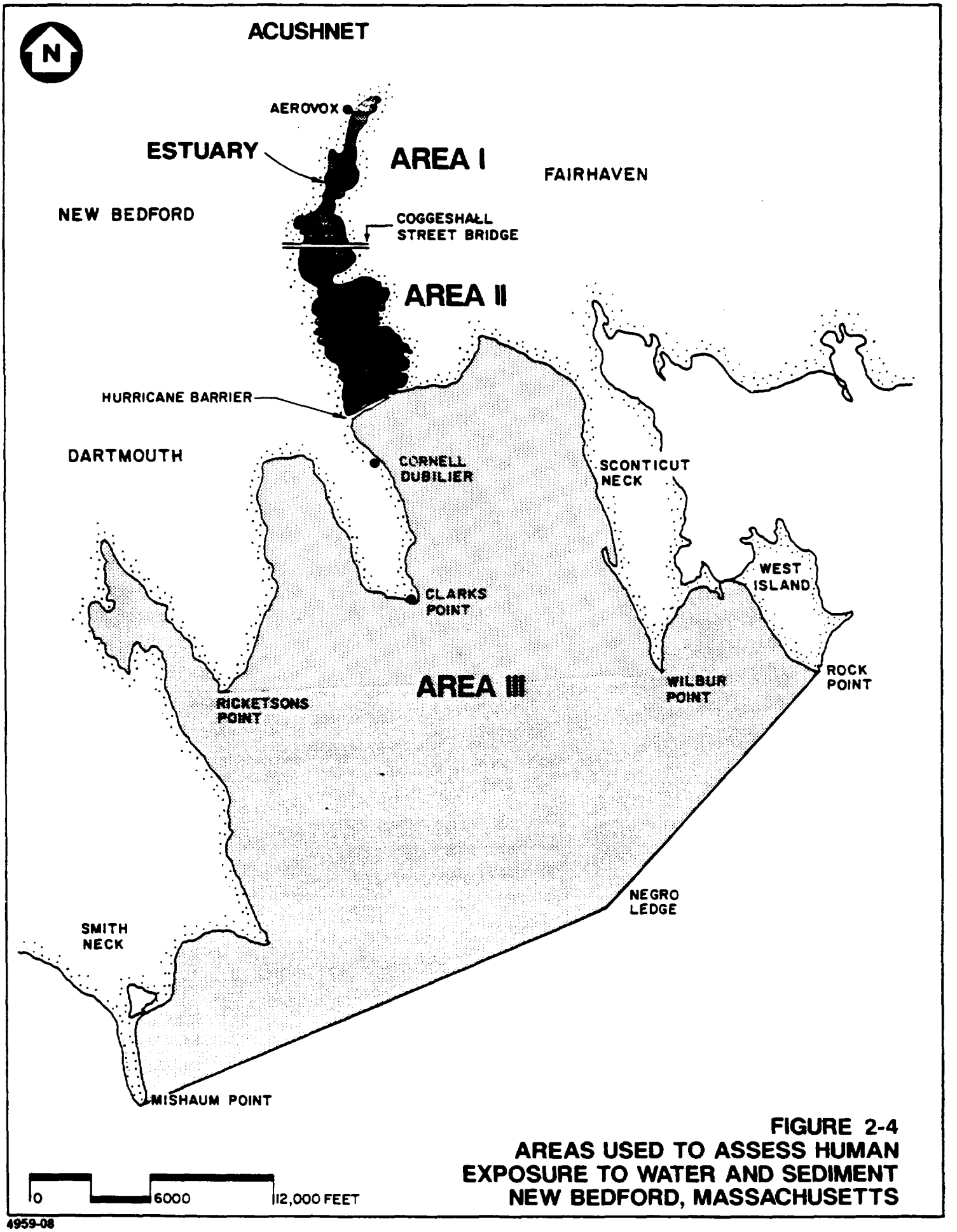


FIGURE 2-4
AREAS USED TO ASSESS HUMAN EXPOSURE TO WATER AND SEDIMENT NEW BEDFORD, MASSACHUSETTS

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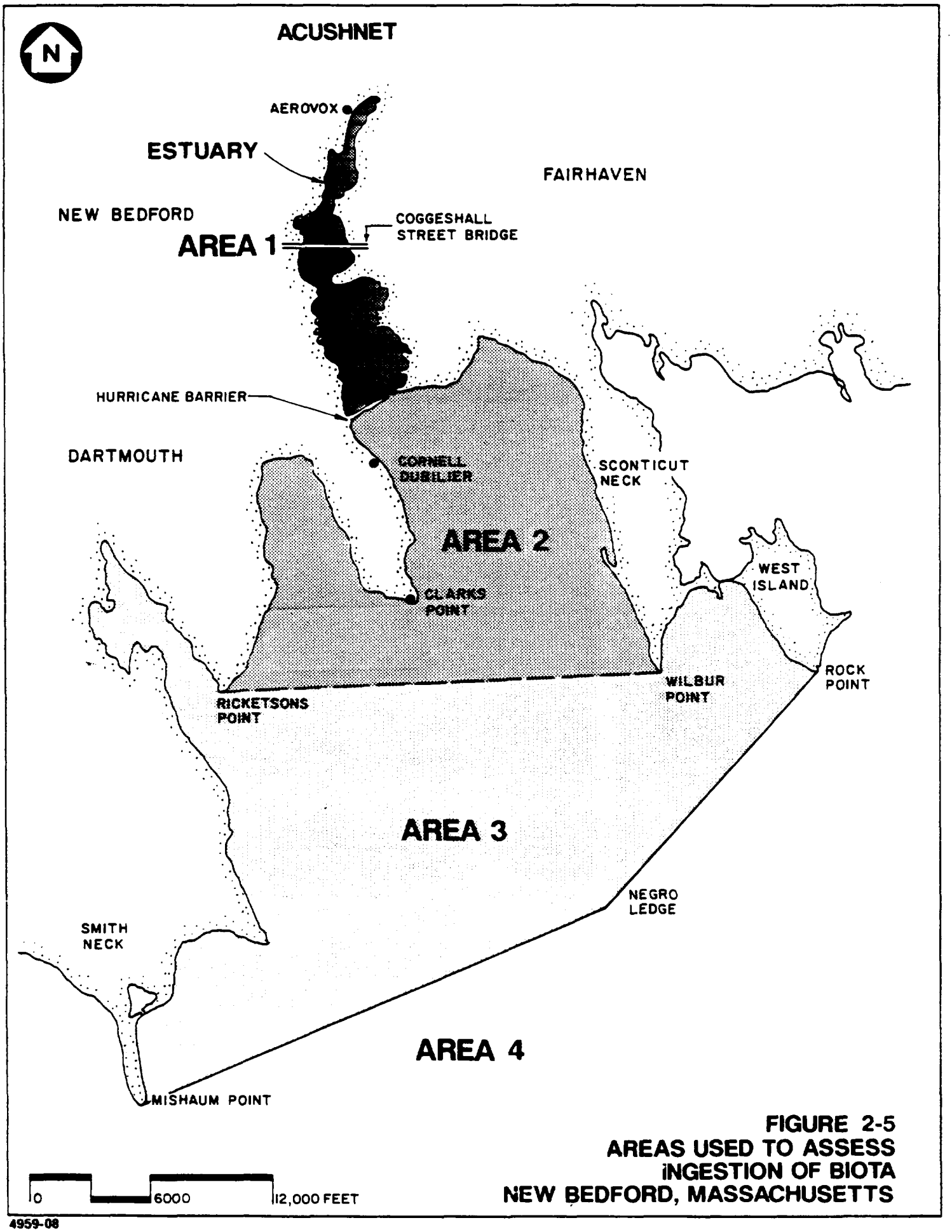

FIGURE 2-5
AREAS USED TO ASSESS
INGESTION OF BIOTA
NEW BEDFORD, MASSACHUSETTS

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2.4 PRINCIPAL EXPOSURE PATHWAYS

Demographic and land-use information indicates a large residential population in the immediate area surrounding the Acushnet River and that people access this site for occupational and recreational purposes. Analytical data for New Bedford Harbor document the presence of elevated levels of PCBs and metals in the sediment, water, biota, and air. Therefore, exposure to contaminants detected in these media is possible through several different pathways, including dermal contact with sediments and water, ingestion of sediment, water, and biota, and/or inhalation of airborne contaminants. To determine the exposure pathways that contribute most significantly to the total contaminant exposures at New Bedford Harbor, a screening evaluation was performed.

The route-specific exposure level (defined as the amount of contaminant taken into the body per unit weight per unit time [mg/kg/day]) attributed to each exposure pathway was determined. These levels were estimated based on extremely conservative exposure assumptions. The route-specific exposure level for each contaminant was estimated assuming the exposure point concentration was the maximum detected concentration of each contaminant. It was also assumed that repetitive exposure, over 70-years duration, occurred at this maximum concentration. The estimated exposure level was then compared to the most appropriate health-based criterion. Exposure pathways were excluded from further consideration only if they contributed a negligible amount to the total exposure dose and if the associated risk was minimal (see Section 4.0). This approach was considered appropriate since the screening evaluation was based on extremely conservative exposure assumptions, with lower exposure levels expected under more realistic exposure conditions.

Exposure to PCBs was evaluated for all routes of exposure. When or if the exposure levels for PCBs were considered insignificant, exposure to cadmium, copper, and lead was then evaluated. This approach prevented the elimination of any route of exposure considered a primary pathway for only one or two contaminants.

Estimated lifetime body doses for the exposure scenarios evaluated in the screening process are in Table 2-2. (The exposure assumptions and body dose calculations appear in Appendix A, Tables A-1 through A-6.)

Based on the screening results, direct contact with sediment, ingestion of aquatic biota and sediment, and inhalation of airborne contaminants were all considered to significantly contribute to the total PCB exposure at the New Bedford Harbor

TABLE 2-2

ESTIMATED LIFETIME BODY DOSES FOR SCREENING SCENARIOS
EXPOSURE TO PCBs
NEW BEDFORD, MASSACHUSETTS

Pathway of Exposure	Exposed Population	Average Daily Dose for PCBs (mg/kg-day)	Percent Contribution to Total Dose[a]	Principal Pathway
Ingestion of Aquatic Biota	Older Child (6-16)	9.5×10^{-4}	1.4	Yes
Direct Contact with Sediments	Older Child (6-16)	5.7×10^{-2}	84	Yes
Direct Contact with Surface Water	Older Child (6-16)	5.3×10^{-7}	7.8×10^{-4}	No
Ingestion of Surface Water	Older Child (6-16)	3.4×10^{-6}	5.0×10^{-3}	No
Inhalation of Airborne Contaminants[b]	Child (0-5)	1.7×10^{-5}	0.025	Yes
Ingestion of Sediments	Child (0-5)	1.0×10^{-2}	14.7	Yes
Total Dose		6.8×10^{-2}	100	

[a] The percent contribution was calculated by: $\frac{\text{Average Daily Dose}}{\text{Total Dose}} \times 100$. It provides a relative measure of exposure.

[b] The maximum concentration was assumed to represent the contaminant in the vapor phase.

NOTE:

The average daily dose was estimated based on conservative exposure assumptions, including repetitive exposure to the maximum detected contaminant concentration. The age-class chosen for each pathway of exposure was that considered most likely to be at risk from exposure due either to low body weight or higher frequency of exposure. For example, exposure to children ages 0 to 6 was evaluated because it is possible that this age class could be exposed 24 hours/day. The low body weight of children puts them at greater potential risk to PCB exposure than older children and adults exposed 24 hours/day with a higher body weight. These screening scenarios represent the upper bound, conservative estimate of potential risk.

The Average Daily Dose values in this table were used to screen exposure pathways and not forthe risk assessment presented in Chapter 4.0.

site. These four exposure pathways result in more than 99 percent of the total exposure and, therefore, were assumed to account for the majority of risk at the site. The screening results also show that direct contact with and/or the incidental ingestion of surface water does not result in a significant contaminant exposure. These exposure routes account for 0.001 percent of the PCB exposure.

Exposure dose levels for metals were estimated for direct contact with and incidental ingestion of surface water because these routes of exposure were considered insignificant for PCB exposure. The exposure dose levels for metals estimated under the same conservative assumptions were also insignificant. Because these scenarios were based on conservative exposure assumptions, lower exposure levels would be expected under more realistic exposure conditions. Therefore, exposure to contaminants through incidental ingestion of and direct contact with surface water was not evaluated further in this risk assessment. In summary, PCB and metals exposures for direct contact with and ingestion of sediment, ingestion of biota, and inhalation of airborne contaminants were carried through the analysis for quantitative evaluation.

2.5 QUANTITATIVE EXPOSURE ASSESSMENT

In this section, the equations used to calculate the route-specific exposure level for the principal exposure pathways are described. In addition, the exposure parameters used in these equations are identified and discussed. Values for these exposure parameters were chosen by the REM III team based on site-specific factors and realistic exposure considerations. For example, sediment deposition factors were chosen to reflect sediment characteristics, and site-specific weather conditions were considered in developing exposure frequencies. In addition, location-specific exposure concentrations were used that allowed exposure to be evaluated separately for areas of high contamination and areas of low contamination. This provided a realistic range of exposure parameters which reflected the exposure conditions in this area. Exposure parameters were obtained from the scientific literature and appear in the tables in Appendix C.

To provide a range of exposure doses, two exposure scenarios were considered in each analysis: one based on "average" or probable or moderate exposure conditions, and the other based on "conservative" exposure conditions. Together, these scenarios provide a range of potential exposure levels, within which the actual exposure for a particular individual would likely fall. Figure 2-6 is an overview of the exposure scenarios evaluated in this section.

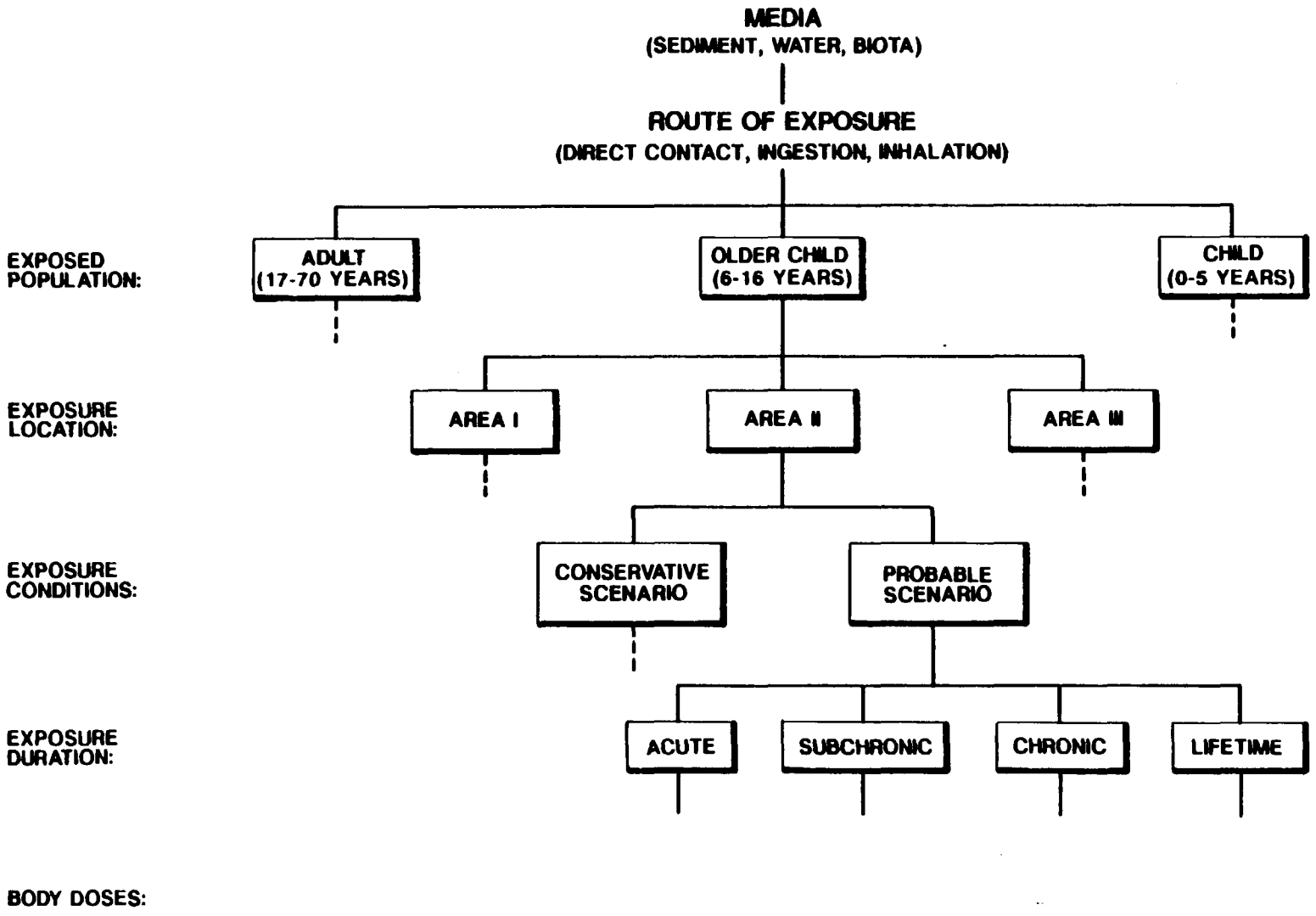

FIGURE 2-6
FLOW CHART OF
EXPOSURE SCENARIOS EVALUATED
NEW BEDFORD, MASSACHUSETTS

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The exposure scenarios evaluated in this report provide a range of possible exposure doses for a "hypothetical individual," rather than for a specific population. These scenarios do not predict the number of people who may be exposed to contaminants in the Greater New Bedford Area, but rather provide an estimate of the magnitude of exposure that could be incurred by an individual receptor under specified exposure conditions.

Exposure to each medium is discussed generally in subsequent subsections, followed by a quantitative exposure analysis for each scenario under review. The equations used to estimate systemic contaminant doses from the various exposure routes are in Table 2-3. The exposure parameters identified in these equations are summarized in Table 2-4, as well as in the text.

2.5.1 Sediment

For sediment, possible exposure pathways include two exposure scenarios: (1) direct contact exposure to sediment, and (2) ingestion of sediment.

Ingestion of sediment is considered limited to children younger than 6 years, while direct contact with sediment is possible for all age groups. Because different exposure parameters govern these two exposure pathways, separate evaluations were performed.

2.5.1.1 Direct Contact Exposure to Sediment

Land-use around the study area and results from NOAA (1986), indicate that the local population uses the beaches along the Acushnet River for recreational purposes. Therefore, persons of all ages may be exposed to contaminated sediment as a result of swimming, wading, and/or fishing in the Acushnet River. The most likely locations for these activities to occur are south of the Coggeshall Street Bridge (Areas II and III). However, because access to the river is not restricted, exposure to sediment in Area I is possible, and therefore, was evaluated in this section.

Direct contact exposure to sediment was assessed separately for Areas I, II, and III. Because of the wide range of PCB contaminant concentrations in Area I (ND to 6,393 ppm), separate exposure scenarios were developed for the Cove Area, and the Upper and Lower Estuary (Figure 2-7). For Areas II and III, exposure was evaluated at specific locations that support recreational activities; these included Popes Island, Palmer Island, and Marsh Island for Area II; and The Fort Rodman and Fort Phoenix state beaches for Area III.

The contaminant concentrations detected or estimated through computer interpolation in the shoreline sediment were used to

TABLE 2-3

EQUATIONS USED TO ESTIMATE SYSTEMIC CONTAMINANT DOSES
NEW BEDFORD, MASSACHUSETTS

Exposure Via Direct Contact:

$$DEX_{DC} = \frac{C_A \times SA \times DF \times TKF \times F \times CF}{BW}$$

Exposure Via Ingestion:

$$DEX_{ING} = \frac{C_A \times Q \times TKF \times F \times CF}{BW}$$

Exposure Via Inhalation:

$$DEX_{INH} = \frac{C_A \times IR \times TKF \times F}{BW}$$

For carcinogens, the average daily exposure over a lifetime is calculated by multiplying DEX by the duration of exposure (D = years) divided by 70-year lifetime.

where:

DEX = Average Daily Exposure Over Period of Exposure (mg/kg-day)

C_A = Contaminant Concentration Detected in Area A (mg/kg, mg/L or mg/m^3)

SA = Exposed Surface Area (cm^2)

DF = Sediment Deposition Factor (mg/cm^2-event)

Q = Quantity of Sediment Ingested (mg/exposure)

IR = Inhalation Rate (m^3/day)

BW = Body Weight (kg)

TKF = Toxicokinetic Factor (unitless)

F = Frequency of Exposure (events/exposure period (days))

D = Duration of Exposure (years)

CF = Correction Factor (1 kg/10^6 mg)

TABLE 2-4

AGE-SPECIFIC EXPOSURE PARAMETERS USED TO ESTIMATE AVERAGE DAILY EXPOSURE DOSES
NEW BEDFORD, MASSACHUSETTS

	Age Category		
	0-5	6-16	17-65
Body Weight[1]	10 kg	40 kg	70 kg
Surface Area Total[1]	6,880 cm^2	11,900 cm^2	18,000 cm^2
Legs and Feet[1]	2,280 cm^2	4,400 cm^2	3,060 cm^2 (lower legs only)
Forearms, Arms, Hands, Lower Legs and Feet[1]	2,525 cm^2	4,415 cm^2	4,990 cm^2
Sediment Deposition[2] Factors	1.5 mg/cm^2	1.5 mg/cm^2	1.5 mg/cm^2
Sediment Ingestion Rates[3]	0.5 grams	N/A	N/A
Inhalation Rates[4]	5 m^3/day	20 m^3/day	20 m^3/day
Biota Ingestion Rates	115 grams/per meal	227 grams/per meal	227 grams/per meal
Toxicokinetic Factor[5]			
Dermal-PCBs	0.07	0.07	0.07
-Metals	0.01-0.001	0.01-0.001	0.01-0.001
Inhalation-PCBs	1.0	1.0	1.0
-Metals	1.0	1.0	1.0
Gastrointestinal-PCBs	1.0	1.0	1.0
-Metals	1.0	1.0	1.0

Notes:

[1] USEPA, 1985
[2] USEPA, 1984
[3] LaGoy, 1987
[4] USEPA, 1986
[5] See Appendix B
N/A = Not Applicable

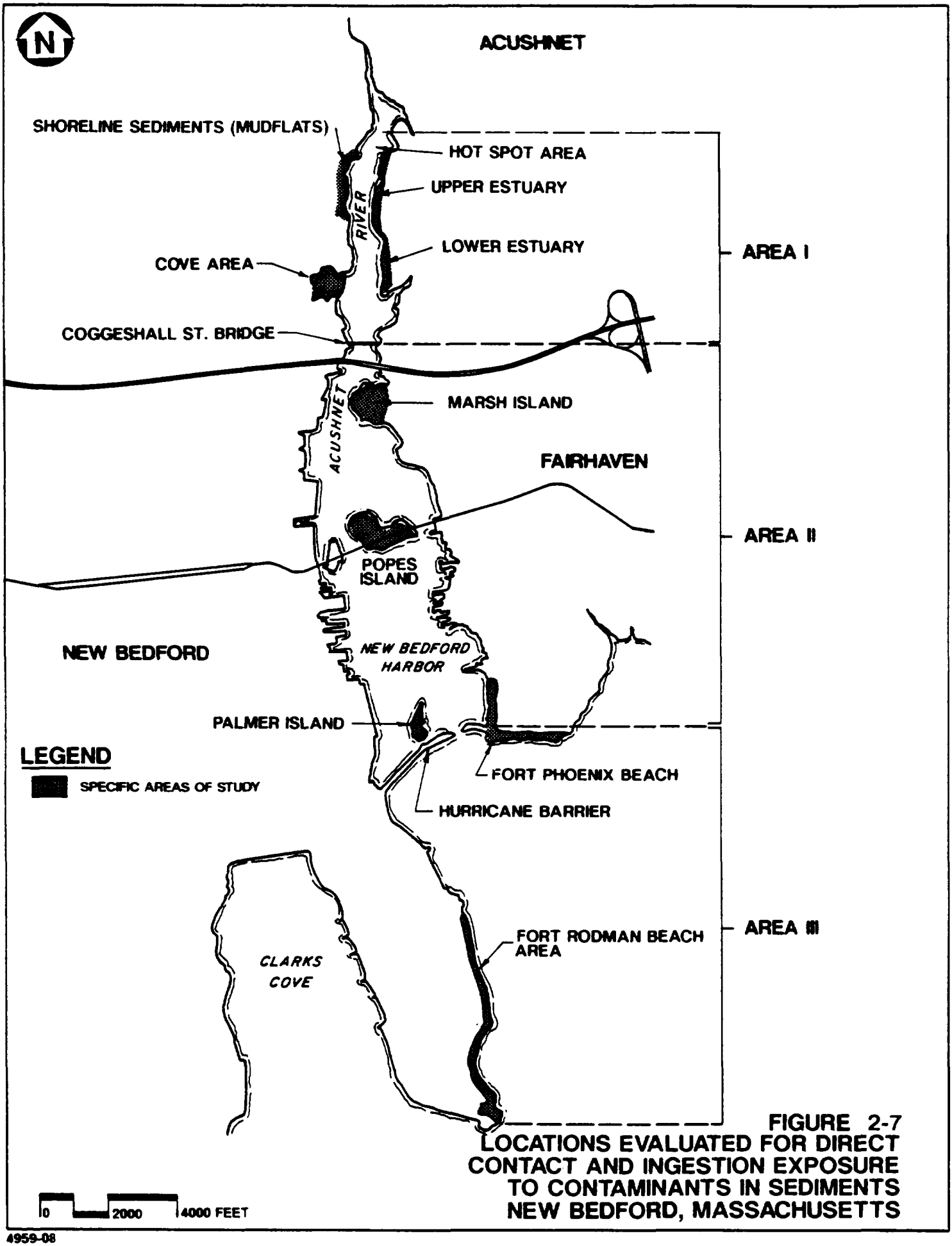

FIGURE 2-7
LOCATIONS EVALUATED FOR DIRECT CONTACT AND INGESTION EXPOSURE TO CONTAMINANTS IN SEDIMENTS NEW BEDFORD, MASSACHUSETTS

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evaluate direct contact exposure when available. (These data are contained in the New Bedford Harbor Data Base and the Administrative Record.) Midchannel sediment concentrations were not included because exposure to this sediment is considered unlikely. In general, midchannel sediment was more contaminated than shoreline sediment. The geometric mean and the maximum PCB concentrations were used to evaluate exposure under probable and conservative exposure conditions, respectively. The arithmetic mean and maximum metal concentrations were used to evaluate exposure under probable and conservative exposure conditions, respectively. (Data were not available to determine the geometric mean concentrations for metals.) Table 2-5 presents the mean and maximum sediment concentrations used to assess direct contact and ingestion exposures.

It was assumed that young children would only be exposed to sediment while playing or swimming at the beach. The frequency of exposure (e.g., the number of trips to the beach per year) was estimated to range between 20 and 100 times per year, which corresponds to one and five days per week during the warmer months.

Although children in this age class are not expected to have access to nonbeach areas of the New Bedford Harbor site, a subsection of Area I (i.e., Cove Area) is located next to a playground and represents a specific area where children may access the shoreline. Because inadvertent exposure is possible, exposure scenarios were developed for this area. The frequency of exposure in this location was considered less than in the beach area, and was estimated to range from 1 to 20 exposures per year.

An older child or adult was assumed to have access to all areas (i.e., Areas I, II, and III) of the New Bedford Harbor site and contact with sediment as a result of swimming, wading, or shellfishing activities. The frequency of contact was estimated to be between 20 and 100 times per year. This range represents exposures occurring one and five days per week during the warmer summer months. Body weights of 10, 40, and 70 kilograms were assumed for children, older children, and adults, respectively (EPA, 1985a).

Exposure was also evaluated assuming acute (single event), subchronic (1- to 5-year), chronic (10-year), and lifetime exposure durations. Lifetime exposure was assessed by summing the exposure dose received during each age period (i.e., zero to 5, 6 to 16, and 17 to 70). These exposure durations were chosen to reflect likely exposure periods for the Greater New Bedford Area population.

TABLE 2-5

PCB and METALS SEDIMENT CONCENTRATIONS (ppm) USED
TO ASSESS DIRECT CONTACT AND INGESTION EXPOSURES
NEW BEDFORD, MASSACHUSETTS

	PCBs		Cadmium		Copper		Lead	
	Mean	Maximum	Mean	Maximum	Mean	Maximum	Mean	Maximum
Area I								
Shoreline Concentrations								
Entire Area	378	6,393	19.2	69	591	3,180	384	1,680
Upper Estuary	378	6,393	18.8	69	588	1,900	445	1,680
Lower Estuary	149	399	20	63	598	3,180	278	1,330
Cove Area	286	399	19.8	48	915	3,180	393	1,330
Area II								
Shoreline Concentrations								
Entire Area	21	125	7.6	14	570	2,790	160	559
Palmer Island	3	11	ND	ND	310	310	139	139
Popes Island	11	34	ND	ND	492	771	156	272
Marsh Island	8	22	ND	ND	300	463	191	323
Area III								
Shoreline Concentrations								
Entire Area	4	29	ND	ND	94	154	55	106
Fort Rodman Beach Area	2	7	NA	NA	NA	NA	NA	NA
Fort Phoenix Beach Area	0.59	0.75	NA	NA	NA	NA	NA	NA

Notes:

Mean concentration for PCBs represents the geometric mean value. The mean concentration for metals represents the arithmetic mean value of the concentrations detected in each area.
Maximum concentration represents the maximum value detected in each area.
NA = Not Available; shoreline sediment data for metals was unavailable.
ND = Not Detected.

3.88.80
0010.0.0

The amount of sediment contacted per exposure event was estimated based on the exposed surface area and the deposition of sediment onto the skin. For wading and swimming activities, the exposed surface area was considered to be the lower legs; for shellfishing activities, both the lower legs and forearms were considered. Surface areas for these body parts were obtained from EPA and are in Table 2-4 (EPA, 1985a). The sediment deposition factor was estimated to be 1.5 mg/cm^2, which represents the upper end of the soil deposition range used by EPA to assess contact with soil (EPA, 1984a). This value was considered appropriate for assessing sediment exposure, given that sediment tends to adhere more to exposed skin than soil. The sediment deposition factor multiplied by the surface area equals the amount of contaminated sediment contacted per exposure event.

The toxicokinetic factor (TKF) is the final parameter necessary to assess direct contact exposure. This factor adjusts for the differences in absorption between the dermally absorbed dose received from exposure to sediment at the site, and the administered dose of the laboratory test from which the cancer potency factor or reference dose was derived. This adjustment allows quantitative dose-response data from animal studies to be applied to human exposure doses. Jordan derived two TKFs for dermal exposure to PCBs. A TKF of 0.5 (50 percent) was used to estimate exposure to highly contaminated sediment (i.e., PCB concentrations greater than 1 percent); 0.07 (7 percent) was used to assess exposure to moderately contaminated sediment (i.e., PCB concentrations less than 1 percent). The TKF and the basis for its development are discussed in Appendix B. The parameters used to assess direct contact exposure appear in Table 2-6; body dose calculations are in Appendix C.

2.5.1.2 Ingestion of Sediment

Exposure to contaminants can also result from the inadvertent or incidental ingestion of sediment deposited on the hands, food items, or objects placed in the mouth. This route of exposure is expected to be most significant for children less than 6 years old. Young children in this age group engage in substantial hand to mouth activities that can result in incidental soil ingestion. Therefore, this route of exposure is expected to be most significant at locations where children play. For the New Bedford Harbor Site Area, these include the public beaches in Area III and recreational areas located in Area II. Because recreational areas in Area I abut the shoreline (Cove Area), exposure via the ingestion of sediment at these locations is considered possible. The high concentration detected in this sediment suggests that even minimal exposure may be significant. Therefore, exposure scenarios were developed to assess incidental ingestion of contaminated sediment from Area I and the recreational and beach areas in Areas II and III (see Figure 2-7).

TABLE 2-6

EXPOSURE PARAMETERS USED TO ASSESS DIRECT CONTACT EXPOSURE TO SEDIMENTS (SUBCHRONIC, CHRONIC, AND LIFETIME EXPOSURES) NEW BEDFORD, MASSACHUSETTS

Exposure Parameter	Child	Older Child	Adult
Average Weight over Period of Exposure	10 kg	40 kg	70 kg
Frequency of Exposure			
Area I			
Probable	1 exp/year	20 exp/year	20 exp/year
Conservative	20 exp/year	100 exp/year	100 exp/year
Area II and III			
Probable	20 exp/year	20 exp/year	20 exp/year
Conservative	100 exp/year	100 exp/year	100 exp/year
Amount of Sediment Contacted			
Probable	Legs and Feet*	Legs and Feet*	Lower Legs and Feet*
Conservative	Forearms, Arms, Hands, Lower Legs and Feet*	Forearms, Arms, Hands, Lower Legs and Feet*	Forearms, Arms, Hands, Lower Legs and Feet*
Dermal Toxicokinetic Factor			
Concentrations <10,000 ppm	7%	7%	7%
Concentrations >10,000 ppm	50%	50%	50%
Duration of Exposure	1 year 5 years	1 year 10 years	1 year 10 years Lifetime

Note:

*See Table 2-4

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A review of the literature indicates that between 100 to 500 mg of sediment per exposure is a reasonable estimate for sediment ingestion by children less than 5 years old (LaGoy, 1987). Recent EPA guidance suggests an ingestion rate of 200 mg/day be applied to exposures concerning children between the ages of 2-6 years (EPA, 1989). In this risk assessment, a value of 500 mg/exposure was assumed as the amount of sediment ingested. This is the upper end of the range of estimated values and will provide a conservative estimate of exposure. The frequency of exposure is assumed to be 1 to 20 days for Area I, and 20 to 100 days per year for Areas II and III. (These are the same frequencies used to assess direct contact exposure.)

The mean and maximum sediment concentrations detected in each area are used in the probable and conservative scenarios, respectively (see Table 2-5). The exposure assumptions used to assess this route of exposure are in Table 2-7; body dose calculations are in Appendix C.

2.5.2 Biota

Exposure to contaminants through ingestion of aquatic biota is considered a primary route of exposure for this area. Aquatic biota are known to bioaccumulate and bioconcentrate PCBs. Therefore, organisms living in contaminated areas may be a direct source of PCB exposure if consumed or contribute to PCB contamination of higher trophic level organism within the food chain. Studies conducted in New Bedford Harbor show elevated levels of PCBs in edible tissue of lobsters, clams, and winter flounder. In general, seafood consumption has been noted as a primary source of PCB exposure in the areas of the U.S. where PCB-contaminated sediment has been observed (ATSDR, 1987).

The FDA identified a number of species likely to have PCB residue if taken from contaminated areas (ATSDR, 1987). These species are listed in Table 2-8, along with the fraction of people participating in the GNBHES who reported consuming these locally caught species (MDPH, 1987). Based on this summary, ingestion of winter flounder, lobster, and softshell clam was considered in this exposure assessment. (Recent analytical data [post-1984] was not available to assess exposure to eel, striped bass, or mackerel.) In addition, exposure to metals via consumption of biota was assessed.

Ingestion of biota was assessed separately for each age class: children, older children, and adults. Body weights of 10, 40, and 70 kilograms were assumed for children, older children, and adults, respectively (EPA, 1985a).

A standard 8-ounce (i.e., 227 grams) portion of fish per meal for older children and adults, and 4 ounces (i.e., 115 grams)

TABLE 2-7

EXPOSURE ASSUMPTIONS USED TO ASSESS INGESTION OF SEDIMENTS
NEW BEDFORD, MASSACHUSETTS

Exposure Parameter	Value
Average Weight Over Period of Exposure	10 kg
Duration of Exposure	5 years
Exposure Locations	Cove Area and upper and lower estuary in Area I; Recreational and Beach Areas in II and III
Frequency of Exposure	
Area I:	
Most Probable	1 exp/year
Conservative	20 exp/year
Areas II and III:	
Most Probable	20 exp/year
Conservative	100 exp/year
Amount Ingested	0.5 grams/exposure
Gastrointestinal Toxicokinetic Factor	1.0
Contaminant Concentrations	See Table 2-5

TABLE 2-8

SEAFOOD CONSUMPTION BY SPECIES
NEW BEDFORD, MASSACHUSETTS

	Percent Consuming Various Species	
	Prevalence[1] n=840	Enrichment[2] n=110
Fish/Shellfish	Local Fish	Local Fish
Clams, quahogs	23.3	70.9
Mussels	2.0	19.1
Eel	1.9	24.5
Bluefish/Striped Bass/Mackerel	13.4	70.0
Scup, tautog, fluke flounder, cod, or sea trout	17.1	59.1
Lobster	13.0	62.7

Notes:

* Self-reported consumption.

Source: The Greater New Bedford PCB Health Effects Study (1984-1987) (MDPH, 1987).

n = number of respondents

[1] Prevalence = The cross-sectional randomly sampled group of residents of Greater New Bedford participating in this study.

[2] Enrichment = The recruited group of residents considered to be at greater risk of exposure participating in this study.

per meal for younger children, was assumed. These values were decided after a review of the literature failed to provide a site-specific value applicable to recreational consumption of fish and shellfish.

Examination of the different sources of data shows that a variety of definitions have been used for "fish consumption." Some studies examine only commercially-caught fish and others do not distinguish between consumption of marine versus freshwater fish, or between finfish and shellfish. Finally, some do not differentiate between consumption of fresh fish versus processed (frozen, canned,smoked, etc.) fish. Thus, it is difficult to draw meaningful comparisons among the various fish consumption values derived from studies or sources (Environ, 1985).

Values cited in the literature range from 6.5 g fish/day used by EPA in its Ambient Water Quality Criteria to 18.7 g fish/day cited by Cordel et al. (1978). (These values correspond to 10.5 and 30 8-ounce fish meals per year, respectively.) The Environ (1985) report discusses the limitations of these values and recommends using 14 g fish/day (22.5 8-ounce fish meals per year) as a reasonable average daily fish consumption by freshwater recreational fishermen. Since there was no widely accepted value for recreational fish and shellfish consumption, the REM team chose to use 8 ounces (i.e., 227 grams) as a standard value for each fish meal, and vary the number of fish meals consumed per year to provide a range of exposure frequencies. The uncertainty associated with the 227- or 115-gram value is well within the ranges of uncertainty for other exposure parameters, indicating that the use of other values would not affect the overall uncertainty of the risk estimated for this route of exposure.

Exposure frequencies of one fish meal per day, per week, and per month were evaluated in this risk assessment because this range reflects reasonable exposure frequencies for both tourists (short-term exposure) and residents (chronic and lifetime exposure). Information on local seafood consumption was reported in GNBHES (MDPH, 1987). The majority of persons eating locally caught lobster reported a frequency of consumption of "less than once/month, at least once/year." However, some people reported consumption frequencies of "two or more times/week." (These data are presented in Table 2-9.) The range of consumption frequencies used in this report were based on likely consumption values of the local population. Acute exposure via ingestion of biota was evaluated to reflect the exposure frequency of less than one fish meal per month, and chronic exposure via ingestion of biota was evaluated to reflect exposure frequencies of greater than one fish meal per month.

Edible-tissue PCB concentrations were used when available, or estimated from the data using an edible tissue:whole body ratio

TABLE 2-9

LOCAL SEAFOOD CONSUMPTION FOR
NEW BEDFORD, MASSACHUSETTS

	Number of Persons Reporting			
PCB Blood Serum Level Range[1]:	0.5 - 2.68 n = 212	2.69 - 3.93 n = 209	3.94 - 6.84 n = 210	6.85 - 60.92 n = 209
FREQUENCY OF EATING LOCAL LOBSTER				
Two or more times/week	2	2	3	1
At least once/week	1	4	7	1
Less than once/week, at least once/month	6	7	13	7
Less than once/month, at least once/year	12	21	11	9
Less than once/year	--	1	--	1

[1] PCB concentrations reported in ppb

Source: MDPH, 1987; Tables 15 and 16

developed by Battelle Ocean Sciences (BOS, 1987). (The edible portion excludes inedible bones, scales, and viscera.) Edible tissue:whole body ratios for metals were not available for any of the species. Therefore, whole-body concentrations were used to assess exposure to metals.

The edible tissue:whole body ratio developed by BOS for the lobster did not include the tomalley (i.e., hepatopancreas) as part of the edible tissue. Since the tomalley is part of the lobster's digestive system and tends to accumulate PCBs, excluding this as part of the ratio underestimates the actual exposure concentration for those persons who consume lobster tomalley.

Inclusion of the tomalley is required by the FDA for calculation of compliance with its tolerance limit of 2 ppm. Analyses which include the tomalley have been prepared from 1981 through 1987 for Area III lobsters and show PCB levels in excess of 2 ppm, with no evidence of decline (see Figure 2-8). Analyses performed by EPA in 1987 included separate analyses of the tomalley and the muscle tissue, and provide data to calculate a ratio of these weights. Using this study, it can be shown that some lobsters analyzed by Battelle (Duxbury) would have exceeded the FDA limit had the tomalley been included.

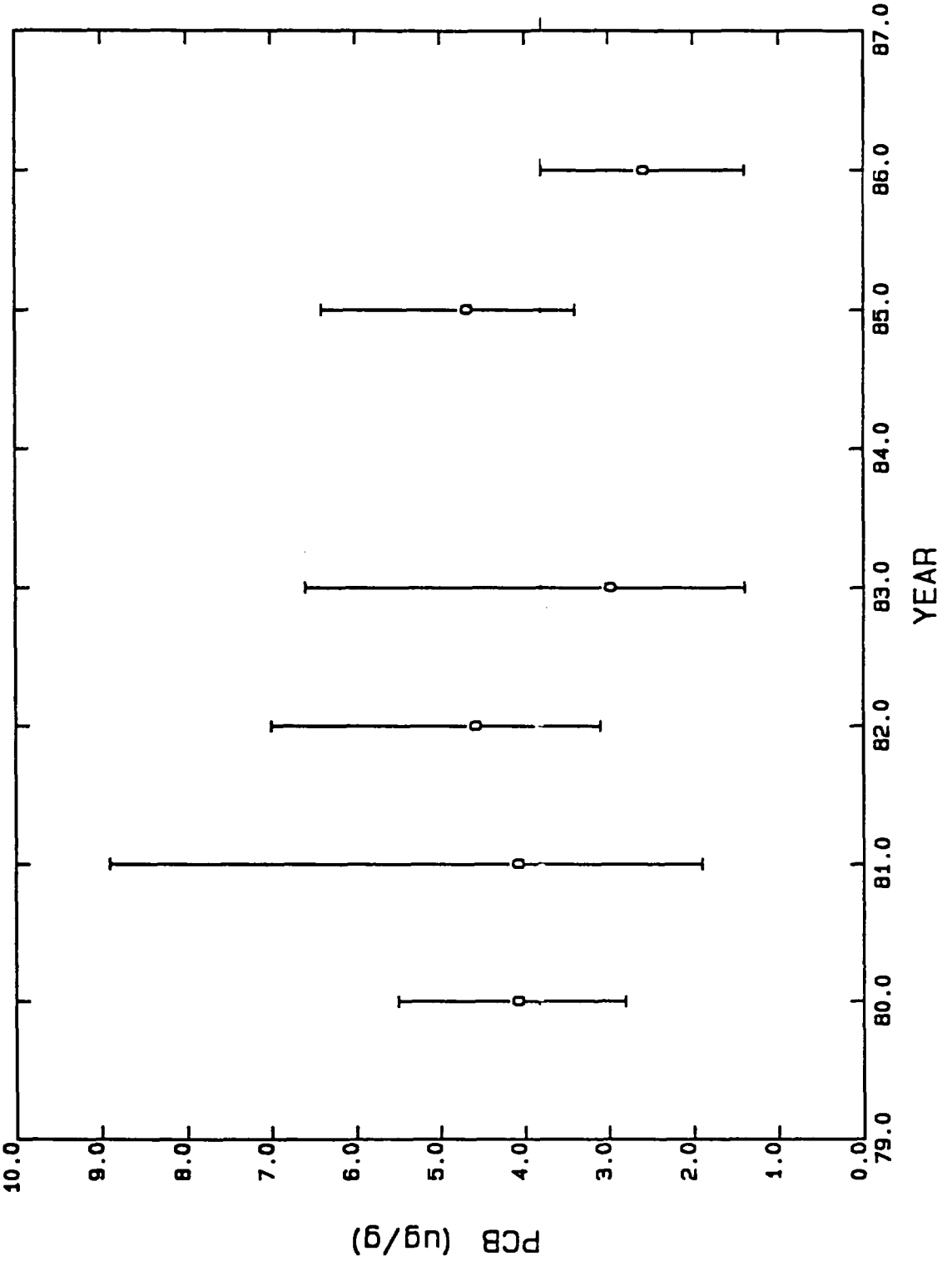
PCB concentrations in the edible portion of the lobster, defined by FDA to include the tomalley, were calculated using data reported by Pruell et al. (1988). The mean weight of the edible tissue and tomalley from lobsters collected in Area 3 were 156 g and 14.4 g, respectively (Pruell, et al., 1988) (see Appendix E). The mean total weight of edible tissue was 170.4 g. Using these values and the PCB concentration detected by BOS (1987), edible tissue (including tomalley) concentrations were derived. The following equations were used:

- PCB concentration in edible tissue x weight of edible tissue = ug PCBs

- PCB concentration in tomalley x weight of tomalley = ug PCBs

- $\frac{\text{Equation (1) + (2) (ug)}}{\text{Total weight of edible tissue (g)}}$ = PCB concentration

An example calculation using the BOS data is presented in Table 2-10. Using the weights reported by Pruell et al., (1988), the PCB concentration in edible tissue for Areas 1 through 4 are: 7.6 ppm, 2.3 ppm, 1.4 ppm, and 0.4 ppm, respectively. Carcinogenic risk calculations have been performed using these data and show that higher levels of risk are associated with consumption of the tomalley. (see Section 4.2.2.2)



Source: Dept. of Marine Fisheries; Spring Sampling

FIGURE 2-8
PCB CONCENTRATIONS (INCLUDING TOMALLEY) FROM LOBSTERS IN AREA 3: 1979-1987

TABLE 2-10

CALCULATION OF EDIBLE TISSUE PCB CONCENTRATIONS FOR LOBSTERS (INCLUDING TOMALLEY)
NEW BEDFORD, MASSACHUSETTS

Mean weight of hepatopancreas[1]: 14.4 grams
Mean weight of edible muscle[1]: 156 grams

Total weight of edible tissue: 170.4 grams

Median PCB concentration in edible muscle from lobsters in Area 3[2]: 0.231 μg/g

Median PCB concentration in hepatopancreas from lobsters in Area 3[2]: 14.414 μ/g

PCB concentration in edible tissue:

Edible Muscle Concentration

0.231 μg/g PCB x 156g = 36.1 μg PCB

Hepatopancreas

14.414 μg/g PCB x 14.4g = 207 μg PCB

Total Edible Tissue Concentration

$$\frac{36.1\ \mu g\ PCB + 207\ \mu g\ PCB}{170.4\ g\ tissue} = 1.43\ ppm\ PCB$$

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[1] Pruell et al., 1988

[2] BOS, 1987

Exposure to contaminants from the consumption of biota was assessed for each of the four areas identified Figure 2-5. Tables 2-11 and 2-12 present the mean and maximum PCB and metal concentrations used to assess exposure via ingestion of lobster, clams, and winter flounder. Other exposure parameters used in this assessment are presented in Table 2-13. Body dose calculations for these exposure scenarios are in Appendix C.

2.5.3 Air

The inhalation of airborne contaminants represents another potentially important route of exposure for the New Bedford Harbor area. However, limited air monitoring was performed in New Bedford and, as such, the data available for this risk assessment are viewed as representing a "snapshot" of contaminant levels in this area (NUS, 1986). Since the sampling locations used to obtain these data were designed to study possible tidal influence on airborne concentrations of PCBs and metals they may not be appropriate to characterize the extent of and potential exposure to airborne contamination at receptor locations.

Monitoring locations were selected to characterize the concentrations at high and low tide around the mudflat near the Aerovox Plant. Therefore, any extrapolation of the magnitude of air contamination at this area to other areas within the Greater New Bedford area may not be appropriate. However, to provide some indication of the potential exposure to airborne contaminants, these data were used (NUS, 1986).

Cadmium and lead were the only metals of concern monitored in the NUS study. Cadmium was not found in any of the samples analyzed, and the concentrations of lead were too low to make a precise determination of the ambient lead concentrations. Therefore, inhalation of airborne contaminants was assessed only for PCB exposure. Because no distinction between particulate and vapor phase PCBs can be made from the available monitoring data, it is assumed that all measured concentrations represent PCBs in the vapor-phase. This is a conservative assumption that may potentially overestimate the actual exposure; however, it is appropriate in the absence of specific data which could differentiate between PCBs in the particulate versus vapor phase.

Jordan evaluated inhalation exposure for each age class using the maximum, mean, and "background" PCB concentrations detected in the 1985 study. (The background PCB air concentration for New Bedford is estimated to be 10 ng/m^3 [NUS, 1986].) Inhalation rates of 5 m^3/day were assumed for a young child and 20 m^3/day for an older child and adult (EPA, 1985a).

TABLE 2-11

CONCENTRATIONS OF TOTAL PCBs (ppm) IN EDIBLE TISSUE OF
BIOTA COLLECTED FROM NEW BEDFORD HARBOR
NEW BEDFORD, MASSACHUSETTS

SPECIES	AREA 1[1]	AREA 2[1]	AREA 3[1]	AREA 4[1]
American Lobster[2]				
Mean	NC	0.568	0.213	0.064
Maximum	NC	1.234	0.351	0.176
Winter Flounder[3]				
Mean	1.039	0.371	0.278	0.101
Maximum	2.629	1.048	0.825	0.340
Clam				
Mean	0.689	0.231	0.156	0.039
Maximum	2.121	1.181	0.478	0.137

Notes:

1 = Areas refers to the division of the Harbor and Bay established by HydroQual.
2 = Lobster concentrations DO NOT include tomalley.
3 = The edible tissue concentration was estimated using a whole body: edible tissue ratio of 0.13 (BOS, 1987).
NA = Not Applicable (shellfish and crustaceans have naturally high levels of copper in their bodies).
NC = Not Collected; lobsters were not collected from Area I.
Mean = Arithmetic mean value of all samples collected.
Maximum = Maximum value detected in each Area.

TABLE 2-12

CONCENTRATIONS OF METALS (ppm) IN BIOTA
COLLECTED FROM NEW BEDFORD HARBOR AREA
NEW BEDFORD, MASSACHUSETTS

SPECIES	AREA 1[1]			AREA 2[1]			Area 3[1]			Area 4[1]		
	Cd	Cu	Pb	Cd	Cu	Pb	Cd	Cu	Pb	Cd	Cu	Pb
Lobster												
Mean	NC	NC	NC	0.38	NA	0.99	0.33	NA	0.38	0.26	NA	0.23
Max	NC	NC	NC	0.7	NA	3.3	0.54	NA	1.12	0.59	NA	0.84
Clam												
Mean	0.17	NA	1.01	0.26	NA	0.76	0.29	NA	1.28	0.32	NA	0.97
Max	0.36	NA	1.9	0.33	NA	0.98	0.38	NA	3.46	0.49	NA	1.72
Flounder												
Mean	0.01	3.1	0.89	0.01	3.7	0.83	0.005	9.7	0.63	0.01	9.6	1.2
Max	0.014	11.1	3.35	0.02	19.8	4.52	0.012	51.6	2.72	0.09	43.9	6.84

Notes:

1 = Areas refers to the division of the Harbor and Bay established by HydroQual.
NA = Not Applicable (shellfish and crustaceans have naturally high levels of copper in their bodies).
NC = Not Collected; lobsters were not collected from Area I.
Mean = Arithmetic mean value of all samples collected.
Maximum = Maximum value detected in each Area.

TABLE 2-13

EXPOSURE PARAMETERS USED TO ASSESS INGESTION OF BIOTA
NEW BEDFORD, MASSACHUSETTS

Exposure Parameter	Child	Older Child	Adult
Average Weight over Period of Exposure	10 kg	40 kg	70 kg
Frequency of Exposure (fish meals)	1 per day 1 per week 1 per month	1 per day 1 per week 1 per month	1 per day 1 per week 1 per month
Amount Ingested	115 grams/ fish meal	227 grams/ fish meal	227 grams/ fish meal
Gastrointestinal Toxicokinetic Factor	1.0	1.0	1.0
Species Consumed	Lobster Winter Flounder Clam	Lobster Winter Flounder Clam	Lobster Winter Flounder Clam
Contaminant Concentrations	See Tables 2-9 and 2-10		See Tables 2-9 and 2-10

Daily exposure durations of 8 and 24 hours per day were assumed for the probable and conservative exposure scenarios, respectively. The pulmonary TKF was assumed to be 1.0.

Table 2-14 presents the parameters used to assess inhalation exposure to PCBs; the body dose calculations are in Appendix C.

2.5.4 Other Exposure Considerations

Other exposure pathways that may be important but which could not be quantitatively evaluated in this risk assessment include neonatal and occupational exposure to PCBs and metals.

PCBs were used in several manufacturing processes in the Greater New Bedford Area over an extended period. Because PCBs are no longer manufactured or used in the U.S., occupational exposures to PCBs in this area are expected to be limited to exposure during the repair of PCB-containing transformers and capacitors, or accidents involving electrical equipment containing PCBs. In an occupational setting, PCB exposure may occur through absorption by the skin or respiratory or alimentary tracts. Because PCBs are highly lipophilic and relatively stable, they tend to rapidly bioaccumulate and distribute into the adipose tissue of humans. These compounds are slowly eliminated from the body and tend to bioaccumulate over time. Therefore, historical and/or current limited occupational exposure to PCBs may result in an increased body burden of these compounds above the general population. Although it is not possible to quantitatively determine the extent of previous exposure from these sources, environmental exposures to PCBs discussed in this section represent an additional contribution of PCBs to existing body dose levels of occupationally exposed individuals.

In-utero and neonatal exposure to PCBs are significant. Neonates, fetuses, and embryos are unable to effectively detoxify and eliminate PCBs from the body (EPA, 1986b). Laboratory studies have demonstrated that PCBs can cross the placental barrier and accumulate in the fetus (ATSDR, 1987). In addition, PCBs are known to be excreted in the breast milk of lactating (i.e., nursing) women. Therefore, frequent and/or high exposure to PCBs may occur through lactation, in which the highly lipophilic PCBs are readily transferred from maternal milk to the neonate. A qualitative discussion of the potential health effects of neonatal and occupational exposures is presented in Appendix D.

2.6 THE GREATER NEW BEDFORD HEALTH EFFECTS STUDY

In the fall of 1987, MDPH released the findings of the GNBHES, a three-year study designed to (1) determine the prevalence of elevated serum PCB levels in a random sample of Greater New

TABLE 2-14

EXPOSURE PARAMETERS USED TO ASSESS INHALATION OF AIRBORNE CONTAMINANTS
NEW BEDFORD, MASSACHUSETTS

Exposure Parameter	Child	Older Child	Adult
Average Weight over Period of Exposure	10 kg	40 kg	70 kg
Duration of Exposure			
Probable	8 hrs/day	8 hrs/day	8 hrs/day
Conservative	24 hrs/day	24 hrs/day	24 hrs/day
Frequency of Exposure	Daily	Daily	Daily
Inhalation Rate	5 m^3/day	20 m^3/day	20 m^3/day
Pulmonary Toxicokinetic Factor	1.0	1.0	1.0
Contaminant Concentration			
Background (NUS, 1985)	10 ng/m^3	10 ng/m^3	10 ng/m^3
Most Probable (NUS, 1985)	84 ng/m^3	84 ng/m^3	84 ng/m^3
Realistic Worst (NUS, 1985)	471 ng/m^3	471 ng/m^3	471 ng/m^3

Bedford Area residents, and (2) test the relationship between serum PCB levels and various health effects. The GNBHES was a collaborative effort of the MDPH, the Massachusetts Health Research Institute (MHRI), and the U.S. Center for Disease Control (CDC).

The GNBHES was conducted in two phases. The purpose of Phase I was to determine the prevalence of elevated serum PCB levels in the Greater New Bedford Area population and whether there was a relationship between serum PCB levels and blood pressure measurements. Phase I required a random selection of 1,784 New Bedford, Acushnet, Dartmouth, and Fairhaven residents between 18 and 64 years of age.

In Phase II, if 150 individuals could be found whose serum PCB level exceeded 30 parts per billion (ppb), the level identified as the 99th percentile of the general U.S. population, the health of those individuals would be compared with a control group.

Of the 1,482 residents considered eligible for inclusion in the study, 840 individuals chose to participate (the "Prevalence Study"). The serum PCB levels for this group were measured. Eleven of the 840 (i.e., 1.3 percent) were identified with PCB levels (greater than or equal to 30 ppb). Blood pressure did not appear correlated with serum PCB levels.

Subsequently, additional participants were recruited. These individuals were not randomly selected and were considered at high risk from exposure to PCBs as a result of ingestion of moderate to high amounts of seafood from contaminated areas (the "Enrichment Group"). Seven of the 110 participants (6.4 percent) in the Enrichment Group had serum PCB levels greater than or equal to 30 ppb (MDPH, 1987). Because the number of individuals with greater than 30 ppb was too small for statistical analysis, Phase II was not conducted.

The geometric mean of PCB serum levels in non-exposed, non-fisheating populations in the U.S. has been found to range between 4.2 and 6.4 ppb. The Prevalence Study subjects had a geometric mean of 5.8 ppb, while the mean of the Enrichment Group was almost three times as high (i.e., 13.34 ppb).

The GNBHES provided retrospective exposure and demographic information for the Greater New Bedford Area, some of which was incorporated into this exposure assessment. Because the GNBHES focused on seafood consumption and occupational exposure, information for either inhalation or direct contact exposure to PCBs was not presented. In addition, the GNBHES provided exposure and demographic information only for persons between 18 and 64 years of age.

The GNBHES provided an assessment of the exposure of the general population several years after issuance of the fishing ban. This assessment focuses on estimating the potential exposures received by hypothetical individuals from all exposure pathways, assuming different levels of consumption and direct contact.

The exposure scenarios developed in this report are not intended to predict the actual number of individuals exposed to PCBs. These scenarios are intended to reflect the possible exposures received by hypothetical individuals in order to assess risks posed by the site. The scenarios are reasonable possibilities and are consistent with information collected in the GNBHES and in studies performed by NOAA of commercial and recreational fishing and recreational beach use.

Results of this risk assessment are being used to determine the need for and evaluation of remedial actions rather than to determine or predict actual health effects. Although the risk assessment and the GNBHES serve separate purposes, they can be viewed jointly to gain a better understanding of actual and potential effects of PCB exposure in this area. Recommendations stated in the GNBHES include the following:

- The current ban on fishing in and around the New Bedford Harbor site should remain in effect until PCB concentrations in aquatic life decline to acceptable standards.

- Residents should refrain from obtaining and consuming recreationally caught seafood from the closure areas.

- Small-scale follow-up studies, including surveillance of high risk individuals, should be designed and conducted by MDPH for health research purposes.

3.0 TOXICITY ASSESSMENT

This section provides appropriate toxicological information necessary to evaluate the potential carcinogenic and noncarcinogenic risks to human health from exposure to PCBs, cadmium, copper, and lead.

A toxicological summary was compiled for each of the four contaminants and are in Appendix D. These evaluations describe the nature and severity of the potential adverse effects associated with exposure to each compound. Information contained in the summaries for each compound includes: physiochemical data, pharmacokinetic and toxicity information, and descriptions of the noncarcinogenic and carcinogenic effects associated with acute, chronic, and lifetime exposures. The information presented in these assessments summarize available research for descriptive purposes. They are not intended to be exclusive reviews of the toxicity of the contaminants of concern. Comprehensive discussions of the most recent research considered by EPA and ATSDR are also presented in EPA (1988a) and ATSDR (1987).

In addition, information on the potency of the four contaminants is presented as part of the dose-response assessment. Included in this assessment are the pertinent standards, criteria, advisories, and guidelines developed for protecting public health. How these values were derived and applied to the risk evaluation of the contaminants for the New Bedford Harbor site is described in the following subsection.

Because some of the standards and guidelines described in this section will be designated as chemical-specific applicable or relevant and appropriate requirements (ARARs) or non-promulgated standards, criteria, and guidance to be considered (TBCs) in the FS, a brief discussion of these values is also presented. These ARARs, and TBCs, however, are not necessarily used to assess the health risks. For example, Maximum Contaminant Levels (MCLs) established under the Safe Drinking Water Act are ARARs, but because the MCLs are not based strictly on health considerations, they are not relevant to the dose-response evaluation (see Section 3.2.2). It should be noted that the FS includes a section identifying and summarizing all ARARs and TBCs associated with New Bedford Harbor.

3.1 TOXICOLOGICAL SUMMARY

Toxicological summaries compiled for PCBs, cadmium, copper, and lead are in Appendix D. These evaluations emphasize the potential health effects associated with the principal routes of exposure at the New Bedford Harbor site. Therefore, the toxicological evaluations for PCBs, cadmium, copper, and lead

focus on (when possible) the oral, dermal, and inhalation routes of exposure. Each evaluation includes background information, an overview of the health effects observed in animals and humans, and a discussion of the toxicokinetics and interactive effects of each contaminant.

3.2 DOSE-RESPONSE EVALUATION

This subsection contains the quantitative indices of toxicity that were used to estimate risks associated with PCBs, cadmium, copper, and lead exposure at the New Bedford Harbor site. These contaminants were identified as the contaminants of concern. Various regulatory agencies have developed standards, guidelines, and criteria to protect public health from the adverse effects of chemical exposure. The NCP identifies these health-based standards/guidelines/criteria and categorizes them, along with other technology-based values, as either "potential ARARs" or "TBCs." Those health-based values relevant to the assessment of potential risk at the New Bedford Harbor site were identified for the four chemicals of concern (Table 3-1).

To compare the estimated body doses developed in Subsection 2.5 to an applicable standard or guideline, it was often necessary to convert the criterion to the same units as the body dose units (i.e., mg/kg-day). To adjust mg/l into mg/kg-day for an adult, the following conversion was used:

$$\frac{mg}{l} \times \frac{2l}{day} \times \frac{1}{70\ kg} = \text{Equivalent Daily Dose (mg/kg-day)}$$

(Two liters of water ingested per day and an average adult body weight of 70 kg are the standard exposure assumptions used by EPA.)

This conversion was used specifically for Health Advisory (HA) criteria and the Maximum Contaminant Level Goals (MCLGs) (see Subsection 3.2.2).

3.2.1 Carcinogens

If toxicological evidence suggests that a chemical may be a potential carcinogen, mathematical models are used to calculate the estimated excess cancer risk associated with exposure to the chemical. Unit cancer risks or carcinogenic potency factors were developed by the EPA Carcinogen Assessment Group (CAG) for approximately 58 chemicals. CAG calculated the unit risks using a linearized multistage model for low-dose extrapolation.

This model leads to a plausible upper limit (upper 95-percent confidence limit) of carcinogenic risk. The risk value obtained

TABLE 3-1. DOSE RESPONSE TABLE FOR THE CONTAMINANTS OF CONCERN AT NEW BEDFORD HARBOR NEW BEDFORD, MASSACHUSETTS.

COMPOUND	CARCINOGENIC GROUP	RfD (a) (mg/kg/day) (oral)	CANCER POTENCY FACTOR (b) (mg/kg/day)-1	MCL (c) (mg/l)	MCLG (d) (mg/l)	HEALTH ADVISORY (e) (mg/l) 1-DAY (10 kg) (child)	10-DAY (10 kg) (child)	LONGER TERM (10 kg) (child)	LONGER TERM (70 kg) (adult)	LIFETIME (70 kg) (adult)
PCBs	B2	NA	7.7 (oral)	0.0005 proposed	0	NA	NA	0.001	0.0035	NA
COPPER	D	NA	NA	1.3 proposed	1.3 proposed	NA	NA	NA	NA	NA
LEAD	C	NA	NA	0.005 proposed	0 proposed	NA (under review)	NA (under review)	NA (under review)	NA (under review)	NA (under review)
CADMIUM	B1	0.0005	6.1 (inhalation)	0.01 final	0.005 proposed	0.04	0.04	0.005	0.02	0.0003

TABLE 3-1 (con't). DOSE RESONSE TABLE FOR THE CONTAMINANTS OF CONCERN AT NEW BEDFORD HARBOR.

COMPOUND	10-DAY HEALTH ADVISORY (mg/kg) (f)	HEAs (g)		FDA ACTION LIMIT (edible portion fish and shellfish) (h)	AWQC (mg/l) (i)			OSHA STANDARD (mg/m3) (j)	NIOSH RECOMMENDED LEVEL (k) (ug/m3)
		AIS mg/kg-day (oral)	AIC mg/kg-day (oral)		Water + Fish Ingestion	Fish Ingestion Only	Drinking Water Ingestion Only		
PCBs	0.01	NA	NA	2	7.9E-8	NA	> 12.6E-6	1.0 (TWA) (1242) 0.5 (TWA) (1254)	1.0 (TWA)
COPPER	NA	0.037	0.037	NA	1 (based on taste)	NA	1 (based on taste)	1 (TWA)	NA
LEAD	NA	NA	0.0014	NA	0.05	NA	NA	0.05 (TWA)	<100 (TWA)
CADMIUM	NA	NA	0.013	NA	NA	NA	NA	0.2 (TWA) 0.6 (C)	Lowest Feasible Limit

TABLE 3-1 (con't). DOSE RESPONSE TABLE FOR THE CONTAMINANTS OF CONCERN AT NEW BEDFORD HARBOR.

COMPOUND	ACGIH TLV (l) (mg/m3)	MASS. AAL (m) (ug/m3)	NAAQS (n) (ug/m3)	MASS. MCL (p) (mg/l)
PCBs	1 (TWA) (1242) 0.5 (TWA) (1254)	0.003 (under review)	NA	NA
COPPER	1 (Dust and Mist)	NA	NA	NA
LEAD	0.15	0.68 (under review)	1.5 (o) (90-day)	0.05
CADMIUM	0.05 (Dust and Salts)	0.0003 (under review)	NA	0.01

TABLE 3-1 (con't). DOSE RESPONSE TABLE FOR CONTAMINANTS OF CONCERN AT NEW BEDFORD HARBOR.

FOOTNOTES

NA = Not Available
(a) RfD = Reference dose, an estimate (with an uncertainty of one order of magnitude or more) of a lifetime dose which is likely to be without significant risk to human populations.
(b) Cancer Potency Factor = A value, established by the USEPA Carcinogen Assessment Group, which is used to calculate the incremental cancer risk that a carcinogen could potentially pose. PCB value obtained from the USEPA DWQC Document, 1988a.
(c) MCL = Maximum Contaminant Level, drinking water regulations that are promulgated under the Safe Drinking Water Act. Proposed MCLs for copper and lead were listed in the Federal Register 8/18/88. The MCL for PCBs is listed in the Federal Register 5/22/89 p.22062.
(d) MCLG = Maximum Contaminant Level Goal, non-enforceable health goals that are instituted under the Safe Drinking Water Act.
(e) Health Advisory = Drinking water guidance issued by the USEPA Office of Drinking Water (USEPA, 1987). PCB values from USEPA DWQC Document, 1988a. Values for lead are currently under review and should not be used per USEPA. Lifetime cadmium value from ODW Health Advisory, 1987.
(f) PCB value developed by USEPA Exposure Assessment Group. (USEPA, 1986).
(g) HEAs = Health Effects Assessments; expressed as AIC (acceptable intakes chronic) and AIS (acceptable intake subchronic); Prepared by USEPA Environmental Criteria and Assessment Office. These values are listed in the Superfund Public Health Manual (USEPA, 1986)
(h) The edible portion of fish excludes the head, scales, viscera and inedible bones
(i) AWQC = Ambient Water Quality Criteria, guidance for the protection of human health set by the USEPA Office of Water, Standard and Criteria Division. Values based on carcinogenesis are listed for 10-6 risk. PCB values from the AWQC Document, 1980; Lead and cadmium values from IRIS.
(j) OSHA Standard = Workplace air regulations promulgated under the Occupational Safety and Health Act. Standards listed are either TWA (Time Weighted Averages) or C (Ceiling values). Values from NIOSH Pocket Guide to Chemical Hazards, 1985.
(k) NIOSH is the National Instituste for Ocupational Safety and Health. The Recommended Level is a Time Weighted Average(TWA) for 10 hrs/day; 40 hrs/wk expsoure. Values from NIOSH Pocket Guide to Chemical Hazards, 1985.
(l) ACGIH = American Conference of Government Industrial Hygienists. Values listed are Time Weighted Averages (TWA).
(m) MASS AAL = Massachusetts Acceptable Ambient Level for contaminants in air. Corresponds to a 10-5 risk level
(n) NAAQS = National Ambient Air Quality Standard, air regulations promulgated under the Clean Air Act.
(o) 3-month arithmetic mean
(p) Mass MCL = Massachusetts Maximum Contaminant Level for contaminants in water. Values from Mass. DEQE.

represents increased carcinogenic risk over a person's lifetime from exposure to a particular chemical. The cancer potency factors are expressed in units of $(mg/kg\text{-}day)^{-1}$.

EPA developed a classification system for the overall weight of evidence for carcinogenicity of chemicals based on human and animal studies, as well as other supporting data. The classification system is divided into five categories: Group A, Carcinogenic in Humans; Group B, Probably Carcinogenic to Humans (B1 and B2 for higher and lower degrees of evidence, respectively); Group C, Possibly Carcinogenic to Humans; Group D, Not Classifiable as to Human Carcinogenicity; and Group E, No Evidence of Carcinogenicity for Humans.

For the contaminants of concern at the New Bedford Harbor site, EPA classified PCBs and cadmium as Group B2 and B1 carcinogens, respectively; lead as a Group C carcinogen; and copper as a Group D carcinogen. However, for lead, the test doses that induce cancer in animals were greater than the lethal dose for humans. Therefore, exposure to lead is not assessed for carcinogenic effects. In addition, there are not sufficient data to consider cadmium to be carcinogenic to humans by the oral route. Therefore, the potential carcinogenic risks for cadmium are assessed only for inhalation exposure. Potency factors were derived by CAG for PCBs and cadmium (see Table 3-1).

The potency factor for PCBs was recently revised from 4.34 $(mg/kg\text{-}day)^{-1}$ to 7.7 $(mg/kg\text{-}day)^{-1}$ (EPA, 1988a). In the past, EPA based risk estimates on a study in which chronic exposure to Aroclor 1260 was shown to cause hepatocellular carcinomas in female Sherman rats (Kimbrough et al., 1975). The revised potency factor (7.7 $(mg/kg\text{-}day)^{-1}$) is based on a study in which chronic dietary administration of Aroclor 1260 was shown to cause hepatocellular carcinomas in male and female Sprague-Dawley rats (Norback and Weltman, 1985). This recent study is preferred because the Sprague-Dawley rat has a low incidence of spontaneous hepatocellular neoplasms and because the study spanned the natural life of the animal. Although the potency estimate is computed based on exposure to Aroclor 1260, it is intended to represent other PCB mixtures as well (EPA, 1988a).

A more recent review of the congener-specific toxicity of PCBs was performed by EPA as part of a risk assessment for Quincy Bay, Massachusetts (EPA, 1988b). In this report, a cancer potency factor specific to Aroclor 1254 was used to evaluate the potential risk from fish consumption. This value was derived based on the 1978 National Cancer Institute (NCI) study of Aroclor 1254 and estimated to be 2.6 $(mg/kg\text{-}day)^{-1}$ (EPA, 1988). The application of this cancer potency factor toward

assessing risk at this site was warranted based on the congener mix detected in Quincy Bay seafood. Analyses of these data showed the congener make-up to more closely resemble Aroclor 1254 than Aroclor 1260.

EPA conducted new congener-specific PCB analyses on lobster and flounder collected from New Bedford Harbor to determine the most appropriate cancer potency factor to apply to this risk assessment (EPA, 1988c). These data were statistically analyzed and the conclusions were summarized as follows: "The PCB mixture of the seafood from New Bedford Harbor cannot be classified as any commercial mixture, although the pattern of PCBs in the seafood appears to lie roughly between Aroclors 1254 and 1260. That the non-ortho-substituted congeners are not depleted but are actually enriched in New Bedford Harbor seafood lends some support for taking a conservative approach to assessing risks from seafood ingestion" (see Appendix E). Based on this review, the revised cancer potency factor of 7.7 $(mg/kg\text{-}day)^{-1}$ was used to evaluate risks from PCB exposure.

3.2.2 Noncarcinogenic

Evaluation of the potential noncarcinogenic effects of a compound is performed by comparing the exposure dose to the most applicable health-based standard or criteria. Because multiple criteria were developed for many compounds, the following list describes the hierarchy followed in this risk assessment. Noncarcinogenic risk for each contaminant was estimated by making the appropriate comparison of the body dose level to the first standard or criteria on this list available for the route-specific exposure. Separate lists exist for the oral/dermal and inhalation routes of exposures. When possible, chronic exposures were evaluated against criteria based on chronic exposure (e.g., derived from a chronic toxicity test) and likewise for acute and lifetime exposures.

The risk evaluation process often requires comparisons between exposure doses received via direct contact with or ingestion of contaminants and criteria developed for drinking water exposure (i.e., MCLs or HAs). This is appropriate and standard procedure for conducting risk assessments (SPHEM, 1986), since these criteria values were developed to provide a level of protection against contaminant exposure. As discussed in Section 2, the use of the TKF corrects for differences between contaminant uptake from the various routes of exposure (see Appendix B). Often it is necessary to convert the criteria values expressed in mg/l to units of mg/kg-day. This is accomplished by incorporating the standard exposure assumptions for drinking water ingestion (see Section 3.2).

It is also possible to estimate the noncarcinogenic effects associated with carcinogenic compounds, because some compounds elicit both carcinogenic and noncarcinogenic effects. However, the noncarcinogenic risk estimates do not account for the potential carcinogenic effects.

To assess the potential toxicity from exposure to Noncarcinogenic from the oral and/or dermal route of exposure, the following standards or criteria were used. Preference was given to the first standard or guideline presented.

EPA Reference Dose. Route-specific Reference Doses (RfDs) are the preferred criteria to evaluate noncarcinogenic effects. These values are based on the assumption that threshold levels exist for the toxic effects elicited by each compound. The RfD is considered to be the level unlikely to cause adverse health effects in humans exposed for a lifetime. These values are expressed in mg/kg body weight/day for a 70-kg person. The degree of uncertainty associated with these values may span one or more orders of magnitude or more.

RfDs are calculated by dividing a NOAEL (no observed adverse effect level) or LOAEL (lowest observed adverse effect level) by an uncertainty factor. The toxic endpoint chosen for calculating RfDs is the most sensitive effect seen in a test animal. RfDs for carcinogenic compounds can also be derived. These values are designed to protect against the noncarcinogenic effects of carcinogens, but should not be considered to provide protection from their carcinogenic effects. RfDs are developed by the EPA Environmental Criteria and Assessment Office (ECAO) in Cincinnati, Ohio. These values are available through the Integrated Risk Information System (IRIS).

An RfD exists only for cadmium; this value was used to evaluate chronic exposure to this contaminant. No RfDs exist for the other contaminants of concern at New Bedford Harbor. Therefore, the health-based criteria and standards that follow were used to assess the potential noncarcinogenic health risks from exposure to these contaminants at this site.

EPA Health Advisories. The EPA Office of Drinking Water (ODW) developed Health Advisories (HAs) for contaminants in drinking water. These HAs are set at levels that are not expected to cause adverse health effects and are expressed in units of mg/l. HA values are developed from data describing noncarcinogenic endpoints of toxicity; therefore, they are not considered protective of the potential carcinogenic effects of carcinogenic compounds.

HA values are derived for 1-day, 10-day, longer-term, and lifetime exposures when applicable information is available.

HAs are based on a 10-kg child drinking 1 liter of water per day, or a 70-kg adult drinking 2 liters per day. Lifetime HA values are developed for adults only.

Because HAs are developed for various exposure durations (1-day, longer-term, and lifetime), these criteria were used (when available) to assess potential risks associated with a specific exposure duration. HAs, developed by the ODW, exist for PCBs and cadmium (see Table 3-1). The HAs developed for lead are currently under review by the ODW and are therefore not listed in Table 3-1. In addition to the HAs developed by the ODW, the EPA Exposure Assessment Group (EAG) developed a 10-day HA for PCBs. This value was used to assess acute exposures to PCBs because it is considered protective against the noncarcinogenic effects of PCBs for an exposure period of 10 days or less. The 10-day HA values were used in this risk assessment to assess acute exposures, and the longer-term HAs were used to assess chronic exposure to PCBs and cadmium. (The longer-term HA and RfD for cadmium are the same value.) These values, expressed as mg/l, were converted to the same units as the exposure dose (mg/kg-day) using the standard exposure assumptions discussed in Section 3.2.

<u>EPA Maximum Contaminant Level Goals (MCLGs) and Maximum Contaminant Level (MCLs)</u>. Pursuant to Section 1412 of the Safe Drinking Water Act, EPA promulgated drinking water standards for certain organic and inorganic substances. These standards establish Maximum Concentration Limits (MCLs) that specify the maximum permissible level of a contaminant in water used as a public water supply. MCLs are enforceable standards and are based in part on economic considerations such as the availability and cost of treatment techniques. Generally, an MCL for a compound represents the maximum allowable lifetime exposure to the compound, assuming a 70 kg adult ingests 2 liters of water per day.

In the process of developing MCLs, EPA also develops MCLGs. MCLGs are nonenforceable health-based goals and are therefore always equal to or less than the MCLs. MCLGs are based on toxicological information and are set at a level at which no adverse health effects are anticipated. For contaminants where no safe threshold is known to exist (i.e., carcinogens), the MCLG is set at zero.

MCLs and/or MCLGs exist for all the contaminants of concern at the New Bedford Harbor site (see Table 3-1). Only the MCL for cadmium is a final value. The MCLs for PCBs, lead, and copper are proposed values (5/22/89 for PCBs and 8/18/88 for lead and copper). MCLGs for copper, lead, and cadmium have also been established. These values are set at levels at which no known or anticipated effects are expected; therefore, the MCLGs can be

used to evaluate potential risk. The proposed MCLG for lead was lowered on August 18, 1988 from 0.005 mg/l to zero. However, because the earlier MCLG value (0.005 mg/l and now the proposed MCL for lead) was the only criteria available to assess noncarcinogenic risks from exposure to lead, it was used in this risk assessment. As such, the noncarcinogenic risks for lead may underestimate the potential risks. The MCLG for PCBs is set at zero because it has been classified by the EPA as a Group B2 carcinogen (54 FR 22064). Since no RfDs or HAs exist for lead and copper, the MCLGs were used to assess the noncarcinogenic risks associated with exposure to these contaminants. These MCLGs, expressed as mg/l, were converted to the same units as the exposure dose (mg/kg-day) using the standard exposure assumptions discussed in Section 3.2.

Health Effects Assessment. Health Effects Assessments, prepared by EPA's ECAO, provide route-specific acceptable exposure levels for contaminants. Two categories are estimated for each systemic toxicant (i.e., toxicants for which cancer is not the endpoint of concern) when sufficient data exist. The Acceptable Intake Subchronic (AIS) is an estimate of an exposure level at which no adverse effects are expected when exposure occurs during a limited time period (subchronic exposure). Animal data used to estimate AIS levels generally include studies with exposure durations of 30 to 90 days. The Acceptable Intake Chronic (AIC), the second category, is an estimate of an exposure level at which no adverse effects are expected when exposure occurs for a significant portion of the lifespan (chronic exposure). Neither AISs nor AICs are derived for compounds for which there is sufficient evidence of carcinogenicity.

For the contaminants at the New Bedford Harbor site, AISs and/or AICs exist for cadmium, copper, and lead. No AIC or AIS exists for PCBs. The AIC for copper (0.037 mg/kg-day) is the same value as the converted MCLG for copper and was used to assess chronic exposure. No other AIC or AIS values were used in this risk assessment.

EPA Ambient Water Quality Criteria. Federal Ambient Water Quality Criteria (AWQC), developed under Section 304(a)(1) of the Clean Water Act, are health-based estimates of the ambient surface water concentration that will not result in adverse health effects.

For most compounds, AWQC are available for two different exposure pathways. One criterion is based on lifetime ingestion of both drinking water and aquatic organisms; the other is based on lifetime ingestion of aquatic organisms alone. These criteria assume a 70-kg adult consumes 2 liters of water and/or 6.5 grams of aquatic organisms daily for 70 years.

For carcinogens, the AWQC are water concentrations corresponding to incremental carcinogenic risks of 10^{-7}, 10^{-6}, and 10^{-5}. AWQC exist for PCBs, cadmium, and lead (See Table 3-1). An AWQC exists for copper but is based on the organoleptic threshold and is therefore not considered a health-based criterion.

Food and Drug Administration Tolerance Level. The FDA is authorized to establish tolerance levels for unavoidable food contaminants which are set to protect public health, as well as to consider other factors such as economic and technical feasibility. The current tolerance for residues of PCBs in fish and shellfish (edible portion) is 2 ppm. The edible portion of fish excludes head, scales, viscera, and inedible bones. FDA tolerance levels do not exist for cadmium, copper, or lead.

Because the FDA tolerance levels are intended to be national standards, they are developed based on the assumptions that not all of an exposed person's diet is from the contaminated food source, and not all of the contaminated food source contains concentrations at the tolerance level. The FDA tolerance levels do not allow the conclusion that lower levels pose no risk, particularly in the New Bedford context, because New Bedford residents that consume seafood caught within the fish and shellfish closure areas may receive a large portion of their total diet from a contaminated source.

Massachusetts Maximum Contaminant Level. The Massachusetts Department of Environmental Quality Engineering (DEQE) Office of Research and Standards adopted the MCLS promulgated by EPA (310 CMR 22.00). As previously described, EPA MCLs are enforceable standards, based in part on economic considerations, which specify the maximum permissible level of a contaminant in water used as a public water supply. Massachusetts MCLS (MMCLs) exist for lead and cadmium (see Table 3-1).

Inhalation Exposure. To assess risk from inhalation exposure, the following criteria and standards may be used.

EPA National Ambient Air Quality Standards. Primary National Ambient Air Quality Standards (NAAQS) were developed by EPA based on air quality criteria for individual pollutants. Primary NAAQS are designed to protect public health, while secondary NAAQS are designed to protect the public welfare (e.g., visibility, property, wildlife, and vegetation). The Clean Air Act, under which NAAQS are promulgated, does not require EPA to consider the costs (economics) of achieving or the technological feasibility of implementing the standards. Standards can be promulgated as annual maximums, annual geometric means, annual arithmetic means, or for other periods that vary from 1 hour to one year.

Primary NAAQS must allow for an adequate margin of safety to account for unidentified hazards and effects. The law requires EPA to set its ambient air standards to protect particularly sensitive populations (e.g., asthmatics). In developing primary NAAQS, EPA must specify the nature and severity of the health effects of each contaminant, characterize the sensitive population involved, determine probable adverse health effect levels in sensitive persons, and estimate the level that provides an adequate margin of safety to protect public health.

For the four contaminants of concern, NAAQS exist only for lead (see Table 3-1).

Massachusetts Acceptable Ambient Level. The DEQE Air Toxics Program established draft Acceptable Ambient Levels (AALs) for certain compounds. AALs are ambient air limits for specific chemicals based on the health effects data. AALs are considered protective against the most sensitive effect elicited by a chemical. For carcinogens, the AAL is set to correspond to an excess lifetime carcinogenic risk of 10^{-5}. AALs were developed for PCBs, lead, and cadmium (see Table 3-1).

Occupational Safety and Health Administration Standard. The Occupational Safety and Health Administration (OSHA) develops standards for workplace exposures to hazardous substances (CFR 29 Section 1910, 1000 Subpart Z). OSHA standards are expressed as 8-hour time-weighted averages (TWA) and are legally enforceable for occupational exposures. Table 3-1 lists OSHA standards for the four contaminants of concern.

National Institute of Occupational Safety and Health Recommended Standard. The National Institute of Occupational Safety and Health (NIOSH) develops recommended standards for workplace exposure to hazardous chemicals, which are then recommended to OSHA. NIOSH recommends standards based on exposures up to 10 hours/day for a 40-hour week. NIOSH-recommended standards exist for PCBs, lead, and chromium (see Table 3-1).

Threshold Limit Values. Threshold limit values (TLVs) are developed by the American Conference of Governmental Industrial Hygienists (ACGIH) and are used in evaluating occupational exposure to a chemical. A TLV is a TWA concentration for a contaminant considered to be without adverse effects, assuming an 8-hour workday and a 40-hour workweek. TLVs refer to airborne concentrations of chemicals, and are typically expressed in units of ppm or mg/m^3. As shown in Table 3-1, TLVs exist for all the contaminants of concern.

3.3 ARARs

Chemical-specific ARARs and TBCs were also identified for the contaminants of concern. ARARs and TBCs can be used to

determine the extent of site cleanup by providing either actual clean-up levels or the basis for calculating medium-specific target concentrations, which can then be used to assess the effectiveness of remedial alternatives. In addition, ARARs can be used to assess the attainment or non-attainment of institutional requirements.

Although the FS will include a section detailing all ARARs pertinent to the New Bedford Harbor remediation efforts, a brief description of ARARs is included herein because chemical-specific ARARs and TBCs are identified for the contaminants of concern. As required by the National Contingency Plan (NCP) and CERCLA as amended by SARA, ARARs are required to be identified and evaluated throughout the CERCLA RI/FS process. ARARs are promulgated and enforceable federal and state requirements that evaluate the appropriate extent of site cleanup, scope and formulate remedial action alternatives, and govern the implementation and operation of a selected action.

Applicable requirements specifically address a hazardous substance, location, or remedial action. Relevant and appropriate requirements address circumstances sufficiently similar to those at a CERCLA site, thus making the requirement relevant. If it is deemed appropriate to use the requirement given the circumstances, the requirement is considered an ARAR. Applicable requirements and relevant and appropriate requirements are given the same weight.

ARARs are identified and considered so that CERCLA responses are consistent with the state and federal environmental laws. ARARs are divided into three categories: chemical-specific (e.g., SDWA, MCLs), location-specific (e.g., wetlands regulations, Endangered Species Act), and action-specific (e.g., hazardous waste rules governing incineration). Federal and state nonregulatory guidance, standards, and criteria such as AWQC, MCLGs, and RfDs are not considered ARARs; however, they may be considered during a CERCLA response when ARARs do not exist. These nonpromulgated standards, guidelines, and criteria are categorized as TBCs.

3.4 SUMMARY

Selected criteria presented previously were used to develop quantitative indices of the potential risks associated with exposure at the New Bedford Harbor site. The revised cancer potency factor of 7.7 ($mg/kg\text{-}day^{-1}$) was used to provide estimates of the incremental carcinogenic risks associated with exposure to PCBs, which was the only contaminant evaluated for carcinogenic risks. (As discussed in Section 2.5.3, it was not

necessary to evaluate the carcinogenic risks associated with the inhalation of cadmium.) Because an RfD exists only for cadmium, other criteria were used to evaluate the noncarcinogenic risks associated with exposure to PCBs, copper, and lead. The converted 10-day and longer-term HAs or the MCLG values were used when appropriate.

4.0 PUBLIC HEALTH RISK CHARACTERIZATION

This section characterizes potential risks associated with exposure to contaminants at the New Bedford Harbor site. The estimated body dose levels of PCBs and selected metals, calculated in Section 2.5, are evaluated in this section using the appropriate health-based standards and criteria identified and discussed in Section 3.1.

Estimates of carcinogenic and noncarcinogenic risks associated with acute, subchronic, chronic, and lifetime exposure durations to PCBs and metals are included in this section, as are individual risk estimates for each contaminant and the overall risks resulting from each route of exposure. The contaminants, exposure routes, and specific locations within the New Bedford Harbor area that present a significant risk are identified and summarized. These results are used in the FS to establish response objectives, indicate impacts associated with the no-action alternative, and evaluate the effectiveness of the proposed remedial alternatives.

4.1 METHODOLOGY

The methodology used to generate the various risk estimates is discussed in the following subsections. Table 4-1 presents the equations used to derive these quantitative risk estimates.

4.1.1 Estimating Noncarcinogenic Risk

Noncarcinogenic effects associated with contaminant exposure include a variety of effects on various tissues and organ systems. These effects are considered to have a threshold value below which toxicant exposure results in no adverse effects. The specific noncarcinogenic effects for PCBs, cadmium, copper, and lead are discussed in Appendix D.

Noncarcinogenic risk estimates for the New Bedford Harbor site were generated by comparing the exposure dose for each contaminant to the most applicable health-based standard or criteria value. The values used in this risk assessment, listed in Table 3-1, represent the best estimate of the maximum contaminant level that will not result in adverse effects. The ratio of the estimated body dose levels to these standard or criteria values is used to evaluate risk. This ratio is referred to in this risk assessment as the risk ratio.

Generally, EPA states that if the risk ratio is less than 1, the predicted body dose level is anticipated to be without lifetime risk to human health. For example, a value of 0.25 implies that a person is receiving an estimated average daily dose equal to 25 percent of the acceptable intake of that contaminant. If the ratio exceeds 1, the estimated average daily dose levels exceed a level considered safe; therefore, the exposure could

TABLE 4-1

EQUATIONS USED TO ESTIMATE RISK
NEW BEDFORD, MASSACHUSETTS

Noncarcinogenic Risk Estimates:

Risk Ratio: $\frac{E}{RL}$

where E = Exposure Level generally in (mg/kg-day).
RL = Reference Level expressed in same units as E.

Carcinogenic Risk Estimates:

Incremental Carcinogenic Risk = CDI x CPF

where CDI = Chronic Daily Intake (mg/kg-day)
CPF = Carcinogenic Potency Factor $(mg/kg\text{-}day)^{-1}$

Multitoxic Risk Estimates:

Noncarcinogenic: $HI = E_1/RL_1 + E_2/RL_2 + E_3/RL_3 \ldots E_i/RL_i$

where E_i = Exposure Level for i^{th} toxicant
RL_i = Reference Level for i^{th} toxicant
HI = Hazard Index

Carcinogenic: $\Sigma\ (CDI_i \times CPF_i)$

where CDI_i = Chronic Daily Intake for i^{th} toxicant
CPF_i = Carcinogenic Potency Factor for i^{th} toxicant

potentially result in adverse health effects. The noncarcinogenic risk estimates developed in this subsection are evaluated against a risk ratio of 1.

The risk ratio best reflects the potential noncarcinogenic risk when comparisons are made to standards or criteria that are based on the same exposure assumptions as the exposure dose. For example, acute exposure doses should be compared to 1- or 10-day health-based criteria and chronic exposure doses to longer-term criteria. However, for many contaminants in this risk assessment, the only criteria available to evaluate noncarcinogenic risks were those based on lifetime exposure. RfDs and MCLGs are criteria that define an acceptable daily exposure of a contaminant, assuming a 70-year exposure duration. Therefore, comparing an average daily dose derived for a chronic (10-year) or acute exposure to the RfD or MCLG may overestimate the actual risk. In such instances, the significance of the risk ratio value requires further evaluation. For this report, the toxicity endpoints and the magnitude of the uncertainty associated with the criteria development were considered in evaluating these potential risks.

4.1.2 Estimating Carcinogenic Risk

Carcinogenic risk estimates for known or probable human carcinogens were calculated by multiplying the potency factor of the chemical (expressed as $(mg/kg\text{-}day)^{-1}$) by the estimated body dose (expressed as (mg/kg-day)). The product of these two values is an estimate of the incremental lifetime cancer risk, which is defined as the excess probability that an individual will develop cancer over a lifetime.

In this risk evaluation, PCBs are the only contaminants assessed for carcinogenic risks. Of the other contaminants, copper and lead are not classified as known or probable human carcinogens, and cadmium is considered carcinogenic only by the inhalation route of exposure. Because cadmium was not detected in any air samples, a risk evaluation for this route of exposure was not necessary.

The incremental carcinogenic risk estimates appear in scientific notation in this report. For example, a 2×10^{-6} incremental risk level implies that an individual's probability of manifesting cancer from the exposure assessed is two in one million.

The method used to estimate carcinogenic risks is based on EPA's linearized, multistage model of carcinogenic dose-response. This model assumes that no threshold value exists below which exposure to a carcinogen can be considered safe or risk-free. Therefore, any positive dose is assumed to result in a finite increment to an individual's lifetime risk of developing cancer.

EPA guidance states that the target total carcinogenic risk for an individual resulting from exposure at a Superfund site may range from 10^{-4} to 10^{-7} (EPA, 1986a and 1988). Response objectives and remedial alternatives should be developed to reduce total carcinogenic risks to levels within or below this range. The carcinogenic risk estimates developed in this subsection are evaluated using this target range.

4.1.3 Estimating Multitoxic Risk

Because most instances of environmental contamination involve concurrent exposure to a variety of compounds, it is necessary to assess the potential adverse effects that exposure to contaminant mixtures may have on public health. EPA proposed guidelines for assessing the effects of exposure to chemical mixtures (51FR:34014, 1986). These guidelines, based on the assumption of dose additivity, recommend estimating a Hazard Index (HI) for a mixture by summing the individual risk ratios for each chemical in the mixture. This approach assumes that multiple subthreshold exposures may result in adverse effects even if no single chemical exceeds its reference level. As with single contaminant exposure, concern over the potential risk increases as the HI approaches unity.

Because of the assumption of dose additivity, the use of the HI is appropriate only if chemicals in the mixture are expected to exert similar toxic effects by the same mechanism. Therefore, the chemicals of concern in this risk assessment were grouped and assessed together based on their critical effect. HI values for multitoxic exposure were calculated for PCB and metal exposure, because these compounds have been shown to exert similar toxic effects (i.e., renal, hepatic, and reproductive) in test animals and humans.

For carcinogens, the multitoxic value is derived by summing the incremental carcinogenic risks associated with each compound in the mixture. Because only one carcinogenic compound (i.e., PCBs) was evaluated in this risk assessment, multitoxic carcinogenic risk estimates were not developed.

As mentioned in Section 1.0, PAH compounds have also been detected in sediment from the New Bedford Harbor area. These compounds tend to be co-located with PCBs, but generally are present at lower concentrations (E.C. Jordan/Ebasco, 1986). Total PAH concentrations ranged from below detection limit to 930 ppm. The carcinogenic risks associated with exposure to PAH compounds were not evaluated in this risk assessment. As such, the risk cited for direct contact with and/or incidental ingestion of sediment may be underestimated. However, because the treatment technologies proposed for remediating PCB contamination would adequately reduce PAH concentrations, no residual risks from exposure to these compounds are anticipated

(E.C. Jordan/Ebasco, 1989). PCBs are the carcinogenic contaminants of concern and were the focus of this risk assessment.

4.1.4 Uncertainties in Estimating Risk

It should be emphasized that the risk estimates in this subsection are based on numerous assumptions, each having uncertainty associated with it. Several types of uncertainties should be considered in any risk evaluation:

- uncertainties associated with estimating the frequency, duration, and magnitude of exposure
- uncertainties associated with assigning exposure parameters to a heterogeneous population (e.g., body weight and ventilation rate)
- uncertainties in estimating carcinogenic potency factors and/or noncarcinogenic measures of toxicity (e.g., RfDs and MCLGs)

The uncertainties associated with estimating exposure result from the variance in sampling and analytical techniques, estimating the extent of contamination, and quantifying parameters that are not directly observed (e.g., frequency and duration of exposure). Because some of these parameters are functions of the behavior patterns and personal habits of the exposed populations, no one value can be assumed representative of all possible exposure conditions. To account for some of this variation, exposure scenarios were developed based on a range of exposure frequencies and durations. For some exposure scenarios, the range of exposure parameters spans two orders of magnitude. It was assumed that the actual exposure encountered by any individual receiving exposure will fall within this range.

There is also uncertainty associated with assigning quantitative values to exposure parameters such as body weight, ventilation rate, surface areas, and absorption or TKFs. The parameters used in this exposure assessment were based on actual or extrapolated values from surveys reported in the literature and professional judgment; therefore, they may not be representative of specific individuals in the New Bedford Harbor site area. However, the parameters are considered representative of the populations described in the exposure scenarios. The uncertainties associated with assigning values to these parameters are estimated to be less than one order of magnitude.

The use of toxicity parameters (e.g., RfDs and MCLGs) and cancer potency factors introduces additional uncertainties into the risk assessment process. These parameters are generally based on animal studies, many of which are performed at high doses relative to the site-specific exposures actually experienced at Superfund sites. These data require interpretation and/or extrapolation in the low dose area of the dose-response curve. Uncertainty factors are often incorporated to account for species-to-species and/or route-to-route extrapolations. The uncertainties associated with the use of toxicity parameters may be as high as three orders of magnitude.

To account for some of the uncertainties described in the previous paragraphs, the approach taken in this risk assessment was to estimate risk based on both most probable and upper-bound exposure conditions. This approach provided risk estimates that were considered appropriately conservative and unlikely to underestimate the actual risk.

4.1.5 Evaluating Risk

As stated previously, EPA established criteria for evaluating both noncarcinogenic and carcinogenic risk estimates at Superfund sites. For noncarcinogenic risks, a risk ratio less than 1 represents an exposure dose considered to be without lifetime risk to public health. For carcinogenic risks, EPA uses a target risk range of 10^{-4} to 10^{-7} to evaluate the need for and effectiveness of various remedial actions. The risk estimates developed in the following subsections are evaluated against these criteria.

In addition, the Commonwealth of Massachusetts enacted legislation parallel to CERCLA authorizing state response to releases of oil or hazardous materials and the assignment of liability, and providing for cost recovery for assessment, remedial response, and damage to natural resources. This legislation is contained in Chapter 21E of the Massachusetts General Laws (MGL.C.21E 1983, amended 1986). Regulations in the form of a state contingency plan were promulgated in October 1988. The portion of the Massachusetts Contingency Plan (MCP) relevant to this risk assessment requires that a permanent solution, which effectively eliminates significant or otherwise unacceptable risks to health, safety, public welfare or the environment, be implemented at all disposal sites. As stated in the MCP, the total site cancer risk will be compared to a cancer risk limit of 1 in 100,000 (1×10^{-5}). The total site noncarcinogenic risk will be compared to a risk limit represented by an HI equal to 0.2. The risk estimates generated in this report are also evaluated against the MCP criteria (see Section 4.3).

4.2 QUANTITATIVE RISK EVALUATION

Numerous risk estimates were derived as part of the risk evaluation for the New Bedford Harbor site. Each risk calculation is in Appendix C and is presented in summary tables throughout this subsection. A strict comparison of these risk estimates to appropriate standards and criteria values or the target range risk levels shows that many of these values exceed levels of risk considered to be of potential concern, under current EPA and state guidance. As such, these risks indicate that remedial actions may be warranted at this site.

Noncarcinogenic and carcinogenic risks were evaluated separately and are presented in the following subsections. The noncarcinogenic evaluation, discussed first, describes risks associated with acute and chronic exposure to PCBs, cadmium, copper, and lead. The carcinogenic evaluation follows and describes the risks from chronic and lifetime exposure to PCBs.

4.2.1 Noncarcinogenic Risk Evaluation

Noncarcinogenic risk ratios were developed for exposure to cadmium, copper, lead, and PCBs under both acute and chronic exposure conditions for the following routes of exposure:

- ingestion of sediment
- direct contact with sediment
- ingestion of aquatic biota

In addition to deriving the individual risk ratio values, Jordan generated multitoxic HI values for concurrent exposure to the three metals and PCBs. These compounds exhibit similar toxic endpoints (see Appendix D); therefore, it was appropriate to sum the individual risk ratios to derive a multitoxic HI value.

4.2.1.1 Sediment

Two routes of exposure (i.e., direct contact with and ingestion of contaminated sediment) were evaluated in this risk assessment. Exposure dose levels of PCBs, cadmium, copper, and lead were estimated separately for both routes of exposure and compared to the most applicable standard or criteria value. The noncarcinogenic risk evaluation for these routes of exposure are discussed separately in the following paragraphs.

Direct Contact with Sediment. The land use and activity patterns for the New Bedford Harbor area suggest that persons of all ages may be exposed to contaminated sediment as a result of swimming, wading, and/or fishing in the Acushnet River. As stated previously, the most likely locations for these activities to occur are south of the Coggeshall Street Bridge in

Areas II and III. Exposure to contaminated sediment in these areas was estimated to occur between 20 and 100 times per year. Because access to the shoreline Area I is not restricted, exposure to sediment in this area was considered possible and also evaluated. For adults and older children, who may access the mudflats in Area I to clam or fish, exposure to sediment was estimated to occur between 20 and 100 times per year. Since there are no recreational areas located within Area I and children (0-5) have limited mobility, exposure to sediment in Area I was estimated to occur between 1 and 20 times per year.

Risk ratio and multitoxic HIs were evaluated for both acute and chronic exposure durations. These values are listed in Table 4-2.

Chronic. Risk ratios for chronic exposure to PCB- and cadmium-contaminated sediment were derived by comparing the estimated exposure dose of each contaminant to the respective longer-term HAs. The HAs, expressed in mg/l, were converted to mg/kg-day by factoring in the standard exposure assumptions of 1 liter of water ingested per day for a 10-kg child or 2 liters of water ingested per day for a 70-kg adult. The converted longer-term HAs are $1x10^{-4}$ mg/kg-day and $5x10^{-4}$ mg/kd-day for PCBs and cadmium, respectively. (Note the converted longer-term HA for cadmium is the same value as the RfD for cadmium.)

Risk ratios for lead and copper exposure were derived by comparing the exposure dose of each contaminant to the respective MCL or MCLG. The MCL and MCLG values were converted to units of mg/kg-day by factoring in the standard exposure assumptions of 2 liters of water ingested per day for a 70-kg adult. The converted MCL for lead is $1.4x10^{-4}$ and MCLG for copper is $3.7x10^{-2}$. (Note the converted MCLG for copper is the same value as the AIC for copper.)

Location-specific exposure concentrations were used when available. However, the metals data could not be segregated by specific locations within an area; therefore, area-wide contaminant concentrations were used to evaluate exposure to metals. As such, the assumed exposure-point concentrations may overestimate actual exposure conditions, because they include data collected from the more-contaminated midchannel sediment.

Risk ratios for chronic exposure by children (0-5) years to sediment in Area I under most-probable conditions were not evaluated since it was assumed that exposure in this area occurs only once per year. The potential risks for this route of exposure is evaluated under acute exposure to sediment. Chronic exposure to contaminated sediment in Area I is evaluated assuming conservative exposure conditions only. Exposure to

TABLE 4-2. NONCARCINOGENIC RISK FROM DIRECT CONTACT WITH SEDIMENTS; CHILDREN OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Location	PCB Risk Ratio (a)	Cadmium Risk Ratio (b)	Copper Risk Ratio (c)	Lead Risk Ratio (d)	Multi-Toxic HI (e)
CHRONIC EXPOSURE:					
AREA I					
Area wide					
Child					
Prob.	NA	NA	NA	NA	NA
Cons.	93	0.003	0.002	0.2	93
Older Child					
Prob.	2.4	0.0003	0.00014	0.025	2
Cons.	200	0.0060	0.0038	0.5	201
Adult					
Prob.	1.0	0.0001	0.00006	0.010	1
Cons.	130	0.0040	0.0025	0.35	130
Upper Estuary					
Child					
Prob.	NA	NA	NA	NA	NA
Cons.	93	0.003	0.001	0.3	93
Older Child					
Prob.	2.4	0.0003	0.00014	0.028	2
Cons.	200	0.0062	0.0023	0.5	201
Adult					
Prob.	1.0	0.0001	0.00006	0.011	1
Cons.	130	0.0040	0.0015	0.35	130
Lower Estuary					
Child					
Prob.	NA	NA	NA	NA	NA
Cons.	6	0.003	0.002	0.2	6
Older Child					
Prob.	0.9	0.0004	0.00015	0.018	1
Cons.	13	0.0057	0.0038	0.43	13
Adult					
Prob.	0.4	0.0001	0.00006	0.007	0.4
Cons.	8	0.0036	0.0025	0.27	8
Cove Area					
Child					
Prob.	NA	NA	NA	NA	NA
Cons.	6	0.002	0.002	0.2	6
Older Child					
Prob.	1.8	0.0004	0.00022	0.025	2
Cons.	13	0.0043	0.0039	0.43	13

TABLE 4-2. NONCARCINOGENIC RISK FROM DIRECT CONTACT WITH SEDIMENTS; CHILDREN OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Location		PCB Risk Ratio (a)	Cadmium Risk Ratio (b)	Copper Risk Ratio (c)	Lead Risk Ratio (d)	Multi-Toxic HI (e)
Cove Area	Adult					
	Prob.	0.7	0.0001	0.00009	0.010	0.7
	Cons.	8	0.0028	0.0025	0.28	8
AREA II						
	Area wide					
	Child					
	Prob.	0.27	0.0003	0.0003	0.021	0.3
	Cons.	9	0.0029	0.008	0.410	9
	Older Child					
	Prob.	0.13	0.0001	0.00013	0.010	0.1
	Cons.	4	0.0012	0.003	0.2	4
	Adult					
	Prob.	0.05	0.00005	0.00005	0.004	0.06
	Cons.	2.60	0.0008	0.002	0.100	3
	Popes Island					
	Child					
	Prob.	0.14	ND	0.00024	0.02	0.2
	Cons.	2.50	ND	0.002	0.2	3
	Older Child					
	Prob.	0.069	ND	0.00012	0.01	0.08
	Cons.	1.10	ND	0.0009	0.087	1
	Adult					
	Prob.	0.027	ND	0.000047	0.004	0.03
	Cons.	0.69	ND	0.0006	0.057	0.7
	Palmer Island					
	Child					
	Prob.	0.039	ND	0.00015	0.018	0.06
	Cons.	0.80	ND	0.0009	0.100	0.9
	Older Child					
	Prob.	0.019	ND	0.000075	0.0089	0.03
	Cons.	0.35	ND	0.0004	0.044	0.4
	Adult					
	Prob.	0.0075	ND	0.00003	0.0035	0.01
	Cons.	0.23	ND	0.0002	0.029	0.3
	Marsh Island					
	Child					
	Prob.	0.1	ND	0.00015	0.025	0.13
	Cons.	1.60	ND	0.0008	0.240	1.8

TABLE 4-2. NONCARCINOGENIC RISK FROM DIRECT CONTACT WITH SEDIMENTS; CHILDREN OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Location	PCB Risk Ratio (a)	Cadmium Risk Ratio (b)	Copper Risk Ratio (c)	Lead Risk Ratio (d)	Multi-Toxic HI (e)
Older Child					
Prob.	0.05	ND	0.00007	0.012	0.06
Cons.	0.70	ND	0.0004	0.100	0.8
Adult					
Prob.	0.02	ND	0.000029	0.0049	0.02
Cons.	0.45	ND	0.0002	0.067	0.5
AREA III					
Area wide					
Child					
Prob.	0.05	ND	0.00005	0.007	0.06
Cons.	2.10	ND	0.0004	0.078	2.2
Older Child					
Prob.	0.02	ND	0.00002	0.004	0.027
Cons.	0.93	ND	0.0002	0.034	1.0
Adult					
Prob.	0.01	ND	0.000009	0.00140	0.011
Cons.	0.59	ND	0.0001	0.022	0.6
Fort Rodman					
Child					
Prob.	0.03	ND	ND	ND	0.03
Cons.	0.50	ND	ND	ND	0.5
Older Child					
Prob.	0.01	ND	ND	ND	0.012
Cons.	0.22	ND	ND	ND	0.2
Adult					
Prob.	0.005	ND	ND	ND	0.005
Cons.	0.14	ND	ND	ND	0.1
Fort Phoenix					
Child					
Prob.	0.008	ND	ND	ND	0.01
Cons.	0.05	ND	ND	ND	0.1
Older Child					
Prob.	0.004	ND	ND	ND	0.004
Cons.	0.02	ND	ND	ND	0.0
Adult					
Prob.	0.001	ND	ND	ND	0.001
Cons.	0.02	ND	ND	ND	0.0

TABLE 4-2. NONCARCINOGENIC RISK FROM DIRECT CONTACT WITH SEDIMENTS; CHILDREN OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Location	PCB Risk Ratio (a)	Cadmium Risk Ratio (b)	Copper Risk Ratio (c)	Lead Risk Ratio (d)	Multi-Toxic HI (e)
ACUTE EXPOSURE					
AREA I					
Cove Area					
Child					
Prob.	0.68	0.0020	NA	NA	0.7
Cons.	1.10	0.0042	NA	NA	1.1
Older Child					
Prob.	0.33	0.0007	NA	NA	0.3
Cons.	0.46	0.0018	NA	NA	0.5
Adult					
Prob.	0.13	0.0003	NA	NA	0.1
Cons.	0.29	0.0012	NA	NA	0.3
Maximum Concentration					
Child					
Prob.	15.00	0.0038	NA	NA	15.0
Cons.	17.00	0.0042	NA	NA	17.0
Older Child					
Prob.	7.30	0.0018	NA	NA	7.3
Cons.	7.30	0.0018	NA	NA	7.3
Adult					
Prob.	2.90	0.0007	NA	NA	2.9
Cons.	4.70	0.0012	NA	NA	4.7

(a) = The modified longer-term HA was used to assess chronic exposure and the modified 10-day HA was used to assess acute exposure.
(b) = The modified longer-term HA was used to assess chronic exposure and the modified 10-day HA was used to assess acute exposure.
(c) = The AIC was used to assess chronic exposure; no appropriate standard or guideline exists to assess acute exposure.
(d) = The modified proposed MCL was used to assess chronic exposure; no appropriate standard or guideline exists to assess acute exposure.
(e) = The Multitoxic Hazard Index (HI) is the sum of the risk ratios for PCBs, cadmium, copper and lead.
NA = Not Applicable
ND = Not Detected

sediment in Area I by older children and adults was evaluated for both most-probable and conservative scenarios.

Metals. The risk ratios based on exposure to cadmium-, copper-, and lead-contaminated sediment were below 1 for all areas and for all exposure conditions. These included risk ratios based on exposure to the maximum contaminant concentration detected in sediment. Because these values fall below 1, direct contact exposure to these contaminants is not considered to present a human health risk.

PCBS. The risks associated with direct contact exposure to PCB-contaminated sediment were greatest for the Upper Estuary in Area I. Risk ratio values for older children and adults under probable exposure conditions ranged from less than 1 to 2.4, and under conservative exposure conditions ranged from 8 to 200. Chronic exposure to sediment by younger children was assessed under conservative exposure assumptions only. The risk ratios for these scenarios ranged from 6 to 93. The magnitude to which these values exceed 1 indicates that exposure to PCB-contaminated sediment in this area presents a public health risk. All age classes appear to be at risk from direct contact exposure to PCBs. Methods to reduce these risks will be addressed in the FS.

Risk ratios based on exposure to PCB-contaminated shoreline sediment from Area II ranged from below 1 to 9. Risks associated with exposure to sediment from specific locations within Area II were lower than those estimated based on area-wide PCB concentrations. Risk ratios based on exposure to PCB concentrations detected in shoreline sediment from the Palmer Island area were all below 1, while risk ratios for Marsh Island ranged from 0.05 to 1.6, and risk ratios for the Popes Island area ranged from 0.03 to 3 (see Table 4-2).

The two risk ratios which exceeded 1 (1.6 and 3) were based on exposure by a young child to the maximum PCB concentration detected in these specific areas. Since it is unlikely that repetitive, long-term exposure to this concentration will occur, the potential risk to young children is considered to be less than the risks indicated by the ratios. Exposure to sediment in Area II is not considered to present a public health risk.

The risk ratios based on exposure to shoreline sediment at Fort Rodman and Fort Phoenix beaches in Area III were below 1 for all scenarios evaluated. The only risk ratio to exceed 1 was based on exposure to the maximum PCB concentration detected in shoreline sediment from all of Area III, and was estimated at 2. Since it is unlikely that repetitive long-term exposure will occur at this concentration, this scenario is considered to be overly conservative. The risks associated with exposure under

more realistic conditions are all less than 1. Direct contact exposure to PCB-contaminated sediment in Area III is not considered to present a public health risk.

Multitoxic. The multitoxic HI values based on concurrent exposure to the three metals was less than 1 for all exposure conditions except one, in which the HI was 1.1. However, this exposure scenario was based on conservative assumptions and is not considered representative of actual exposure conditions. Therefore, concurrent exposure to cadmium, copper, and lead is not considered to present a risk to human health.

The multitoxic HI based on concurrent exposure to all four contaminants slightly exceeded 1 in Area I (most-probable case) and exceeded 1 only under conservative exposure conditions for other areas within the New Bedford Harbor area. The major contribution to the HI value was the individual risk associated with exposure to PCBs.

Because exposure to all four contaminants at the maximum concentration is unlikely, these exposure scenarios are considered to be overly conservative. Actual exposure conditions are more likely to be represented by the conditions assumed under the probable exposure scenarios. The multitoxic HI values associated with these scenarios were below 1. Exposure through direct contact with metal-contaminated sediment is therefore not considered to present a risk to public health.

Acute. An acute exposure scenario was evaluated to determine if intermittent or once-in-a-lifetime contact with contaminated sediment posed a risk to public health. To provide an estimate of potential risk, body dose levels were calculated using the mean and maximum contaminant levels detected in shoreline sediment from Area I. This area was the most widely contaminated and had the highest shoreline PCB concentrations. The body dose levels estimated under this scenario were compared to appropriate short-term criterion. For PCBs and cadmium, the converted 10-day HAs were used to evaluate risk; however, there were no appropriate short-term criteria available to assess lead or copper exposure. The risk ratios are listed in Table 4-2.

The risk ratios associated with acute exposure to cadmium-contaminated sediment were below 1 for all scenarios. The risk ratios associated with acute exposure to PCB-contaminated sediment ranged from 0.2 to 17. The ratios that exceeded 1 were all based on exposure to the maximum PCB concentration detected in this area (6,393 ppm). A distribution of the PCB concentration in sediment from this area estimates the 90th percentile to be 1,800 ppm and the 75th percentile to be 390 ppm PCB (Battelle Sediment Data Base, 1988), suggesting that it is unlikely for exposure to occur at the maximum PCB

concentration. Risk ratios based on acute exposure via direct contact exposure to 1,800 or 390 ppm PCB were below 1 for all subpopulations. Since shoreline PCB concentrations in Areas II and III are less than 390 ppm, acute exposure to sediment in all three areas is not considered to present a public health risk.

Ingestion of Sediment. Ingestion of sediment is considered an age-related activity and most significant for children less than six years old. Exposure through ingestion of sediment was, therefore, assessed for the zero to 5-year age class only, and focused on areas where exposure by this age group was likely. Risk ratios for PCBs and metals were generated for exposure to sediment in the Upper and Lower Estuary and the Cove Area of Area I, and the recreational and beach areas within Areas II and III (see Figure 2-7). Location-specific concentrations of these contaminants were used when available. Given the nature of the metals data, cadmium, copper, and lead concentrations could not be estimated for specific recreational areas. Since the exposure concentrations used to derive the risk ratios for metals are based on area-wide concentrations, they may be greater than the location-specific exposure concentrations.

The areas chosen in these exposure scenarios represent locations where young children may have access to shoreline sediment. Children were expected to frequent the recreational and beach areas more often than areas in Area I. Therefore, differentfrequencies of ingestion were assumed for Area I than Areas II and III. Risk ratio values for exposure via ingestion of sediment are in Table 4-3.

Chronic. Risk ratios based on exposure to cadmium- and copper-contaminated sediment were below 1 for all scenarios. Risk ratios based on exposure to PCB- and lead-contaminated sediment exceeded 1 under certain scenarios. For Area I, risk ratios were derived assuming only conservative exposure assumptions, since the probable exposure scenarios assumed only 1 exposure per year which represents an acute versus chronic exposure. Assuming chronic exposure, both PCB and lead risk ratios exceeded 1 for all areas within Area I and ranged from 11 to 175 and 26 to 33, respectively. The multitoxic HI for these scenarios ranged from 37 to 209. Although these risk ratios were based on conservative exposure assumptions, the magnitude to which they exceed 1 indicates that ingestion of sediment from Area I presents a potential health risk.

The risk ratios for ingestion of lead-contaminated sediment from Area II ranged from 2.7 (Palmer Island) to 55 (area-wide). The highest risk ratios were based on conservative exposure conditions. Because the maximum lead concentrations used to derive these ratios were detected in midchannel sediment, they may overestimate the potential exposure and subsequent risk from

TABLE 4-3. NONCARCINOGENIC RISK FROM CHRONIC INGESTION OF SEDIMENTS; CHILDREN
NEW BEDFORD, MASSACHUSETTS.

Location	PCB Risk Ratio (a)	Cadmium Risk Ratio (b)	Copper Risk Ratio (c)	Lead Risk Ratio (d)	Multi-Toxic HI (e)
AREA I					
Area wide					
Child					
Prob.	NA	NA	NA	NA	NA
Cons.	175	0.380	0.23	33	209
Upper Estuary					
Child					
Prob.	NA	NA	NA	NA	NA
Cons.	175	0.380	0.140	33.0	209
Lower Estuary					
Child					
Prob.	NA	NA	NA	NA	NA
Cons.	11	0.340	0.230	26.0	38
Cove Area					
Child					
Prob.	NA	NA	NA	NA	NA
Cons.	11	0.260	0.230	26.0	37
AREA II					
Area wide					
Child					
Prob.	0.57	0.0410	0.0420	3.1	3.8
Cons.	17	0.3800	1.000	55	73
Popes Island					
Child					
Prob.	0.3	ND	0.04	3	3.3
Cons.	4.70	ND	0.280	27.0	32
Palmer Island					
Child					
Prob.	0.08	ND	0.02	2.7	2.8
Cons.	1.50	ND	0.1100	14	15.6

TABLE 4-3. NONCARCINOGENIC RISK FROM CHRONIC INGESTION OF SEDIMENTS; CHILDREN
NEW BEDFORD, MASSACHUSETTS.

Location	PCB Risk Ratio (a)	Cadmium Risk Ratio (b)	Copper Risk Ratio (c)	Lead Risk Ratio (d)	Multi-Toxic HI (e)
Marsh Island					
Child					
Prob.	0.22	ND	0.02	3.7	3.9
Cons.	3.00	ND	0.1100	32	35
AREA III					
Area wide					
Child					
Prob.	0.11	ND	0.00700	1.100	1.2
Cons.	4.00	ND	0.0600	10.000	14
Fort Rodman					
Child					
Prob.	0.06	ND	ND	ND	0.06
Cons.	0.90	ND	ND	ND	0.9
Fort Phoenix					
Child					
Prob.	0.020	ND	ND	ND	0.02
Cons.	0.10	ND	ND	ND	0.1

(a) = The modified longer-term HA was used to assess chronic exposure.
(b) = The modified longer-term HA was used to assess chronic exposure.
(c) = The modified MCLG (AIC) was used to assess chronic exposure.
(d) = The modified proposed MCL was used to assess chronic exposure.
(e) = The Multitoxic Hazard Index (HI) is the sum of the risk ratios for PCBs, cadmium, copper and lead.
ND = Not Detected

this route of exposure. Midchannel sediment, in general, was more contaminated than shoreline sediment.

The risk ratios developed for ingestion of lead-contaminated sediment in specific areas, under probable exposure conditions, were considered more representative of the potential risks from this route of exposure. These values slightly exceed 1 and ranged from 2.7 to 3.7, suggesting that chronic exposure to lead through ingestion of sediment is not significant for Area II. Lead was not detected in sediment from the Fort Rodman and Fort Phoenix beach areas in Area III.

The risk ratios based on ingestion of PCB-contaminated sediment in Areas II and III ranged from below 1 to 17. However, the risk ratios based on probable exposure conditions and location-specific PCB concentrations were all below 1. Since these scenarios are considered to be most representative of actual exposure conditions, ingestion of sediment from Areas II and III is not considered to present a noncarcinogenic public health risk.

Acute. Acute exposure to contaminants from ingestion of sediment was evaluated to determine if intermittent or once-in-a-lifetime exposure to sediment in New Bedford Harbor presented a risk to children, older children, and adults. The acute scenario was based on exposure to the maximum contaminant level detected in shoreline sediment. Risk ratios could only be derived for PCBs and cadmium because no appropriate standards or criteria exist to evaluate acute exposure to copper or lead. The body dose levels for PCBs and cadmium were compared to converted 10-day HAs. These risk ratios appear in Table 4-4.

The risk ratios based on ingestion of cadmium-contaminated sediment were below 1 for all subpopulations and areas. The risk ratios based on ingestion of PCB-contaminated sediment exceeded 1 only in Area I and ranged from 0.28 to 2 based on exposure to the mean PCB concentration and 4.6 to 32 for the maximum PCB concentration. Children are considered to be at greater risk than older children and adults. Risk ratios for this age class exceeded 1 under both most probable and conservative scenarios.

Summary. The noncarcinogenic risks associated with direct contact and ingestion exposures to sediment were evaluated by comparing the estimated exposure dose to the most appropriate standard or criterion. The risk ratios developed based on these evaluations indicate a potential risk to public health fr chronic exposure via ingestion and/or direct contact wi sediment in Area I. Children may be at risk from acute exposure via ingestion of sediment in Area I. PCBs are the major contaminant of concern in this area, and methods to reduce these

TABLE 4-4. NONCARCINOGENIC RISK FROM ACUTE EXPOSURE TO CONTAMINANTS VIA INGESTION OF SEDIMENTS; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Location	PCB Risk Ratio (a)	Cadmium Risk Ratio (b)	Copper Risk Ratio (c)	Lead Risk Ratio (d)	Multi-Toxic HI (e)
AREA I (Maximum Concentration)					
Child	32	0.8	NA	NA	33
Older Child	8	0.2	NA	NA	8
Adult	4.6	0.11	NA	NA	5
AREA I (Mean Concentration)					
Child	2	0.23	NA	NA	2
Older Child	0.47	0.056	NA	NA	0.5
Adult	0.28	0.03	NA	NA	0.3
AREA II (Maximum Concentration)					
Child	0.6	0.16	NA	NA	0.76
Older Child	0.16	0.04	NA	NA	0.20
Adult	0.086	0.023	NA	NA	0.11
AREA III (Maximum Concentration)					
Child	0.14	ND	ND	ND	0.1
Older Child	0.03	ND	ND	ND	0.03
Adult	0.021	ND	ND	ND	0.02

(a) = The modified 10-day HA was used to assess acute exposure.
(b) = The modified 10-day HA was used to assess acute exposure.
(c) = No appropriate criterion was available to assess acute exposure.
(d) = No appropriate criterion was available to assess acute exposure.
(e) = The Multitoxic Hazard Index (HI) is the sum of the risk ratios.
ND = Not Detected
NA = Not Applicable

risks will be evaluated in the FS. Chronic and acute exposure to PCB-, cadmium-, copper-, or lead-contaminated sediment in other locations of the New Bedford Harbor site area were not considered to present a significant noncarcinogenic risk to public health.

4.2.1.2 Biota

Risk ratios were generated for acute and chronic exposures to PCBs, cadmium, copper, and lead through ingestion of aquatic biota and are listed on Table 4-5. Because copper occurs at naturally high levels in shellfish and crustaceans (due to their copper-based blood), it is not possible to determine the copper concentration in these organisms resulting from contaminant exposure. Because copper data for lobsters and clams were not suitable for describing contaminant exposure, exposure to copper was only assessed for the ingestion of winter flounder. As discussed in Section 2.5, exposure to aquatic biota was assessed for the same four areas (Areas 1 through 4) established by HydroQual for their food-chain model.

Exposure through the ingestion of aquatic biota by younger children, older children, and adults was evaluated for both weekly and daily exposure frequencies, assuming an ingestion amount of 4 ounces (i.e., 115 grams) for younger children and 8 ounces (i.e., 227 grams) of fish per meal for older children and adults. Separate exposure scenarios were developed for each of the three species. Therefore, each scenario assumes that 100 percent of the seafood diet is comprised of the species evaluated.

<u>Chronic</u>. Chronic exposure to PCBs and metals via ingestion of biota was based on daily and weekly consumption frequencies and evaluated against criteria based on toxicity studies of chronic but less than lifetime exposure duration, when available. The most appropriate criterion for assessing chronic exposure to PCBs and cadmium is the converted longer-term HA. No appropriate criteria are available to evaluate chronic exposure to lead or copper; therefore, these contaminants were evaluated using the converted MCL and MCLG, respectively. Because the MCL and MCLGs are developed to be protective for lifetime exposure, using them to assess chronic exposure (i.e., 10-year) may overestimate potential risks.

<u>Metals</u>. Chronic exposure to cadmium and copper by older children and adults was not considered to present a public health risk. Risk ratios based on both weekly and daily ingestion frequencies for these subpopulations ranged from less than 1 to 7.9. Ratios in excess of 1 were based on daily ingestion frequencies and whole body tissue concentrations. These factors may result in conservative estimates of risk. The

TABLE 4-5. NONCARCINOGENIC RISK FROM INGESTION OF BIOTA; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

		Younger Child					Older Child					Adult				
			Risk Ratios			Multi Toxic		Risk Ratios			Multi Toxic		Risk Ratios			Multi Toxic
Species	Area	Cadmium	Copper	Lead	PCBs	HI (a)	Cadmium	Copper	Lead	PCBs	HI (a)	Cadmium	Copper	Lead	PCBs	HI (a)
PROBABLE SCENARIO																
Weekly Ingestion																
Lobster	1	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Clam	1	0.5	NA	11.0	11.1	23	0.3	NA	5.7	5.6	11.5	0.2	NA	3.3	3.2	6.7
Flounder	1	0.03	0.1	11.0	16.8	28	0.01	0.1	5.0	8.4	13.5	0.008	0.0	2.9	4.8	7.7
Lobster	2	1.2	NA	11.0	9.2	21	0.6	NA	5.6	4.6	10.8	0.3	NA	3.2	2.6	6.1
Clam	2	0.8	NA	8.7	3.7	13.2	0.4	NA	4.3	1.9	6.6	0.2	NA	2.5	1.1	3.8
Flounder	2	0.03	0.2	9.4	6.0	15.6	0.014	0.1	4.7	3.0	7.8	0.001	0.0	2.7	1.7	4.4
Lobster	3	1.0	NA	4.3	3.4	8.7	0.5	NA	2.1	1.7	4.3	0.3	NA	1.2	1.0	2.5
Clam	3	0.9	NA	14.0	2.6	17.5	0.5	NA	7.2	1.3	9.0	0.3	NA	4.1	0.7	5.1
Flounder	3	0.02	0.4	7.1	4.4	11.9	0.008	0.2	3.6	2.2	6.0	0.0005	0.1	2.0	1.3	3.4
Lobster	4	0.8	NA	2.6	1.0	4.4	0.4	NA	1.3	0.5	2.2	0.2	NA	0.7	0.3	1.3
Clam	4	1.0	NA	11.0	0.6	12.6	0.5	NA	5.5	0.3	6.3	0.3	NA	3.1	0.2	3.6
Flounder	4	0.3	0.4	13.0	1.6	15.4	0.16	0.2	6.8	0.8	8.0	0.090	0.1	3.9	0.5	4.6

TABLE 4-5. NONCARCINOGENIC RISK FROM INGESTION OF BIOTA; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

		Younger Child					Older Child					Adult				
			Risk Ratios			Multi Toxic		Risk Ratios			Multi Toxic		Risk Ratios			Multi Toxic
Species	Area	Cadmium	Copper	Lead	PCBs	HI (a)	Cadmium	Copper	Lead	PCBs	HI (a)	Cadmium	Copper	Lead	PCBs	HI (a)
Daily Ingestion																
Lobster	1	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Clam	1	4.0	NA	80	78	162	1.9	NA	40.0	39.0	80.9	1.1	NA	23.0	22	46
Flounder	1	0.2	0.9	70	118	189	0.088	0.5	35.0	59.0	94.6	0.050	0.3	20.0	34	54
Lobster	2	8.4	NA	79	64	151	4.2	NA	39.0	32.0	75.2	2.4	NA	22.0	18	42
Clam	2	5.3	NA	60	26	91	2.6	NA	30.0	13.0	45.6	1.6	NA	17.0	7.5	26.1
Flounder	2	0.2	1.1	65	42	108	0.099	0.6	32.0	21.0	53.7	0.056	0.3	19.0	12.0	31.4
Lobster	3	7.3	NA	30	24	61	3.6	NA	15.0	12.0	30.6	2.1	NA	9.0	6.9	18.0
Clam	3	6.5	NA	101	18	125	3.3	NA	51.0	8.8	63.1	1.9	NA	29.0	5.1	36.0
Flounder	3	0.1	3.0	50	32	85	0.055	1.5	25.0	16.0	42.5	0.030	0.9	14.0	9.0	23.9
Lobster	4	5.7	NA	18.0	7.2	31	2.9	NA	9.0	3.6	15.5	1.6	NA	5.0	2.0	8.6
Clam	4	7.1	NA	77	4.4	88	3.5	NA	38.0	2.2	43.7	2.0	NA	22.0	3.2	27.2
Flounder	4	2.2	2.9	95	11	112	1.1	1.5	48.0	5.7	56.3	0.6	0.8	27.0	3.2	31.7

TABLE 4-5. NONCARCINOGENIC RISK FROM INGESTION OF BIOTA; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

		Younger Child					Older Child					Adult				
			Risk Ratios			Multi Toxic		Risk Ratios			Multi Toxic		Risk Ratios			Multi Toxic
Species	Area	Cadmium	Copper	Lead	PCBs	HI (a)	Cadmium	Copper	Lead	PCBs	HI (a)	Cadmium	Copper	Lead	PCBs	HI (a)
CONSERVATIVE SCENARIO																
Weekly Ingestion																
Lobster	1	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Clam	1	1.1	NA	21.0	34	56	0.6	NA	11.0	17.0	28.6	0.3	NA	6.0	9.8	16.1
Flounder	1	0.04	0.5	38.0	42	81	0.020	0.2	19.0	21.0	40.3	0.010	0.1	11.0	12.0	23.2
Lobster	2	2.2	NA	37.0	20	59	1.1	NA	19.0	9.9	30.0	0.6	NA	11.0	5.7	17.3
Clam	2	1.0	NA	11.0	19	31	0.5	NA	5.0	9.6	15.1	0.3	NA	3.0	5.5	8.8
Flounder	2	0.06	0.9	51.0	17	69	0.030	0.4	26.0	8.5	35.0	0.020	0.3	15.0	4.8	20.1
Lobster	3	1.7	NA	13.0	5.6	20	0.9	NA	6.0	2.8	9.7	0.5	NA	4.0	1.6	6.1
Clam	3	1.2	NA	39.0	7.8	48	0.6	NA	20.0	3.9	24.5	0.3	NA	11.0	2.2	13.5
Flounder	3	0.04	2.2	31.0	13	47	0.020	1.1	15.0	6.7	22.8	0.1	0.6	9.0	3.8	13.5
Lobster	4	1.9	NA	9.0	2.8	13.7	0.9	NA	5.0	1.4	7.3	0.5	NA	3.0	0.8	4.3
Clam	4	1.6	NA	19.0	2.2	22.8	0.8	NA	10.0	1.1	11.9	0.4	NA	6.0	0.6	7.1
Flounder	4	0.3	1.9	77	5.4	85	0.1	1.0	39.0	2.7	42.8	0.080	0.6	22.0	1.6	24.2

TABLE 4-5. NONCARCINOGENIC RISK FROM INGESTION OF BIOTA; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

		Younger Child					Older Child					Adult				
			Risk Ratios			Multi Toxic		Risk Ratios			Multi Toxic		Risk Ratios			Multi Toxic
Species	Area	Cadmium	Copper	Lead	PCBs	HI (a)	Cadmium	Copper	Lead	PCBs	HI (a)	Cadmium	Copper	Lead	PCBs	HI (a)
Daily Ingestion																
Lobster	1	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Clam	1	7.9	NA	150	240	398	4.0	NA	75	120	199	2.3	NA	43	69	114
Flounder	1	0.3	3.4	270	298	572	0.2	1.7	130	149	281	0.088	1.0	76	85	162
Lobster	2	15.5	NA	260	140	415	7.7	NA	131	70	209	4.4	NA	75	40	119
Clam	2	7.3	NA	78	134	219	3.6	NA	39	67	110	2.1	NA	22.0	38	62
Flounder	2	0.4	6.1	360	118	485	0.2	3.0	180	59	242	0.1	1.7	102	34	138
Lobster	3	11.9	NA	89	40	141	6.0	NA	45	20	71	3.4	NA	25.0	11	39
Clam	3	8.4	NA	275	54	337	4.2	NA	137	27	168	2.4	NA	78	15	95
Flounder	3	0.3	15.8	216	92	324	0.1	7.9	108	46	162	0.080	4.5	62	27	94
Lobster	4	13.0	NA	67	20	100	6.5	NA	33.0	10	50	3.7	NA	19.0	5.7	28
Clam	4	10.9	NA	137	15	163	5.5	NA	68	7.7	81	3.1	NA	39	4.4	47
Flounder	4	2.0	13.4	540	38	593	1.0	6.7	270	19	297	0.6	3.8	155	11	170

(a) The Multi HI is the sum of the risk ratios for cadmium, copper, lead and PCBs.
A Longer-term Health Advisory was used to estimate the risk ratio for cadmium and PCB exposure.
The MCLG and proposed MCL were used to estimate the risk ratio for copper and lead respectively.
NA = Not Applicable

risk ratios for cadmium and copper generated under weekly exposure conditions are considered more reflective of actual exposure conditions, and these values were less than 1.

Chronic exposure to cadmium and copper through the ingestion of fish by children (zero to 5 years) resulted in risk ratios ranging from below 1 to 16. Of the 70 scenarios evaluated for children, 35 had corresponding risk ratios greater than 1. Although many of these scenarios were based on conservative assumptions (i.e., daily ingestion and whole-body contaminant concentrations), the frequency and magnitude to which these values exceed 1 suggest a potential health risk. In addition, young children are more sensitive to contaminant exposure than adults. Therefore, exposure to cadmium and copper through ingestion of biota may pose a risk to a child's health.

The risk ratio based on exposure to lead through the ingestion of biota by all age classes exceed 1 under both sets of exposure conditions and for all areas. These risk ratios were based on both weekly and daily ingestion frequencies and were as high as 540 (see Table 4-5). The frequency and magnitude by which the risk ratio values exceeded 1 indicate a potential risk to human health from lead exposure.

No one area or species appeared to consistently present a greater risk for exposure to lead. The mean lead concentration detected in winter flounder, clams, and lobsters from all four areas ranged from 0.23 to 1.28 ppm, and the maximum concentrations ranged from 0.84 to 6.84 ppm. The relatively low variance in concentrations indicates that chronic ingestion of any species from any area presents a potential risk to public health.

PCBs. The noncarcinogenic risks associated with PCB exposure were estimated by comparing the intake contaminant level to the longer-term HA established for PCBs. The risk ratio based on all sets of exposure conditions ranged from below 1 to 298. Elevated risk ratios were observed even under probable exposure conditions, suggesting that exposure to PCBs via ingestion of biota presents a potential health risk for all age classes.

As with lead, no one species or area appeared to consistently present a greater risk for PCB exposure. The mean PCB concentration in all three species (edible portion) ranged from 0.064 to 1.039 ppm, and the maximum PCB concentration ranged from 0.137 to 2.629 ppm. The low variance in concentrations indicates that ingestion of any species from any area presents a potential noncarcinogenic risk to public health.

These risk estimates only address the potential noncarcinogenic effects associated with PCB exposure and do not reflect the

potential carcinogenic risks. The carcinogenic risks associated with PCB exposure are evaluated in the next subsection.

Multitoxic. The combined HI values generated by summing individual risk ratios for the four contaminants exceed 1 for most exposure conditions evaluated (see Table 4-5). Concurrent exposure to these contaminants may therefore result in exposure levels in excess of those recommended in health-based criteria. The majority of the risk described by the multitoxic HI value is derived from the contribution of lead and PCB exposure. As indicated, the ingestion of biota may result in exposure to lead and PCBs above recommended levels. Because cadmium and copper exhibit similar toxic effects, the concurrent exposure to these contaminants may increase this risk.

Acute. Acute exposure via ingestion of biota was evaluated to reflect the potential risks associated with consumption frequencies of less than one fish meal per month. As discussed in Section 2.5.2 and presented in Table 2-9, the majority of residents in the Greater New Bedford area consume seafood less than once per month but greater than once per year. Because of the infrequent exposure, a larger portion of fish per meal was assumed. The exposure scenario was based on a single meal consisting of 400 grams of fish containing the maximum contaminant level detected in each species. The 10-day HAs for PCBs and cadmium were used to derive risk ratios (Table 4-6). Currently, no appropriate standard or criteria values are available to assess acute exposures to lead or copper.

Risk ratios based on cadmium exposure are equal to or less than 1 for all species and for all areas, indicating that acute exposures do not exceed the acceptable daily intake for this contaminant. These risk ratio values represent the upper-bound risk estimates because they were based on the maximum cadmium concentration detected in each species. Therefore, lower risks would be associated with more probable exposure conditions (i.e., lower contaminant concentrations).

The risk ratios based on acute exposure to PCBs slightly exceeded 1. However, the probability of ingesting fish contaminated with the maximum concentration of PCBs is low, suggesting that these risk ratios are overly conservative. Lower risk ratio values based on the ingestion of 400 grams of fish contaminated at the mean PCB concentration were below 1. These values are considered more reflective of potential risks from acute exposure via ingestion of biota. Therefore, noncarcinogenic risks associated with acute exposure via ingestion of aquatic biota are not considered to present a public health risk.

TABLE 4-6. NONCARCINOGENIC RISK FROM INGESTION OF BIOTA; ACUTE EXPOSURE
NEW BEDFORD, MASSACHUSETTS.

Species/ Contaminant	Maximum Concentration (PPM)	Acute Criteria (mg/kg)	Risk Ratios Child	Older Child	Adult
Lobster					
PCBs	1.23	0.01	4.92	1.23	0.70
Cd	0.7	0.004	7	1.75	1
Cu	N/A				
Pb	16				
Flounder					
PCBs	2.63	0.01	10.52	2.63	1.50
Cd	0.1	0.004	1	0.25	0.14
Cu	51.64				
Pb	6.89				
Clam					
PCBs	2.12	0.01	8.48	2.12	1.21
Cd	0.5	0.004	5	1.25	0.71
Cu	N/A				
Pb	6.34				

N/A = Data Not Available due to the naturally high level of copper in blood of these organisms.

No appropriate criteria or standards are available to assess acute exposure to copper or lead.

The converted 10-day HA values were used to assess acute exposure to PCBs and cadmiumn.

4.2.1.3 Air

The noncarcinogenic risks associated with inhalation of airborne contaminants were not developed because of the limited amount of available data (see Section 2.5). Carcinogenic risk estimates associated with this route of exposure were developed to provide a conservative estimate of the potential risks (see Subsection 4.2.2.3).

4.2.2 Carcinogenic Risk Evaluation

A major focus of this risk assessment was on the carcinogenic risks associated with exposure to PCB-contaminated sediment (ingestion and direct contact), biota, and air. As discussed in Section 3.0, exposure to copper, lead, and cadmium was not evaluated for potential carcinogenic risks.

Incremental carcinogenic risk estimates were developed based on subchronic, chronic, and lifetime exposures to PCBs and are presented in summary tables throughout this subsection. Chronic exposures to PCBs were considered most representative of probable exposure durations for the population within the New Bedford Harbor site area, given that a relatively large percentage of the population reported living in this area for more than five years (see Section 2.1). Therefore, risk estimates based on chronic exposure were the focus of the carcinogenic risk evaluation. The lifetime and subchronic risk estimates were used as upper and lower bounds of potential risks and to strengthen conclusions regarding risks associated with a particular route of exposure. The lifetime risks were estimated by summing the incremental risks associated with exposure during 0-5 years, 6-16 years and 17-70 years.

The carcinogenic risk estimates are based on environmental conditions as they exist in 1986 and assume that contaminant concentrations remain constant over the period of time evaluated. Therefore, the lifetime incremental carcinogenic risk estimates assume that PCB concentrations in sediment and biota remain constant over 70 years. This assumption may overestimate the actual exposure dose and subsequent risk.

Carcinogenic risk estimates developed for each route of exposure were evaluated with reference to the Superfund target range of 10^{-4} to 10^{-7}. Additional criteria used to evaluate the significance of these risk estimates included the contaminant distribution for both the general areas (Areas I, II, and III) and the specific exposure locations within each area; the ease of access to and the physical conditions at exposure locations; and the assumed exposure parameters, including frequency and duration of exposure. The discussion of carcinogenic risks for the New Bedford Harbor site is presented by medium for the significant routes of exposure in the following subsections.

4.2.2.1 Sediment

Two routes of exposure (i.e., direct contact with and ingestion of contaminated sediment) were evaluated in the exposure assessment. The risks associated with these routes of exposure are presented in the following paragraphs.

Direct Contact with Sediment. Risks from direct contact exposure to PCB-contaminated sediment were assessed separately for area-wide mean contaminant concentrations in Areas I, II, and III, and for location-specific mean and maximum concentrations within these areas (see Figure 2-7). Wading, shellfishing, and fishing were activities considered most likely to result in contaminant exposure. Because these activities occur in shoreline areas, the exposure concentrations used to assess direct contact exposure were based on contaminant levels detected in the shoreline sediment. Concentrations of PCBs detected in midchannel sediment were not included as part of this evaluation. The incremental carcinogenic risks associated with these exposure scenarios are in Table 4-7 and summarized by area in the following paragraphs.

Area I. Exposure to sediment in Area I was considered likely for all age classes based on the ease of access to the shoreline, the large mudflat areas suitable for clamming, and the high population density around this area. Because of the large range of contaminant concentrations detected in shoreline sediment from this area (ND to 6,393 ppm), separate evaluations were made for the upper and lower halves of the estuary and the Cove Area.

The incremental carcinogenic risks associated with direct contact exposure were greatest for children and older children. The risk estimates for these age classes range from within to greater than the target range for all subdivisions of Area I even under probable exposure conditions. The risk estimates for adults also exceeded the 10^{-4} risk level. Under conservative exposure assumptions, these risks were as high as $2x10^{-2}$ for chronic exposures by children and older children. The relatively high risk estimates generated for all three areas, in addition to the ease of access and likely land-use indicates a potential risk to public health. Methods to reduce these risks will be addressed in the FS.

Area II. The risk associated with direct contact exposure to sediment from Area II focused on locations where recreational activities were likely to occur. A majority of the shoreline in Area II is not readily accessible since the private property abutting the shoreline is fenced off. In addition, much of the land use in this area is classified as industrial. However, three locations within Area II are accessible and support

TABLE 4-7. CARCINOGENIC RISK ESTIMATES FOR DIRECT CONTACT WITH SEDIMENTS; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Location of Exposure	PCB Concen-tration (ppm)	CHILD Incremental Risks Sub-Chronic (1 year)	CHILD Incremental Risks Sub-Chronic (5 years)	OLDER CHILD Incremental Risks Sub-Chronic (1 year)	OLDER CHILD Incremental Risks Chronic (10 year)	ADULT Incremental Risks Sub-Chronic (1 year)	ADULT Incremental Risks Chronic (10 year)	Life time (70 yrs)
AREA I								
Upper Est.								
Prob.	378	2.7E-06	1.4E-05	2.6E-05	2.6E-04	1.0E-05	1.0E-04	8.2E-04
Cons.	6393	1.0E-03	5.1E-03	2.2E-03	2.2E-02	1.4E-03	1.4E-02	1.0E-01
Lower Est.								
Prob.	149	1.1E-06	5.4E-06	1.0E-05	1.0E-04	4.1E-06	4.1E-05	3.3E-04
Cons.	399	6.4E-05	3.2E-04	1.4E-04	1.4E-03	9.0E-05	9.0E-04	6.7E-03
Cove Area								
Prob.	286	2.1E-06	1.0E-05	2.0E-05	2.0E-04	7.9E-06	7.9E-05	6.4E-04
Cons.	399	6.4E-05	3.2E-04	1.4E-04	1.4E-03	9.0E-05	9.0E-04	6.7E-03
AREA II								
Entire Area								
Prob.	21	3.0E-06	1.5E-05	1.5E-06	1.5E-05	5.6E-07	5.6E-06	6.1E-05
Cons.	125	9.9E-05	4.9E-04	4.3E-05	4.3E-04	2.8E-05	2.8E-04	2.5E-03
Palmer Is.								
Prob.	3	4.4E-07	2.2E-06	2.2E-07	2.2E-06	8.4E-08	8.4E-07	9.0E-06
Cons.	11	9.1E-06	4.5E-05	4.0E-06	4.0E-05	2.6E-06	2.6E-05	2.3E-04
Popes Is.								
Prob.	11	1.6E-06	8.0E-06	7.6E-07	7.6E-06	3.0E-07	3.0E-06	3.2E-05
Cons.	34	2.7E-05	1.4E-04	1.2E-05	1.2E-04	7.7E-06	7.7E-05	6.8E-04
Marsh Island								
Prob.	8	1.2E-06	5.6E-06	5.5E-07	5.5E-06	2.2E-07	2.2E-06	2.3E-05
Cons.	22	1.7E-05	8.7E-05	7.8E-06	7.8E-05	4.9E-06	4.9E-05	4.3E-04
AREA III								
Entire Area								
Prob.	3.7	5.3E-07	2.7E-06	2.6E-07	2.6E-06	1.0E-07	1.0E-06	1.1E-05
Cons.	29	2.3E-05	1.2E-04	1.0E-05	1.0E-04	6.6E-06	6.6E-05	5.8E-04
Ft. Phoenix								
Prob.	0.6	8.5E-08	4.2E-07	4.1E-08	4.1E-07	1.6E-08	1.6E-07	3.3E-05
Cons.	0.75	6.0E-07	3.0E-06	2.6E-07	2.6E-06	1.7E-07	1.7E-06	9.4E-05
Ft. Rodman								
Prob.	2.1	3.1E-07	1.5E-06	1.5E-07	1.5E-06	5.9E-07	5.9E-06	3.9E-06
Cons.	7.1	5.7E-06	2.8E-05	2.5E-06	2.5E-05	1.6E-06	1.6E-05	6.3E-05

The cancer potency factor for PCBs is 7.7 (mg/kg-day)-1
Prob. = Probable exposure conditions.
Cons. = Conservative exposure conditions.
Lifetime = Incremental carcinogenic risks for a 70 year exposure.

recreational land uses. These are: Popes Island, Marsh Island, and Palmer Island.

The PCB concentration in shoreline sediment was lowest for the Palmer Island area (3 ppm mean; 11 ppm maximum) than for Marsh Island (8 ppm mean; 22 ppm maximum) or Popes Island (11 ppm mean; 34 ppm maximum). The incremental carcinogenic risks associated with contaminant exposure around Palmer were greatest for children and older children. Risk estimates based on realistic exposure conditions for these age classes ranged from $2x10^{-7}$ to $2x10^{-6}$. Under more conservative exposure conditions, the risk estimates increased and ranged from $4x10^{-6}$ to $4x10^{-5}$. Lower risks were associated with contaminant exposure by adults.

The concentration distribution of PCBs in sediment from Palmer Island show that 93 percent of the concentrations fall below 5 ppm (Figure 4-1), indicating that the actual exposure in this area is reflected by the assumptions used in the probable exposure scenario (mean concentration 3 ppm; 93 percentile is 5 ppm). Since these risk estimates fall at or below the lower end of the target range, exposure in this area is not considered to present a significant health risk.

The risk estimates generated for exposure to sediment around Marsh Island were greatest for children and older children, and ranged from $5x10^{-7}$ to $5x10^{-6}$ under probable exposure conditions and $8x10^{-6}$ to $8x10^{-5}$ under conservative exposure conditions. Risk estimates for adults were lower than those for children. All risk estimates, however, fall within the target range of 10^{-4} to 10^{-7}.

The concentration distribution of PCBs in sediment from the Marsh Island area indicates that 77 percent of the PCB concentrations are less than 8 ppm and similar to the concentration used to assess risk under probable exposure conditions (Figure 4-2). As stated, risk estimates based on exposure by all age classes to 8 ppm PCBs and probable exposure parameters range from $2x10^{-7}$ to $6x10^{-6}$ (Table 4-7). These risk estimates fall within the lower end of the target range and are considered reflective of the likely exposure conditions in this area.

The concentrations of PCBs in sediment from Pope's Island are higher than those detected at either Marsh Island or Palmer Island (Figure 4-3). The risks associated with exposure to this sediment are within or slightly above the target range with two scenarios exceeding a 10^{-4} risk ($1.2x10^{-4}$ and $1.3x10^{-4}$). As with exposure around Palmer and Marsh Island, the incremental carcinogenic risks were greatest for children and older children. These risks ranged from $8x10^{-7}$ to $8x10^{-6}$ under

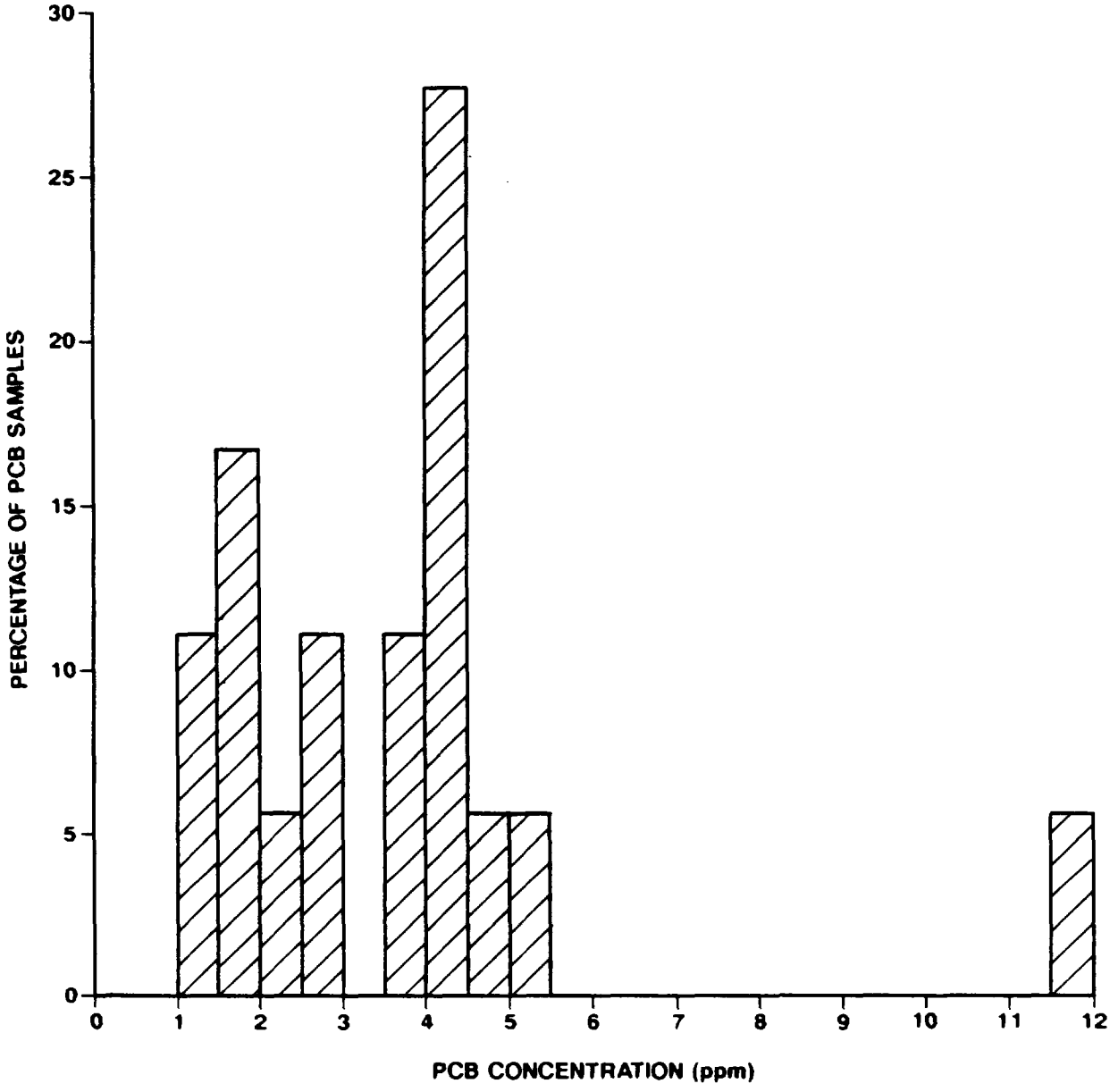

FIGURE 4-1
PCB DISTRIBUTION IN SEDIMENT IN THE PALMER ISLAND AREA (AREA II) NEW BEDFORD, MASSACHUSETTS

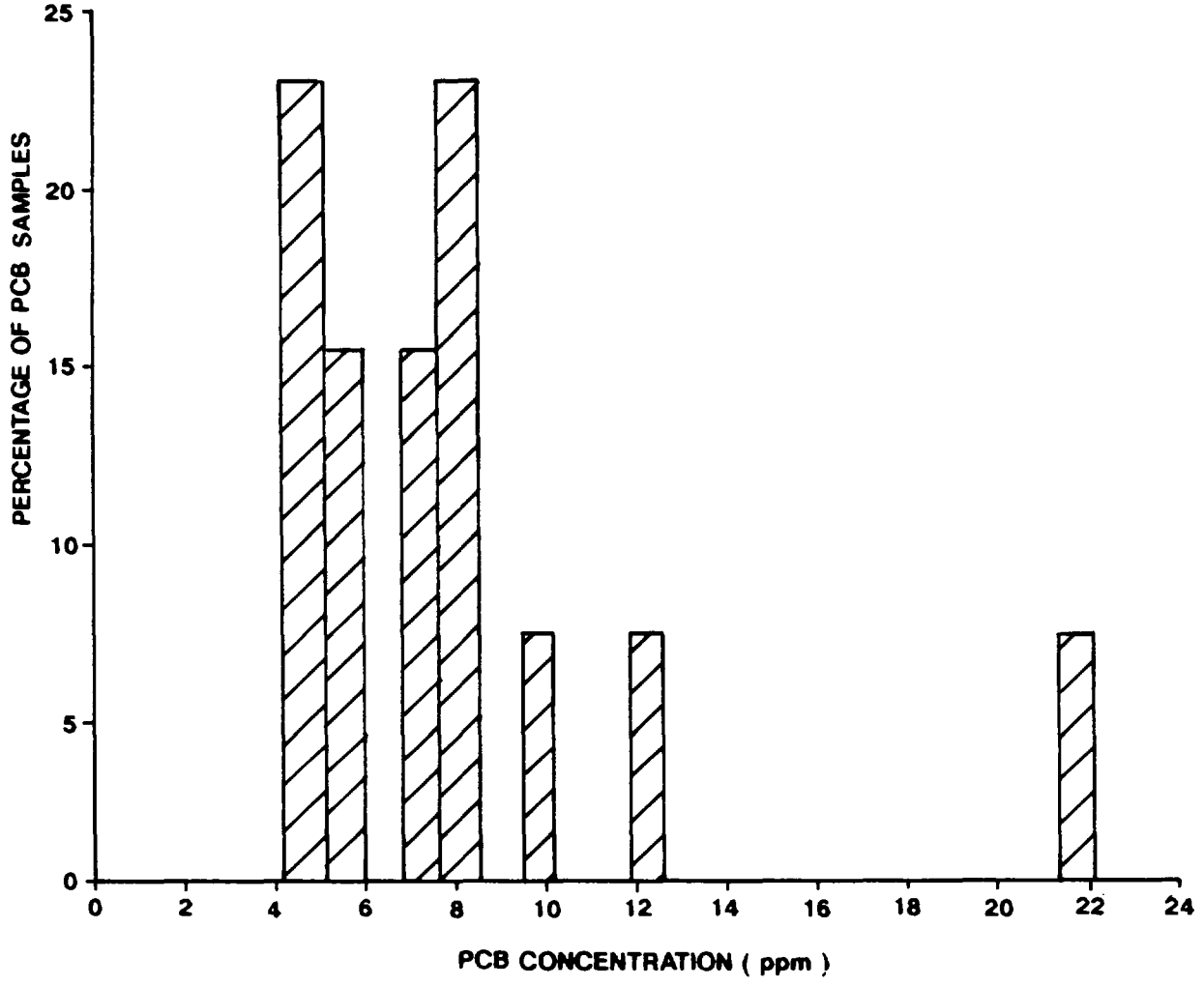

FIGURE 4-2
PCB DISTRIBUTION IN SEDIMENT
IN THE MARSH ISLAND AREA (AREA II)
NEW BEDFORD, MASSACHUSETTS

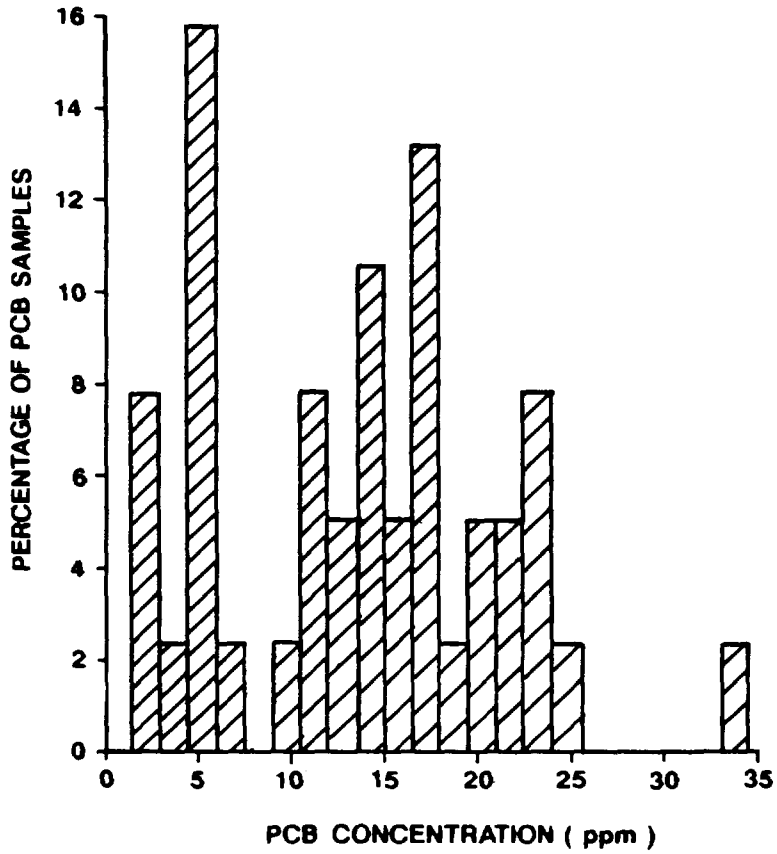

FIGURE 4-3
PCB DISTRIBUTION IN SEDIMENT
IN THE POPES ISLAND AREA (AREA II)
NEW BEDFORD, MASSACHUSETTS

probable exposure conditions and $1x10^{-5}$ to $1x10^{-4}$ under conservative exposure conditions. Because the 50th percentile of PCB concentrations from this area is greater than the mean concentration used to evaluate risk under probable exposure conditions, the risks estimated under conservative exposure conditions are considered to reflect likely exposure conditions in this area. Because these risk estimates span the target range with two scenarios exceeding a 10^{-4} risk, methods to reduce these risks will be addressed in the FS.

Area III. Direct contact exposure to sediment in Area III was assessed separately for the Fort Rodman (2.1 ppm mean; 7.1 ppm maximum) and Fort Phoenix (0.6 ppm mean; 0.8 ppm maximum) state park areas. The incremental risks estimated for all age classes for these locations range from $2x10^{-8}$ to $3x10^{-5}$. Under the probable exposure conditions, risks ranged from $2x10^{-8}$ to $2x10^{-6}$.

The concentration distribution of PCBs in sediment from the beach areas indicates that exposure is likely to occur at concentrations similar to those assumed under the probable exposure conditions. Seventy-five percent of samples had PCB concentrations less than 5 ppm from the Fort Rodman area, and less than 0.65 ppm for Fort Phoenix area (Figures 4-4 and 4-5). Risks associated with exposure to sediment from these areas are reflected by those calculated under probable exposure conditions. The low frequency of detection of highly contaminated sediment, combined with carcinogenic risks that are less than $2x10^{-6}$ suggests minimal public health risks from exposure to this sediment.

Ingestion of Sediment. Ingestion of sediment is considered an age-related exposure pathway that is most significant for ages 2 through 5. For the New Bedford Harbor site area, exposure through the ingestion of contaminated sediment is considered likely for the Cove Area of Area I and the beaches (Fort Rodman and Fort Phoenix) located in Area III (see Figure 2-7). These locations represent areas where children may play. Access to shoreline sediment in other locations in Areas I and II is considered unlikely given that industrial land use accounts for the majority of shoreline, and that children ages 2 through 5 are generally not unsupervised or sufficiently mobile to gain access to such areas. However, because access to these other areas is not restricted, exposure is possible. Therefore, the carcinogenic risks associated with exposure to sediment in all locations were evaluated. The incremental carcinogenic risks to young children are listed in Table 4-8 and summarized by area in the following paragraphs.

Area I. The incremental carcinogenic risks associated with the ingestion of sediment were greatest for exposure to sediment in

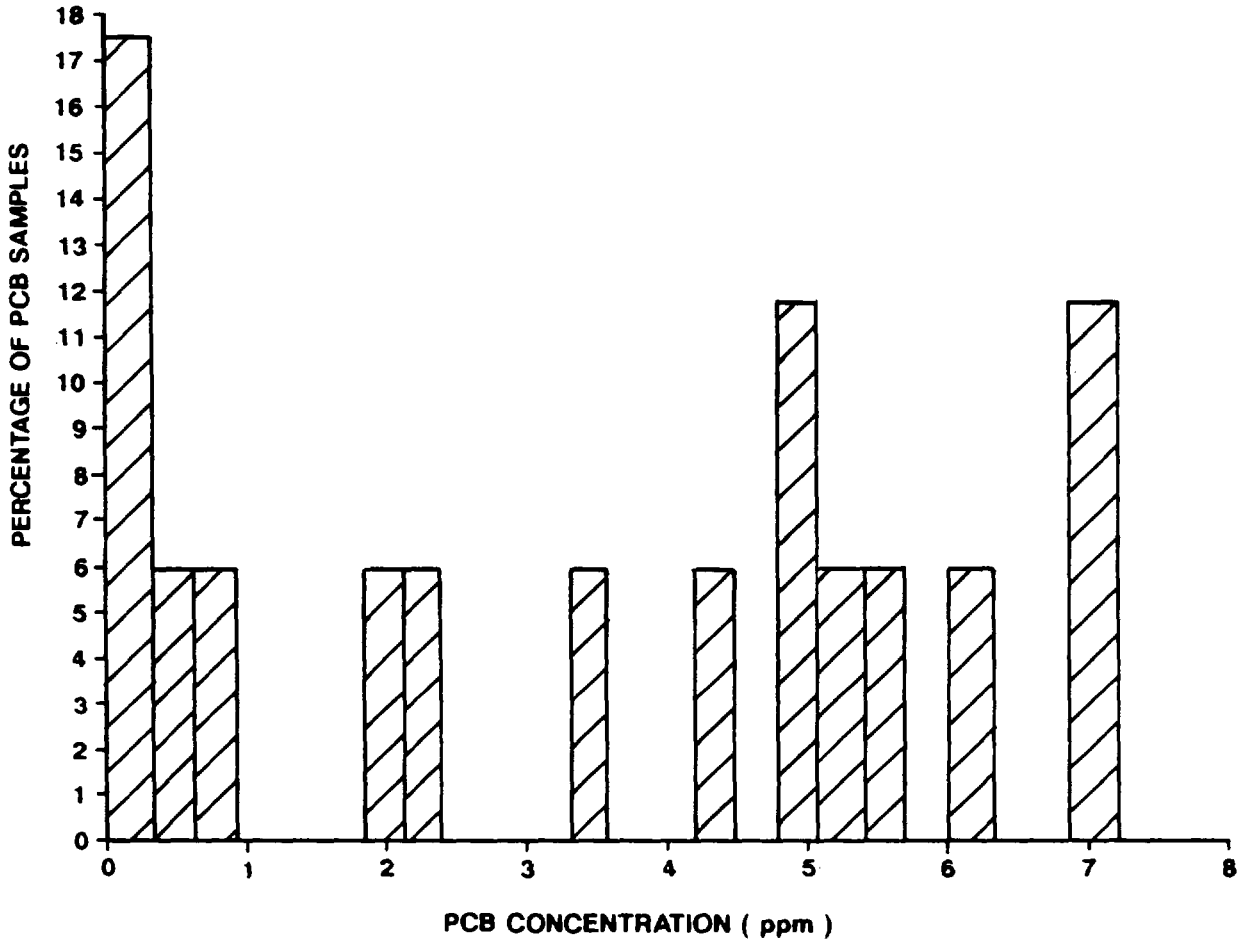

FIGURE 4-4
PCB DISTRIBUTION IN SEDIMENT IN THE FORT RODMAN BEACH AREA (AREA III)
NEW BEDFORD, MASSACHUSETTS

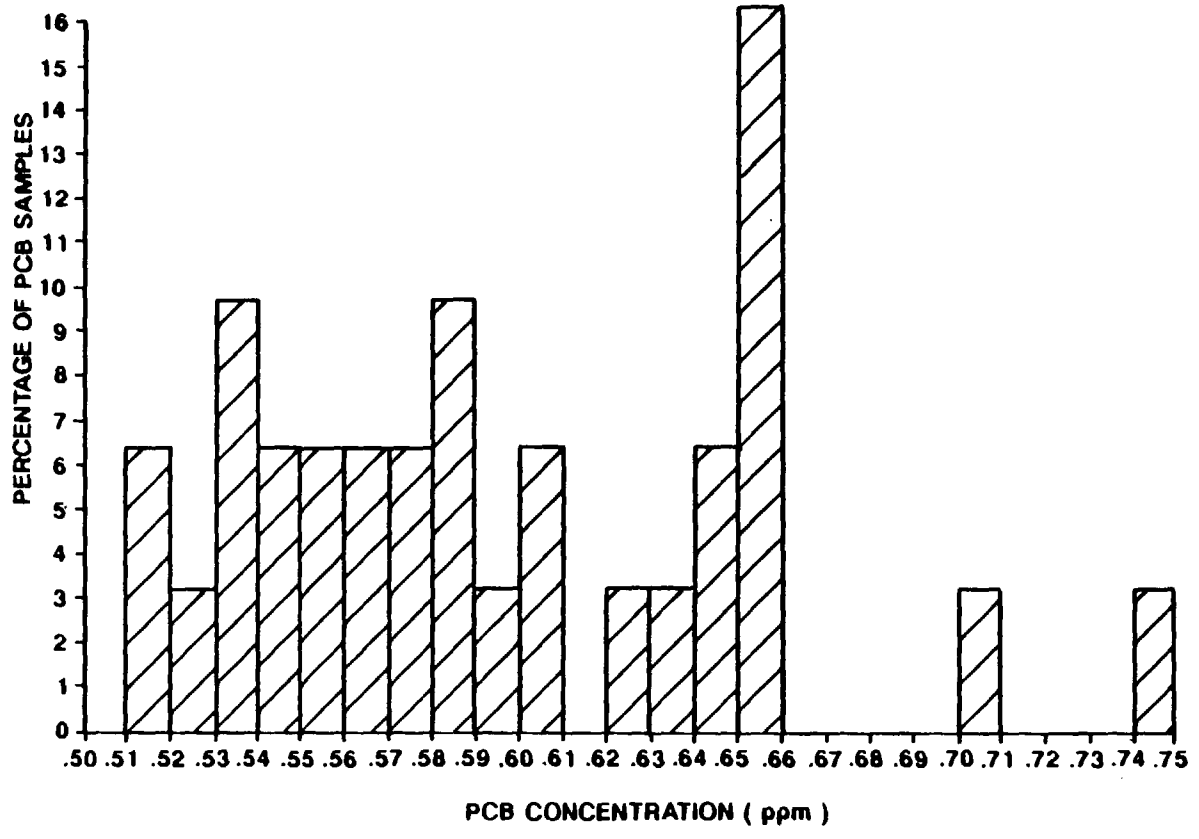

FIGURE 4-5
PCB DISTRIBUTION IN SEDIMENT IN THE FORT PHOENIX BEACH AREA (AREA III) NEW BEDFORD, MASSACHUSETTS

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TABLE 4-8. CARCINOGENIC RISK ESTIMATES FROM THE INGESTION OF SEDIMENTS; CHILDREN; NEW BEDFORD, MASSACHUSETTS.

Location of Exposure	PCB Concen-tration (ppm)	CHILD Incremental Risks Sub-Chronic (1 year)	CHILD Incremental Risks Sub-Chronic (5 years)	OLDER CHILD Sub-Chronic (1 year)	OLDER CHILD Chronic (10 year)	ADULT Sub-Chronic (1 year)	ADULT Chronic (10 year)	Life time (70 yrs)
AREA I								
Upper Estuary								
Prob.	378	5.60E-06	2.80E-05	NA	NA	NA	NA	2.80E-05
Cons.	6393	1.90E-03	9.60E-03	NA	NA	NA	NA	9.60E-03
Lower Estuary								
Prob.	149	2.20E-06	1.10E-05	NA	NA	NA	NA	1.10E-05
Cons.	399	1.20E-04	6.00E-04	NA	NA	NA	NA	6.00E-04
Cove Area								
Prob.	286	4.30E-06	2.10E-05	NA	NA	NA	NA	2.10E-05
Cons.	399	1.20E-04	6.00E-04	NA	NA	NA	NA	6.00E-04
AREA II								
Popes Island								
Prob.	11	3.20E-06	1.60E-05	NA	NA	NA	NA	1.60E-05
Cons.	34	5.10E-05	2.50E-04	NA	NA	NA	NA	2.50E-04
Palmer Island								
Prob.	3	9.00E-07	4.50E-06	NA	NA	NA	NA	4.50E-06
Cons.	11	1.70E-05	8.30E-05	NA	NA	NA	NA	8.30E-05
Marsh Island								
Prob.	8	2.40E-06	1.20E-05	NA	NA	NA	NA	1.20E-05
Cons.	22	3.30E-05	1.60E-04	NA	NA	NA	NA	1.60E-04
AREA III								
Ft.Rodman								
Prob.	2.1	6.30E-07	3.10E-06	NA	NA	NA	NA	3.10E-06
Cons.	7.1	1.00E-05	5.30E-05	NA	NA	NA	NA	5.30E-05
Ft. Phoenix								
Prob.	0.6	1.80E-07	9.00E-07	NA	NA	NA	NA	9.00E-07
Cons.	0.7	1.00E-06	5.20E-06	NA	NA	NA	NA	5.20E-06

The cancer potency factor for PCBs is 7.7 (mg/kg-day)-1
Prob. = Probable exposure conditions.
Cons. = Conservative exposure conditions.
Lifetime = Incremental carcinogenic risks for a 70 year exposure.

the Upper Estuary Area of Area I. The risks estimated based on exposure in this area were within or exceeded the target range of 10^{-4} to 10^{-7} ($6x10^{-6}$ to $1x10^{-2}$). The PCB exposure-point concentrations were 378 and 6,393 ppm. However, since young children are not expected to have access to these areas the risks estimated may not reflect actual exposure conditions.

The risk estimates for exposure to sediment from the Cove Area are considered more representative of potential exposure conditions because this area is located near a playground. The risk estimates based on ingestion of sediment from the Cove Area fall within or exceed the target range ($4x10^{-6}$ to $6x10^{-4}$). The assumed exposure concentrations in this area were 286 and 399 ppm of PCBs. The PCB distribution in shoreline sediment from the Cove Area shows that over 80 percent of this sediment have concentrations between 250 and 400 ppm (Figure 4-6), indicating that exposure to sediment in this area is likely to occur at concentrations similar to those used to assess risk. Because these risk estimates are based on realistic exposure conditions, they are considered to represent a public health risk; methods to reduce these risks will be developed in the FS.

Area II. The risk estimates based on ingestion of sediment from Area II ranged from $9x10^{-7}$ to $2x10^{-4}$, with the majority of risk values falling between 10^{-5} and 10^{-6}. Risks associated with exposure to sediment were lower at the Palmer Island area ($9x10^{-7}$ to $8x10^{-5}$) than Marsh Island ($2x10^{-6}$ to $2.x10^{-4}$) or Popes Island ($3x10^{-6}$ x $2x10^{-4}$). The higher risk estimates are associated with exposure under conservative conditions.

The highest risk estimates for this route of exposure are associated with chronic exposure to sediment from the Pope Island and Marsh Island area. Because these values exceed the target range, they may present a public health risk. As such, methods to reduce these risks will be evaluated in the FS.

Area III. The risk estimates generated based on ingestion exposure to sediment in the southern portion of New Bedford Harbor are lower than those estimated for Areas I or II. Specific locations within Area III, where exposure was considered likely to occur, included the beaches at Fort Rodman and Fort Phoenix state parks. The concentrations used to assess exposure at these areas ranged from 0.6 to 7.1 ppm PCBs. The risk estimates generated for these areas are below or within the lower end of the target range (the highest risk estimate was $5x10^{-5}$).

The concentration distribution in sediment from these areas suggests that exposure is more likely to occur at concentrations similar to those evaluated under the probable exposure scenario

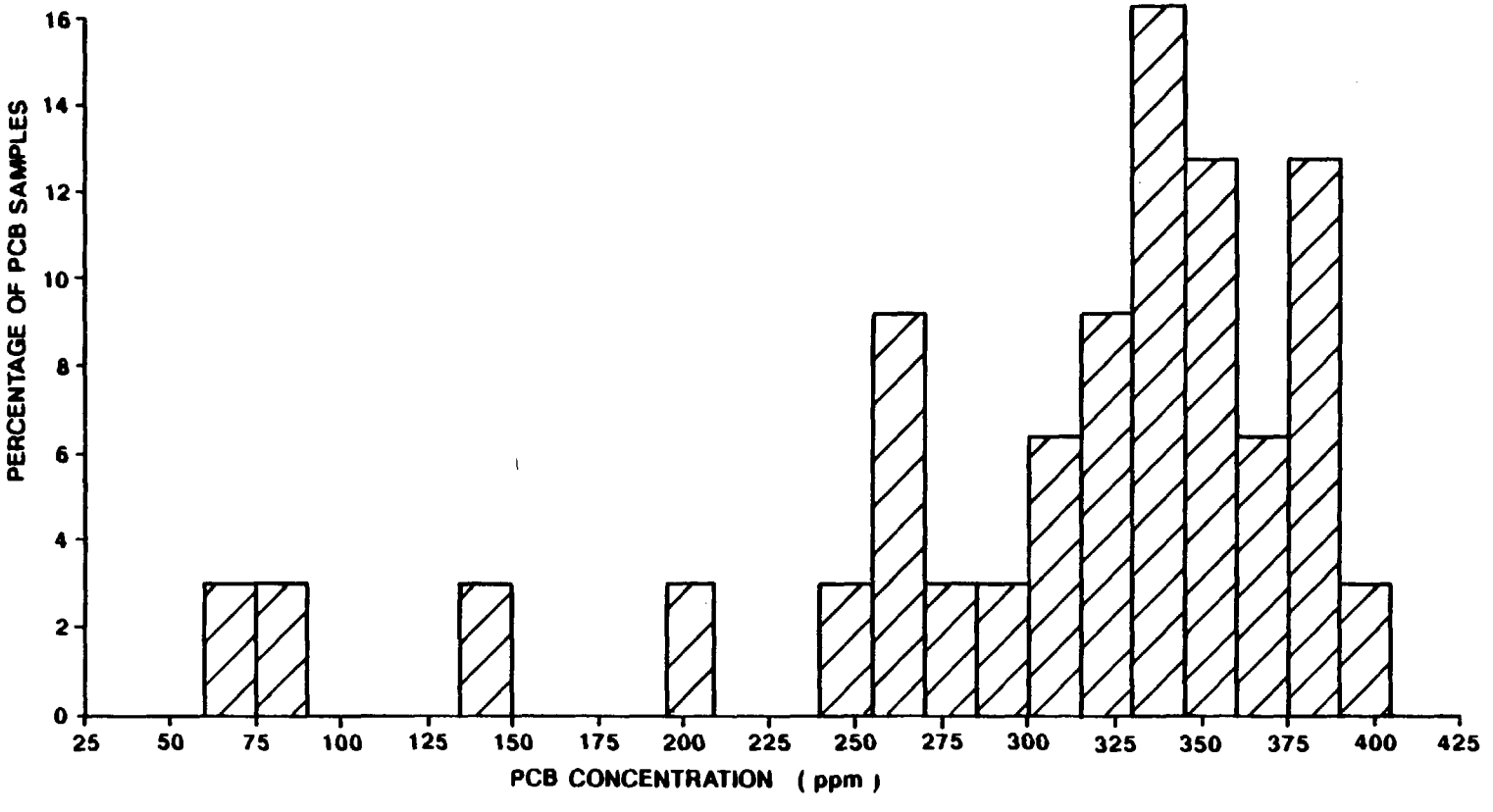

FIGURE 4-6
PCB DISTRIBUTION IN SEDIMENT IN THE COVE AREA (AREA I)
NEW BEDFORD, MASSACHUSETTS

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(3 to 5 ppm) (see Figures 4-4 and 4-5). Therefore, the risk estimates generated under probable exposure conditions are considered to best reflect the potential risks associated with this route of exposure. These values fall within the lower end of the target range and are between $2x10^{-7}$ and $3x10^{-6}$.

Summary. Risk from direct contact and ingestion of contaminated shoreline sediment is greatest for Area I. Exposure to sediment in all three subdivisions of this area (i.e., Upper Estuary, Lower Estuary, and Cove Area) resulted in risks for all age classes exceeding the target range of 10^{-4}. Risks were high even under probable exposure conditions (i.e., mean concentrations and probable exposure parameters). Exposure through direct contact to and ingestion of sediment around the Popes Island area was within or above the target range. Young children were considered to be at greater risk from contaminant exposure in this area than older children or adults. Ingestion of sediment from the Marsh Island area was associated within or above the target range. Methods to reduce risks associated with these exposure scenarios will be addressed in the FS. Exposure to sediment from other locations in Areas II and III was not considered to present a public health risk.

4.2.2.2 Biota

Exposure to PCBs through the ingestion of biota was assessed separately for lobster, winter flounder, and clams. These species were considered representative of biota most commonly consumed in the New Bedford Harbor site area. Exposure frequencies of one fish meal per day, per week, and per month were assessed. As discussed in Section 2.5.2 and presented in Table 2-9, the majority of the population in this area consumes fish less than once per month but greter than once per year. Each scenario assumes that the particular species evaluated comprises total seafood consumption. Incremental carcinogenic risk estimates for this route of exposure are in Table 4-9.

Risk estimates were derived for subchronic, chronic, and lifetime exposure durations, to the mean and maximum PCB concentrations detected in these species. As discussed in Section 2.5.2, the edible-tissue PCB concentration was used when available. PCB concentrations in the winter flounder, lobster (without tomalley), and clams ranged from 0.039 to 2.7 ppm, with only two concentrations greater than 2 ppm (see Table 2-9). Lobster concentration in edible tissue including tomalley ranged from 0.4 to 2.3 ppm (Pruell, 1988). Risks from ingestion of biota were evaluated separately for each area.

Area 1. Risk estimates based on exposure to biota obtained from Area 1 exceed the 10^{-4} risk level for the majority of exposure conditions evaluated. The best indicator of potential risks

TABLE 4-9. CARCINOGENIC RISK ESTIMATES FOR THE INGESTION OF BIOTA; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Area of Exposure	PCB Concen-tration (ppm)	CHILD Incremental Risks Sub-Chronic (1 year)	CHILD Incremental Risks Chronic (5 year)	OLDER CHILD Incremental Risks Sub-Chronic (1 year)	OLDER CHILD Incremental Risks Chronic (10 year)	ADULT Incremental Risks Sub-Chronic (1 year)	ADULT Incremental Risks Chronic (10 year)	Life time (70 yrs)
AREA 1								
Lobster								
Prob. daily	0.52	NA	NA	NA	NA	NA	NA	NA
weekly		NA	NA	NA	NA	NA	NA	NA
monthly		NA	NA	NA	NA	NA	NA	NA
Cons. daily	0.52	NA	NA	NA	NA	NA	NA	NA
weekly		NA	NA	NA	NA	NA	NA	NA
monthly		NA	NA	NA	NA	NA	NA	NA
Clam								
Prob. daily	0.689	8.6E-04	4.3E-03	4.3E-04	4.3E-03	2.4E-04	2.4E-03	2.2E-02
weekly		1.2E-04	6.1E-04	6.1E-05	6.1E-04	3.5E-05	3.5E-04	3.1E-03
monthly		2.8E-05	1.4E-04	1.4E-05	1.4E-04	8.1E-06	8.1E-05	7.3E-04
Cons. daily	2.121	2.6E-03	1.3E-02	1.3E-03	1.3E-02	7.6E-03	7.6E-02	4.4E-01
weekly		3.8E-04	1.9E-03	1.9E-04	1.9E-03	1.1E-04	1.1E-03	9.9E-03
monthly		8.6E-05	4.3E-04	4.3E-05	4.3E-04	2.5E-05	2.5E-04	2.2E-03
Flounder								
Prob. daily	1.039	1.3E-03	6.5E-03	6.5E-04	6.5E-03	3.7E-04	3.7E-03	3.3E-02
weekly		1.8E-04	9.2E-04	9.2E-05	9.2E-04	5.3E-05	5.3E-04	4.8E-03
monthly		4.2E-05	2.1E-04	2.1E-05	2.1E-04	1.2E-05	1.2E-04	1.1E-03
Cons. daily	2.629	3.2E-03	1.6E-02	1.6E-03	1.6E-02	9.3E-04	9.3E-03	8.3E-02
weekly		4.6E-04	2.3E-03	2.3E-04	2.3E-03	1.3E-04	1.3E-03	1.2E-02
monthly		1.0E-04	5.2E-04	5.2E-05	5.4E-04	3.1E-05	3.1E-04	2.8E-03
AREA 2								
Lobster (w/o tomalley)								
Prob. daily	0.57	7.0E-04	3.5E-03	3.5E-04	3.5E-03	2.0E-04	2.0E-03	1.8E-02
weekly		1.0E-04	5.0E-04	5.0E-05	5.0E-04	2.9E-05	2.9E-04	2.6E-03
monthly		2.4E-05	1.2E-04	1.2E-05	1.2E-04	6.7E-06	6.7E-05	6.1E-04
Cons. daily	1.234	1.5E-03	7.7E-03	7.7E-04	7.7E-03	4.4E-04	4.4E-03	4.0E-02
weekly		2.2E-04	1.1E-03	1.1E-04	1.1E-03	6.3E-05	6.3E-04	5.7E-03
monthly		5.0E-05	2.5E-04	2.5E-05	2.5E-04	1.5E-05	1.5E-04	1.3E-03
Lobster (tomalley)								
daily	2.3	2.8E-03	1.4E-02	1.4E-03	1.4E-02	8.1E-04	8.1E-03	7.3E-02
weekly		4.0E-04	2.0E-03	2.0E-04	2.0E-03	1.2E-04	1.2E-03	1.0E-02
monthly		9.7E-05	4.8E-04	4.8E-05	4.8E-04	2.7E-05	2.7E-04	2.5E-03

TABLE 4-9. CARCINOGENIC RISK ESTIMATES FOR THE INGESTION OF BIOTA; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Area of Exposure	PCB Concen-tration (ppm)	CHILD Incremental Risks Sub-Chronic (1 year)	CHILD Incremental Risks Chronic (5 year)	OLDER CHILD Incremental Risks Sub-Chronic (1 year)	OLDER CHILD Incremental Risks Chronic (10 year)	ADULT Incremental Risks Sub-Chronic (1 year)	ADULT Incremental Risks Chronic (10 year)	Life time (70 yrs)
Clam								
Prob. daily	0.231	2.8E-04	1.4E-03	1.4E-04	1.4E-03	8.2E-05	8.2E-04	7.3E-03
weekly		4.0E-05	2.0E-04	2.0E-05	2.0E-04	1.2E-05	1.2E-04	1.1E-03
monthly		9.4E-06	4.7E-05	4.7E-06	4.7E-05	2.7E-06	2.7E-05	2.4E-04
Cons. daily	1.181	1.5E-03	7.4E-03	7.4E-04	7.4E-03	4.2E-04	4.2E-03	3.8E-02
weekly		2.0E-04	1.0E-03	1.0E-04	1.0E-03	6.0E-05	6.0E-04	5.3E-03
monthly		4.8E-05	2.4E-04	2.4E-05	2.4E-04	1.4E-05	1.4E-04	1.3E-03
Flounder								
Prob. daily	0.371	4.6E-04	2.3E-03	2.3E-04	2.3E-03	1.3E-04	1.3E-03	1.2E-02
weekly		6.6E-05	3.3E-04	3.3E-05	3.3E-04	1.9E-05	1.9E-04	1.7E-03
monthly		1.5E-05	7.6E-05	7.6E-06	7.6E-05	4.3E-06	4.3E-05	3.9E-04
Cons. daily	1.048	1.3E-03	6.5E-03	6.5E-04	6.5E-03	3.7E-04	3.7E-03	3.3E-02
weekly		1.9E-04	9.3E-04	9.3E-05	9.3E-04	5.3E-05	5.3E-04	4.8E-03
monthly		4.4E-05	2.2E-04	2.2E-05	2.2E-04	1.2E-05	1.2E-04	1.1E-03
AREA 3								
Lobster (w/o tomalley)								
Prob. daily	0.213	2.6E-04	1.3E-03	1.3E-04	1.3E-03	7.6E-05	7.6E-04	6.8E-03
weekly		3.8E-05	1.9E-04	1.9E-05	1.9E-04	1.1E-05	1.1E-04	9.8E-04
monthly		8.8E-06	4.4E-05	4.4E-06	4.4E-05	2.5E-06	2.5E-05	2.3E-04
Cons. daily	0.351	4.4E-04	2.2E-03	2.2E-04	2.2E-03	1.2E-04	1.2E-03	1.1E-02
weekly		6.2E-05	3.1E-04	3.1E-05	3.1E-04	1.8E-05	1.8E-04	1.6E-03
monthly		1.4E-05	7.2E-05	7.2E-06	7.2E-05	4.1E-06	4.1E-05	3.7E-04
Lobster (tomalley)								
daily	1.4	1.7E-03	8.5E-03	8.5E-04	8.5E-03	5.0E-04	5.0E-03	4.5E-02
weekly		2.3E-04	1.2E-03	1.2E-04	1.2E-03	7.2E-05	7.2E-04	6.3E-03
monthly		5.8E-06	2.9E-05	2.9E-06	2.9E-05	1.6E-05	1.6E-04	9.6E-04
Clam								
Prob. daily	0.156	1.9E-04	9.7E-04	9.7E-05	9.7E-04	5.6E-05	5.6E-04	5.0E-03
weekly		2.8E-05	1.4E-04	1.4E-05	1.4E-04	7.9E-06	7.9E-05	7.1E-04
monthly		6.4E-06	3.2E-05	3.2E-06	3.2E-05	1.8E-06	1.8E-05	1.6E-04
Cons. daily	0.478	6.0E-04	3.0E-03	3.0E-04	3.0E-03	1.7E-04	1.7E-03	1.5E-02
weekly		8.4E-05	4.2E-04	4.2E-05	4.2E-04	2.4E-05	2.4E-04	2.2E-03
monthly		2.0E-05	9.8E-05	9.8E-06	9.8E-05	5.6E-06	5.6E-05	5.0E-04

TABLE 4-9. CARCINOGENIC RISK ESTIMATES FOR THE INGESTION OF BIOTA; CHILDREN; OLDER CHILDREN AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Area of Exposure	PCB Concen-tration (ppm)	CHILD Incremental Risks Sub-Chronic (1 year)	CHILD Incremental Risks Chronic (5 year)	OLDER CHILD Incremental Risks Sub-Chronic (1 year)	OLDER CHILD Incremental Risks Chronic (10 year)	ADULT Incremental Risks Sub-Chronic (1 year)	ADULT Incremental Risks Chronic (10 year)	Life time (70 yrs)
Flounder								
Prob. daily	0.278	3.4E-04	1.7E-03	1.7E-04	1.7E-03	9.9E-05	9.9E-04	8.8E-03
weekly		5.0E-05	2.5E-04	2.5E-05	2.5E-04	1.4E-05	1.4E-04	1.3E-03
monthly		1.1E-05	5.7E-05	5.7E-06	5.7E-05	3.3E-06	3.3E-05	3.0E-04
Cons. daily	0.825	1.0E-03	5.1E-03	5.1E-04	5.1E-03	2.9E-04	2.9E-03	2.6E-02
weekly		1.5E-04	7.3E-04	7.3E-05	7.3E-04	4.2E-05	4.2E-04	3.8E-03
monthly		3.4E-05	1.7E-04	1.7E-05	1.7E-04	9.7E-05	9.7E-04	5.7E-03
AREA 4								
Lobster (w/o tomalley)								
Prob. daily	0.069	8.0E-05	4.0E-04	4.0E-05	4.0E-04	2.3E-05	2.3E-04	2.1E-03
weekly		1.1E-05	5.7E-05	5.7E-06	5.7E-05	3.2E-06	3.2E-05	2.9E-04
monthly		2.6E-06	1.3E-05	1.3E-06	1.3E-05	7.5E-07	7.5E-06	6.7E-05
Cons. daily	0.176	2.2E-04	1.1E-03	1.1E-04	1.1E-03	6.3E-05	6.3E-04	5.7E-03
weekly		3.2E-05	1.6E-04	1.6E-05	1.6E-04	8.9E-06	8.9E-05	8.1E-04
monthly		7.2E-06	3.6E-05	3.6E-06	3.6E-05	2.1E-06	2.1E-05	1.9E-04
Lobster (tomalley)								
daily	0.4	4.6E-04	2.3E-03	2.3E-04	2.3E-03	1.3E-04	1.3E-03	1.2E-02
weekly		6.6E-05	3.3E-04	3.3E-05	3.3E-04	1.9E-05	1.9E-04	1.7E-03
monthly		1.5E-05	7.5E-05	7.5E-06	7.5E-05	4.3E-06	4.3E-05	3.9E-04
Clam								
Prob. daily	0.039	5.0E-05	2.5E-04	2.5E-05	2.5E-04	1.4E-05	1.4E-04	1.3E-03
weekly		7.0E-06	3.5E-05	3.5E-06	3.5E-05	2.0E-06	2.0E-05	1.8E-04
monthly		1.6E-06	8.1E-06	8.1E-07	8.1E-06	4.7E-07	4.7E-06	4.2E-05
Cons. daily	0.137	1.7E-04	8.5E-04	8.5E-05	8.5E-04	4.9E-05	4.9E-04	4.4E-03
weekly		2.4E-05	1.2E-04	1.2E-05	1.2E-04	6.9E-06	6.9E-05	6.2E-04
monthly		5.6E-06	2.8E-05	2.8E-06	2.8E-05	1.6E-06	1.6E-05	1.4E-04
Flounder								
Prob. daily	0.101	1.3E-04	6.3E-04	6.3E-05	6.3E-04	3.6E-05	3.6E-04	3.2E-03
weekly		1.8E-05	9.0E-05	9.0E-06	9.0E-05	5.1E-06	5.1E-05	4.6E-04
monthly		4.2E-06	2.1E-05	2.1E-06	2.1E-05	1.2E-06	1.2E-05	1.1E-04
Cons. daily	0.339	4.2E-04	2.1E-03	2.1E-04	2.1E-03	1.2E-04	1.2E-03	1.1E-02
weekly		6.0E-05	3.0E-04	3.0E-05	3.0E-04	1.7E-05	1.7E-04	1.5E-03
monthly		1.4E-05	7.0E-05	7.0E-06	7.0E-05	4.0E-06	4.0E-05	3.6E-04

The cancer potency factor for PCBs is 7.7 (mg/kg-day)-1
Prob. = Probable exposure conditions.
Cons. = Conservative exposure conditions.
Lifetime = Incremental carcinogenic risk for 70 year exposure.
NA = Data not available.

from exposure to biota from this area is the clam because this organism is sessile and lives its entire life within the contaminated sediment from this area. (Winter flounder is a migratory species and spends a portion of its life cycle outside the contaminated area; lobster is not expected to inhabit this area because of the physical and chemical conditions of the Upper Estuary.) Risk estimates based on ingestion of clams fall within or exceed the target range of 10^{-4} to 10^{-7}. These estimates range from $8x10^{-6}$ to $2x10^{-2}$. Risk estimates for ingestion of winter flounder range from $1x10^{-5}$ to $2x10^{-2}$. Because of the frequency and magnitude to which these values exceed the target range, methods to reduce risks associated with ingestion of biota will be addressed in the FS.

Area 2. Incremental carcinogenic risk estimates based on consumption of biota obtained for Area 2 were within or exceeded the target range of 10^{-4} to 10^{-7}. Chronic exposure through the daily or weekly ingestion of any species (i.e., clam, lobster, or winter flounder) containing the mean PCB concentration resulted in risk estimates that exceed $3x10^{-5}$. Ingestion of lobster (including the tomalley) presented the highest risks. These risks ranged from $3x10^{-5}$ to $2x10^{-2}$. Methods to reduce risks from ingestion of biota from this area will be considered in the FS.

Area 3. Exposure through the consumption of biota obtained from Area 3 results in incremental risks in excess of 10^{-4} for most scenarios. Risks in excess of $6x10^{-5}$ are noted even when assuming probable exposure conditions. Methods to reduce these risks will be addressed in the FS. As in Area 2, ingestion of lobsters (including the tomalley) presented the greatest risk.

Area 4. Biota concentrations detected in Area 4 were lower than other areas (0.039 to 0.4 ppm). However, risk estimates based on exposure to PCB concentrations observed in biota from this area still fall within or above the target range ($2x10^{-6}$ to $2x10^{-3}$). Methods to reduce these risks will be addressed in the FS. The highest risks were associated with ingestion of lobster (including the tomalley).

Summary. Risks from ingestion of contaminated biota, when assessed for all species and areas, fall within or exceed the target range for most scenarios, even when assuming probable exposure conditions (see Table 4-9). The highest risks were associated with the ingestion of lobster including the tomalley. Risks associated with ingestion of lobster excluding tomalley were consistently lower, indicating that persons who consume tomalley are potentially at greater risk from PCB exposure than persons who do not consume tomalley. In addition, high incremental carcinogenic risks are estimated for lifetime exposure to PCBs from this route of exposure. Many of the lifetime exposure scenarios exceed the $1x10^{-3}$ risk level.

Methods to reduce the risks from contaminant exposure via ingestion of biota will be addressed in the FS.

4.2.2.3 Air

Limited data were available to assess risks associated with inhalation exposure to PCBs. Risk estimates associated with the probable and conservative scenarios for subchronic and chronic exposures ranged from $1x10^{-7}$ to $3x10^{-4}$ and are in Table 4-10. The data available for risk characterization were taken from areas distant from receptor locations and were considered indicative only of maximum concentrations from certain point source areas (i.e., the Hot Spot Area). Therefore, it was difficult to interpret the potential risk to public health from this route of exposure.

An interpretation of the assumed background PCB concentrations of 10 ng/m^3 was also made in this risk assessment (NUS, 1986). Assessing exposure to PCBs at this concentration results in risk estimates at the lower end of the target range (10^{-6} to 10^{-7}). The conservative nature of the exposure assumptions (i.e., continual exposure, complete absorption, and PCBs exclusively in the vapor phase) in this analysis suggests that actual risks from a background exposure of 10 ng/m^3 may be even lower. The lifetime risk associated with a 70-year exposure duration to the estimated 10 ng/m^3 background level is $8x10^{-6}$.

4.2.3 Risk Summary

The noncarcinogenic and carcinogenic risks associated with exposure to PCBs, cadmium, copper, and lead are summarized by route of exposure in the following subsections.

4.2.3.1 Direct Contact with Sediment

Noncarcinogenic and carcinogenic risks associated with direct contact exposure to PCB-, cadmium-, copper-, and lead-contaminated sediment were evaluated separately for Areas I, II, and III, and focused on locations within these areas where exposure was likely to occur. Contaminant concentrations detected in shoreline sediments were used when available.

Noncarcinogenic risk estimates for exposure to sediment in Area I exceeded 1 under the majority of scenarios evaluated and ranged from 0.7 to 200. Exposure to PCBs accounted for the majority of the risk. Individual risk ratios for cadmium, copper, and lead were all below 1. The noncarcinogenic risk ratios associated with PCB exposure in Area I indicate a potential public health risk. Young children were considered to be at greatest risk.

Exposure to sediment from Areas II and III was associated with noncarcinogenic risk ratios ranging from less than 1 to 3. The

TABLE 4-10. CARCINOGENIC RISK ESTIMATES FOR INHALATION OF AIR; CHILDREN, OLDER CHILDREN, AND ADULTS; NEW BEDFORD, MASSACHUSETTS.

Location of Exposure	PCB Concen-tration (ng/m3)	CHILD		OLDER CHILD		ADULT		
		Sub-Chronic (1 year)	Sub-Chronic (5 years)	Sub-Chronic (1 year)	Chronic (10 year)	Sub-Chronic (1 year)	Chronic (10 year)	Life time (70 yrs)
ALL AREAS								
Background	10	1.80E-07	9.20E-07	1.80E-07	1.80E-06	1.00E-07	1.00E-06	8.00E-06
Prob.	85	1.60E-06	7.80E-06	1.60E-06	1.60E-06	8.80E-07	8.80E-06	6.00E-05
Cons.	471	2.60E-05	1.30E-04	2.60E-05	2.60E-04	1.50E-05	1.50E-04	1.00E-03

The cancer potency factor for PCBs is 7.7 (mg/kg-day)-1
Prob. = Probable exposure conditions.
Cons. = Conservative exposure conditions.
Lifetime = Incremental carcinogenic risks from 70 year exposure.

only risk ratios to exceed 1 were based on conservative exposure assumptions which were not considered representative of likely exposure conditions for these areas. These include long-term repetitive exposure to the maximum detected contaminant concentration. The risk ratios based on more realistic exposure conditions were less than 1. Based on this evaluation, the noncarcinogenic risk for direct contact exposure in Areas II and III was not considered to pose a risk to public health.

The carcinogenic risks associated with direct contact exposure to sediment was greatest for Area I. The risk estimates based on exposure by a child, older child and adult, ranged from $1x10^{-6}$ to $2x10^{-2}$, with the majority of scenarios associated with risks in excess of EPA's target risk range of 10^{-4} to 10^{-7}. Based on this evaluation, methods to reduce these risks will be addressed in the FS.

The carcinogenic risks estimated for Area II assuming probable exposure conditions ranged from $2x10^{-7}$ to $5x10^{-6}$. The only risk estimates exceeding the target range were those associated with exposure to PCBs under conservative exposure conditions. Since these conditions assume repetitive, long-term exposure to the maximum PCB concentration, the associated risks were considered to be overly conservative. As stated, exposure under more realistic conditions were associated with risks in the lower end of the target range.

In Area III, the carcinogenic risks ranged from $1x10^{-8}$ to $2x10^{-6}$ under probable exposure conditions, and from $2x10^{-7}$ to $1x10^{-4}$ under conservative exposure conditions. No risk estimates exceeded EPA's target risk range.

4.2.3.2 Ingestion of Sediment

Exposure through ingestion of sediment was considered to be an age-related activity and most significant for children less than six years. Both noncarcinogenic and carcinogenic risks associated with this route of exposure were evaluated.

Noncarcinogenic risk associated with exposure to cadmium- and copper-contaminated sediment in all three areas was below 1 for all scenarios evaluated. Risk ratios based on exposure to PCB- and lead-contaminated sediment exceeded 1 under certain scenarios. For Area I, risk ratios for PCBs and lead ranged from 11 to 175 and 26 to 33, respectively. The magnitude and extent to which these values exceed 1 indicates that ingestion of sediment from Area I presents a potential health risk to children.

Risk ratios based on ingestion of PCB-contaminated sediment in Area II and III ranged from below 1 to 17. However, risk ratios based on exposure at recreational locations and under probable

exposure conditions within these areas were all below 1. Since these scenarios were considered representative of actual exposure conditions, ingestion of sediment from Areas II and III was not considered to present a noncarcinogenic health risk.

The incremental carcinogenic risks associated with exposure through ingestion of sediment were greatest for Area I and ranged from $6x10^{-6}$ to $1x10^{-2}$. These risk estimates were based on exposure to sediment in areas where access by children is considered possible. These risk fall within and exceeded the EPA's target range of 10^{-4} to 10^{-7}. As such, methods to reduce these risks will be addressed in the FS.

The risk estimates based on exposure in Area II ranged from $9x10^{-7}$ to $2x10^{-4}$, with the majority of risk values falling between 10^{-5} and 10^{-6}. Risk estimates based on probable exposure conditions ranged from $9x10^{-7}$ to $2x10^{-5}$. The risks based on exposure in Area III fall within the lower end of the target range and are between $2x10^{-7}$ to $3x10^{-6}$.

<u>Summary of Sediment Exposure</u>. The risks associated with exposure via direct contact with and ingestion of contaminated shoreline sediment are greatest for Area I. Both the carcinogenic and noncarcinogenic risk estimates based on exposure to PCBs in this area exceeded the criteria levels established by EPA. Noncarcinogenic risks based on exposure to metals in this area were below levels considered to represent a public health risk. Methods to reduce these carcinogenic risks from PCB exposure will be evaluated in the FS.

Risk estimates based on exposure to sediment from other areas in the New Bedford Harbor were less than those developed for Area I. Noncarcinogenic risks based on exposure to PCBs and metals were below levels considered to represent a public health concern. Carcinogenic risks associated with probable exposure conditions via direct contact with and ingestion of sediment from Areas II and III ranged from less than 10^{-7} to $8x10^{-5}$. The majority of risks were between 10^{-6} to 10^{-5}. Young children were considered to be at a greater risk from contaminant exposure than either older children or adults.

Risk estimates based on acute exposure to sediment, representing intermittent or once-in-a-lifetime exposure, were not considered to present a public health risk.

4.2.3.3 Ingestion of Aquatic Biota

Exposure to PCBs and metals via ingestion of biota was evaluated for potential noncarcinogenic and carcinogenic risks. Three species were considered in this evaluation: winter flounder, clam, and lobster. Separate scenarios were developed for each species and assumed that 100 percent of the seafood diet was

comprised of these species. A standard 8-ounce (i.e., 227 grams) fish meal was assumed for older children and adults and 4-ounce (i.e., 115 grams) fish meal was assumed for younger children.

Risk ratios based on exposure to cadmium and copper by older children and adults ranged from below 1 to 7.9. Ratios in excess of 1 were based on daily ingestion frequencies and whole body tissue concentrations. These conservative assumptions may overestimate the actual risks, suggesting that exposure to cadmium and copper may not present a public health concern. However, exposure to cadmium and copper by children resulted in risk ratios ranging from below 1 to 16. Since young children are more susceptible to contaminant exposure than older children and adults, this route of exposure was considered to present a greater risk to a child's health.

Risk ratios based on exposure to lead and PCBs via ingestion of biota for all age classes exceeded 1 for the majority of scenarios evaluated. No one area or species appeared to consistently present a greater risk from exposure to these compounds. Based on this evaluation, exposure to lead and PCBs through ingestion of biota presents a public health risk.

Incremental carcinogenic risks associated with ingestion of biota fall within or exceed EPA's target range. Many of the scenarios evaluated had associated lifetime risks in excess of 10^{-3}. The risk estimates based on chronic exposure range from $1x10^{-5}$ to $9x10^{-3}$ for Area 1; from $4x10^{-6}$ to $1x10^{-2}$ for Area 2; from $6x10^{-6}$ to $9x10^{-3}$ for Area 3; and from $1x10^{-6}$ to $2x10^{-3}$ for Area 4. Ingestion of lobster, including tomalley, presents the greatest risk from exposure to PCBs.

Methods to reduce the noncarcinogenic risks from exposure to cadmium, copper, lead, and PCBs and carcinogenic risks from exposure to PCBs will be assessed in the FS.

4.2.3.4 Inhalation of Airborne Contaminants

Limited air data were available to assess risks associated with inhalation exposure to PCBs. The data available for risk evaluation were collected from sampling stations distant from receptor location. These areas were chosen to provide a measure of the maximum PCB concentrations in the air above the mudflats in Area I. Using these concentrations to assess potential risk was considered to be overly conservative.

Lifetime exposure to the assumed "background" concentration of 10 ng/m^3 for the New Bedford area was assessed and associated with incremental carcinogenic risks in the 10^{-6} range. These risk estimates were based on conservative exposure conditions, suggesting that actual risks from this route of exposure are less than 10^{-6}.

4.3 OVERALL SITE RISKS

The risk evaluation performed in Section 4.2 focused on the carcinogenic and noncarcinogenic risks from a single exposure pathway. Based on this evaluation, exposure to contaminants through ingestion of and direct contact with sediment in Area I and ingestion of biota from all areas may result in potential risks to human health. PCBs were identified as the major contaminant of concern. Noncarcinogenic risks in excess of EPA's criterion were also attributed to lead exposure through the ingestion of biota (all age classes). In addition, young children (zero to 5 years) were considered to be at a higher risk from cadmium and copper exposure through the ingestion of biota than older children and adults.

The total site risk associated with multimedia and multitoxic exposure was generated by summing the individual risk estimates developed for the ingestion of and direct contact with sediment, ingestion of biota, and inhalation of air. This scenario represents the risks associated with concurrent or sequential exposure to contaminants through multiple exposure pathways. These risk estimates are listed in Table 4-11. Total site risk estimates were evaluated against the MCP criteria of $1x10^{-5}$ incremental carcinogenic risk level and 0.2 noncarcinogenic HI.

The total site risks evaluated in this report were based on chronic exposure via ingestion of, direct contact with, and inhalation of PCBs, cadmium, copper, and lead under probable exposure conditions. The carcinogenic and noncarcinogenic risk estimates for each age class and area assessed exceed 10^{-5} and 0.2, respectively. Based on this evaluation, methods to reduce the overall site risk will be addressed in the FS.

Table 4-11. SUMMARY TABLE OF TOTAL SITE CARCINOGENIC AND NONCARCINOGENIC RISKS - PROBABLE EXPOSURE SCENARIO; NEW BEDFORD, MASSACHUSETTS.

AREA 1 (1)

	Cancer Risk	Hazard Index
YOUNG CHILD		
Ingestion of biota	7.65E-04 (2)	17.00 (3)
Ingestion of sediments (4/5)	1.50E-05	3.40
Direct Contact/Sediments (5)	7.50E-06	0.17
Total	7.88E-04	20.57
OLDER CHILD		
Ingestion of biota	7.65E-04	8.50
Ingestion of sediments	NE	NE
Direct Contact/Sediments	9.50E-05	0.06
Total	8.60E-04	8.56
ADULT		
Ingestion of biota	4.40E-04	4.90
Ingestion of sediments	NE	NE
Direct Contact/Sediments	3.75E-05	0.02
Total	4.78E-04	4.92
LIFETIME		
Ingestion of biota	3.18E-03	
Ingestion of sediments	4.02E-05	
Direct Contact/Sediments	4.29E-04	
Total	3.65E-03	

AREA 2

	Cancer Risk	Hazard In
YOUNG CHILD		
Ingestion of biota	3.43E-04 (2)	9.43
Ingestion of sediments (4/6)	2.00E-06	1.20
Direct Contact/Sediments (6)	2.65E-06	0.02
Total	3.48E-04	10.65
OLDER CHILD		
Ingestion of biota	3.43E-04	4.77
Ingestion of sediments	NE	NE
Direct Contact/Sediments	2.60E-06	0.01
Total	3.46E-04	4.77
ADULT		
Ingestion of biota	2.00E-04	2.67
Ingestion of sediments	NE	NE
Direct Contact/Sediments	1.00E-06	0.01
Total	2.01E-04	2.68
LIFETIME		
Ingestion of biota	1.45E-03	
Ingestion of sediments	2.00E-06	
Direct Contact/Sediments	1.08E-05	
Total	1.46E-03	

Table 4-11. SUMMARY TABLE OF TOTAL SITE CARCINOGENIC AND NONCARCINOGENIC RISKS - PROBABLE EXPOSURE SCENARIO; NEW BEDFORD, MASSACHUSETTS.

AREA 3

	Cancer Risk	Hazard Index
YOUNG CHILD		
Ingestion of biota (2)	1.93E-04	6.40
Ingestion of sediments	NA	NA
Direct Contact/Sediments	NA	NA
Total	1.93E-04	6.40
OLDER CHILD		
Ingestion of biota	1.93E-04	3.23
Ingestion of sediments	NE	NE
Direct Contact/Sediments	NA	NA
Total	1.93E-04	3.23
ADULT		
Ingestion of biota	1.10E-04	1.83
Ingestion of sediments	NE	NE
Direct Contact/Sediments	NA	NA
Total	1.10E-04	1.83
LIFETIME		
Ingestion of biota	4.91E-04	
Ingestion of sediments	NA	
Direct Contact/Sediments	NA	
Total	4.91E-04	

AREA 4

	Cancer Risk	Hazard Index
YOUNG CHILD		
Ingestion of biota (2)	6.07E-05	4.23
Ingestion of sediments	NA	NA
Direct Contact/Sediments	NA	NA
Total	6.07E-05	4.23
OLDER CHILD		
Ingestion of biota	6.07E-05	2.13
Ingestion of sediments	NE	NE
Direct Contact/Sediments	NA	NA
Total	6.07E-05	2.13
ADULT		
Ingestion of biota	3.43E-05	1.23
Ingestion of sediments	NE	NE
Direct Contact/Sediments	NA	NA
Total	3.43E-05	1.23
LIFETIME		
Ingestion of biota	2.50E-04	
Ingestion of sediments	NA	
Direct Contact/Sediments	NA	
Total	2.50E-04	

1. These Areas correspond geographically to the subdivision of the New Bedford Harbor depicted in Figure 2-5.
2. Cancer risks for ingestion of biota reflect the mean values for the three species evaluated under the weekly ingestion, chronic exposure, probable scenario.
3. Hazard indices for ingestion of biota reflect the mean values for the three species evaluated.
4. Ingestion of sediments was only evaluated for young children.
5. Hazard indices and carcinogenic risk for direct contact with and ingestion of sediments in Area 1 represent the mean values estimated for chronic exposure to sediments from Areas I and II in Tables 4-2, 4-3, 4-7 and 4-8.
6. Hazard indices and carcinogenic risk for direct contact with and ingestion of sediments in Area 1 represent the mean values estimated for chronic exposure to sediments from Areas III in Tables 4-2, 4-3, 4-7 and 4-8.

NE - not evaluated.
NA - data not available.

Area 1 corresponds geographically to Areas I and II as depicted in Figure 2-4.
Area 2 corresponds geographically to Area III as depicted in Figure 2-4.
Exposure to sediments in Areas 3 and 4 were not evaluated in this risk assessment.

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APPENDIX A

EXPOSURE ASSUMPTIONS AND BODY DOSE CALCULATIONS FOR THE SCREENING SCENANRIOS

TABLE A-1

EXPOSURE ASSUMPTIONS FOR INGESTION OF BIOTA: SCREENING SCENARIO

Exposure Parameter	Value
Average Weight Over Period of Exposure	40 kg
Duration of Exposure	10 years
Frequency of Exposure	52 exp/year
Amount Ingested	227 grams
Gastrointestinal Toxicokinetic Factor	1.0

Contaminant Concentration	Average Daily Exposure Dose
8.2 ug/g (reported by Battelle)	9.5×10^{-4} (mg/kg-day)

3.88.80
0017.0.0

TABLE A-2

EXPOSURE ASSUMPTIONS FOR DIRECT CONTACT WITH SEDIMENTS: SCREENING SCENARIO

Exposure Parameter	Value
Average Weight Over Period of Exposure	40 kg
Duration of Exposure	10 years
Frequency of Exposure	100 exp/year
Amount of Sediment Contacted*	6.6 grams/exp
Dermal Toxicokinetic Factor	0.5

Contaminant Concentration	Average Daily Exposure Dose
17,404 mg/kg (reported by Battelle)	5.7×10^{-2} (mg/kg-day)

* Surface Area (4,415 cm^2) x Deposition Factor (1.5 mg/cm^2) = 6.6 grams/exposure.

3.88.80
0018.0.0

TABLE A-3

EXPOSURE ASSUMPTIONS FOR DIRECT CONTACT WITH SURFACE WATER: SCREENING SCENARIO

Exposure Parameter	Value
Average Weight Over Period of Exposure	40 kg
Duration of Exposure	10 years
Frequency of Exposure	100 exp/year
Hours Exposed	2.6 hrs/exp
Exposed Surface Area	11,900 cm^2
Flux Rate of Contaminant Across Skin	0.5 $mg/cm^2/hr$

Contaminant Concentration	Average Daily Exposure Dose
Weight Fraction of Penetrant in H_2O	
$\frac{0.035\ mg}{1000\ gm\ H_2O}$ = PCBs	5.3×10^{-7} (mg/kg-day)
$\frac{0.00029\ mg}{1000\ mg\ H_2O}$ = Cadmium	4.3×10^{-4} (mg/kg-day)
$\frac{0.004\ mg}{1000\ mg\ H_2O}$ = Lead	6.0×10^{-8} (mg/kg-day)
$\frac{0.0094\ mg}{1000\ mg\ H_2O}$ = Copper	1.4×10^{-7} (mg/kg-day)

TABLE A-4

EXPOSURE ASSUMPTIONS FOR INCIDENTAL INGESTION OF SURFACE WATER: SCREENING SCENARIO

Exposure Parameter	Value
Average Weight Over Period of Exposure	40 kg
Duration of Exposure	10 years
Frequency of Exposure	100 exp/year
Amount Ingested	100 mls/exp
Gastrointestinal Toxicokinetic Factor	1.0

Contaminant Concentration	Average Daily Exposure Dose
35 μg/ℓ (reported by Battelle)-PCB	3.4×10^{-6} (mg/kg-day)
0.29 μg/ℓ Cadmium	2.8×10^{-11} (mg/kg-day)
4 μg/ℓ Lead	3.9×10^{-7} (mg/kg-day)
9.9 μg/ℓ Copper	9.1×10^{-7} (mg/kg-day)

TABLE A-5

EXPOSURE ASSUMPTIONS FOR INHALATION OF AIRBORNE CONTAMINANTS: SCREENING SCENARIO

Exposure Parameter	Value
Average Weight Over Period of Exposure	10 kg
Duration of Exposure	5 years
Frequency of Exposure	24 hours/day
Amount Inhaled	5 m^3/day
Respiratory Toxicokinetic Factor	1.0

Contaminant Concentration	Average Daily Exposure Dose
471 ng/m^3 (reported by NUS)	1.7×10^{-5} (mg/kg-day)

3.88.80
0021.0.0

TABLE A-6

EXPOSURE ASSUMPTIONS FOR INGESTION OF SEDIMENTS: SCREENING SCENARIO

Exposure Parameter	Value
Average Weight Over Period of Exposure	10 kg
Duration of Exposure	3 years
Frequency of Exposure	100 exp/year
Amount Ingested	0.5 grams/exp
Gastrointestinal Toxicokinetic Factor	1.0

Contaminant Concentration	Average Daily Exposure Dose
17,404 mg/kg (reported by Battelle)	1.0×10^{-2} (mg/kg-day)

3.88.80
0022.0.0

APPENDIX B

DERIVATION OF THE TOXICOKINETIC FACTORS FOR PCBs

APPENDIX B
PCB TOXICOKINETIC FACTORS FOR USE IN NEW BEDFORD HARBOR RISK ASSESSMENT

Because the dose/response information employed in risk assessments is derived from toxicological studies that are based on administered doses, it is important in a quantitative risk assessment to estimate an administered dose, not an absorbed dose. In comparing two administered doses, it may be necessary to make adjustments if the efficiency of absorption is known or expected to differ because of physiological effects and/or matrix or vehicle effects. The toxicokinetic factor (TKF) is used for this purpose. The TFK is defined as the ratio of the estimated dermal absorption factor for contaminated soil or sediment to the absorption factor for the laboratory toxicology study from which the cancer potency factor or reference dose was derived. Most commonly, this will be a study where the test compound was administered orally. For PCBs, the cancer potency factor was derived from a long-term feeding study with laboratory animals.

PCB TKFs have been developed for two types of contaminated sediment: heavily contaminated sediments in which the concentration of total PCBs exceeds 1 percent; and less contaminated sediments. The two approaches are required because PCBs not adsorbed to matrix matter are present in samples that are contaminated in the percent range. In lesser contaminated sediments, PCBs are adsorbed to matrix components. It is appropriate to consider the effects of matrix components in reducing the bioavailability of PCBs only when the ratio of sediment to PCBs is large, such as when PCBs are present at ppm levels.

To estimate the two TKFs for dermal exposure to contaminated sediments, the following three factors have been derived:

(1) The gastrointestinal absorption factor for the study from which the EPA cancer potency factor was derived (Norback and Weltman, 1985) is estimated at <u>80%</u>.

(2) The dermal absorption factor for pure PCBs is estimated at <u>41%</u>.

(3) The dermal absorption factor for PCBs in sediments contaminated with PCBs at levels below 1% is estimated at <u>5.4%</u>.

The supporting documentation for these estimates appears in the accompanying appendices.

The TKFs for use in risk assessment are derived below:

(A) TKF for highly contaminated sediments ([PCBs]>1%):

$$TKF = \frac{\text{Absorption, dermal, pure compound}}{\text{Absorption, oral, diet}} = \frac{41\%}{80\%} = 50\% \ (0.50)$$

(B) TKF for moderately contaminated sediments ([PCBs]<1%):

$$TKF = \frac{\text{Absorption, dermal sediment}}{\text{Absorption, oral, diet}} = \frac{5.4\%}{80\%} = 7\% \ (0.07)$$

For risk assessment, the <u>administered</u> doses from contaminated sediment should appear in the equations for estimated body dose levels, and they should be multiplied by the appropriate TKF before computation of carcinogenic risk. The relevant exposure level of PCB-contaminated sediment is estimated by multiplying surface area (cm^2) by the deposition factor (mg/cm^2). Because of the nature of the experiments from which the above absorption factors were derived, the estimated deposition factor should not exceed 15 mg/cm^2. Optimal accuracy will be achieved for moderately contaminated sediments (<1% PCBs), with deposition factors of 1.5-15 mg/cm^2, because this range of deposited sediment per unit area is similar to the amount of pure PCB administered to experimental animals per unit area. For highly contaminated sediments (>1% PCBs), optimal accuracy will be achieved with deposition factors of 1.5 mg/cm^2 or less. This is because a smaller fraction of the sediment-adsorbed PCBs in contact with the skin is available for absorption compared to pure PCBs.

Supporting Documentation for PCB Toxicokinetic Factor

Gastrointestinal Absorption Factor for Norback and Weltman (1985) Study

This study, from which the current EPA carcinogenic potency factor is derived, is a chronic feed study using Sprague-Dawley rats. PCBs (Arochlor 1260) were administered in the diet. Arochlor 1260 was mixed in corn oil and then added to Purina Rat Chow. No information on the efficiency of gastrointestinal absorption was available from the study. To estimate the efficiency of gastrointestinal absorption, the toxicological literature was searched for appropriate studies on PCBs. Six studies were identified that contained relevant absorption information:

(1) Allen, et. al. (1975) gave single oral doses of 2,5,2',5'-tetrachlorobiphenyl (18 mg/kg bw) to four adult rhesus monkeys by gastric intubation. PCBs were given in 2.5 mL of corn oil on an empty stomach. Unmetabolized PCBs were analyzed in the feces by GC. Minimum gastrointestinal absorption was found to be 88%. PCBs found in the feces over specified post-dosing times were presumed to be unabsorbed material. Because PCB metabolites are known to be eliminated in the bile, the possibility exists that some of the PCBs present in the feces were absorbed and then eliminated. As such, only minimum absorption efficiencies can be determined from this and similar studies.

(2) Allen, et. al. (1974) gave single oral doses of PCBs (Arochlor 1248) (1.5 or 3.0 g/kg bw) to two adult rhesus monkeys by gastric intubation. The vehicle was not specified but is presumed to be corn oil. Dosing was done on an empty stomach. Unmetabolized PCBs were analyzed for in feces by GC. Recovery was reported to be high. Minimum gastrointestinal absorption was reported to be 94%.

(3) Norback, et. al. (1978) gave single oral doses of 2,4,5,2',4',5'-hexachlorobiphenyl (mg/kg bw) to two adult rhesus monkeys by nasogastric intubation. Corn oil was the vehicle. No information was available concerning the animals' stomach contents at the time of dosing. Total radioactivity was measured in the feces and the bile (bile duct cannulated). Minimum absorption was 13% in one animal and 41% in the other (average = 27%).

Because these investigators were measuring both parent and metabolized species in the feces (total radioactivity), the degree of absorption may be underestimated if metabolites were present in the feces during the first week. In addition, according to the limited experimental details available in this abstract, bile was returned from the cannulated bile duct to the duodenum. If so, not all of the radioactivity in the feces may be due to unabsorbed material. Thus, the reported absorption figures are minimum values.

(4) Albro and Fishbein (1972) gave single oral doses of 20 different PCB congeners (5-100 mg/kg bw) and the unabsorbed marker compound, squalene, to CD rats. The mixture was given by stomach tube to feed animals who were allowed food and water ad libitum. No vehicle was specified. Although this was not a diet study, per se, it is possible that dietary components were present in the stomach at the same time as were the test compounds. Minimum absorption was reported to be 90% for all congeners.

(5) Tanabe, et. al. (1981) gave repeated oral doses of Kanechlors (300, 400, 500, 600) (c.30 mg/kg bw/day x 5 days) to Wistar rats. The dose was given in corn oil.

Commercial diet was given ad libitum. No information on the animals' stomach contents was reported. Parent compounds were analyzed in the feces by GC/MS. Minimal gastrointestinal absorption was reported to be 85% for total PCBs. Cl_5 to Cl_7 congeners had 75-90% absorption.

(6) Berlin, et. al. (1974) gave a single oral dose of 2,4,5,2',4',5'-pentachlorobiphenyl (7 mg/kg bw) to three CBA mice. The PCBs were given as an aqueous emulsion. No information on the animals' stomach contents was given. Minimal gastrointestinal absorption was reported to be 93%.

These studies, which involve both rodents and primates and various PCB mixtures and purified congeners, all show that PCBs are very effectively absorbed from the gastrointestinal tract. It is possible that absorption of PCBs that are thoroughly mixed in the diet is lower than absorption from these studies in which PCBs are dissolved in corn oil and given by gastric intubation. In the chronic feeding study of Norback and Weltman (1985), however, PCBs were added to the diet as a corn oil solution. Jordan has determined that the above studies do yield reasonable

estimates of the degree of absorption expected in the Norback and Weltman study. This six absorption factors were averaged to yield the estimate of 80%.

Dermal Absorption of Pure PCBs

Several studies have investigated the efficiency of dermal absorption of pure PCBs or PCBs given in aqueous solution.

(A) Shah, et. al. (1981) place 2,4,5,2',4',5',-hexachlorobiphenyl on the shaved backs of Dulpin ICR mice for various times. The PCB was administered in 100 mL of acetone, which was quickly evaporated. Total radioactivity was determined in specific tissues, organs, excretory products, and the carcass. Radioactivity at the application site was analyzed to determine the quantity of unabsorbed chemical. 45% of the administered dose was systemically absorbed in 30 minutes (n=3), and 55% was absorbed in 1 hour (n=3). After evaporation, the quantity of PCB on the skin surface was a film 0.0005 mm thick (assuming that the density is 1 g/cm^3).

(B) Wester, et. al. (1983) placed 42% PCB (4.1 and 19.3 ug/cm^2) on the shaved abdomens of four rhesus monkeys for 24 hours. The PCB was administered in 50 uL of hexane/benzene (1:1) that evaporated quickly. The efficiency of dermal absorption was determined by comparing the total urinary excretion of radioactivity following topical administration to the following parenteral administration. 15-34% of the administered dose was systemically absorbed. The average absorption for the four animals was 21.5 $\pm$ 8.5%. After evaporation, the dose of PCB corresponded to a thin film of 4-19 x 10^{-5} mm thickness (assuming that the density is 1 g/cm^3).

Guinea pigs were dosed with 42% PCB (4.6 ug/cm^2) or 54% PCB (5.2 ug/cm^2) on the skin on the back of the ear for 24 hours. Dermal absorption was 33.2 $\pm$ 6.3% (n=3) for 42% PCB and 55.6 $\pm$ 2.6% (n=3) for 54% PCB. These values indicate the dermal absorption that was observed after 24 hours. No earlier time points were determined. Shah, et. al. (1981), however, found that dermal absorption of PCBs in mice was not linear over time. Instead, it plateaued after only a short periodof time (approximately 1 hour). Thus, the absorption observed by Wester, et. al. (1983) over 24 hours was probably virtually complete after 1-2 hours.

(C) Wester, et al. (1987) found that 96% of the 54% PCB in dilute aqueous solution (1.6 ug/mL) bound to powdered human skin (stratum corneum). In this experiment, 1.5 mL of aqueous solution was mixed with 1.5 mg of powdered skin. The fraction of chemical bound was determined by measuring the amount of radioactivity on the skin and in the supernatant. In another in vitro experiment, 12% of the PCBs in the same aqueous solution were bound to and absorbed through a section of fresh human skin from surgical reduction. The administered dose corresponds to a thin film of aqueous solution 1.6 mm in depth above the skin surface.

For purposes of estimating the dermal absorption of pure PCBs from the available data based on several experiments, the results of Wester, et. al.'s in vitro powdered skin experiment was excluded. The absorption may have been abnormally high due to the very high surface area of the skin. The four results for mice, guinea pigs, rhesus monkeys, and humans from the three studies were averaged to yield a value of 41.3 ± 16.8% absorption for pure PCBs.

Dermal Absorption of PCBs for Contaminated Sediments

There are no experiments in which the dermal absorption for PCB contaminated soils or sediments is measured. In one study, however, the absorption of structurally similar TCDD was compared for a TCDD solution in methanol and for TCDD-contaminated soil. Poiger and Schlatter (1980) dosed hairless rats (Naked ex Back-Cross and Holzman strain) with radiolabelled TCDD. The percent of the administered dose in the liver after 24 hours was compared for two situations:

(1) 26 mg TCDD in 50 uL of methanol per 3 cm^2 of skin; and

(2) 350 or 1,300 mg TCDD in a soil/water paste of 75 mg per 3-4 cm^2 of skin (50 mg dry soil/3-4 cm^2)

The percent dose in the liver after administration of the soil paste was the same for the two dose levels. Jordan averaged the values and compared them to the percent dose in the liver following administration of pure PCB from a methanol solution:

$$\frac{\text{dermal absorption, soil}}{\text{dermal absorption, solvent}} = \frac{1.95}{14.8} = 13\% \ (0.13)$$

It is assumed that PCBs are absored to soil and retarded in their dermal absorption to the same degree as is TCDD. Thus, the

dermal absorption factor for PCBs in contaminated sediments is derived by multiplying the dermal absorption factor for pure PCBs by the expected ratio for the dermal absorption from sediments to the dermal absorption of pure PCBs.

The dermal absorption factor for PCBs in contaminated sediment = 0.13 x 1.413 = 0.054 (5.4%). This value agrees well with the dermal absorption factor used by EPA for PCB contaminated soil in contact with human skin (5%) (EPA, 1986).

APPENDIX B

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APPENDIX C

EXPOSURE DOSE CALCULATIONS

TABLE C-1. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; UPPER ESTUARY; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER: TABLE 1

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Direct Contact with Sediment	Area I Upper Estuary						
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	378.0000	6393.0000	0.07	0.07	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 16-Mar-89

MOST PROBABLE

DIRECTORY: EXPOSURES DAYS

DISK: NEW BEDFORD

FILE: SEDAI

AMT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	6.6	40
ADULT:	4.59	70

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	6.6225	55
ADULT:	7.495	

New Bedford
Area I Upper Estuary
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	378.0000	6.6	0.07	40.0	1.75E-01	NA	2.4E-04	2.4E-03	7.70E+00	2.63E-05	2.63E-04	NA

New Bedford
Area I Upper Estuary
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	378.0000	4.6	0.07	70.0	1.21E-01	NA	9.5E-05	9.5E-04	7.70E+00	1.05E-05	1.05E-04	5.75E-04

TABLE C-1. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; UPPER ESTUARY; ADULT AND OLDER CHILD.

New Bedford
Area I Upper Estuary
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	6393.0000	6.6	0.07	40.0	2.96E+00	NA	2.0E-02	2.0E-01	7.70E+00	2.23E-03	2.23E-02	NA

New Bedford
Area I Upper Estuary
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	6393.0000	7.5	0.07	70.0	3.35E+00	NA	1.3E-02	1.3E-01	7.70E+00	1.44E-03	1.44E-02	7.93E-02

TABLE C-2. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; LOWER ESTUARY; ADULT AND OLDER CHILD

SUMMARY TABLE HEADER:

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY	NONCARCINOGEN STANDARD/G	
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE	ACUTE	SUB-CHRONIC
EXPOSURE:	Direct Contact with Sediment	Area I Lower Estuary	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1	(mg/kg/day)	(mg/kg/day)
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	149.0000	399.0000	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Older Child	Adult	' ' ' '	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
				0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE: 16-Mar-89

DIRECTORY:
DISK: NEW BEDFORD
FILE: SEDAIA

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	6.6		CHILD:	40
ADULT:	4.59		ADULT:	70
	REALISTIC WORST		ADULT LIFETIME EXPOSURE:	
CHILD:	6.6225		55	
ADULT:	7.485			

New Bedford
Area I Lower Estuary
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	149.0000	6.6	0.07	40.0	6.88E-02	NA	9.4E-05	9.4E-04	7.70E+00	1.04E-05	1.04E-04	NA

New Bedford
Area I Lower Estuary
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	149.0000	4.6	0.07	70.0	4.79E-02	NA	3.7E-05	3.7E-04	7.70E+00	4.12E-06	4.12E-05	2.27E-04

New Bedford

TABLE C-2. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; LOWER ESTUARY; ADULT AND OLDER CHILD

Area I Lower Estuary
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	399.0000	6.6	0.07	40.0	1.85E-01	NA	1.3E-03	1.3E-02	7.70E+00	1.39E-04	1.39E-03	NA

New Bedford
Area I Lower Estuary
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	399.0000	7.5	0.07	70.0	2.09E-01	NA	8.2E-04	8.2E-03	7.70E+00	9.00E-05	9.00E-04	4.95E-03

TABLE C-3. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; COVE AREA (AREA 1); ADULTS AND OLDER CHILDREN.
SUMMARY TABLE HEADER:

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Direct Contact with Sediment	Area I Cove Area	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	286.0000	399.0000	0.07	0.07	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 16-Mar-89

DIRECTORY:
DISK: NEW BEDFORD
FILE: SEDA1B

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	6.6	CHILD:	40
ADULT:	4.59	ADULT:	70

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	6.6225	35
ADULT:	7.485	

New Bedford
Area I Cove Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	286.0000	6.6	0.07	40.0	1.32E-01	NA	1.8E-04	1.8E-03	7.70E+00	1.99E-05	1.99E-04	NA

New Bedford
Area I Cove Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-t body du (mg/kg-			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	286.0000	4.6	0.07	70.0	9.19E-02	NA	7.2E-05	7.2E-04	7.70E+00	7.91E-06	7.91E-05	4.35E-04

TABLE C-3. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; COVE AREA (AREA I); ADULTS AND OLDER CHILDREN.

New Bedford
Area I Cove Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	399.0000	6.6	0.07	40.0	1.85E-01	NA	1.3E-03	1.3E-02	7.70E+00	1.39E-04	1.39E-03	NA

New Bedford
Area I Cove Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	399.0000	7.5	0.07	70.0	2.09E-01	NA	8.2E-04	8.2E-03	7.70E+00	9.00E-05	9.00E-04	4.95E-03

TABLE C-4. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; AREA II; ADULTS AND OLDER CHILDREN.

SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Direct Contact with Sediment	Area II						
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	20.9100	124.5900	0.07	0.07	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 16-Mar-89

DIRECTORY:
DISK: NEW BEDFORD
FILE: SEDAII

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	6.6	CHILD: 40
ADULT:	4.59	ADULT: 70

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	6.6225	55
ADULT:	7.485	

New Bedford
Area II
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	20.9100	6.6	0.07	40.0	9.66E-03	NA	1.3E-05	1.3E-04	7.70E+00	1.46E-06	1.46E-05	NA

New Bedford
Area II
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	20.9100	4.6	0.07	70.0	6.72E-03	NA	5.3E-06	5.3E-05	7.70E+00	5.78E-07	5.78E-06	3.18E-05

TABLE C-4. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; AREA II; ADULTS AND OLDER CHILDREN.

New Bedford
Area II
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Shor body (mg/			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	124.5900	6.6	0.07	40.0	5.78E-02	NA	4.0E-04	4.0E-03	7.70E+00	4.35E-05	4.35E-04	NA

New Bedford
Area II
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)	(s		CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	124.5900	7.5	0.07	70.0	6.53E-02	NA	2.6E-04	2.6E-03	7.70E+00	2.81E-05	2.81E-04	1.55E-03

TABLE C-5. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; PALMER ISLAND (AREA II); ADULTS AND OLDER CHILDREN.
SUMMARY TABLE HEADER:

SITE: New Bedford
EXPOSURE: Direct Contact with Sediment — Palmer Island Area II
Carcinogenic Effects — Noncarcinogenic Effect
Most Probable Case — Realistic Worst Case
Older Child — Adult

DATE: 16-[illegible]-[illegible]

DIRECTORY:
DISK: NEW BEDF
FILE: SEDAIIA

CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/B ACUTE (mg/kg/day)	NONCARCINOGEN STANDARD/B SUB-CHRONIC (mg/kg/day)
PCB's	3.0400	11.4200	0.07	0.07	7.70E+00	0.00	0.00
Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

% SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	6.6	40
ADULT:	4.59	70

	REALISTIC WORST
CHILD:	6.6225
ADULT:	7.485

ADULT LIFETIME EXPOSURE: 53

New Bedford
Palmer Island Area II
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-ter body dose (mg/kg-d.			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	3.0400	6.6	0.07	40.0	1.40E-03	NA	1.9E-06	1.9E-05	7.70E+00	2.12E-07	2.12E-06	NA

New Bedford
Palmer Island Area II
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	3.0400	4.6	0.07	70.0	9.77E-04	NA	7.6E-07	7.6E-06	7.70E+00	8.41E-08	8.41E-07	4.63E-06

TABLE C-5. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; PALMER ISLAND (AREA II); ADULTS AND OLDER CHILDREN.

New Bedford
Palmer Island Area II
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-te body dos (mg/kg-d			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	11.4200	6.6	0.07	40.0	5.29E-03	NA	3.6E-05	3.6E-04	7.70E+00	3.99E-06	3.99E-05	NA

New Bedford
Palmer Island Area II
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short body (mg/k			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	11.4200	7.5	0.07	70.0	5.90E-03	NA	2.3E-05	2.3E-04	7.70E+00	2.58E-06	2.58E-05	1.42E-04

TABLE C-6. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; POPES ISLAND (AREA II); ADULTS AND OLDER CHILDREN.
SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	NONCARCINOGEN STANDARD/G SUB-CHRONIC (mg/kg/day)
SITE:	New Bedford									
EXPOSURE:	Direct Contact with Sediment	Popes Island Area II								
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	10.8600	34.0100	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Older Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
				0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE: 16-Mar-89

DIRECTORY:
DISK: NEW BEDFORD
FILE: SEDAIIB

WT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	6.6	CHILD: 40
ADULT:	4.59	ADULT: 70

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	6.6225	53
ADULT:	7.485	

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New Bedford
Popes Island Area II
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	10.8600	6.6	0.07	40.0	5.02E-03	NA	6.9E-06	6.9E-05	7.70E+00	7.56E-07	7.56E-06	NA

New Bedford
Popes Island Area II
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	10.8600	4.6	0.07	70.0	3.49E-03	NA	2.7E-06	2.7E-05	7.70E+00	3.00E-07	3.00E-06	1.65E-05

TABLE C-6. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; POPES ISLAND (AREA II); ADULTS AND OLDER CHILDREN.

New Bedford
Popes Island Area II
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	34.0100	6.6	0.07	40.0	1.58E-02	NA	1.1E-04	1.1E-03	7.70E+00	1.19E-05	1.19E-04	NA

New Bedford
Popes Island Area II
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	34.0100	7.5	0.07	70.0	1.78E-02	NA	7.0E-05	7.0E-04	7.70E+00	7.67E-06	7.67E-05	4.22E-04

TABLE C-7. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; AREA III; ADULTS AND OLDER CHILDREN.
SUMMARY TABLE HEADER:

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY	NONCARCINOGEN STANDARD/G	
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE	ACUTE	SUB-CHRONIC
EXPOSURE:	Direct Contact with Sediment	Area III	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1	(mg/kg/day)	(mg/kg/day)
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	3.7000	29.2200	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Older Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
			Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE: 16-Mar-[illegible]

DIRECTORY:
DISK: NEW BEDFORD
FILE: SEDAIII

SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	6.6	CHILD:	40
ADULT:	4.59	ADULT:	70

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	6.6225	55
ADULT:	7.485	

New Bedford
Area III
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	3.7000	6.6	0.07	40.0	1.71E-03	NA	2.3E-06	2.3E-05	7.70E+00	2.58E-07	2.58E-06	NA

New Bedford
Area III
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	3.7000	4.6	0.07	70.0	1.19E-03	NA	9.3E-07	9.3E-06	7.70E+00	1.02E-07	1.02E-06	5.63E-06

TABLE C-7. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; AREA III; ADULTS AND OLDER CHILDREN.

New Bedford
Area III
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	29.2200	6.6	0.07	40.0	1.35E-02	NA	9.3E-05	9.3E-04	7.70E+00	1.02E-05	1.02E-04	NA

New Bedford
Area III
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	29.2200	7.5	0.07	70.0	1.53E-02	NA	6.0E-05	6.0E-04	7.70E+00	6.59E-06	6.59E-05	3.63E-04

TABLE C-8. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; FORT RODMAN (AREA III); ADULTS AND OLDER CHILDREN.
SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	NONCARCINOGEN STANDARD/G SUB-CHRONIC (mg/kg/day)
SITE:	New Bedford									
EXPOSURE:	Direct Contact with Sediment	Fort Rodman Beach Area								
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	2.1500	7.1300	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Older Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
			Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE:

DIRECTORY:
DISK:
FILE:

T OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	6.6	40
ADULT:	4.59	70

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	6.6225	53
ADULT:	7.485	

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New Bedford
Fort Rodman Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	2.1500	6.6	0.07	40.0	9.93E-04	NA	1.4E-06	1.4E-05	7.70E+00	1.50E-07	1.50E-06	NA

New Bedford
Fort Rodman Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	2.1500	4.6	0.07	70.0	6.91E-04	NA	5.4E-07	5.4E-06	7.70E+00	5.95E-08	5.95E-07	3.27E-06

TABLE C-8. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; FORT RODMAN (AREA III); ADULTS AND OLDER CHILDREN.

New Bedford
Fort Rodman Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	7.1300	6.6	0.07	40.0	3.31E-03	NA	2.3E-05	2.3E-04	7.70E+00	2.49E-06	2.49E-05	NA

New Bedford
Fort Rodman Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	7.1300	7.5	0.07	70.0	3.74E-03	NA	1.5E-05	1.5E-04	7.70E+00	1.61E-06	1.61E-05	8.85E-05

TABLE C-9. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS: FORT PHOENIX (AREA III); ADULTS AND OLDER CHILDREN.
SUMMARY TABLE HEADER:

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY	NONCARCINOGEN STANDARD/B	
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE	ACUTE	SUB-CHRONIC
EXPOSURE:	Direct Contact with Sediment	Fort Phoenix Beach Area	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1	(mg/kg/day)	(mg/kg/day)
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.5900	0.7500	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Older Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
			Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE:

DIRECTORY
DISK:
FILE:

DUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	6.6	CHILD:	40
ADULT:	4.59	ADULT:	70

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	6.6225	55
ADULT:	7.485	

New Bedford
Fort Phoenix Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	0.5900	6.6	0.07	40.0	2.73E-04	NA	3.7E-07	3.7E-06	7.70E+00	4.11E-08	4.11E-07	NA

New Bedford
Fort Phoenix Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	0.5900	4.6	0.07	70.0	1.90E-04	NA	1.5E-07	1.5E-06	7.70E+00	1.63E-08	1.63E-07	8.98E-07

TABLE C-9. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; FORT PHOENIX (AREA III); ADULTS AND OLDER CHILDREN.

New Bedford
Fort Phoenix Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	0.7500	6.6	0.07	40.0	3.48E-04	NA	2.4E-06	2.4E-05	7.70E+00	2.62E-07	2.62E-06	NA

New Bedford
Fort Phoenix Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	0.7500	7.5	0.07	70.0	3.93E-04	NA	1.5E-06	1.5E-05	7.70E+00	1.69E-07	1.69E-06	9.30E-06

TABLE C-10. BODY DOSE CALCULATIONS: DIRECT CONTACT WITH SEDIMENTS; COVE AREA (AREA I); CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Direct Contact with Sediment	Area I Cove Area	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	286.0000	399.0000	0.07	0.07	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Child	Adult						

DATE: 11-Mar-90

DIRECTORY:
DISK: NEW B
FILE: SEDIB

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	3.42		CHILD:	10
ADULT:	N/A		ADULT:	N/A
	REALISTIC WORST		ADULT LIFETIME EXPOSURE:	
CHILD:	3.7875			55
ADULT:	N/A			

New Bedford
Area I Cove Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	286.0000	3.4	0.07	10.0	6.85E-02	NA	1.9E-05	9.4E-05	7.70E+00	2.06E-06	1.03E-05	NA

New Bedford
Area I Cove Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	399.0000	3.8	0.07	10.0	1.06E-01	NA	5.8E-04	2.9E-03	7.70E+00	6.38E-05	3.19E-04	NA

TABLE C-11. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; UPPER ESTUARY (AREA I); CHILD

SUMMARY TABLE HEADER:

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY	NONCARCINOGEN STANDARD/G	
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE	ACUTE	SUB-CHRONIC
EXPOSURE:	Direct Contact with Sediment	Area I Upper Estuary	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1	(mg/kg/day)	(mg/kg/day)
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	378.0000	6393.0000	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
			Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE: 16-Mar-89

DIRECTORY:
DISK: NEW BEI
FILE: SEDI-C

AMT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	3.42	10
ADULT:	N/A	N/A

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	3.7875	55
ADULT:	N/A	

New Bedford
Area I Upper Estuary
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	378.0000	3.4	0.07	10.0	9.05E-02	NA	2.5E-05	1.2E-04	7.70E+00	2.73E-06	1.36E-05	NA

New Bedford
Area I Upper Estuary
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	6393.0000	3.8	0.07	10.0	1.69E+00	NA	9.3E-03	4.6E-02	7.70E+00	1.02E-03	5.11E-03	NA

TABLE C-12. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; LOWER ESTUARY (AREA 1); CHILD.
SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	NONCARCINOGEN STANDARD/G SUB-CHRONIC (mg/kg/day)
SITE:	New Bedford									
EXPOSURE:	Direct Contact with Sediment	Area I Lower Estuary								
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	149.0000	399.0000	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
			Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DAT

DIR
DIS
FIL

SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE		REALISTIC WORST
ILD:	3.42		3.7875
ULT:	N/A		N/A

BODY WEIGHT (kg)	
CHILD:	10
ADULT:	N/A

ADULT LIFETIME EXPOSURE:
55

New Bedford
Area I Lower Estuary
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	149.0000	3.4	0.07	10.0	3.57E-02	NA	9.8E-06	4.9E-05	7.70E+00	1.08E-06	5.38E-06	NA

New Bedford
Area I Lower Estuary
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body d (mg/kg			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	399.0000	3.8	0.07	10.0	1.06E-01	NA	5.8E-04	2.9E-03	7.70E+00	6.38E-05	3.19E-04	NA

TABLE C-13. BODY DOSE CALCULATIONS: DIRECT CONTACT WITH SEDIMENTS; AREA II: CHILD.
SUMMARY TABLE HEADER: TABLE 1

SITE: New Bedford
EXPOSURE: Direct Contact with Sediment — Area II
Carcinogenic Effects — Noncarcinogenic Effect
Most Probable Case — Realistic Worst Case
Child — Adult

DATE:

DIREI
DISK:
FILE:

CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	NONCARCINOGEN STANDARD/G SUB-CHRONIC (mg/kg/day)
PCB's	20.9100	124.5900	0.07	0.07	7.70E+00	0.00	0.00
Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

EDIMENT CONTACTED (g/exp)
MOST PROBABLE
D: 3.42
T: N/A

REALISTIC WORST
D: 3.7875
T: N/A

BODY WEIGHT (kg)
CHILD: 10
ADULT: N/A

ADULT LIFETIME EXPOSURE:
55

New Bedford
Area II
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	20.9100	3.4	0.07	10.0	5.01E-03	NA	2.7E-05	1.4E-04	7.70E+00	3.02E-06	1.51E-05	NA

New Bedford
Area II
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	124.5900	3.8	0.07	10.0	3.30E-02	NA	9.0E-04	4.5E-03	7.70E+00	9.95E-05	4.98E-04	NA

TABLE C-13. BODY DOSE CALCULATIONS: DIRECT CONTACT WITH SEDIMENTS; AREA II; CHILD.
SUMMARY TABLE HEADER: TABLE 1

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY	NONCARCINOGEN STANDARD/G	
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE	ACUTE	SUB-CHRONIC
EXPOSURE:	Direct Contact with Sediment	Area II	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1	(mg/kg/day)	(mg/kg/day)
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	20.9100	124.5900	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
				0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE:

DIRECTORY:
DISK: NEW :
FILE: SED1:

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	3.42		CHILD:	10
ADULT:	N/A		ADULT:	N/A
	REALISTIC WORST		ADULT LIFETIME EXPOSURE:	
CHILD:	3.7875		53	
ADULT:	N/A			

New Bedford
Area II
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	20.9100	3.4	0.07	10.0	5.01E-03	NA	2.7E-05	1.4E-04	7.70E+00	3.02E-06	1.51E-05	NA

New Bedford
Area II
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount c Contamin Absorbed (m				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	124.5900	3.8	0.07	10.0	3.30E-02	NA	9.0E-04	4.5E-03	7.70E+00	9.95E-05	4.98E-04	NA

TABLE C-14. BODY DOSE CALCULATIONS: DIRECT CONTACT WITH SEDIMENTS; PALMER ISLAND (AREA II); CHILD.
SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (1) MOST PROBABLE	FRACTION ABSORBED (1) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/S ACUTE (mg/kg/day)	NONCARCINOGEN STANDARD/S SUB-CHRONIC (mg/kg/day)
SITE:	New Bedford									
EXPOSURE:	Direct Contact with Sediment	Area II Palmer Island								
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	3.0400	11.4200	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
				0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE:

DIRECTORY:
DISK: NEW BE
FILE: SEDIIA

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	3.42	10
ADULT:	N/A	N/A

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	3.7875	55
ADULT:	N/A	

New Bedford
Area II Palmer Island
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	3.0400	3.4	0.07	10.0	7.28E-04	NA	4.0E-06	2.0E-05	7.70E+00	4.39E-07	2.19E-06	NA

New Bedford
Area II Palmer Island
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	11.4200	3.8	0.07	10.0	3.03E-03	NA	8.3E-05	4.1E-04	7.70E+00	9.12E-06	4.56E-05	NA

TABLE C-15. BODY DOSE CALCULATIONS: DIRECT CONTACT WITH SEDIMENTS; POPES ISLAND (AREA II); CHILD.
SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	NONCARCINOGEN STANDARD/G SUB-CHRONIC (mg/kg/day)
SITE:	New Bedford									
EXPOSURE:	Direct Contact with Sediment	Area II Popes Island	PCB's	10.8600	34.0100	0.07	0.07	7.70E+00	0.00	0.00
	Carcinogenic Effects	Noncarcinogenic Effect	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Child	Adult	[illegible]	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE: 20-

DIRECTORY:
DISK: NEW BEDF
FILE: SEDIIB-C

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	3.42	CHILD:	10
ADULT:	N/A	ADULT:	N/A

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	3.7875	55
ADULT:	N/A	

0
5

5
5

New Bedford
Area II Popes Island
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	10.8600	3.4	0.07	10.0	2.60E-03	NA	1.4E-05	7.1E-05	7.70E+00	1.57E-06	7.84E-06	NA

New Bedford
Area II Popes Island
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	34.0100	3.8	0.07	10.0	9.02E-03	NA	2.5E-04	1.2E-03	7.70E+00	2.72E-05	1.36E-04	NA

TABLE C-16. BODY DOSE CALCULATIONS: DIRECT CONTACT WITH SEDIMENTS; AREA III; CHILD.
SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	NONCARCINOGEN STANDARD/G SUB-CHRONIC (mg/kg/day)
SITE:	New Bedford									
EXPOSURE:	Direct Contact with Sediment	Area III								
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	3.7100	29.2200	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
			Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
DATE:	[illegible]									

DIRECTOR
DISK:
FILE:

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	3.42	10
ADULT:	N/A	N/A

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	3.7875	55
ADULT:	N/A	

New Bedford
Area III
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	3.7100	3.4	0.07	10.0	8.88E-04	NA	4.9E-06	2.4E-05	7.70E+00	5.35E-07	2.68E-06	NA

New Bedford
Area III
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-body C (mg/k			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	29.2200	3.8	0.07	10.0	7.75E-03	NA	2.1E-04	1.1E-03	7.70E+00	2.33E-05	1.17E-04	NA

TABLE C-17. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; FORT RODMAN (AREA III); CHILD.
SUMMARY TABLE HEADER:

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY	NONCARCINOGEN STANDARD/G	
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE	ACUTE	SUB-CHRONIC
EXPOSURE:	Direct Contact with Sediment	Fort Rodman Beach Area	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1	(mg/kg/day)	(mg/kg/day)
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	2.1500	7.1300	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
			Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE:

DIRECTOR
DISK:
FILE:

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	3.42	10
ADULT:	N/A	N/A

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	3.7875	55
ADULT:	N/A	

New Bedford
Fort Rodman Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	2.1500	3.4	0.07	10.0	5.15E-04	NA	2.8E-06	1.4E-05	7.70E+00	3.10E-07	1.55E-06	NA

New Bedford
Fort Rodman Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	7.1300	3.8	0.07	10.0	1.89E-03	NA	5.2E-05	2.6E-04	7.70E+00	5.70E-06	2.85E-05	NA

TABLE C-18. BODY DOSE CALCULATIONS: DIRECT CONTACT WITH SEDIMENTS; FORT PHOENIX (AREA III); CHILD.
SUMMARY TABLE HEADER:

				CONCENTRATION (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY	NONCARCINOGEN STANDARD/G	
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE	ACUTE	SUB-CHRONIC
EXPOSURE:	Direct Contact with Sediment	Fort Phoenix Beach Area	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1	(mg/kg/day)	(mg/kg/day)
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.5900	0.7500	0.07	0.07	7.70E+00	0.00	0.00
	Most Probable Case	Realistic Worst Case	Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
	Child	Adult	Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
			Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

DATE:

DIREC
DISK:
FILE:

AMOUNT OF SEDIMENT CONTACTED (g/exp)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	3.42	CHILD:	10
ADULT:	N/A	ADULT:	N/A

REALISTIC WORST		ADULT LIFETIME EXPOSURE:
CHILD:	3.7875	55
ADULT:	N/A	

New Bedford
Fort Phoenix Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-t body dc (mg/kg-			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	0.5900	3.4	0.07	10.0	1.41E-04	NA	7.7E-07	3.9E-06	7.70E+00	8.51E-08	4.26E-07	NA

New Bedford
Fort Phoenix Beach Area
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-tc body dos (mg/kg-u			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	0.7500	3.8	0.07	10.0	1.99E-04	NA	5.4E-06	2.7E-05	7.70E+00	5.99E-07	3.00E-06	NA

TABLE C-19. BODY DOSE CALCULATIONS; INHALATION OF AIR; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

SITE: New Bedford
EXPOSURE: Inhalation of Air
Carcinogenic Effects
Most Probable Case
Older Child

Zone 1
Noncarcinogenic Effect
Realistic Worst Case
Adult

DATE: 01-Jan-80

DIRECTORY:
DISK: NEW BEDFORD
FILE: AIR

CONTAMINANTS OF CONCERN:	CONTAMINANT CONCENTRATION IN AIR (ug/m^3) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	SUB-CHRONIC (mg/kg/day)
PCB's	0.0100	0.4710	1.00	1.00	7.70E+00	0.01	0.01
Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

VENTILATION RATE (m^3/exp)

	MOST PROBABLE	REALISTIC WORST
CHILD:	20	20
ADULT:	20	20

BODY WEIGHT (kg)

CHILD:	40
ADULT:	70

NUMBER OF HOURS OF EXPOSURE PER 24 HOUR PERIOD

MOST PROBABLE	8
REALISTIC WORST	24

New Bedford
Zone 1
Inhalation of Air
Carcinogenic Effects
Most Probable Case
Older Child

1 year exp. 10 year exp.

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Chronic body dose (mg/kg-day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0100	20.0	1.00	40.0	2.00E-04	NA	1.7E-06	1.7E-06	7.70E+00	NA	1.83E-07	1.83E-06
Cadmium	0.0000	20.0	0.00	40.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	1.83E-07	1.83E-06

New Bedford
Zone 1
Inhalation of Air
Carcinogenic Effects
Most Probable Case
Adult

1 year exp. 10 years exp.

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Chronic body dose (mg/kg-day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0100	20.0	1.00	70.0	2.00E-04	NA	9.5E-07	9.5E-07	7.70E+00	NA	1.05E-07	1.05E-06
Cadmium	0.0000	20.0	0.00	70.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	1.05E-07	1.05E-06

New Bedford

TABLE C-19. BODY DOSE CALCULATIONS; INHALATION OF AIR; ADULT AND OLDER CHILD.

Zone 1
Inhalation of Air
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day) 1 year exp	Chronic body dose (mg/kg-day) 10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4710	20.0	1.00	40.0	9.42E-03	NA	2.4E-04	2.4E-04	7.70E+00	NA	2.59E-05	2.59E-04
Cadmium	0.0000	20.0	0.00	40.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	2.59E-05	2.59E-04

New Bedford

Zone 1
Inhalation of Air
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day) 1 year exp.	Chronic body dose (mg/kg-day) 10 years exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4710	20.0	1.00	70.0	9.42E-03	NA	1.3E-04	1.3E-04	7.70E+00	NA	1.48E-05	1.48E-04
Cadmium	0.0000	20.0	0.00	70.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	1.48E-05	1.48E-04

New Bedford

TABLE C-20. BODY DOSE CALCULATIONS; INHALATION OF AIR; CHILD.

SUMMARY TABLE HEADER:

SITE: New Bedford
EXPOSURE: Inhalation of Air
Carcinogenic Effects
Most Probable Case
Child

All Areas
Noncarcinogenic Effect
Realistic Worst Case
Adult

DATE: 01-Jan-80

DIRECTORY:
DISK: NEW BEDFORD
FILE: AIRCHLD

CONTAMINANTS OF CONCERN:	CONTAMINANT CONCENTRATION IN AIR (ug/m^3) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	SUB-CHRONIC (mg/kg/day)
PCB's	0.0100	0.4710	1.00	1.00	7.70E+00	0.01	0.01
Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

VENTILATION RATE (m^3/exp)

	MOST PROBABLE	REALISTIC WORST
CHILD:	5	5
ADULT:	20	20

BODY WEIGHT (kg)

CHILD:	10
ADULT:	70

NUMBER OF HOURS OF EXPOSURE PER 24 HOUR PERIOD

MOST PROBABLE	8
REALISTIC WORST	24

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Lifetime body dose (mg/kg-day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0100	5.0	1.00	10.0	5.00E-05	NA	1.7E-06	1.7E-06	7.70E+00	NA	1.83E-07	9.17E-07
Cadmium	0.0000	5.0	0.00	10.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	1.83E-07	9.17E-07

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Lifetime body dose (mg/kg-day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4710	5.0	1.00	10.0	2.35E-03	NA	2.4E-04	2.4E-04	7.70E+00	NA	2.59E-05	1.30E-04
Cadmium	0.0000	5.0	0.00	10.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	2.59E-05	1.30E-04

New Bedford

TABLE C-31. BODY DOSE CALCULATIONS: INHALATION OF AIR: ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

SITE: New Bedford
EXPOSURE: Inhalation of Air
Carcinogenic Effects
Most Probable Case
Older Child

All Areas
Noncarcinogenic Effect
Realistic Worst Case
Adult

DATE: 16-Mar-89

DIRECTORY:
DISK: NEW BEDFORD
FILE: AIF

CONTAMINANTS OF CONCERN:	CONTAMINANT CONCENTRATION IN AIR (ug/m 3) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAS POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARDS ACUTE (mg/kg/day)	SUB-CHRONIC (mg/kg/day)
PCB's	0.0845	0.4710	1.00	1.00	7.70E+00	0.01	0.01
Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

VENTILATION RATE (m'3/exp)

	MOST PROBABLE
CHILD:	20
ADULT:	20

	REALISTIC WORST
CHILD:	20
ADULT:	20

BODY WEIGHT (kg)

CHILD:	40
ADULT:	70

NUMBER OF HOURS OF EXPOSURE PER 24 HOUR PERIOD

MOST PROBABLE	8
REALISTIC WORST	24

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/m'3)	Ventilation Rate (m'3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Chronic body dose (mg/kg-day)	CAS Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
						1 year exp.	10 year exp.					
PCB's	0.0845	20.0	1.00	40.0	1.69E-03	NA	1.4E-05	1.4E-05	7.70E+00	NA	1.55E-06	1.55E-05
Cadmium	0.0000	20.0	0.00	40.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	1.55E-06	1.55E-05

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/m'3)	Ventilation Rate (m'3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Chronic body dose (mg/kg-day)	CAS Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
						1 year exp.	10 years exp.					
PCB's	0.0845	20.0	1.00	70.0	1.69E-03	NA	8.0E-06	8.0E-06	7.70E+00	NA	6.85E-07	6.85E-06
Cadmium	0.0000	20.0	0.00	70.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	6.85E-07	6.85E-06

New Bedford

TABLE C-21. BODY DOSE CALCULATIONS: INHALATION OF AIR: ADULT AND OLDER CHILD.
All Areas
Inhalation of Air
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day) 1 year exp	Chronic body dose (mg/kg-day) 10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4710	20.0	1.00	40.0	9.42E-02	NA	2.4E-04	2.4E-04	7.70E+00	NA	2.59E-05	2.59E-04
Cadmium	0.0000	20.0	0.00	40.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	2.59E-05	2.59E-04

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day) 1 year exp.	Chronic body dose (mg/kg-day) 10 years exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4710	20.0	1.00	70.0	9.42E-02	NA	1.3E-04	1.3E-04	7.70E+00	NA	1.48E-05	1.48E-04
Cadmium	0.0000	20.0	0.00	70.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	1.48E-05	1.48E-04

New Bedford

TABLE C-23. BODY DOSE CALCULATIONS; INHALATION OF AIR; CHILD.

SUMMARY TABLE HEADER:

SITE: New Bedford
EXPOSURE: Inhalation of Air
Carcinogenic Effects
Most Probable Case
Child

All Areas
Noncarcinogenic Effect
Realistic Worst Case
Adult

DATE: 01-Jan-80

DIRECTORY:
DISK: NEW BEDFORD
FILE: AIRCHLD

CONTAMINANTS OF CONCERN:	CONTAMINANT CONCENTRATION IN AIR (ug/m^3) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	SUB-CHRONIC (mg/kg/day)
PCB's	0.0845	0.4710	1.00	1.00	7.70E+00	0.01	0.01
Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

VENTILATION RATE (m^3/exp)

	MOST PROBABLE	REALISTIC WORST
CHILD:	5	5
ADULT:	20	20

	BODY WEIGHT (kg)
CHILD:	10
ADULT:	70

NUMBER OF HOURS OF EXPOSURE PER 24 HOUR PERIOD

MOST PROBABLE	8
REALISTIC WORST	24

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Lifetime body dose (mg/kg-day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0845	5.0	1.00	10.0	4.23E-04	NA	1.4E-05	1.4E-05	7.70E+00	NA	1.55E-06	7.75E-06
Cadmium	0.0000	5.0	0.00	10.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	1.55E-06	7.75E-06

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Lifetime body dose (mg/kg-day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4710	5.0	1.00	10.0	2.35E-03	NA	2.4E-04	2.4E-04	7.70E+00	NA	2.59E-05	1.30E-04
Cadmium	0.0000	5.0	0.00	10.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	2.59E-05	1.30E-04

New Bedford

TABLE C-23. BODY DOSE CALCULATIONS: INHALATION OF AIR; CHILD.

SUMMARY TABLE HEADER:

SITE: New Bedford
EXPOSURE: Inhalation of Air
Carcinogenic Effects
Most Probable Case
Child

All Areas
Noncarcinogenic Effect
Realistic Worst Case
Adult

DATE: 21-Mar-89

DIRECTORY:
DISK: NEW BEDFORD
FILE: AIRCHLD

CONTAMINANTS OF CONCERN:	CONTAMINANT CONCENTRATION IN AIR (ug/m^3) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1	NONCARCINOGEN STANDARD/G ACUTE (mg/kg/day)	SUB-CHRONIC (mg/kg/day)
PCB's	0.0845	0.4710	1.00	1.00	7.70E+00	0.01	0.01
Copper	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Lead	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00
Cadmium	0.0000	0.0000	0.00	0.00	0.00E+00	0.00	0.00

VENTILATION RATE (m^3/exp)

	MOST PROBABLE
CHILD:	5
ADULT:	20

	REALISTIC WORST
CHILD:	5
ADULT:	20

BODY WEIGHT (kg)

CHILD:	10
ADULT:	70

NUMBER OF HOURS OF EXPOSURE PER 24 HOUR PERIOD

MOST PROBABLE	8
REALISTIC WORST	24

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Lifetime body dose (mg/kg-day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0845	5.0	1.00	10.0	4.23E-04	NA	1.4E-05	1.4E-05	7.70E+00	NA	1.55E-06	7.75E-06
Cadmium	0.0000	5.0	0.00	10.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	1.55E-06	7.75E-06

New Bedford
All Areas
Inhalation of Air
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/m^3)	Ventilation Rate (m^3/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg-day)	Subchronic body dose (mg/kg-day)	Lifetime body dose (mg/kg-day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4710	5.0	1.00	10.0	2.35E-03	NA	2.4E-04	2.4E-04	7.70E+00	NA	2.59E-05	1.30E-04
Cadmium	0.0000	5.0	0.00	10.0	0.00E+00	0.0E+00	0.0E+00	0.0E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
								SUMMARY INCREMENTAL RISK		0.00E+00	2.59E-05	1.30E-04

New Bedford

TABLE C-24. BODY DOSE CALCULATIONS: INGESTION OF SEDIMENTS: FORT RODMAN (AREA III): CHILD.

SITE: New Bedford	CONTAMINANTS	WORST CASE	WORST CASE	WORST CASE	WORST CASE	ESTIMATE
EXPOSURE: Ingestion of Sediment	OF CONCERN:	ONE	TWO	ONE	TWO	(mg/kg/day)-1
Carcinogenic Effects	PCB's	2.1500	7.1300	1.00	1.00	7.70E+00
Area III:Fort Rodman						
Child						

DATE: 21-Mar-89

	Most Probable Case	NUMBER OF		
DIRECTORY:	FREQUENCY OF CONTACT (days)	EXPOSURES	DAYS	AMOUNT OF SEDIMENT CONSUMED (g/exp)
DISK: 205				WORST CASE ONE
FILE: INGAIIIa				LD: 0.5
				WORST CASE TWO
				LD: 0.5

BODY WEIGHT (kg)
CHILD: 10

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg/day)	Subchronic body dose (mg/kg/day)	Chronic body dose (mg/kg/day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.1500	0.5	1.00	10.0	1.08E-03	NA	5.9E-06	5.9E-06	7.70E+00	NA	6.48E-07	3.24E-06

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	Short-term body dose (mg/kg/day)	Subchronic body dose (mg/kg/day)	Chronic body dose (mg/kg/day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	7.1300	0.5	1.00	10.0	3.57E-03	NA	9.8E-05	9.8E-05	7.70E+00	NA	1.07E-05	5.37E-05

TABLE C-25. BODY DOSE CALCULATIONS; INGESTION OF SEDIMENTS; FORT PHOENIX (AREA III); CHILD.

			(ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford	CONTAMINANTS	WORST CASE	WORST CASE	WORST CASE	WORST CASE	ESTIMATE
EXPOSURE:	Ingestion of Sediment	OF CONCERN:	ONE	TWO	ONE	TWO	(mg/kg/day)-1
	Carcinogenic Effects	PCB's	0.5900	0.7500	1.00	1.00	7.70E+00
	Area III:Fort Phoenix						
	Child						

DATE: 21-Mar-89

DIRECTORY:
DISK: 205
FILE: IMGAIIIb

	JNT OF SEDIMENT CONSUMED (g/exp)		BODY WEIGHT (kg)
	WORST CASE ONE		
.HILD:	0.5	CHILD:	10
	WORST CASE TWO		
CHILD:	0.5		

(years)

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)	Short-term body dose (mg/kg/day)	Subchronic body dose (mg/kg/day)	Chronic body dose (mg/kg/day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.5900	0.5	1.00	10.0	2.95E-04	NA	1.6E-06	1.6E-06	7.70E+00	NA	1.78E-07	8.89E-07

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	Short-term body dose (mg/kg/day)	Subchronic body dose (mg/kg/day)	Chronic body dose (mg/kg/day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.7500	0.5	1.00	10.0	3.75E-04	NA	1.0E-05	1.0E-05	7.70E+00	NA	1.13E-06	5.65E-06

TABLE C-26. BODY DOSE CALCULATIONS: INGESTION OF BIOTA (CLAM); AREA 1; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER: TABLE 1

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Clam (daily)	Area 1	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.6890	2.1210	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

MOST PROBABLE NUMBER OF

DIRECTORY:

DISK: NEW BEDFORD

FILE: CLAMI

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	227	CHILD: 40
ADULT:	227	ADULT: 70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 1
Ingestion of Clam (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.6890	227.0	1.00	40.0	1.56E-01	3.9E-03	5.6E-04	7.70E+00	NA	4.30E-04	4.30E-03

New Bedford
Area 1
Ingestion of Clam (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.6890	227.0	1.00	70.0	1.56E-01	2.2E-03	3.2E-04	7.70E+00	NA	2.46E-04	2.45E-03

TABLE C-26. BODY DOSE CALCULATIONS; INGESTION OF BIOTA (CLAM); AREA 1; ADULT AND OLDER CHILD.

New Bedford
Area 1
Ingestion of Clam (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.1210	227.0	1.00	40.0	4.81E-01	1.2E-02	1.7E-03	7.70E+00	NA	1.32E-03	1.32E-02

New Bedford
Area 1
Ingestion of Clam (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.1210	227.0	1.00	70.0	4.81E-01	6.9E-03	9.8E-04	7.70E+00	NA	7.57E-04	7.54E-03

TABLE C-27. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (CLAM); AREA 1; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER: TABLE 1

			CONTAMINANTS OF CONCERN:	CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Ingestion of Clam (weekly)	Area 1	PCB's	0.6890	2.1210	1.00	1.00	7.70E+00
	Carcinogenic Effects	Noncarcinogenic Effect						
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

MOST PROBABLE

DIRECTORY:

DISK: NEW BEDFORD

FILE: CLAM1a

NUMBER OF FISH MEALS 10

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	227	40
ADULT:	227	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

- No. yrs exp chronic
- No yrs exp subchronic

New Bedford
Area 1
Ingestion of Clam (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.6890	227.0	1.00	40.0	1.56E-01	5.6E-04	8.0E-05	7.70E+00	NA	6.13E-05	6.13E-04

New Bedford
Area 1
Ingestion of Clam (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.6890	227.0	1.00	70.0	1.56E-01	3.2E-04	4.5E-05	7.70E+00	NA	3.50E-05	3.50E-04

TABLE C-27. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (CLAM); AREA 1; ADULT AND OLDER CHILD.

New Bedford
Area 1
Ingestion of Clam (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.1210	227.0	1.00	40.0	4.81E-01	1.7E-03	2.4E-04	7.70E+00	NA	1.89E-04	1.88E-03

New Bedford
Area 1
Ingestion of Clam (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.1210	227.0	1.00	70.0	4.81E-01	9.8E-04	1.4E-04	7.70E+00	NA	1.08E-04	1.08E-03

TABLE C-28. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (CLAM); AREA 1; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER: TABLE 1

			CONTAMINANTS OF CONCERN:	CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Ingestion of Clam (monthly)	Area 1						
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.6890	2.1210	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:
DISK: NEW BEDFORD
FILE: CLAM1b

	MOST PROBABLE	REALISTIC WORST
NUMBER OF FISH CONSUMED (g/meal)	227	227
	227	227

BODY WEIGHT (kg)	
CHILD:	40
ADULT:	70

New Bedford
Area 1
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	Subchronic bod. dose (mg/kg/day)	[illegible]	[illegible]	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.6890	227.0	1.00	40.0	1.56E-01	1.3E-04	1.8E-05	7.70E+00	NA	1.41E-05	1.41E-04

New Bedford
Area 1
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	Subchronic body dose (mg/kg/day)	[illegible]	[illegible]	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.6890	227.0	1.00	70.0	1.56E-01	7.3E-05	1.0E-05	7.70E+00	NA	8.08E-06	8.08E-05

TABLE C-28. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (CLAM); AREA 1; ADULT AND OLDER CHILD.

New Bedford
Area 1
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.1210	227.0	1.00	40.0	4.81E-01	4.0E-04	5.7E-05	7.70E+00	NA	4.35E-05	4.35E-04

New Bedford
Area 1
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.1210	227.0	1.00	70.0	4.81E-01	2.3E-04	3.2E-05	7.70E+00	NA	2.49E-05	2.49E-04

TABLE C-29. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (CLAM); AREA 2; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER: TABLE 1

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Clam (daily)	Area 1	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2310	1.1810	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS		AMOUNT OF FISH CONSUMED (g/meal)			
DIRECTORY:					MOST PROBABLE		BODY WEIGHT (kg)	
DISK:	NEW BEDFORD			10	CHILD:	227	CHILD:	40
FILE:	CLAMII	No. meals/year	365	365	ADULT:	227	ADULT:	70
			1					
		- No. yrs exp chronic		10				
		- No yrs exp subchronic		1	REALISTIC WORST			
					CHILD:	227		
					ADULT:	227		
			365	365				
			1					
		- No. yrs exp chronic		10				
		- No yrs exp subchronic		1				

New Bedford
Area 1
Ingestion of Clam (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2310	227.0	1.00	40.0	5.24E-02		1.3E-03	1.9E-04	7.70E+00	NA	1.44E-04	1.44E-03

New Bedford
Area 1
Ingestion of Clam (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2310	227.0	1.00	70.0	5.24E-02		7.5E-04	1.1E-04	7.70E+00	NA	8.24E-05	8.24E-04

TABLE C-29. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (CLAM); AREA 2; ADULT AND OLDER CHILD.

New Bedford
Area 1
Ingestion of Clam (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.1810	227.0	1.00	40.0	2.68E-01	6.7E-03	9.6E-04	7.70E+00	NA	7.37E-04	7.35E-03

New Bedford
Area 1
Ingestion of Clam (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.1810	227.0	1.00	70.0	2.68E-01	3.8E-03	5.5E-04	7.70E+00	NA	4.21E-04	4.20E-03

TABLE C-29a. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (CLAM); AREA 2; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER: TABLE 1

SITE:	New Bedford		CONTAMINANTS OF CONCERN:	CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
EXPOSURE:	Ingestion of Clam (weekly)	Area 1						
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2310	1.1810	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:
DISK: NEW BEDF.
FILE: CLAMI1a

	MOST PROBABLE	NUMBER OF FISH MEALS
- No. yrs exp chronic		10
- No yrs exp subchronic		1
	52	365
	1	
- No. yrs exp chronic		10
- No yrs exp subchronic		1

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 1
Ingestion of Clam (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (µg/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2310	227.0	1.00	40.0	5.24E-02	1.9E-04	2.7E-05	7.70E+00	NA	2.05E-05	2.05E-04

New Bedford
Area 1
Ingestion of Clam (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2310	227.0	1.00	70.0	5.24E-02	1.1E-04	1.5E-05	7.70E+00	NA	1.17E-05	1.17E-04

TABLE C-29a. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (CLAM); AREA 2: ADULT AND OLDER CHILD.

New Bedford
Area 1
Ingestion of Clam (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.1B10	227.0	1.00	40.0	2.6BE-01		9.5E-04	1.4E-04	7.70E+00	NA	1.05E-04	1.05E-03

New Bedford
Area 1
Ingestion of Clam (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.1B10	227.0	1.00	70.0	2.6BE-01		5.5E-04	7.8E-05	7.70E+00	NA	6.00E-05	6.00E-04

TABLE C-30. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (CLAM); AREA 2; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Clam (monthly)	Area 2	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2310	1.1810	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:

DISK: NEW BEDFORD

FILE: CLAMIIb

MOST PROBABLE

- No. yrs exp chronic 10
- No yrs exp subchronic 1

NUMBER OF FISH MEALS

NUMBER OF

1

- No. yrs exp chronic 10
- No yrs exp subchronic 1

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE

CHILD: 227

ADULT: 227

REALISTIC WORST

CHILD: 227

ADULT: 227

BODY WEIGHT (kg)

CHILD: 40

ADULT: 70

New Bedford
Area 2
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

1

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2310	227.0	1.00	40.0	5.24E-02		4.3E-05	6.2E-06	7.70E+00	NA	4.74E-06	4.74E-05

New Bedford
Area 2
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2310	227.0	1.00	70.0	5.24E-02		2.5E-05	3.5E-06	7.70E+00	NA	2.71E-06	2.71E-05

TABLE C-30. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (CLAM); AREA 2; ADULT AND OLDER CHILD.

New Bedford
Area 2
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.1B10	227.0	1.00	40.0	2.6BE-01	2.2E-04	3.1E-05	7.70E+00	NA	2.42E-05	2.42E-04

New Bedford
Area 2
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.1B10	227.0	1.00	70.0	2.68E-01	1.3E-04	1.8E-05	7.70E+00	NA	1.39E-05	1.39E-04

TABLE C-31. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (CLAM); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Clam (monthly)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.1560	0.4780	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:

DISK: NEW BEDFORD

FILE: CLAMIII

	MOST PROBABLE NUMBER OF FISH MEALS	AMOUNT OF FISH CONSUMED (g/meal) MOST PROBABLE	BODY WEIGHT (kg)
CHILD:		227	40
ADULT:		227	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1560	227.0	1.00	40.0	3.54E-02	8.9E-04	1.3E-04	7.70E+00	NA	9.74E-05	9.74E-04

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	5		CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1560	227.0	1.00	70.0	3.54E-02	5.1E-04	7.2E-05	7.70E+00	NA	5.56E-05	5.56E-04

TABLE C-31. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (CLAM); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4780	227.0	1.00	40.0	1.09E-01	2.7E-03	3.9E-04	7.70E+00	NA	2.98E-04	2.98E-03

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4780	227.0	1.00	70.0	1.09E-01	1.6E-03	2.2E-04	7.70E+00	NA	1.71E-04	1.70E-03

TABLE C-32. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (CLAM); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Clam (monthly)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.1560	0.4780	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY: MOST PROBABLE

DISK: NEW BEDFORD

FILE: CLAMIIIa

NUMBER OF FISH MEALS

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1560	227.0	1.00	40.0	3.54E-02	1.3E-04	1.8E-05	7.70E+00	NA	1.39E-05	1.39E-04

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1560	227.0	1.00	70.0	3.54E-02	7.2E-05	1.0E-05	7.70E+00	NA	7.93E-06	7.93E-05

TABLE C-32. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (CLAM); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4780	227.0	1.00	40.0	1.09E-01	3.9E-04	5.5E-05	7.70E+00	NA	4.25E-05	4.25E-04

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4780	227.0	1.00	70.0	1.09E-01	2.2E-04	3.2E-05	7.70E+00	NA	2.43E-05	2.43E-04

TABLE C-33. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (CLAM); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Clam (monthly)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.1560	0.4780	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

MOST PROBABLE

DIRECTORY:

DISK: NEW BEDFO

FILE: CLAMIIIb

NUMBER OF FISH MEALS

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1560	227.0	1.00	40.0	3.54E-02	2.9E-05	4.2E-06	7.70E+00	NA	3.20E-06	3.20E-05

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1560	227.0	1.00	70.0	3.54E-02	1.7E-05	2.4E-06	7.70E+00	NA	1.83E-06	1.83E-05

TABLE C-33. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (CLAM); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4780	227.0	1.00	40.0	1.09E-01	8.9E-05	1.3E-05	7.70E+00	NA	9.81E-06	9.81E-05

New Bedford
Area 3
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.4780	227.0	1.00	70.0	1.09E-01	5.1E-05	7.3E-06	7.70E+00	NA	5.61E-06	5.61E-05

TABLE C-34. BODY DOSE CALCULATIONS: DAILY INGESTION OF BIOTA (CLAM): AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Clam (daily)	Area 4	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.0397	0.1370	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

	MOST PROBABLE	NUMBER OF FISH MEALS
DIRECTORY:		
DISK:	NEW BEDFORD	
FILE:	CLAMIV	

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 4
Ingestion of Clam (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0397	227.0	1.00	40.0	9.01E-03	2.3E-04	3.2E-05	7.70E+00	NA	2.48E-05	2.48E-04

New Bedford
Area 4
Ingestion of Clam (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0397	227.0	1.00	70.0	9.01E-03	1.3E-04	1.8E-05	7.70E+00	NA	1.42E-05	1.42E-04

TABLE C-34. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (CLAM); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Clam (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1370	227.0	1.00	40.0	3.11E-02		7.8E-04	1.1E-04	7.70E+00	NA	8.55E-05	8.55E-04

New Bedford
Area 4
Ingestion of Clam (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1370	227.0	1.00	70.0	3.11E-02		4.4E-04	6.3E-05	7.70E+00	NA	4.89E-05	4.89E-04

TABLE C-35. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (CLAM); AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (I) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Ingestion of Clam (weekly)	Area 4	PCB's	0.0397	0.1370	1.00	1.00	7.70E+00
	Carcinogenic Effects	Noncarcinogenic Effect						
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY: MOST PROBABLE

DISK: NEW BEDFORD

FILE: CLAMIVa No. meals/year

NUMBER OF FISH MEALS

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	227	40
ADULT:	227	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 4
Ingestion of Clam (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (I)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0397	227.0	1.00	40.0	9.01E-03		3.2E-05	4.6E-06	7.70E+00	NA	3.53E-06	3.53E-05

New Bedford
Area 4
Ingestion of Clam (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (I)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0397	227.0	1.00	70.0	9.01E-03		1.8E-05	2.6E-06	7.70E+00	NA	2.02E-06	2.02E-05

TABLE C-35. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (CLAM); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Clam (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1370	227.0	1.00	40.0	3.11E-02		1.1E-04	1.6E-05	7.70E+00	NA	1.22E-05	1.22E-04

New Bedford
Area 4
Ingestion of Clam (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1370	227.0	1.00	70.0	3.11E-02		6.3E-05	9.0E-06	7.70E+00	NA	6.96E-06	6.96E-05

TABLE C-36. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (CLAM); AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Clam (monthly)	Area 4	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.0397	0.1370	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-99

	MOST PROBABLE	NUMBER OF
DIRECTORY:		FISH MEALS
DISK:	NEW BEDFORD	
FILE:	CLAMIVb	No. meals/year

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE		BODY WEIGHT (kg)
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 4
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0397	227.0	1.00	40.0	9.01E-03	7.4E-06	1.1E-06	7.70E+00	NA	8.15E-07	8.15E-06

New Bedford
Area 4
Ingestion of Clam (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		y)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0397	227.0	1.00	70.0	9.01E-03	4.2E-06	6.0E-07	7.70E+00	NA	4.66E-07	4.66E-06

TABLE C-36. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF RIDTA (CLAM); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1370	227.0	1.00	40.0	3.11E-02	2.6E-05	3.7E-06	7.70E+00	NA	2.81E-06	2.81E-05

New Bedford
Area 4
Ingestion of Clam (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1370	227.0	1.00	70.0	3.11E-02	1.5E-05	2.1E-06	7.70E+00	NA	1.61E-06	1.61E-05

TABLE C-77. BODY DOSE CALCULATIONS: DAILY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 1; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST PROBABLE	REALISTIC WORST	MOST PROBABLE	REALISTIC WORST	(mg/kg/day)-1
EXPOSURE:	Ingestion of Flounder (daily)	Area 1	OF CONCERN:					
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	1.0390	2.6290	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS
DIRECTORY:			
DISK:	NEW BEDFORD		0
FILE:	WFI	No. meals/year	5

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 1
Ingestion of Flounder (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	.c .e day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0390	227.0	1.00	40.0	2.36E-01		5.9E-03	8.4E-04	7.70E+00	NA	6.49E-04	6.49E-03

New Bedford
Area 1
Ingestion of Flounder (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		Subchronic		CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0390	227.0	1.00	70.0	2.36E-01		3.4E-03	4.8E-04	7.70E+00	NA	3.71E-04	3.70E-03

TABLE C-37. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 1; ADULT AND OLDER CHILD.

New Bedford
Area 1
Ingestion of Flounder (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.6290	227.0	1.00	40.0	5.97E-01	1.5E-02	2.1E-03	7.70E+00	NA	1.64E-03	1.63E-02

New Bedford
Area 1
Ingestion of Flounder (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.6290	227.0	1.00	70.0	5.97E-01	8.5E-03	1.2E-03	7.70E+00	NA	9.38E-04	9.33E-03

TABLE C-38. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 1; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g) MOST PROBABLE	CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Ingestion of Flounder (weekly)	Area 1						
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	1.0390	2.6290	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:

DISK: NEW BEDFORD

FILE: WF1a

	MOST PROBABLE	NUMBER OF FISH MEALS
No. meals/year		0
		5

- ;
- ;

No.

-
-

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	REALISTIC WORST
CHILD:	227	227
ADULT:	227	227

BODY WEIGHT (kg)

CHILD:	40
ADULT:	70

New Bedford
Area 1
Ingestion of Flounder (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0390	227.0	1.00	40.0	2.36E-01		8.4E-04	1.2E-04	7.70E+00	NA	9.24E-05	9.24E-04

New Bedford
Area 1
Ingestion of Flounder (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0390	227.0	1.00	70.0	2.36E-01	4.8E-04	6.9E-05	7.70E+00	NA	5.28E-05	5.28E-04

TABLE C-38. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 1; ADULT AND OLDER CHILD.

New Bedford
Area 1
Ingestion of Flounder (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.6290	227.0	1.00	40.0	5.97E-01	2.1E-03	3.0E-04	7.70E+00	NA	2.34E-04	2.34E-03

New Bedford
Area 1
Ingestion of Flounder (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		y)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.6290	227.0	1.00	70.0	5.97E-01	1.2E-03	1.7E-04	7.70E+00	NA	1.34E-04	1.34E-03

TABLE C-39. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 1; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Flounder (monthly)	Area 1	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	1.0390	2.6290	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:

DISK: NEW BEDFORD

FILE: WF1b

	MOST PROBABLE No. meals/year	NUMBER OF FISH MEALS

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	227	CHILD: 40
ADULT:	227	ADULT: 70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 1
Ingestion of Flounder (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0390	227.0	1.00	40.0	2.36E-01	1.9E-04	2.8E-05	7.70E+00	NA	2.13E-05	2.13E-04

New Bedford
Area 1
Ingestion of Flounder (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0390	227.0	1.00	70.0	2.36E-01	1.1E-04	1.6E-05	7.70E+00	NA	1.22E-05	1.22E-04

TABLE C-39. BODY DOSE CALCULATIONS: MONTHLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 1: ADULT AND OLDER CHILD.

New Bedford
Area 1
Ingestion of Flounder (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	[illegible]	[illegible]	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.6290	227.0	1.00	40.0	5.97E-01	4.9E-04	7.0E-05	7.70E+00	NA	5.40E-05	5.40E-04

New Bedford
Area 1
Ingestion of Flounder (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	[illegible]	[illegible]	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	2.6290	227.0	1.00	70.0	5.97E-01	2.8E-04	4.0E-05	7.70E+00	NA	3.08E-05	3.08E-04

TABLE C-40. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 2; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Flounder (daily)	Area 2	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.3710	1.0480	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS
DIRECTORY:			
DISK:	NEW BEDFORD		10
FILE:	WFII	No. meals/year	365

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 2
Ingestion of Flounder (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	y)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3710	227.0	1.00	40.0	8.42E-02		2.1E-03	3.0E-04	7.70E+00	NA	2.32E-04	2.32E-03

New Bedford
Area 2
Ingestion of Flounder (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3710	227.0	1.00	70.0	8.42E-02		1.2E-03	1.7E-04	7.70E+00	NA	1.32E-04	1.32E-03

TABLE C-40. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 2; ADULT AND OLDER CHILD.

New Bedford
Area 2
Ingestion of Flounder (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0480	227.0	1.00	40.0	2.38E-01	5.9E-03	8.5E-04	7.70E+00	NA	6.54E-04	6.52E-03

New Bedford
Area 2
Ingestion of Flounder (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		(mg/kg/day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0480	227.0	1.00	70.0	2.38E-01	3.4E-03	4.9E-04	7.70E+00	NA	3.74E-04	3.73E-03

TABLE C-41. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 2; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Flounder (weekly)	Area 2	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.3710	1.0480	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS	
DIRECTORY:				
DISK:	NEW BEDFORD			10
FILE:	NFIla	No. meals/year	32	365
			1	

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 2
Ingestion of Flounder (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3710	227.0	1.00	40.0	8.42E-02	3.0E-04	4.3E-05	7.70E+00	NA	3.30E-05	3.30E-04

New Bedford
Area 2
Ingestion of Flounder (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	(y)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3710	227.0	1.00	70.0	8.42E-02	1.7E-04	2.4E-05	7.70E+00	NA	1.89E-05	1.89E-04

TABLE C-41. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 2; ADULT AND OLDER CHILD.

New Bedford
Area 2
Ingestion of Flounder (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0480	227.0	1.00	40.0	2.38E-01	8.5E-04	1.2E-04	7.70E+00	NA	9.32E-05	9.32E-04

New Bedford
Area 2
Ingestion of Flounder (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		(mg/kg/day)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0480	227.0	1.00	70.0	2.38E-01	4.8E-04	6.9E-05	7.70E+00	NA	5.33E-05	5.33E-04

TABLE C-42. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIDTA (WINTER FLOUNDER); AREA 2; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Flounder (monthly)	Area 2	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.3710	1.0480	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS		AMOUNT OF FISH CONSUMED (g/meal)			
DIRECTORY:					MOST PROBABLE		BODY WEIGHT (kg)	
DISK:	NEW BEDFORD			10				
FILE:	WFT1b	No. meals/year	12	365	CHILD:	227	CHILD:	40
			1		ADULT:	227	ADULT:	70
		-						
		-			REALISTIC WORST			
					CHILD:	227		
					ADULT:	227		
		No.						
		-						
		-						

New Bedford
Area 2
Ingestion of Flounder (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

1 year exp 10 year exp

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3710	227.0	1.00	40.0	8.42E-02		6.9E-05	9.9E-06	7.70E+00	NA	7.61E-06	7.61E-05

New Bedford
Area 2
Ingestion of Flounder (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3710	227.0	1.00	70.0	8.42E-02		4.0E-05	5.7E-06	7.70E+00	NA	4.35E-06	4.35E-05

TABLE C-42. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 2; ADULT AND OLDER CHILD.

New Bedford
Area 2
Ingestion of Flounder (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0480	227.0	1.00	40.0	2.38E-01		2.0E-04	2.8E-05	7.70E+00	NA	2.15E-05	2.15E-04

New Bedford
Area 2
Ingestion of Flounder (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.0480	227.0	1.00	70.0	2.38E-01		1.1E-04	1.6E-05	7.70E+00	NA	1.23E-05	1.23E-04

TABLE C-43. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Flounder (daily)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2780	0.8250	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:		MOST PROBABLE	NUMBER OF FISH MEALS	
DISK:	NEW BEDFORD			10
FILE:	WFIII	No. meals/year	365	365

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 3
Ingestion of Flounder (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2780	227.0	1.00	40.0	6.31E-02	1.6E-03	2.3E-04	7.70E+00	NA	1.74E-04	1.74E-03

New Bedford
Area 3
Ingestion of Flounder (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absor (mg/fish meal)			CAE Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2780	227.0	1.00	70.0	6.31E-02	9.0E-04	1.3E-04	7.70E+00	NA	9.92E-05	9.92E-04

TABLE C-43. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Flounder (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.8250	227.0	1.00	40.0	1.87E-01	4.7E-03	6.7E-04	7.70E+00	NA	5.15E-04	5.14E-03

New Bedford
Area 3
Ingestion of Flounder (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.8250	227.0	1.00	70.0	1.87E-01	2.7E-03	3.8E-04	7.70E+00	NA	2.94E-04	2.94E-03

TABLE C-44. BODY BOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Flounder (weekly)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2780	0.8250	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS	
DIRECTORY:				
DISK:	NEW BEDFORD			10
FILE:	WFIIIa	No. meals/year	52	365
			1	

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227		CHILD:	40
ADULT:	227		ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 3
Ingestion of Flounder (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2780	227.0	1.00	40.0	6.31E-02	2.2E-04	3.2E-05	7.70E+00	NA	2.47E-05	2.47E-04

New Bedford
Area 3
Ingestion of Flounder (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2780	227.0	1.00	70.0	6.31E-02	1.3E-04	1.8E-05	7.70E+00	NA	1.41E-05	1.41E-04

TABLE C-44. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Flounder (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	[illegible]	[illegible]	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.8250	227.0	1.00	40.0	1.87E-01	[illegible]	6.7E-04	9.5E-05	7.70E+00	NA	7.34E-05	7.34E-04

New Bedford
Area 3
Ingestion of Flounder (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	[illegible]	[illegible]	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.8250	227.0	1.00	70.0	1.87E-01	[illegible]	3.8E-04	5.4E-05	7.70E+00	NA	4.19E-05	4.19E-04

TABLE C-45. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Flounder (monthly)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2780	0.8250	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS	
DIRECTORY:				
DISK:	NEW BEDFORD			10
FILE:	WFIIIb	No. meals/year	12	365

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 3
Ingestion of Flounder (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2780	227.0	1.00	40.0	6.31E-02	5.2E-05	7.4E-06	7.70E+00	NA	5.71E-06	5.71E-05

New Bedford
Area 3
Ingestion of Flounder (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2780	227.0	1.00	70.0	6.31E-02	3.0E-05	4.2E-06	7.70E+00	NA	3.26E-06	3.26E-05

TABLE C-45. BODY DOSE CALCULATIONS: MONTHLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Flounder (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.8250	227.0	1.00	40.0	1.87E-01		1.5E-04	2.2E-05	7.70E+00	NA	1.69E-05	1.69E-04

New Bedford
Area 3
Ingestion of Flounder (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.8250	227.0	1.00	70.0	1.87E-01		8.8E-05	1.3E-05	7.70E+00	NA	9.68E-06	9.68E-05

TABLE C-46. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Ingestion of Flounder (daily)	Area 4						
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.1010	0.3390	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS	
DIRECTORY:				
DISK:	NEW BEDFORD			10
FILE:	WF1V	No. meals/year	365	365
			1	

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 4
Ingestion of Flounder (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1010	227.0	1.00	40.0	2.29E-02		5.7E-04	8.2E-05	7.70E+00	NA	6.30E-05	6.30E-04

New Bedford
Area 4
Ingestion of Flounder (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1010	227.0	1.00	70.0	2.29E-02		3.3E-04	4.7E-05	7.70E+00	NA	3.60E-05	3.60E-04

TABLE C-46. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Flounder (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3390	227.0	1.00	40.0	7.70E-02	1.9E-03	2.7E-04	7.70E+00	NA	2.12E-04	2.11E-03

New Bedford
Area 4
Ingestion of Flounder (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3390	227.0	1.00	70.0	7.70E-02	1.1E-03	1.6E-04	7.70E+00	NA	1.21E-04	1.21E-03

TABLE C-47. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Flounder (weekly)	Area 4	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.1010	0.3390	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS		AMOUNT OF FISH CONSUMED (g/meal)		BODY WEIGHT (kg)	
DIRECTORY:					MOST PROBABLE			
DISK:	NEW BEDFORD			10	CHILD:	227	CHILD:	40
FILE:	NFIVa	No. meals/year	52	365	ADULT:	227	ADULT:	70
			1					

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 4
Ingestion of Flounder (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1010	227.0	1.00	40.0	2.29E-02	8.2E-05	1.2E-05	7.70E+00	NA	8.98E-06	8.98E-05

New Bedford
Area 4
Ingestion of Flounder (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1010	227.0	1.00	70.0	2.29E-02	4.7E-05	6.7E-06	7.70E+00	NA	5.13E-06	5.13E-05

TABLE C-47. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Flounder (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3390	227.0	1.00	40.0	7.70E-02		2.7E-04	3.9E-05	7.70E+00	NA	3.01E-05	3.01E-04

New Bedford
Area 4
Ingestion of Flounder (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3390	227.0	1.00	70.0	7.70E-02		1.6E-04	2.2E-05	7.70E+00	NA	1.72E-05	1.72E-04

TABLE C-48. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Flounder (monthly)	Area 4	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.1010	0.3390	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-

		NUMBER OF FISH MEALS	
DIRECTORY:			
DISK:	NEW BEDFORD		10
FILE:	WF1Vb	12	365
		1	

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 4
Ingestion of Flounder (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1010	227.0	1.00	40.0	2.29E-02	1.9E-05	2.7E-06	7.70E+00	NA	2.07E-06	2.07E-05

New Bedford
Area 4
Ingestion of Flounder (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1010	227.0	1.00	70.0	2.29E-02	1.1E-05	1.5E-06	7.70E+00	NA	1.18E-06	1.18E-05

TABLE C-48. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (WINTER FLOUNDER); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Flounder (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3390	227.0	1.00	40.0	7.70E-02		6.3E-05	9.0E-06	7.70E+00	NA	6.96E-06	6.96E-05

New Bedford
Area 4
Ingestion of Flounder (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3390	227.0	1.00	70.0	7.70E-02		3.6E-05	5.2E-06	7.70E+00	NA	3.98E-06	3.98E-05

TABLE [illegible] [illegible]; DAILY INGESTION OF DIETA (LOBSTER), AREA 2; ADULT AND OLDER CHILD.
[illegible] TABLE REPORT

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Lobster (daily)	Area 2	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.5680	1.2340	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS	
DIRECTORY:				
DISK:	NEW BEDFORD			10
FILE:	LOBI	No. meals/year	365	365

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 2
Ingestion of Lobster (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.5680	227.0	1.00	40.0	1.29E-01	3.2E-03	4.6E-04	7.70E+00	NA	3.55E-04	3.55E-03

New Bedford
Area 2
Ingestion of Lobster (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.5680	227.0	1.00	70.0	1.29E-01	1.8E-03	2.6E-04	7.70E+00	NA	2.03E-04	2.02E-03

TABLE C-49. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (LOBSTER); AREA 2; ADULT AND OLDER CHILD.

New Bedford
Area 2
Ingestion of Lobster (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.2340	227.0	1.00	40.0	2.80E-01		7.0E-03	1.0E-03	7.70E+00	NA	7.70E-04	7.67E-03

New Bedford
Area 2
Ingestion of Lobster (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.2340	227.0	1.00	70.0	2.80E-01		4.0E-03	5.7E-04	7.70E+00	NA	4.40E-04	4.39E-03

TABLE C-50. BODY DOSE CALCULATIONS: WEEKLY INGESTION OF BIOTA (LOBSTER); AREA 2; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Lobster (weekly)	Area 2	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.5680	1.2340	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:		NUMBER OF FISH MEALS		AMOUNT OF FISH CONSUMED (g/meal)		BODY WEIGHT (kg)	
DISK:	NEW BEDFORD		10	MOST PROBABLE			
FILE:	LOBIa	52	365	CHILD:	227	CHILD:	40
				ADULT:	227	ADULT:	70
				REALISTIC WORST			
				CHILD:	227		
				ADULT:	227		

New Bedford
Area 2
Ingestion of Lobster (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.5680	227.0	1.00	40.0	1.29E-01		4.6E-04	6.6E-05	7.70E+00	NA	5.05E-05	5.05E-04

New Bedford
Area 2
Ingestion of Lobster (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.5680	227.0	1.00	70.0	1.29E-01		2.6E-04	3.7E-05	7.70E+00	NA	2.89E-05	2.89E-04

TABLE C-50. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (LOBSTER); AREA 2; ADULT AND OLDER CHILD.

New Bedford
Area 2
Ingestion of Lobster (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.2340	227.0	1.00	40.0	2.80E-01	1.0E-03	1.4E-04	7.70E+00	NA	1.10E-04	1.10E-03

New Bedford
Area 2
Ingestion of Lobster (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.2340	227.0	1.00	70.0	2.80E-01	5.7E-04	8.1E-05	7.70E+00	NA	6.27E-05	6.27E-04

TABLE C-51. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (LOBSTER); AREA 2; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Lobster (monthly)	Area 2	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.5680	1.2340	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

MOST PROBABLE

DIRECTORY:

DISK: NEW BEDFORD

FILE: LOB1b

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 2
Ingestion of Lobster (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.5680	227.0	1.00	40.0	1.29E-01	1.1E-04	1.5E-05	7.70E+00	NA	1.17E-05	1.17E-04

New Bedford
Area 2
Ingestion of Lobster (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.5680	227.0	1.00	70.0	1.29E-01	6.1E-05	8.7E-06	7.70E+00	NA	6.66E-06	6.66E-05

TABLE C-51. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (LOBSTER); AREA 2; ADULT AND OLDER CHILD.

New Bedford
Area 2
Ingestion of Lobster (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.2340	227.0	1.00	40.0	2.80E-01	2.3E-04	3.3E-05	7.70E+00	NA	2.53E-05	2.53E-04

New Bedford
Area 2
Ingestion of Lobster (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	1.2340	227.0	1.00	70.0	2.80E-01	1.3E-04	1.9E-05	7.70E+00	NA	1.45E-05	1.45E-04

TABLE C-52. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (LOBSTER); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Lobster (daily)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2130	0.3510	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		NUMBER OF FISH MEALS		AMOUNT OF FISH CONSUMED (g/meal)			
DIRECTORY:				MOST PROBABLE		BODY WEIGHT (kg)	
DISK:	NEW BEDFORD		10				
FILE:	LOBIII	365	365	CHILD:	227	CHILD:	40
		1		ADULT:	227	ADULT:	70
				REALISTIC WORST			
				CHILD:	227		
				ADULT:	227		

New Bedford
Area 3
Ingestion of Lobster (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2130	227.0	1.00	40.0	4.84E-02	1.2E-03	1.7E-04	7.70E+00	NA	1.33E-04	1.33E-03

New Bedford
Area 3
Ingestion of Lobster (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		c e ay)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2130	227.0	1.00	70.0	4.84E-02	6.9E-04	9.9E-05	7.70E+00	NA	7.60E-05	7.60E-04

TABLE C-52. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (LOBSTER); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Lobster (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		: e ay)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3510	227.0	1.00	40.0	7.97E-02	2.0E-03	2.8E-04	7.70E+00	NA	2.19E-04	2.19E-03

New Bedford
Area 3
Ingestion of Lobster (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		: e ay)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3510	227.0	1.00	70.0	7.97E-02	1.1E-03	1.6E-04	7.70E+00	NA	1.25E-04	1.25E-03

TABLE C-53. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (LOBSTER); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY ESTIMATE
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	
EXPOSURE:	Ingestion of Lobster (weekly)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2130	0.3510	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-8

DIRECTORY:		NUMBER OF FISH MEALS		AMOUNT OF FISH CONSUMED (g/meal)			
DISK:	NEW BEDFORD		10	MOST PROBABLE		BODY WEIGHT (kg)	
FILE:	LOBIIIa	52	365	CHILD:	227	CHILD:	40
		1		ADULT:	227	ADULT:	70
				REALISTIC WORST			
				CHILD:	227		
				ADULT:	227		

New Bedford
Area 3
Ingestion of Lobster (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2130	227.0	1.00	40.0	4.84E-02	1.7E-04	2.5E-05	7.70E+00	NA	1.89E-05	1.89E-04

New Bedford
Area 3
Ingestion of Lobster (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2130	227.0	1.00	70.0	4.84E-02	9.8E-05	1.4E-05	7.70E+00	NA	1.08E-05	1.08E-04

TABLE C-53. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (LOBSTER); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Lobster (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3510	227.0	1.00	40.0	7.97E-02		2.8E-04	4.1E-05	7.70E+00	NA	3.12E-05	3.12E-04

New Bedford
Area 3
Ingestion of Lobster (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3510	227.0	1.00	70.0	7.97E-02		1.6E-04	2.3E-05	7.70E+00	NA	1.78E-05	1.78E-04

TABLE C-54. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (LOBSTER); AREA 3; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Lobster (monthly)	Area 3	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.2130	0.3510	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY: MOST PROBABLE

DISK: NEW BEDFORD

FILE: LOBIIIb

	NUMBER OF FISH MEALS	
		10
	12	365
	1	

AMOUNT OF FISH CONSUMED (g/meal)

MOST PROBABLE		BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

REALISTIC WORST	
CHILD:	227
ADULT:	227

New Bedford
Area 3
Ingestion of Lobster (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2130	227.0	1.00	40.0	4.84E-02	4.0E-05	5.7E-06	7.70E+00	NA	4.37E-06	4.37E-05

New Bedford
Area 3
Ingestion of Lobster (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.2130	227.0	1.00	70.0	4.84E-02	2.3E-05	3.2E-06	7.70E+00	NA	2.50E-06	2.50E-05

TABLE C-54. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (LOBSTER); AREA 3; ADULT AND OLDER CHILD.

New Bedford
Area 3
Ingestion of Lobster (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			y)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3510	227.0	1.00	40.0	7.97E-02		6.5E-05	9.4E-06	7.70E+00	NA	7.20E-06	7.20E-05

New Bedford
Area 3
Ingestion of Lobster (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.3510	227.0	1.00	70.0	7.97E-02		3.7E-05	5.3E-06	7.70E+00	NA	4.12E-06	4.12E-05

TABLE C-55. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (LOBSTER); AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g) MOST PROBABLE	REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Ingestion of Lobster (daily)	Area 4						
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.0640	0.1760	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY: MOST PROBABLE

DISK: NEW BEDFORD

FILE: LOBIV

NUMBER OF FISH MEALS: 10

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	227	40
ADULT:	227	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

1

New Bedford
Area 4
Ingestion of Lobster (daily)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0640	227.0	1.00	40.0	1.45E-02	3.6E-04	5.2E-05	7.70E+00	NA	4.00E-05	4.00E-04

New Bedford
Area 4
Ingestion of Lobster (daily)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	[illegible]	[illegible]	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0640	227.0	1.00	70.0	1.45E-02	2.1E-04	3.0E-05	7.70E+00	NA	2.20E-05	2.20E-04

TABLE C-55. BODY DOSE CALCULATIONS; DAILY INGESTION OF BIOTA (LOBSTER); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Lobster (daily)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1760	227.0	1.00	40.0	4.00E-02	1.0E-03	1.4E-04	7.70E+00	NA	1.10E-04	1.10E-03

New Bedford
Area 4
Ingestion of Lobster (daily)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		c e ay)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1760	227.0	1.00	70.0	4.00E-02	5.7E-04	8.2E-05	7.70E+00	NA	6.28E-05	6.28E-04

TABLE C-56. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (LOBSTER); AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Lobster (weekly)	Area 4	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.0640	0.1760	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

		MOST PROBABLE	NUMBER OF FISH MEALS	
DIRECTORY:				
DISK:	NEW BEDFORD			10
FILE:	LDB1Va		52	365
			1	

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	227	CHILD: 40
ADULT:	227	ADULT: 70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 4
Ingestion of Lobster (weekly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp Subchronic	10 year exp Chronic	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0640	227.0	1.00	40.0	1.45E-02	5.2E-05	7.4E-06	7.70E+00	NA	5.69E-06	5.69E-05

New Bedford
Area 4
Ingestion of Lobster (weekly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0640	227.0	1.00	70.0	1.45E-02	3.0E-05	4.2E-06	7.70E+00	NA	3.25E-06	3.25E-05

TABLE C-56. BODY DOSE CALCULATIONS; WEEKLY INGESTION OF BIOTA (LOBSTER); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Lobster (weekly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1760	227.0	1.00	40.0	4.00E-02		1.4E-04	2.0E-05	7.70E+00	NA	1.57E-05	1.57E-04

New Bedford
Area 4
Ingestion of Lobster (weekly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			c ? ay)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1760	227.0	1.00	70.0	4.00E-02		8.1E-05	1.2E-05	7.70E+00	NA	8.94E-06	8.94E-05

TABLE C-57. BODY DOSE CALCULATIONS: MONTHLY INGESTION OF BIOTA (LOBSTER); AREA 4; ADULT AND OLDER CHILD.

SUMMARY TABLE HEADER:

				CONCENTRATION IN EDIBLE PORTION OF FISH (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE:	New Bedford		CONTAMINANTS	MOST	REALISTIC	MOST	REALISTIC	ESTIMATE
EXPOSURE:	Ingestion of Lobster (monthly)	Area 4	OF CONCERN:	PROBABLE	WORST	PROBABLE	WORST	(mg/kg/day)-1
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	0.0640	0.1760	1.00	1.00	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

DIRECTORY:		MOST PROBABLE NUMBER OF FISH MEALS	
DISK:	NEW BEDFORD		10
FILE:	LOBIV6	12	365

AMOUNT OF FISH CONSUMED (g/meal)

	MOST PROBABLE	BODY WEIGHT (kg)	
CHILD:	227	CHILD:	40
ADULT:	227	ADULT:	70

	REALISTIC WORST
CHILD:	227
ADULT:	227

New Bedford
Area 4
Ingestion of Lobster (monthly)
Carcinogenic Effects
Most Probable Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	1 year exp	10 year exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0640	227.0	1.00	40.0	1.45E-02	1.2E-05	1.7E-06	7.70E+00	NA	1.31E-06	1.31E-05

New Bedford
Area 4
Ingestion of Lobster (monthly)
Carcinogenic Effects
Most Probable Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)	[illegible]	[illegible]	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.0640	227.0	1.00	70.0	1.45E-02	6.8E-06	9.7E-07	7.70E+00	NA	7.51E-07	7.51E-06

TABLE C-57. BODY DOSE CALCULATIONS; MONTHLY INGESTION OF BIOTA (LOBSTER); AREA 4; ADULT AND OLDER CHILD.

New Bedford
Area 4
Ingestion of Lobster (monthly)
Carcinogenic Effects
Realistic Worst Case
Older Child

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)		.c .e Jay)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1760	227.0	1.00	40.0	4.00E-02	3.3E-05	4.7E-06	7.70E+00	NA	3.61E-06	3.61E-05

New Bedford
Area 4
Ingestion of Lobster (monthly)
Carcinogenic Effects
Realistic Worst Case
Adult

Compound	Concentration (ug/g)	Amount of Fish Consumed (g/fish meal)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)			CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	0.1760	227.0	1.00	70.0	4.00E-02	1.9E-05	2.7E-06	7.70E+00	NA	2.06E-06	2.06E-05

TABLE C-58. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; MARSH ISLAND (AREA II); CHILD

SUMMARY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Direct Contact with Sediment	Area II; Marsh Island	PCB's	8.0000	22.0000	0.07	0.07	7.70E+00
	Carcinogenic Effects	Noncarcinogenic Effect						
	Most Probable Case	Realistic Worst Case						
	Child	Adult						

DATE: 20-Mar-89

MOST PROBABLE

DIRECTORY: EXP

DISK: NEW BEDFORD

FILE: SEDIM

Frequency of contact

Frequency of contact

AMOUNT OF SEDIMENT CONTACTED (g/exp)

	MOST PROBABLE	BODY WEIGHT (kg)
CHILD:	3.42	10
ADULT:	N/A	N/A

	REALISTIC WORST	ADULT LIFETIME EXPOSURE:
CHILD:	3.7875	55
ADULT:	N/A	

New Bedford
Area II; Marsh Island
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)	1 year exp	10 yrs exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	8.0000	3.4	0.07	10.0	1.92E-03	NA	1.0E-05	5.2E-05	7.70E+00	1.15E-06	5.77E-06	NA

New Bedford
Area II; Marsh Island
Direct Contact with Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)	1 year exp	10 yrs exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
PCB's	22.0000	3.8	0.07	10.0	5.83E-03	NA	1.6E-04	8.0E-04	7.70E+00	1.76E-05	8.79E-05	NA

'A: ..-58. BODY DOSE CALCULATIONS; DIRECT CONTACT WITH SEDIMENTS; MARSH ISLAND (AREA II); ADULTS AND OLDER CHILDREN.
SU RY TABLE HEADER:

			CONTAMINANTS OF CONCERN:	CONCENTRATION (ug/g) MOST PROBABLE	CONCENTRATION (ug/g) REALISTIC WORST	FRACTION ABSORBED (%) MOST PROBABLE	FRACTION ABSORBED (%) REALISTIC WORST	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford							
EXPOSURE:	Direct Contact with Sediment	Area II; Marsh Island						
	Carcinogenic Effects	Noncarcinogenic Effect	PCB's	8.0000	22.0000	0.07	0.07	7.70E+00
	Most Probable Case	Realistic Worst Case						
	Older Child	Adult						

DATE: 20-Mar-89

MOST PROBABLE

D TORY:		EXPOSURES	DAYS	AMOUNT OF SEDIMENT CONTACTED (g/exp) MOST PROBABLE		BODY WEIGHT (kg)	
D	NEW BEDFORD	1	10				
FILE:	SEDIIMA	20	365		CHILD:	6.6	CHILD: 40
		20			ADULT:	4.59	ADULT: 70
				REALISTIC WORST		ADULT LIFETIME EXPOSURE:	
					CHILD:	6.6225	55
					ADULT:	7.485	

Bedford
II: Marsh Island
Direct Contact with Sediment
Carcinogenic Effects
Most Probable Case
r Child

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day)	1 year exp (mg/kg-day)	10 yrs exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
's	8.0000	6.6	0.07	40.0	3.70E-03	NA	5.1E-06	5.1E-05	7.70E+00	5.57E-07	5.57E-06	NA

Bedford
a II: Marsh Island
ect Contact with Sediment
Carcinogenic Effects
st Probable Case
lt

Compound	Concentration (ug/g)	Amount of Sediment Contacted (g/exp)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exp)	Short-term body dose (mg/kg-day	1 year exp	10 yrs exp	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Sub-chronic	Risk Estimate Chronic	Risk Estimate Lifetime
's	8.0000	4.6	0.07	70.0	2.57E-03	NA	2.0E-06	2.0E-05	7.70E+00	2.21E-07	2.21E-06	1.22E-05

TABLE C-59. BODY DOSE CALCULATIONS; INGESTION OF BIOTA; NONCARCINOGENIC RISK ESTIMATES FOR PCB EXPOSURE (MEAN CONCENTRATION).

Species	Mean PCB Conc. (ppm)	Area	Body Dose Teen (mg/kg-day)	Body Dose Adult (mg/kg-day)	Longer-Term HA (a) (mg/kg-day)	Risk Ratio (Teen)	Risk Ratio (Adult)	Body Dose Child (mg/kg-day)	Risk Ratio (Child)
Lobster									
Weekly Ingestion	0.568	2	4.59E-04	2.62E-04	0.0001	4.59	2.62	9.18E-04	9.18
	0.213	3	1.72E-04	9.84E-05	0.0001	1.72	0.98	3.44E-04	3.44
	0.064	4	5.17E-05	2.96E-05	0.0001	0.52	0.30	1.03E-04	1.03
Daily Ingestion	0.568	2	3.22E-03	1.84E-03	0.0001	32.23	18.42	6.45E-03	64.47
	0.213	3	1.21E-03	6.91E-04	0.0001	12.09	6.91	2.42E-03	24.18
	0.064	4	3.63E-04	2.08E-04	0.0001	3.63	2.08	7.26E-04	7.26
Flounder									
Weekly Ingestion	1.039	1	8.40E-04	4.80E-04	0.0001	8.40	4.80	1.68E-03	16.80
	0.371	2	3.00E-04	1.71E-04	0.0001	3.00	1.71	6.00E-04	6.00
	0.278	3	2.25E-04	1.28E-04	0.0001	2.25	1.28	4.50E-04	4.50
	0.101	4	8.17E-05	4.67E-05	0.0001	0.82	0.47	1.63E-04	1.63
Daily Ingestion	1.039	1	5.90E-03	3.37E-03	0.0001	58.96	33.69	1.18E-02	117.93
	0.371	2	2.11E-03	1.20E-03	0.0001	21.05	12.03	4.21E-03	42.11
	0.278	3	1.58E-03	9.02E-04	0.0001	15.78	9.02	3.16E-03	31.55
	0.101	4	5.73E-04	3.28E-04	0.0001	5.73	3.28	1.15E-03	11.46
Clam									
Weekly Ingestion	0.689	1	5.57E-04	3.18E-04	0.0001	5.57	3.18	1.11E-03	11.14
	0.231	2	1.87E-04	1.07E-04	0.0001	1.87	1.07	3.74E-04	3.74
	0.156	3	1.26E-04	7.21E-05	0.0001	1.26	0.72	2.52E-04	2.52
	0.039	4	3.15E-05	1.80E-05	0.0001	0.32	0.18	6.31E-05	0.63
Daily Ingestion	0.689	1	3.91E-03	2.23E-03	0.0001	39.10	22.34	7.82E-03	78.20
	0.231	2	1.31E-03	7.49E-04	0.0001	13.11	7.49	2.62E-03	26.22
	0.156	3	8.85E-04	5.06E-04	0.0001	8.85	5.06	1.77E-03	17.71
	0.039	4	2.21E-04	1.26E-04	0.0001	2.21	1.26	4.43E-04	4.43

(a) The longer term HA for PCBs (0.001 mg/l - Child) was converted to mg/kg-day as follows:
0.001 mg/l X 1 l/day X 1/10 kg = 0.0001 mg/kg-day
The longer term HA for PCBs (0.0035 mg/l - Adult) was converted to mg/kg-day as follows:
0.0035 mg/l X 2 l/day X 1/70 kg = 0.0001 mg/kg-day

NOTE: The mean value represents the arithmetic mean.

Exposure assumptions used to estimate body dose levels:

Amount Ingested (g)	TKF unitless	Bodyweight (Teen) (kg)	Bodyweight (Adult) (kg)	Weekly Ingestion (meals/yr)	Daily Ingestion (meals/yr)
227	1	40	70	52	365

TABLE C-60. BODY DOSE CALCULATIONS; INGESTION OF BIOTA; NONCARCINOGENIC RISK ESTIMATES FOR PCB EXPOSURE (MAXIMUM CONCENTRATION).

Species	Max PCB Conc. (ppm)	Area	Body Dose Teen (mg/kg-day)	Body Dose Adult (mg/kg-day)	Longer-Term HA (a) (mg/kg-day)	Risk Ratio (Teen)	Risk Ratio (Adult)	Body Dose Child (mg/kg-day)	Risk Ratio (Child)
Lobster									
Weekly Ingestion	1.234	2	9.98E-04	5.70E-04	0.0001	9.98	5.70	2.00E-03	19.95
	0.351	3	2.84E-04	1.62E-04	0.0001	2.84	1.62	5.68E-04	5.68
	0.176	4	1.42E-04	8.13E-05	0.0001	1.42	0.81	2.85E-04	2.85
Daily Ingestion	1.234	2	7.00E-03	4.00E-03	0.0001	70.03	40.02	1.40E-02	140.06
	0.351	3	1.99E-03	1.14E-03	0.0001	19.92	11.38	3.98E-03	39.84
	0.176	4	9.99E-04	5.71E-04	0.0001	9.99	5.71	2.00E-03	19.98
Flounder									
Weekly Ingestion	2.629	1	2.13E-03	1.21E-03	0.0001	21.26	12.15	4.25E-03	42.51
	1.048	2	8.47E-04	4.84E-04	0.0001	8.47	4.84	1.69E-03	16.95
	0.825	3	6.67E-04	3.81E-04	0.0001	6.67	3.81	1.33E-03	13.34
	0.34	4	2.75E-04	1.57E-04	0.0001	2.75	1.57	5.50E-04	5.50
Daily Ingestion	2.629	1	1.49E-02	8.53E-03	0.0001	149.20	85.25	2.98E-02	298.39
	1.048	2	5.95E-03	3.40E-03	0.0001	59.47	33.99	1.19E-02	118.95
	0.825	3	4.68E-03	2.68E-03	0.0001	46.82	26.75	9.36E-03	93.64
	0.34	4	1.93E-03	1.10E-03	0.0001	19.30	11.03	3.86E-03	38.59
Clam									
Weekly Ingestion	2.121	1	1.71E-03	9.80E-04	0.0001	17.15	9.80	3.43E-03	34.30
	1.181	2	9.55E-04	5.46E-04	0.0001	9.55	5.46	1.91E-03	19.10
	0.478	3	3.86E-04	2.21E-04	0.0001	3.86	2.21	7.73E-04	7.73
	0.137	4	1.11E-04	6.33E-05	0.0001	1.11	0.63	2.22E-04	2.22
Daily Ingestion	2.121	1	1.20E-02	6.88E-03	0.0001	120.37	68.78	2.41E-02	240.73
	1.181	2	6.70E-03	3.83E-03	0.0001	67.02	38.30	1.34E-02	134.04
	0.478	3	2.71E-03	1.55E-03	0.0001	27.13	15.50	5.43E-03	54.25
	0.137	4	7.77E-04	4.44E-04	0.0001	7.77	4.44	1.55E-03	15.55

(a) The longer term HA for PCBs (0.001 mg/l - Child) was converted to mg/kg-day as follows:
0.001 mg/l X 1 l/day X 1/10 kg = 0.0001 mg/kg-day
The longer term HA for PCBs (0.0035 mg/l - Adult) was converted to mg/kg-day as follows:
0.0035 mg/l X 2 l/day X 1/70 kg = 0.0001 mg/kg-day

NOTE: The mean value represents the arithmetic mean.

Exposure assumptions used to estimate body dose levels:

Amount Ingested (g)	TKF unitless	Bodyweight (Teen) (kg)	Bodyweight (Adult) (kg)	Weekly Ingestion (meals/yr)	Daily Ingestion (meals/yr)
227	1	40	70	52	365

TABLE C-61. BODY DOSE CALCULATIONS; INGESTION OF BIOTA; NONCARCINOGENIC RISK ESTIMATES FOR CADMIUM EXPOSURE (MEAN CONCENTRATIONS).

31-May-89

Species	Mean Cd Conc. (ppm)	Area	Body Dose Teen (mg/kg-day)	Body Dose Adult (mg/kg-day)	Converted HA (a) (mg/kg-day)	LONGER-TERM HA Risk Ratio (Teen)	LONGER-TERM HA Risk Ratio (Adult)	Body Dose Child (mg/kg-day)	Risk Ratio (Child)
Lobster									
Weekly Ingestion	0.38	2	3.07E-04	1.76E-04	0.0005	0.598	0.342	0.0006	1.20
	0.33	3	2.67E-04	1.52E-04	0.0005	0.519	0.297	0.0005	1.04
	0.26	4	2.10E-04	1.20E-04	0.0005	0.409	0.234	0.0004	0.82
Daily Ingestion	0.38	2	2.16E-03	1.23E-03	0.0005	4.196	2.397	0.0043	8.39
	0.33	3	1.87E-03	1.07E-03	0.0005	3.643	2.082	0.0037	7.29
	0.26	4	1.48E-03	8.43E-04	0.0005	2.871	1.640	0.0030	5.74
Flounder									
Weekly Ingestion	0.0085	1	6.87E-06	3.93E-06	0.0005	0.013	0.008	0.000014	0.03
	0.009	2	7.28E-06	4.16E-06	0.0005	0.014	0.008	0.000015	0.03
	0.005	3	4.04E-06	2.31E-06	0.0005	0.008	0.004	0.000008	0.02
	0.099	4	8.00E-05	4.57E-05	0.0005	0.156	0.089	0.0002	0.31
Daily Ingestion	0.0085	1	4.82E-05	2.76E-05	0.0005	0.094	0.054	0.00010	0.19
	0.009	2	5.11E-05	2.92E-05	0.0005	0.099	0.057	0.00010	0.20
	0.005	3	2.84E-05	1.62E-05	0.0005	0.055	0.032	0.00006	0.11
	0.099	4	5.62E-04	3.21E-04	0.0005	1.093	0.625	0.0011	2.19
Clam									
Weekly Ingestion	0.172	1	1.39E-04	7.95E-05	0.0005	0.271	0.155	0.0003	0.54
	0.256	2	2.07E-04	1.18E-04	0.0005	0.403	0.230	0.0004	0.81
	0.295	3	2.39E-04	1.36E-04	0.0005	0.464	0.265	0.0005	0.93
	0.32	4	2.59E-04	1.48E-04	0.0005	0.503	0.288	0.0005	1.01
Daily Ingestion	0.172	1	9.76E-04	5.58E-04	0.0005	1.899	1.085	0.0020	3.80
	0.256	2	1.45E-03	8.30E-04	0.0005	2.826	1.615	0.0029	5.65
	0.295	3	1.67E-03	9.57E-04	0.0005	3.257	1.861	0.0033	6.51
	0.32	4	1.82E-03	1.04E-03	0.0005	3.533	2.019	0.0036	7.07

(a) The Longer-term HA for Cd (0.018 mg/l) was converted to mg/kg-day using the following equation:
0.018 mg/l X 2 l/day X 1/70 kg = 0.000514 mg/kg-day (Adult and Teen Exposure)
The Longer-term HA for Cd (.005 mg/l) was converted to mg/kg-day using the following equation:
0.005 mg/l X 1 l/day X 1/10 kg = .0005

(b) The 10-day HA for Cd (0.04 mg/l) was converted to mg/kg-day using the following equation:
0.04 mg/l X 1 l/day X 1/10kg = 0.004 mg/kg-day

NOTE: The mean value represents the arithmetic mean.

TABLE C-62. BODY DOSE CALCULATIONS; INGESTION OF BIOTA; NONCARCINOGENIC RISK ESTIMATES FOR CADMIUM EXPOSURE (MAXIMUM CONCENTRATIONS).

31-May-89

Species	Max Cd Conc. (ppm)	Area	Body Dose Teen (mg/kg-day)	Body Dose Adult (mg/kg-day)	Converted HA (a) (mg/kg-day)	LONGER-TERM HA Risk Ratio (Teen)	LONGER-TERM HA Risk Ratio (Adult)	Body Dose Child (mg/kg-day)	Risk Ratio (Child)
Lobster									
Weekly Ingestion	0.7	2	5.66E-04	3.23E-04	0.0005	1.10	0.629	0.0011	2.20
	0.54	3	4.37E-04	2.49E-04	0.0005	0.85	0.485	0.0009	1.70
	0.59	4	4.77E-04	2.73E-04	0.0005	0.93	0.530	0.0010	1.86
Daily Ingestion	0.7	2	3.97E-03	2.27E-03	0.0005	7.73	4.416	0.0079	15.46
	0.54	3	3.06E-03	1.75E-03	0.0005	5.96	3.407	0.0061	11.92
	0.59	4	3.35E-03	1.91E-03	0.0005	6.51	3.722	0.0067	13.03
Flounder									
Weekly Ingestion	0.014	1	1.13E-05	6.47E-06	0.0005	0.02	0.013	0.000023	0.04
	0.02	2	1.62E-05	9.24E-06	0.0005	0.03	0.018	0.000032	0.06
	0.012	3	9.70E-06	5.54E-06	0.0005	0.02	0.011	0.000019	0.04
	0.09	4	7.28E-05	4.16E-05	0.0005	0.14	0.081	0.0001	0.28
Daily Ingestion	0.014	1	7.95E-05	4.54E-05	0.0005	0.15	0.088	0.00016	0.31
	0.02	2	1.14E-04	6.49E-05	0.0005	0.22	0.126	0.00023	0.44
	0.012	3	6.81E-05	3.89E-05	0.0005	0.13	0.076	0.00014	0.26
	0.09	4	5.11E-04	2.92E-04	0.0005	0.99	0.568	0.0010	1.99
Clam									
Weekly Ingestion	0.36	1	2.91E-04	1.66E-04	0.0005	0.57	0.324	0.0006	1.13
	0.33	2	2.67E-04	1.52E-04	0.0005	0.52	0.297	0.0005	1.04
	0.38	3	3.07E-04	1.76E-04	0.0005	0.60	0.342	0.0006	1.20
	0.495	4	4.00E-04	2.29E-04	0.0005	0.78	0.445	0.0008	1.56
Daily Ingestion	0.36	1	2.04E-03	1.17E-03	0.0005	3.97	2.271	0.0041	7.95
	0.33	2	1.87E-03	1.07E-03	0.0005	3.64	2.082	0.0037	7.29
	0.38	3	2.16E-03	1.23E-03	0.0005	4.20	2.397	0.0043	8.39
	0.495	4	2.81E-03	1.61E-03	0.0005	5.47	3.123	0.0056	10.93

(a) The Longer-term HA for Cd (0.018 mg/l) was converted to mg/kg-day using the following equation:
0.018 mg/l X 2 l/day X 1/70 kg = 0.000514 mg/kg-day (Adult and Teen Exposure)
The Longer-term HA for Cd (.005 mg/l) was converted to mg/kg-day using the following equation:
0.005 mg/l X 1 l/day X 1/10 kg = .0005

(b) The 10-day HA for Cd (0.04 mg/l) was converted to mg/kg-day using the following equation:
0.04 mg/l X 1 l/day X 1/10kg = 0.004 mg/kg-day

NOTE: The mean value represents the arithmetic mean.

TABLE C-63. BODY DOSE CALCULATIONS; INGESTION OF BIOTA; NONCARCINOGENIC RISK ESTIMATES FOR COPPER (MEAN CONCENTRATIONS).

Species	Mean Cu Conc. (ppm)	Area	Body Dose Teen (mg/kg-day)	Body Dose Adult (mg/kg-day)	Converted MCLG (a) (mg/kg-day)	Risk Ratio (Teen)	Risk Ratio (Adult)	Body Dose Child (mg/kg-day)	Risk Ratio (Child)
Flounder									
Weekly Ingestion	3.1	1	2.51E-03	1.43E-03	0.0371	0.07	0.04	0.005	0.14
	3.7	2	2.99E-03	1.71E-03	0.0371	0.08	0.05	0.006	0.16
	9.7	3	7.84E-03	4.48E-03	0.0371	0.21	0.12	0.016	0.42
	9.6	4	7.76E-03	4.44E-03	0.0371	0.21	0.12	0.016	0.42
Daily Ingestion	3.1	1	1.76E-02	1.01E-02	0.0371	0.47	0.27	0.035	0.95
	3.7	2	2.10E-02	1.20E-02	0.0371	0.57	0.32	0.042	1.13
	9.7	3	5.50E-02	3.15E-02	0.0371	1.48	0.85	0.110	2.97
	9.6	4	5.45E-02	3.11E-02	0.0371	1.47	0.84	0.109	2.94

(a) The MCLG for copper (1.3 mg/l) was converted to mg/kg-day using the following formula:

1.3 mg/l X 2l/day X 1/70 kg = 0.0371 mg/kg-day

NOTE: The mean value represents the arithmetic mean.

TABLE C-64. BODY DOSE CALCULATIONS; INGESTION OF BIOTA; NONCARCINOGENIC RISK ESTIMATES FOR COPPER (MAXIMUM CONCENTRATIONS).

Species	Max. Cu Conc. (ppm)	Area	Body Dose Teen (mg/kg-day)	Body Dose Adult (mg/kg-day)	Converted MCLG (a) (mg/kg-day)	Risk Ratio (Teen)	Risk Ratio (Adult)	Body Dose Child (mg/kg-day)	Risk Ratio (Child)
Flounder									
Weekly Ingestion	11.1	1	8.97E-03	5.13E-03	0.0371	0.24	0.14	0.018	0.48
	19.8	2	1.60E-02	9.15E-03	0.0371	0.43	0.25	0.032	0.86
	51.6	3	4.17E-02	2.38E-02	0.0371	1.12	0.64	0.083	2.25
	43.9	4	3.55E-02	2.03E-02	0.0371	0.96	0.55	0.071	1.91
Daily Ingestion	11.1	1	6.30E-02	3.60E-02	0.0371	1.70	0.97	0.126	3.40
	19.8	2	1.12E-01	6.42E-02	0.0371	3.03	1.73	0.225	6.06
	51.6	3	2.93E-01	1.67E-01	0.0371	7.89	4.51	0.586	15.79
	43.9	4	2.49E-01	1.42E-01	0.0371	6.72	3.84	0.498	13.43

(a) The MCLG for copper (1.3 mg/l) was converted to mg/kg-day using the following formula:
1.3 mg/l X 2l/day X 1/70 kg = 0.0371 mg/kg-day

NOTE: The mean value represents the arithmetic mean.

TABLE C-65. BODY DOSE CALCULATIONS; INGESTION OF BIOTA; NONCARCINOGENIC RISK ESTIMATES
LEAD EXPOSURE (MEAN CONCENTRATION).

Species	Mean Pb Conc. (ppm)	Area	Body Dose Teen (mg/kg-day)	Body Dose Adult (mg/kg-day)	Converted MCL (a) (mg/kg-day)	Risk Ratio (Teen)	Risk Ratio (Adult)	Body Dose Child (mg/kg-day)	Risk Ratio (Child)
Lobster									
Weekly Ingestion	0.99	2	8.00E-04	4.57E-04	0.00014	5.60	3.20	1.62E-03	11.35
	0.38	3	3.07E-04	1.76E-04	0.00014	2.15	1.23	6.14E-04	4.30
	0.23	4	1.86E-04	1.06E-04	0.00014	1.30	0.74	3.72E-04	2.60
Daily Ingestion	0.99	2	5.62E-03	3.21E-03	0.00014	39.33	22.47	1.12E-02	78.66
	0.38	3	2.16E-03	1.23E-03	0.00014	15.10	8.63	4.31E-03	30.19
	0.23	4	1.31E-03	7.46E-04	0.00014	9.14	5.22	2.61E-03	18.27
Flounder									
Weekly Ingestion	0.89	1	7.20E-04	4.11E-04	0.00014	5.04	2.88	1.44E-03	10.07
	0.83	2	6.71E-04	3.83E-04	0.00014	4.70	2.68	1.34E-03	9.39
	0.63	3	5.09E-04	2.91E-04	0.00014	3.57	2.04	1.02E-03	7.13
	1.2	4	9.70E-04	5.54E-04	0.00014	6.79	3.88	1.94E-03	13.58
Daily Ingestion	0.89	1	5.05E-03	2.89E-03	0.00014	35.36	20.20	1.01E-02	70.71
	0.83	2	4.71E-03	2.69E-03	0.00014	32.97	18.84	9.42E-03	65.94
	0.63	3	3.58E-03	2.04E-03	0.00014	25.03	14.30	7.15E-03	50.05
	1.2	4	6.81E-03	3.89E-03	0.00014	47.67	27.24	1.36E-02	95.34
Clam									
Weekly Ingestion	1.01	1	8.17E-04	4.67E-04	0.00014	5.72	3.27	1.63E-03	11.43
	0.76	2	6,14E-04	3.51E-04	0.00014	4.30	2.46	1.23E-03	8.60
	1.28	3	1.03E-03	5.91E-04	0.00014	7.24	4.14	2.07E-03	14.49
	0.97	4	7.84E-04	4.48E-04	0.00014	5.49	3.14	1.57E-03	10.98
Daily Ingestion	1.01	1	5.73E-03	3.28E-03	0.00014	40.12	22.93	1.15E-02	80.24
	0.76	2	4.31E-03	2.46E-03	0.00014	30.19	17.25	8.63E-03	60.38
	1.28	3	7.26E-03	4.15E-03	0.00014	50.85	29.06	1.45E-02	101.70
	0.97	4	5.50E-03	3.15E-03	0.00014	38.53	22.02	1.10E-02	77.07

(a) The proposed MCL for lead (0.005 mg/l) was converted to mg/kg-day using the following equation:

0.005 mg/l X 2l/day X 1/70 kg = 0.00014 mg/kg-day

Exposure assumptions used to estimate body dose levels:

	Amount Ingested (g)	TKF unitless	Bodyweight (Teen) (kg)	Bodyweight (Adult) (kg)	Weekly Ingestion (meals/yr)	Daily Ingestion (meals/yr)
	227	1	40	70	52	365
(Child)	115	1	13		52	365

NOTE: The mean value represents the arithmetic mean

TABLE C-66. BODY DOSE CALCULATIONS; INGESTION OF BIOTA; NONCARCINOGENIC RISK ESTIMATES FOR LEAD EXPOSURE (MAXIMUM CONCENTRATION).

Species	Max. Pb Conc. (ppm)	Area	Body Dose Teen (mg/kg-day)	Body Dose Adult (mg/kg-day)	Converted MCL (a) (mg/kg-day)	Risk Ratio (Teen)	Risk Ratio (Adult)	Body Dose Child (mg/kg-day)	Risk Ratio (Child)
Lobster									
Weekly Ingestion	3.3	2	2.67E-03	1.52E-03	0.00014	18.68	10.67	5.34E-03	37.35
	1.12	3	9.06E-04	5.17E-04	0.00014	6.34	3.62	1.81E-03	12.68
	0.84	4	6.79E-04	3.88E-04	0.00014	4.75	2.72	1.36E-03	9.51
Daily Ingestion	3.3	2	1.87E-02	1.07E-02	0.00014	131.09	74.91	3.75E-02	262.18
	1.12	3	6.36E-03	3.63E-03	0.00014	44.49	25.42	1.27E-02	88.98
	0.84	4	4.77E-03	2.72E-03	0.00014	33.37	19.07	9.53E-03	66.74
Flounder									
Weekly Ingestion	3.35	1	2.71E-03	1.55E-03	0.00014	18.96	10.83	5.42E-03	37.92
	4.52	2	3.65E-03	2.09E-03	0.00014	25.58	14.62	7.31E-03	51.16
	2.72	3	2.20E-03	1.26E-03	0.00014	15.39	8.80	4.40E-03	30.79
	6.84	4	5.53E-03	3.16E-03	0.00014	38.71	22.12	1.11E-02	77.42
Daily Ingestion	3.35	1	1.90E-02	1.09E-02	0.00014	133.08	76.03	3.80E-02	266.16
	4.52	2	2.57E-02	1.47E-02	0.00014	179.56	102.60	5.13E-02	359.11
	2.72	3	1.54E-02	8.82E-03	0.00014	108.05	61.74	3.09E-02	216.10
	6.84	4	3.88E-02	2.22E-02	0.00014	271.72	155.27	7.76E-02	543.44
Clam									
Weekly Ingestion	1.9	1	1.54E-03	8.78E-04	0.00014	10.75	6.14	3.07E-03	21.51
	0.98	2	7.92E-04	4.53E-04	0.00014	5.55	3.17	1.58E-03	11.09
	3.46	3	2.80E-03	1.60E-03	0.00014	19.58	11.19	5.59E-03	39.16
	1.72	4	1.39E-03	7.95E-04	0.00014	9.73	5.56	2.78E-03	19.47
Daily Ingestion	1.9	1	1.08E-02	6.16E-03	0.00014	75.48	43.13	2.16E-02	150.95
	0.98	2	5.56E-03	3.18E-03	0.00014	38.93	22.25	1.11E-02	77.86
	3.46	3	1.96E-02	1.12E-02	0.00014	137.45	78.54	3.93E-02	274.90
	1.72	4	9.76E-03	5.58E-03	0.00014	68.33	39.04	1.95E-02	136.65

(a) The proposed MCL for lead (0.005 mg/l) was converted to mg/kg-day using the following formulas:

0.005 mg/l X 2l/day X 1/70 kg = 0.00014 mg/kg-day

Exposure assumptions used to estimate body dose levels:

	Amount Ingested (g)	TKF unitless	Bodyweight (Teen) (kg)	Bodyweight (Adult) (kg)	Weekly Ingestion (meals/yr)	Daily Ingestion (meals/yr)
	227	1	40	70	52	365
(Child)	115	1	10		52	365

Table C-66a. NONCARCINOGENIC RISKS ASSOCIATED WITH ACUTE EXPOSURE TO BIOTA.

Species/Contaminant	Maximum Concentration	BODY DOSE Child	Teen	Adult	Acute Criteria	RISK RATIOS Child	Teen	Adult
Lobster								
PCBs	1.23	0.0492	0.0123	0.007028	0.01	4.92	1.23	0.70
Cd	0.7	0.028	0.007	0.004	0.004	7	1.75	1
Cu	N/A	0	0	0				
Pb	16	0.64	0.16	0.091428				
Flounder								
PCBs	2.63	0.1052	0.0263	0.015028	0.01	10.52	2.63	1.50
Cd	0.1	0.004	0.001	0.000571	0.004	1	0.25	0.14
Cu	51.64	2.0656	0.5164	0.295085				
Pb	6.89	0.2756	0.0689	0.039371				
Clam								
PCBs	2.12	0.0848	0.0212	0.012114	0.01	8.48	2.12	1.21
Cd	0.5	0.02	0.005	0.002857	0.004	5	1.25	0.71
Cu	N/A	0	0	0				
Pb	6.34	0.2536	0.0634	0.036228				

TABLE C-67. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA 1; CHILD)

New Bedford Harbor
Direct Contact with Sediments: Area I (Child)
Noncarcinogenic Effects

NC Child Acute
Direct Contact
Acute/Chronic

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Dermal TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	378.00		3.4		7.00E-02	10	1	2.48E-05		NA	
Cadmium	19.20		3.4		1.00E-03	10	1	1.80E-08		NA	
Copper	591.00		3.4		1.00E-03	10	1	5.54E-07		NA	
Lead	384.00		3.4		1.00E-03	10	1	3.60E-07			
										TOTAL	
REALISTIC WORST CASE											
PCBs	6393.00		3.8		7.00E-02	10	20	9.29E-03		1.00E-04	9.29E+01
Cadmium	69.00		3.8		1.00E-03	10	20	1.43E-06		5.00E-04	2.87E-03
Copper	3180.00		3.8		1.00E-03	10	20	6.60E-05		3.70E-02	1.78E-03
Lead	1680.00		3.8		1.00E-03	10	20	3.49E-05		1.40E-04	2.49E-01
										TOTAL	9.32E+01

TABLE C-68. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; OLDER CHILD)

28-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	378.00	6.6	7.00E-02	40	20	2.39E-04	1.00E-04	2.39E+00
Cadmium	19.20	6.6	1.00E-03	40	20	1.74E-07	5.00E-04	3.47E-04
Copper	591.00	6.6	1.00E-03	40	20	5.34E-06	3.70E-02	1.44E-04
Lead	384.00	6.6	1.00E-03	40	20	3.47E-06	1.40E-04	2.48E-02
							TOTAL	2.42E+00
REALISTIC WORST CASE								
PCBs	6393.00	6.6	7.00E-02	40	100	2.02E-02	1.00E-04	2.02E+02
Cadmium	69.00	6.6	1.00E-03	40	100	3.12E-06	5.00E-04	6.24E-03
Copper	3180.00	6.6	1.00E-03	40	100	1.44E-04	3.70E-02	3.89E-03
Lead	1660.00	6.6	1.00E-03	40	100	7.59E-05	1.40E-04	5.42E-01
							TOTAL	2.03E+02

TABLE C-69. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; ADULT)

28-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE									
PCBs	378.00		4.6	7.00E-02	70	20	9.51E-05	1.00E-04	9.51E-01
Cadmium	19.20		4.6	1.00E-03	70	20	6.96E-08	5.00E-04	1.38E-04
Copper	591.00		4.6	1.00E-03	70	20	2.12E-06	3.70E-02	5.74E-05
Lead	384.00		4.6	1.00E-03	70	20	1.38E-06	1.40E-04	9.85E-03
								TOTAL	9.61E-01
REALISTIC WORST CASE									
PCBs	6393.00		7.5	7.00E-02	70	100	1.31E-02	1.00E-04	1.31E+02
Cadmium	69.00		7.5	1.00E-03	70	100	2.02E-06	5.00E-04	4.05E-03
Copper	3180.00		7.5	1.00E-03	70	100	9.32E-05	3.70E-02	2.52E-03
Lead	1680.00		7.5	1.00E-03	70	100	4.92E-05	1.40E-04	3.52E-01
								TOTAL	1.32E+02

TABLE C-70. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; COVE AREA; CHILD)

New Bedford Harbor
Direct Contact with Sediments: Area I; Cove Area (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Dermal TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	286.00	3.4	7.00E-02	10	1	1.88E-05	NA	
Cadmium	19.80	3.4	1.00E-03	10	1	1.86E-08	NA	
Copper	915.00	3.4	1.00E-03	10	1	8.57E-07	NA	
Lead	393.00	3.4	1.00E-03	10	1	3.68E-07		
							TOTAL	0.00E+00
REALISTIC WORST CASE								
PCBs	399.00	3.8	7.00E-02	10	20	5.80E-04	1.00E-04	5.80E+00
Cadmium	48.00	3.8	1.00E-03	10	20	9.97E-07	5.00E-04	1.99E-03
Copper	3180.00	3.8	1.00E-03	10	20	6.60E-05	3.70E-02	1.78E-03
Lead	1330.00	3.8	1.00E-03	10	20	2.76E-05	1.40E-04	1.97E-01
							TOTAL	6.00E+00

TABLE C-71. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; COVE AREA; OLDER CHILD)

28-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I; Cove Area (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal Tk Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion Tk Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)	Ingestion Tk Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE									
PCBs	286.00		6.6	7.00E-02	40	20	1.81E-04	1.00E-04	1.81E+00
Cadmium	19.80		6.6	1.00E-03	40	20	1.79E-07	5.00E-04	3.58E-04
Copper	915.00		6.6	1.00E-03	40	20	8.27E-06	3.70E-02	2.24E-04
Lead	393.00		6.6	1.00E-03	40	20	3.55E-06	1.40E-04	2.54E-02
								TOTAL	1.84E+00
REALISTIC WORST CASE									
PCBs	399.00		6.6	7.00E-02	40	100	1.26E-03	1.00E-04	1.26E+01
Cadmium	48.00		6.6	1.00E-03	40	100	2.17E-06	5.00E-04	4.34E-03
Copper	3180.00		6.6	1.00E-03	40	100	1.44E-04	3.70E-02	3.89E-03
Lead	1330.00		6.6	1.00E-03	40	100	6.01E-05	1.40E-04	4.29E-01
								TOTAL	1.31E+01

TABLE C-72. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; COVE AREA; ADULT)

New Bedford Harbor
Direct Contact with Sediments: Area I; Cove Area(Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\underset{(mg/kg/day)}{\text{Body Dose}} = \underset{(ug/g)}{\text{Sediment Concentration}} \times \left[\left(\underset{(g/event)}{\text{Amount Contacted}} \times \text{Dermal Tk Factor}\right) + \left(\underset{(g/event)}{\text{Amount Ingested}} \times \text{Ingestion Tk Factor}\right)\right] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Ingestion TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	286.00		4.6		7.00E-02	70	20	7.19E-05		1.00E-04	7.19E-01
Cadmium	19.80		4.6		1.00E-03	70	20	7.11E-08		5.00E-04	1.42E-04
Copper	915.00		4.6		1.00E-03	70	20	3.29E-06		3.70E-02	8.89E-05
Lead	393.00		4.6		1.00E-03	70	20	1.41E-06		1.40E-04	1.01E-02
										TOTAL	7.30E-01
REALISTIC WORST CASE											
PCBs	399.00		7.5		7.00E-02	70	100	8.19E-04		1.00E-04	8.19E+00
Cadmium	48.00		7.5		1.00E-03	70	100	1.41E-06		5.00E-04	2.81E-03
Copper	3160.00		7.5		1.00E-03	70	100	9.32E-05		3.70E-02	2.52E-03
Lead	1330.00		7.5		1.00E-03	70	100	3.90E-05		1.40E-04	2.78E-01
										TOTAL	8.47E+00

TABLE C-72a. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; UPPER ESTUARY; CHILD)

15-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I; Upper Estuary (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Dermal TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	378.00	3.4	7.00E-02	10	1	2.48E-05	NA	
Cadmium	18.80	3.4	1.00E-03	10	1	1.76E-08	NA	
Copper	588.00	3.4	1.00E-03	10	1	5.51E-07	NA	
Lead	445.00	3.4	1.00E-03	10	1	4.17E-07		
							TOTAL	0.00E+00
REALISTIC WORST CASE								
PCBs	6393.00	3.8	7.00E-02	10	20	9.29E-03	1.00E-04	9.29E+01
Cadmium	69.00	3.8	1.00E-03	10	20	1.43E-06	5.00E-04	2.87E-03
Copper	1900.00	3.8	1.00E-03	10	20	3.95E-05	3.70E-02	1.07E-03
Lead	1680.00	3.8	1.00E-03	10	20	3.49E-05	5.00E-04	6.98E-02
							TOTAL	9.30E+01

TABLE C-72B. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; UPPER ESTUARY; ADULT)

c-72B
28-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I;Upper Estuary (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\underset{(mg/kg/day)}{\text{Body Dose}} = \underset{(ug/g)}{\text{Sediment Concentration}} \times \left[\left(\underset{(g/event)}{\text{Amount Contacted}} \times \text{Dermal Tk Factor}\right) + \left(\underset{(g/event)}{\text{Amount Ingested}} \times \text{Ingestion Tk Factor}\right)\right] \times \frac{1}{\underset{(kg)}{\text{Body Weight}}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)	Ingestion Tk Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE									
PCBs	378.00		4.6	7.00E-02	70	20	9.51E-05	1.00E-04	9.51E-01
Cadmium	18.80		4.6	1.00E-03	70	20	6.75E-08	5.00E-04	1.35E-04
Copper	588.00		4.6	1.00E-03	70	20	2.11E-06	3.70E-02	5.71E-05
Lead	445.00		4.6	1.00E-03	70	20	1.60E-06	1.40E-04	1.14E-02
								TOTAL	9.62E-01
REALISTIC WORST CASE									
PCBs	6393.00		7.5	7.00E-02	70	100	1.31E-02	1.00E-04	1.31E+02
Cadmium	69.00		7.5	1.00E-03	70	100	2.02E-06	5.00E-04	4.05E-03
Copper	1900.00		7.5	1.00E-03	70	100	5.[illegible]E-05	3.70E-02	1.51E-03
Lead	1630.00		7.5	1.00E-03	70	100	4.[illegible]E-05	1.40E-04	3.52E-01
								TOTAL	1.32E+02

TABLE C-72C. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; UPPER ESTUARY; OLDER CHILD)

C-72C
28-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I; Upper Estuary (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\underset{(mg/kg/day)}{\text{Body Dose}} = \underset{(ug/g)}{\text{Sediment Concentration}} \times \left[\left(\underset{(g/event)}{\text{Amount Contacted}} \times \text{Dermal Tk Factor}\right) + \left(\underset{(g/event)}{\text{Amount Ingested}} \times \text{Ingestion Tk Factor}\right)\right] \times \frac{1}{\underset{(kg)}{\text{Body Weight}}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Ingestion Tk Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	378.00		6.6		7.00E-02	40	20	2.39E-04		1.00E-04	2.39E+00
Cadmium	18.60		6.6		1.00E-03	40	20	1.70E-07		5.00E-04	3.40E-04
Copper	588.00		6.6		1.00E-03	40	20	5.32E-06		3.70E-02	1.44E-04
Lead	445.00		6.6		1.00E-03	40	20	4.02E-06		1.40E-04	2.87E-02
REALISTIC WORST CASE										TOTAL	2.42E+00
PCBs	6393.00		6.6		7.00E-02	40	100	2.02E-02		1.00E-04	2.02E+02
Cadmium	69.00		6.6		1.00E-03	40	100	3.12E-06		5.00E-04	6.24E-03
Copper	1900.00		6.6		1.00E-03	40	100	8.59E-05		3.70E-02	2.32E-03
Lead	1680.00		6.6		1.00E-03	40	100	7.59E-05		1.40E-04	5.42E-01
										TOTAL	2.03E+02

TABLE C-72d. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; LOWER ESTUARY; CHILD)

c-125

15-Jun-86

New Bedford Harbor
Direct Contact with Sediments: Area I; Lower Estuary (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Dermal TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	149.00	3.4	7.00E-02	10	1	9.77E-06	NA	
Cadmium	20.00	3.4	1.00E-03	10	1	1.87E-08	NA	
Copper	598.00	3.4	1.00E-03	10	1	5.60E-07	NA	
Lead	278.00	3.4	1.00E-03	10	1	2.60E-07		
							TOTAL	0.00E+00
REALISTIC WORST CASE								
PCBs	399.00	3.8	7.00E-02	10	20	5.80E-04	1.00E-04	5.80E+00
Cadmium	63.00	3.8	1.00E-03	10	20	1.31E-06	5.00E-04	2.62E-03
Copper	3180.00	3.8	1.00E-03	10	20	6.60E-05	3.70E-02	1.78E-03
Lead	1330.00	3.8	1.00E-03	10	20	2.76E-05	1.40E-04	1.97E-01
							TOTAL	6.00E+00

TABLE C-72E. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA 1; LOWER ESTUARY; OLDER CHILD)

C-72E
28-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area 1; Lower Estuary (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\underset{(mg/kg/day)}{\text{Body Dose}} = \underset{(ug/g)}{\text{Sediment Concentration}} \times \underset{(g/event)}{\{(\text{Amount Contacted} \times \text{Dermal Tk Factor})} + \underset{(g/event)}{(\text{Amount Ingested} \times \text{Ingestion Tk Factor})\}} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Ingestion Tk Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	149.00	6.6	7.00E-02	40	20	9.43E-05	1.00E-04	9.43E-01
Cadmium	20.00	6.6	1.00E-01	40	20	1.81E-07	5.00E-04	3.62E-04
Copper	598.00	6.6	1.00E-03	40	20	5.41E-06	3.70E-02	1.46E-04
Lead	278.00	6.6	1.00E-03	40	20	2.51E-06	1.40E-04	1.80E-02
							TOTAL	9.61E-01
REALISTIC WORST CASE								
PCBs	399.00	6.6	7.00E-02	40	100	1.26E-03	1.00E-04	1.26E+01
Cadmium	63.00	6.6	1.00E-01	40	100	2.85E-06	5.00E-04	5.70E-03
Copper	3160.00	6.6	1.00E-03	40	100	1.44E-04	3.70E-02	3.89E-03
Lead	1330.00	6.6	1.00E-03	40	100	6.01E-05	1.40E-04	4.29E-01
							TOTAL	1.31E+01

TABLE C-72F. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; LOWER ESTUARY; ADULT)

C-72F
28-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I;Lower Estuary (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times \left[\left(\text{Amount Contacted (g/event)} \times \text{Dermal Tk Factor}\right) + \left(\text{Amount Ingested (g/event)} \times \text{Ingestion Tk Factor}\right)\right] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Ingestion Tk Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE										
PCBs	149.00		4.6		7.00E-02	70	20	3.75E-05	1.00E-04	3.75E-01
Cadmium	20.00		4.6		1.00E-03	70	20	7.19E-08	5.00E-04	1.44E-04
Copper	598.00		4.6		1.00E-03	70	20	2.15E-06	3.70E-02	5.81E-05
Lead	276.00		4.6		1.00E-03	70	20	9.97E-07	1.40E-04	7.13E-03
									TOTAL	3.82E-01
REALISTIC WORST CASE										
PCBs	399.00		7.5		7.00E-02	70	100	8.19E-04	1.00E-04	8.19E+00
Cadmium	63.00		7.5		1.00E-03	70	100	1.35E-06	5.00E-04	3.67E-03
Copper	3180.00		7.5		1.00E-03	70	100	6.82E-05	3.70E-02	2.52E-03
Lead	1330.00		7.5		1.00E-03	70	100	3.59E-05	1.40E-04	2.78E-01
									TOTAL	8.47E+00

TABLE C-73. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II; CHILD)

tabc-73
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Contact TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	21.00	3.4	7.00E-02	10	20	2.75E-05	1.00E-04	2.75E-01
Cadmium	7.60	3.4	1.00E-03	10	20	1.42E-07	5.00E-04	2.85E-04
Copper	570.00	3.4	1.00E-03	10	20	1.07E-05	3.70E-02	2.89E-04
Lead	160.00	3.4	1.00E-03	10	20	3.00E-06	1.40E-04	2.14E-02
							TOTAL	2.97E-01
REALISTIC WORST CASE								
PCBs	125.00	3.8	7.00E-02	10	100	9.09E-04	1.00E-04	9.09E+00
Cadmium	14.00	3.8	1.00E-03	10	100	1.45E-06	5.00E-04	2.91E-03
Copper	2790.00	3.8	1.00E-03	10	100	2.90E-04	3.70E-02	7.83E-03
Lead	559.00	3.8	1.00E-03	10	100	5.80E-05	1.40E-04	4.15E-01
							TOTAL	9.51E+00

Note: TK - Toxicokinetic Factor

TABLE C-74. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II; OLDER CHILD)

tabc-74
15-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area II (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \frac{\text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times}{}$$

$$\frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct Contact TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	21.00	6.6	7.00E-02	40	20	1.33E-05	1.00E-04	1.33E-01
Cadmium	7.60	6.6	1.00E-03	40	20	6.87E-08	5.00E-04	1.37E-04
Copper	570.00	6.6	1.00E-03	40	20	5.15E-06	3.70E-02	1.39E-04
Lead	160.00	6.6	1.00E-03	40	20	1.45E-06	1.40E-04	1.03E-02
							TOTAL	1.44E-01
REALISTIC WORST CASE								
PCBs	125.00	6.6	7.00E-02	40	100	3.96E-04	1.00E-04	3.96E+00
Cadmium	14.00	6.6	1.00E-03	40	100	6.33E-07	5.00E-04	1.27E-03
Copper	2790.00	6.6	1.00E-03	40	100	1.26E-04	3.70E-02	3.41E-03
Lead	559.00	6.6	1.00E-03	40	100	2.53E-05	1.40E-04	1.80E-01
							TOTAL	4.14E+00

Note: TK - Toxicokinetic Factor

TABLE C-75. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II; ADULT)

TABC-75
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	21.00	4.6	7.00E-02	70	20	5.28E-06	1.00E-04	5.28E-02
Cadmium	7.00	4.6	1.00E-03	70	20	2.52E-08	5.00E-04	5.03E-05
Copper	570.00	4.6	1.00E-03	70	20	2.05E-06	3.70E-02	5.54E-05
Lead	160.00	4.6	1.00E-03	70	20	5.75E-07	1.40E-04	4.11E-03
							TOTAL	5.70E-02
REALISTIC WORST CASE								
PCBs	125.00	7.5	7.00E-02	70	100	2.57E-04	1.00E-04	2.57E+00
Cadmium	14.00	7.5	1.00E-03	70	100	4.10E-07	5.00E-04	8.21E-04
Copper	2770.00	7.5	1.00E-03	70	100	8.18E-05	3.70E-02	2.21E-03
Lead	559.00	7.5	1.00E-03	70	100	1.64E-05	1.40E-04	1.17E-01
							TOTAL	2.69E+00

Note: TK - Toxicokinetic Factor

TABLE C-76. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II; PALMER ISLAND; CHILD)

tabc-76
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Palmer Island (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Contact TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	3.00		3.4		7.00E-02	10	20	3.94E-06		1.00E-04	3.94E-02
Cadmium	0.00		3.4		1.00E-03	10	20	0.00E+00		5.00E-04	0.00E+00
Copper	310.00		3.4		1.00E-03	10	20	5.81E-06		3.70E-02	1.57E-04
Lead	139.00		3.4		1.00E-03	10	20	2.60E-06		1.40E-04	1.86E-02
										TOTAL	5.81E-02
REALISTIC WORST CASE											
PCBs	11.00		3.8		7.00E-02	10	100	6.00E-05		1.00E-04	8.00E-01
Cadmium	0.00		3.8		1.00E-03	10	100	0.00E+00		5.00E-04	0.00E+00
Copper	310.00		3.8		1.00E-03	10	100	3.22E-05		3.70E-02	8.70E-04
Lead	139.00		3.8		1.00E-03	10	100	1.44E-05		1.40E-04	1.03E-01
										TOTAL	9.03E-01

Note: TK - Toxicokinetic Factor

TABLE C-77. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II PALMER ISLAND; OLDER CHILD)

tabc-77
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Palmer Island (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\underset{(mg/kg/day)}{\text{Body Dose}} = \underset{(ug/g)}{\text{Sediment Concentration}} \times \left[\left(\underset{(g/event)}{\text{Amount Contacted}} \times \text{Dermal TK Factor}\right) + \left(\underset{(g/event)}{\text{Amount Ingested}} \times \text{Ingestion TK Factor}\right)\right] \times \frac{1}{\underset{(kg)}{\text{Body Weight}}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)	Direct contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE									
PCBs	3.00		6.6	7.00E-02	40	20	1.99E-06	1.00E-04	1.90E-02
Cadmium	0.00		6.6	1.00E-03	40	20	0.00E+00	5.00E-04	0.00E+00
Copper	310.00		6.6	1.00E-03	40	20	2.80E-06	3.70E-02	7.57E-05
Lead	139.00		6.6	1.00E-03	40	20	1.26E-06	1.40E-04	8.98E-03
								TOTAL	2.80E-02
REALISTIC WORST CASE									
PCBs	11.00		6.6	7.00E-02	40	100	3.48E-05	1.00E-04	3.48E-01
Cadmium	0.00		6.6	1.00E-03	40	100	0.00E+00	5.00E-04	0.00E+00
Copper	310.00		6.6	1.00E-03	40	100	1.40E-05	3.70E-02	3.79E-04
Lead	139.00		6.6	1.00E-03	40	100	6.28E-06	1.40E-04	4.49E-02
								TOTAL	3.93E-01

Note: TK - Toxicokinetic Factor

TABLE C-78. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II FALMER ISLAND; ADULT)

TABC-78
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Falmer Island (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}},$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	3.00		4.6		7.00E-02	70	20	7.55E-07		1.00E-04	7.55E-03
Cadmium	0.00		4.6		1.00E-03	70	20	0.00E+00		5.00E-04	0.00E+00
Copper	310.00		4.6		1.00E-03	70	20	1.11E-06		3.70E-02	3.01E-05
Lead	139.00		4.6		1.00E-03	70	20	4.99E-07		1.40E-04	3.57E-03
										TOTAL	1.11E-02
REALISTIC WORST CASE											
PCBs	11.00		7.5		7.00E-02	70	100	2.26E-05		1.00E-04	2.26E-01
Cadmium	0.00		7.5		1.00E-03	70	100	0.00E+00		5.00E-04	0.00E+00
Copper	310.00		7.5		1.00E-03	70	100	9.07E-06		3.70E-02	2.46E-04
Lead	139.00		7.5		1.00E-03	70	100	4.07E-06		1.40E-04	2.91E-02
										TOTAL	2.55E-01

Note: TK = Toxicokinetic Factor

TABLE C-79. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II; FOPES ISLAND; CHILD)

tabc-79
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Fopes Island (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Contact TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	11.00	3.4	7.00E-02	10	20	1.44E-05	1.00E-04	1.44E-01
Cadmium	0.00	3.4	1.00E-03	10	20	0.00E+00	5.00E-04	0.00E+00
Copper	492.00	3.4	1.00E-03	10	20	9.22E-06	3.70E-02	2.49E-04
Lead	156.00	3.4	1.00E-03	10	20	2.92E-06	1.40E-04	2.09E-02
							TOTAL	1.65E-01
REALISTIC WORST CASE								
PCBs	34.00	3.8	7.00E-02	10	100	2.47E-04	1.00E-04	2.47E+00
Cadmium	0.00	3.8	1.00E-03	10	100	0.00E+00	5.00E-04	0.00E+00
Copper	771.00	3.8	1.00E-03	10	100	8.01E-05	3.70E-02	2.16E-03
Lead	272.00	3.8	1.00E-03	10	100	2.82E-05	1.40E-04	2.02E-01
							TOTAL	2.68E+00

Note: TK - Toxicokinetic Factor

TABLE C-80. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II POPES ISLAND; OLDER CHILD)

tabc-80
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Popes Island (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	11.00		6.6		7.00E-02	40	20	6.96E-06		1.00E-04	6.96E-02
Cadmium	0.00		6.6		1.00E-03	40	20	0.00E+00		5.00E-04	0.00E+00
Copper	492.00		6.6		1.00E-03	40	20	4.45E-06		3.70E-02	1.20E-04
Lead	156.00		6.6		1.00E-03	40	20	1.41E-06		1.40E-04	1.01E-02
										TOTAL	7.98E-02
REALISTIC WORST CASE											
PCBs	34.00		6.6		7.00E-02	40	100	1.08E-04		1.00E-04	1.08E+00
Cadmium	0.00		6.6		1.00E-03	40	100	0.00E+00		5.00E-04	0.00E+00
Copper	771.00		6.6		1.00E-03	40	100	3.49E-05		3.70E-02	9.42E-04
Lead	272.00		6.6		1.00E-03	40	100	1.23E-05		1.40E-04	8.78E-02
										TOTAL	1.16E+00

Notes: TK = Toxicokinetic Factor

TABLE C-81. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II POPES ISLAND; ADULT)

TABC-81
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Popes Island (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	11.00	4.6	7.00E-02	70	20	2.77E-06	1.00E-04	2.77E-02
Cadmium	0.00	4.6	1.00E-03	70	20	0.00E+00	5.00E-04	0.00E+00
Copper	492.00	4.6	1.00E-03	70	20	1.77E-06	3.70E-02	4.78E-05
Lead	156.00	4.6	1.00E-03	70	20	5.61E-07	1.40E-04	4.00E-03
							TOTAL	3.17E-02
REALISTIC WORST CASE								
PCBs	34.00	7.5	7.00E-02	70	100	6.98E-05	1.00E-04	6.98E-01
Cadmium	0.00	7.5	1.00E-03	70	100	0.00E+00	5.00E-04	0.00E+00
Copper	771.00	7.5	1.00E-03	70	100	2.25E-05	3.70E-02	6.11E-04
Lead	272.00	7.5	1.00E-03	70	100	7.97E-06	1.40E-04	5.70E-02
							TOTAL	7.55E-01

Note: TK - Toxicokinetic Factor

TABLE C-B2. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II; MARSH ISLAND; CHILD)

tabc-B2
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Marsh Island (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)	Contact TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE									
PCBs	8.00		3.4	7.00E-02	10	20	1.05E-05	1.00E-04	1.05E-01
Cadmium	0.00		3.4	1.00E-03	10	20	0.00E+00	5.00E-04	0.00E+00
Copper	300.00		3.4	1.00E-03	10	20	5.62E-06	3.70E-02	1.52E-04
Lead	191.00		3.4	1.00E-03	10	20	3.58E-06	1.40E-04	2.56E-02
								TOTAL	1.31E-01
REALISTIC WORST CASE									
PCBs	22.00		3.8	7.00E-02	10	100	1.60E-04	1.00E-04	1.60E+00
Cadmium	0.00		3.8	1.00E-03	10	100	0.00E+00	5.00E-04	0.00E+00
Copper	300.00		3.8	1.00E-03	10	100	3.12E-05	3.70E-02	8.42E-04
Lead	323.00		3.8	1.00E-03	10	100	3.35E-05	1.40E-04	2.40E-01
								TOTAL	1.84E+00

Note: TK - Toxicokinetic Factor

TABLE C-83. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II MARSH ISLAND; OLDER CHILD)

tabc-B3
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Marsh Island (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	8.00	6.6	7.00E-02	40	20	5.06E-06	1.00E-04	5.06E-02
Cadmium	0.00	6.6	1.00E-03	40	20	0.00E+00	5.00E-04	0.00E+00
Copper	300.00	6.6	1.00E-03	40	20	2.71E-06	3.70E-02	7.33E-05
Lead	191.00	6.6	1.00E-03	40	20	1.73E-06	1.40E-04	1.23E-02
							TOTAL	6.30E-02
REALISTIC WORST CASE								
PCBs	22.00	6.6	7.00E-02	40	100	6.96E-05	1.00E-04	6.96E-01
Cadmium	0.00	6.6	1.00E-03	40	100	0.00E+00	5.00E-04	0.00E+00
Copper	300.00	6.6	1.00E-03	40	100	1.35E-05	3.70E-02	3.67E-04
Lead	323.00	6.6	1.00E-03	40	100	1.46E-05	1.40E-04	1.04E-01
							TOTAL	8.01E-01

Note: TK - Toxicokinetic Factor

TABLE C-84. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA II MARSH ISLAND; ADULT)

TABC-84
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area II-Marsh Island (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	8.00	4.6	7.00E-02	70	20	2.01E-06	1.00E-04	2.01E-02
Cadmium	0.00	4.6	1.00E-03	70	20	0.00E+00	5.00E-04	0.00E+00
Copper	300.00	4.6	1.00E-03	70	20	1.08E-06	3.70E-02	2.91E-05
Lead	191.00	4.6	1.00E-03	70	20	6.86E-07	1.40E-04	4.90E-03
							TOTAL	2.51E-02
REALISTIC WORST CASE								
PCBs	22.00	7.5	7.00E-02	70	100	4.51E-05	1.00E-04	4.51E-01
Cadmium	0.00	7.5	1.00E-03	70	100	0.00E+00	5.00E-04	0.00E+00
Copper	300.00	7.5	1.00E-03	70	100	8.79E-06	3.70E-02	2.38E-04
Lead	323.00	7.5	1.00E-03	70	100	9.47E-06	1.40E-04	6.76E-02
							TOTAL	5.19E-01

Note: TK - Toxicokinetic Factor

TABLE C-85. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; CHILD)

tabc-85
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area III (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times \{(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})\} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)	Contact TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE									
PCBs	4.00		3.4	7.00E-02	10	20	5.25E-06	1.00E-04	5.25E-02
Cadmium	0.00		3.4	1.00E-03	10	20	0.00E+00	5.00E-04	0.00E+00
Copper	94.00		3.4	1.00E-03	10	20	1.76E-06	3.70E-02	4.76E-05
Lead	55.00		3.4	1.00E-03	10	20	1.03E-06	1.40E-04	7.36E-03
								TOTAL	5.99E-02
REALISTIC WORST CASE									
PCBs	29.00		3.8	7.00E-02	10	100	2.11E-04	1.00E-04	2.11E+00
Cadmium	0.00		3.8	1.00E-03	10	100	0.00E+00	5.00E-04	0.00E+00
Copper	154.00		3.8	1.00E-03	10	100	1.86E-05	3.70E-02	4.32E-04
Lead	106.00		3.8	1.00E-03	10	100	1.14E-05	1.40E-04	7.86E-02
								TOTAL	2.19E+00

Note: TK - Toxicokinetic Factor

TABLE C-86. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; OLDER CHILD)

tabc-86 31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area III (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	4.00	6.6	7.00E-02	40	20	2.53E-06	1.00E-04	2.53E-02
Cadmium	0.00	6.6	1.00E-03	40	20	0.00E+00	5.00E-04	0.00E+00
Copper	94.00	6.6	1.00E-03	40	20	8.50E-07	3.70E-02	2.30E-05
Lead	55.00	6.6	1.00E-03	40	20	4.97E-07	1.40E-04	3.55E-03
							TOTAL	2.89E-02
REALISTIC WORST CASE								
PCBs	29.00	6.6	7.00E-02	40	100	9.18E-05	1.00E-04	9.18E-01
Cadmium	0.00	6.6	1.00E-03	40	100	0.00E+00	5.00E-04	0.00E+00
Copper	154.00	6.6	1.00E-03	40	100	6.76E-06	3.70E-02	1.88E-04
Lead	106.00	6.6	1.00E-03	40	100	4.75E-06	1.40E-04	3.42E-02
							TOTAL	9.52E-01

Note: TK - Toxicokinetic Factor

TABLE C-87. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; ADULT)

TABC-87
31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area III (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	4.00		4.6		7.00E-02	70	20	1.01E-06		1.00E-04	1.01E-02
Cadmium	0.00		4.6		1.00E-03	70	20	0.00E+00		5.00E-04	0.00E+00
Copper	94.00		4.6		1.00E-03	70	20	3.38E-07		3.70E-02	9.13E-06
Lead	55.00		4.6		1.00E-03	70	20	1.98E-07		1.40E-04	1.41E-03
										TOTAL	1.15E-02
REALISTIC WORST CASE											
PCBs	29.00		7.5		7.00E-02	70	100	5.95E-05		1.00E-04	5.95E-01
Cadmium	0.00		7.5		1.00E-03	70	100	0.00E+00		5.00E-04	0.00E+00
Copper	154.00		7.5		1.00E-03	70	100	4.51E-06		3.70E-02	1.22E-04
Lead	106.00		7.5		1.00E-03	70	100	3.11E-06		1.40E-04	2.22E-02
										TOTAL	6.17E-01

Note: TK - Toxicokinetic Factor

TABLE C-87a BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; FORT RODMAN;CHILD)

31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area III; Fort Rodman (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times \left[\left(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}\right) + \left(\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor}\right)\right] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Contact TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	2.00	3.4	7.00E-02	10	20	2.62E-06	1.00E-04	2.62E-02
Cadmium	0.00	3.4	1.00E-03	10	20	0.00E+00	5.00E-04	0.00E+00
Copper	0.00	3.4	1.00E-03	10	20	0.00E+00	3.70E-02	0.00E+00
Lead	0.00	3.4	1.00E-03	10	20	0.00E+00	1.40E-04	0.00E+00
							TOTAL	2.62E-02
REALISTIC WORST CASE								
PCBs	7.00	3.8	7.00E-02	10	100	5.09E-05	1.00E-04	5.09E-01
Cadmium	0.00	3.8	1.00E-03	10	100	0.00E+00	5.00E-04	0.00E+00
Copper	0.00	3.8	1.00E-03	10	100	0.00E+00	3.70E-02	0.00E+00
Lead	0.00	3.8	1.00E-03	10	100	0.00E+00	1.40E-04	0.00E+00
							TOTAL	5.09E-01

Note: TK - Toxicokinetic Factor

TABLE C-87b. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; OLDER CHILD)

New Bedford Harbor
Direct Contact with Sediments: Area III; Fort Rodman (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	2.00	6.6	7.00E-02	40	20	1.27E-06	1.00E-04	1.27E-02
Cadmium	0.00	6.6	1.00E-03	40	20	0.00E+00	5.00E-04	0.00E+00
Copper	0.00	6.6	1.00E-03	40	20	0.00E+00	3.70E-02	0.00E+00
Lead	0.00	6.6	1.00E-03	40	20	0.00E+00	1.40E-04	0.00E+00
							TOTAL	1.27E-02
REALISTIC WORST CASE								
PCBs	7.00	6.6	7.00E-02	40	100	2.22E-05	1.00E-04	2.22E-01
Cadmium	0.00	6.6	1.00E-03	40	100	0.00E+00	5.00E-04	0.00E+00
Copper	0.00	6.6	1.00E-03	40	100	0.00E+00	3.70E-02	0.00E+00
Lead	0.00	6.6	1.00E-03	40	100	0.00E+00	1.40E-04	0.00E+00
							TOTAL	2.22E-01

Note: TK - Toxicokinetic Factor

TABLE C-87c. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; FORT RODMAN; ADULT)

31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area III; Fort Rodman (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	2.00	4.6	7.00E-02	70	20	5.03E-07	1.00E-04	5.03E-03
Cadmium	0.00	4.6	1.00E-03	70	20	0.00E+00	5.00E-04	0.00E+00
Copper	0.00	4.6	1.00E-03	70	20	0.00E+00	3.70E-02	0.00E+00
Lead	0.00	4.6	1.00E-03	70	20	0.00E+00	1.40E-04	0.00E+00
							TOTAL	5.03E-03
REALISTIC WORST CASE								
PCBs	7.00	7.5	7.00E-02	70	100	1.44E-05	1.00E-04	1.44E-01
Cadmium	0.00	7.5	1.00E-03	70	100	0.00E+00	5.00E-04	0.00E+00
Copper	0.00	7.5	1.00E-03	70	100	0.00E+00	3.70E-02	0.00E+00
Lead	0.00	7.5	1.00E-03	70	100	0.00E+00	1.40E-04	0.00E+00
							TOTAL	1.44E-01

Note: TK - Toxicokinetic Factor

TABLE C-87d BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; FORT PHOENIX;CHILD)

31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area III; Fort Phoenix (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Contact TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	0.59		3.4		7.00E-02	10	20	7.74E-07		1.00E-04	7.74E-03
Cadmium	0.00		3.4		1.00E-03	10	20	0.00E+00		5.00E-04	0.00E+00
Copper	0.00		3.4		1.00E-03	10	20	0.00E+00		3.70E-02	0.00E+00
Lead	0.00		3.4		1.00E-03	10	20	0.00E+00		1.40E-04	0.00E+00
										TOTAL	7.74E-03
REALISTIC WORST CASE											
PCBs	0.75		3.8		7.00E-02	10	100	5.45E-06		1.00E-04	5.45E-02
Cadmium	0.00		3.8		1.00E-03	10	100	0.00E+00		5.00E-04	0.00E+00
Copper	0.00		3.8		1.00E-03	10	100	0.00E+00		3.70E-02	0.00E+00
Lead	0.00		3.8		1.00E-03	10	100	0.00E+00		1.40E-04	0.00E+00
										TOTAL	5.45E-02

Note: TK - Toxicokinetic Factor

TABLE C-[illegible]. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; FORT PHOENIX OLDER CHILD)

C-135

31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area III; Fort Phoenix (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Contacted (g/event)		Direct Contact ~~Ingestion~~ TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	0.59		6.6		7.00E-02	40	20	3.73E-07		1.00E-04	3.73E-03
Cadmium	0.00		6.6		1.00E-03	40	20	0.00E+00		5.00E-04	0.00E+00
Copper	0.00		6.6		1.00E-03	40	20	0.00E+00		3.70E-02	0.00E+00
Lead	0.00		6.6		1.00E-03	40	20	0.00E+00		1.40E-04	0.00E+00
										TOTAL	3.73E-03
REALISTIC WORST CASE											
PCBs	0.75		6.6		7.00E-02	40	100	2.37E-06		1.00E-04	2.37E-02
Cadmium	0.00		6.6		1.00E-03	40	100	0.00E+00		5.00E-04	0.00E+00
Copper	0.00		6.6		1.00E-03	40	100	0.00E+00		3.70E-02	0.00E+00
Lead	0.00		6.6		1.00E-03	40	100	0.00E+00		1.40E-04	0.00E+00
										TOTAL	2.37E-02

Note: TK - Toxicokinetic Factor

TABLE C-87f. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA III; FORT PHOENIX; ADULT)

c-133a

31-May-89

New Bedford Harbor
Direct Contact with Sediments: Area III; Fort Rodman (Adult)
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Direct Contact ~~Ingestion~~ Tk Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	0.59	4.6	7.00E-02	70	20	1.48E-07	1.00E-04	1.48E-03
Cadmium	0.00	4.6	1.00E-03	70	20	0.00E+00	5.00E-04	0.00E+00
Copper	0.00	4.6	1.00E-03	70	20	0.00E+00	3.70E-02	0.00E+00
Lead	0.00	4.6	1.00E-03	70	20	0.00E+00	1.40E-04	0.00E+00
							TOTAL	1.48E-03
REALISTIC WORST CASE								
PCBs	0.75	7.5	7.00E-02	70	100	1.54E-06	1.00E-04	1.54E-02
Cadmium	0.00	7.5	1.00E-03	70	100	0.00E+00	5.00E-04	0.00E+00
Copper	0.00	7.5	1.00E-03	70	100	0.00E+00	3.70E-02	0.00E+00
Lead	0.00	7.5	1.00E-03	70	100	0.00E+00	1.40E-04	0.00E+00
							TOTAL	1.54E-02

Note: TK - Toxicokinetic Factor

TABLE C-88. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; COVE AREA; CHILD)
ACUTE EXPOSURE

15-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I; Cove Area (Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Dermal TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	286.00	3.4	7.00E-02	10	1	6.85E-03	1.00E-02	6.85E-01
Cadmium	19.80	3.4	1.00E-03	10	1	6.77E-06	4.30E-03	1.57E-03
Copper	915.00	3.4	1.00E-03	10	1	3.13E-04	NA	
Lead	393.00	3.4	1.00E-03	10	1	1.34E-04	NA	
							TOTAL	6.86E-01
REALISTIC WORST CASE								
PCBs	399.00	3.8	7.00E-02	10	1	1.06E-02	1.00E-02	1.06E+00
Cadmium	48.00	3.8	1.00E-03	10	1	1.82E-05	4.30E-03	4.23E-03
Copper	3180.00	3.8	1.00E-03	10	1	1.21E-03	NA	
Lead	1330.00	3.8	1.00E-03	10	1	5.04E-04	NA	
							TOTAL	1.06E+00

Note: TK - Toxicokinetic Factor
Criteria used to evaluat risk are the 1 day or 10-day Health Advisories for Cd and Pb.
No appropriate standards or criteria are available to assess acute exposure to copper or lead.

TABLE C-89. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; COVE AREA; OLDER CHILD)
ACUTE EXPOSURE

15-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I; Cove Area (Older Child)
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times \{(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})\} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Dermal TK Factor	Body Weight (kg)	No. of Events per year	Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE							
PCBs	286.00	6.6	7.00E-02	40	1	1.00E-02	3.30E-01
Cadmium	19.80	6.6	1.00E-03	40	1	4.30E-03	7.60E-04
Copper	915.00	6.6	1.00E-03	40	1	NA	
Lead	393.00	6.6	1.00E-03	40	1	NA	
						TOTAL	3.31E-01
REALISTIC WORST CASE							
PCBs	399.00	6.6	7.00E-02	40	1	1.00E-02	4.61E-01
Cadmium	48.00	6.6	1.00E-03	40	1	4.30E-03	1.84E-03
Copper	3180.00	6.6	1.00E-03	40	1	NA	
Lead	1330.00	6.6	1.00E-03	40	1	NA	
						TOTAL	4.63E-01

Note: TK - Toxicokinetic Factor
Criteria used to evaluate risk are the 1 day or 10 day Health Advisories for Cd and PCBs.
No appropriate criteria are available to assess acute exposure to copper or lead.

TABLE C-90. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I; COVE AREA; ADULT)
ACUTE EXPOSURE

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\underset{(mg/kg/day)}{\text{Body Dose}} = \underset{(ug/g)}{\text{Sediment Concentration}} \times \underset{(g/event)}{[(\text{Amount Contacted} \times \text{Dermal TK Factor})} + \underset{(g/event)}{(\text{Amount Ingested} \times \text{Ingestion TK Factor})]} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Dermal TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	286.00	4.6	7.00E-02	70	1	1.32E-03	1.00E-02	1.32E-01
Cadmium	19.80	4.6	1.00E-03	70	1	1.30E-06	4.30E-03	3.03E-04
Copper	915.00	4.6	1.00E-03	70	1	6.01E-05	NA	
Lead	393.00	4.6	1.00E-03	70	1	2.58E-05	NA	
							TOTAL	1.32E-01
REALISTIC WORST CASE								
PCBs	399.00	7.5	7.00E-02	70	1	2.99E-03	1.00E-02	2.99E-01
Cadmium	48.00	7.5	1.00E-03	70	1	5.14E-06	4.30E-03	1.20E-03
Copper	3160.00	7.5	1.00E-03	70	1	3.41E-04	NA	
Lead	1330.00	7.5	1.00E-03	70	1	1.42E-04	NA	
							TOTAL	3.00E-01

Note: TK - Toxicokinetic Factor
Criteria used to evaluated Most Probable Case are the 1-day or 10-day Health Advisories for Cd and PCBs.
No appropriate criteria are available to assess acute exposure to copper or lead.

TABLE C-90a. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I)
ACUTE EXPOSURE

c-90a
15-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Dermal TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE (Child)								
PCBs	6393.00	3.4	7.00E-02	10	1	1.52E-01	1.00E-02	1.52E+01
Cadmium	48.00	3.4	1.00E-03	10	1	1.63E-05	4.30E-03	3.80E-03
REALISTIC WORST CASE								
PCBs	6393.00	3.8	7.00E-02	10	1	1.70E-01	1.00E-02	1.70E+01
Cadmium	48.00	3.8	1.00E-03	10	1	1.82E-05	4.30E-03	4.24E-03
MOST PROBABLE CASE (Older Child)								
PCBs	6393.00	6.6	7.00E-02	40	1	7.38E-02	1.00E-02	7.38E+00
Cadmium	48.00	6.6	1.00E-03	40	1	7.92E-06	4.30E-03	1.84E-03
REALISTIC WORST CASE								
PCBs	6393.00	8.60	7.00E-02	40	1	9.68E-02	1.00E-02	7.38E+00
Cadmium	48.00	8.60	1.00E-03	40	1	9.92E-06	4.30E-03	1.84E-03

TABLE C-90a con't. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; DIRECT CONTACT WITH SEDIMENTS (AREA I)
ACUTE EXPOSURE

c-90acont
15-Jun-89

New Bedford Harbor
Direct Contact with Sediments: Area I
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Dermal TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE (Adult)								
PCBs	6393.00	4.6	7.00E 02	70	1	2.94E-02	1.00E-02	2.94E+00
Cadmium	48.00	4.6	1.00E-03	70	1	3.15E-06	4.30E-03	7.34E-04
REALISTIC WORST CASE								
PCBs	6393.00	7.5	7.00E-02	70	1	4.79E-02	1.00E-02	4.79E+00
Cadmium	48.00	7.5	1.00E 03	70	1	5.14E-06	4.30E-03	1.20E-03

TABLE C-91. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

31-May-89

New Bedford Harbor
Ingestion of Sediments: Area 1
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal IK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion IK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion IK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	378.00	0.5	1.00	10	1			
Cadmium	19.20	0.5	1.00	10	1			
Copper	591.00	0.5	1.00	10	1			
Lead	384.00	0.5	1.00	10	1			
REALISTIC WORST CASE							TOTAL	
PCBs	6393.00	0.5	1.00	10	20	1.75E-02	1.00E-04	1.75E+02
Cadmium	69.00	0.5	1.00	10	20	1.89E-04	5.00E-04	3.78E-01
Copper	3180.00	0.5	1.00	10	20	8.71E-03	3.70E-02	2.35E-01
Lead	1680.00	0.5	1.00	10	20	4.60E-03	1.40E-04	3.29E+01
							TOTAL	2.09E+02

TABLE C-91a. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

c-91a
31-May-89

New Bedford Harbor
Ingestion of Sediments: Area I; Upper Estuary; Child
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Ingested (g/event)		Ingestion TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	378.00		0.5		1.00	10	1				
Cadmium	18.80		0.5		1.00	10	1				
Copper	588.00		0.5		1.00	10	1				
Lead	445.00		0.5		1.00	10	1				
REALISTIC WORST CASE										TOTAL	
PCBs	6393.00		0.5		1.00	10	20	1.75E-02		1.00E-04	1.75E+02
Cadmium	69.00		0.5		1.00	10	20	1.89E-04		5.00E-04	3.78E-01
Copper	1900.00		0.5		1.00	10	20	5.21E-03		3.70E-02	1.41E-01
Lead	1680.00		0.5		1.00	10	20	4.60E-03		1.40E-04	3.29E+01
										TOTAL	2.09E+02

TABLE C-91b. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

c-91b
31-May-89

New Bedford Harbor
Ingestion of Sediments: Area I; Lower Estuary; Child
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	149.00	0.5	1.00	10	1			
Cadmium	20.00	0.5	1.00	10	1			
Copper	598.00	0.5	1.00	10	1			
Lead	278.00	0.5	1.00	10	1			
REALISTIC WORST CASE							TOTAL	
PCBs	359.00	0.5	1.00	10	20	1.09E-03	1.00E-04	1.09E+01
Cadmium	63.00	0.5	1.00	10	20	1.73E-04	5.00E-04	3.45E-01
Copper	3180.00	0.5	1.00	10	20	8.71E-03	3.70E-02	2.35E-01
Lead	1330.00	0.5	1.00	10	20	3.64E-03	1.40E-04	2.60E+01
							TOTAL	3.75E+01

TABLE C-91c. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

c-91c
31-May-89

New Bedford Harbor
Ingestion of Sediments: Area 1; Cove Area; Child
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	236.00	0.5	1.00	10	1			
Cadmium	19.80	0.5	1.00	10	1			
Copper	915.00	0.5	1.00	10	1			
Lead	393.00	0.5	1.00	10	1			
REALISTIC WORST CASE								
PCBs	399.00	0.5	1.00	10	20	1.09E-03	1.00E-04	1.09E+01
Cadmium	48.00	0.5	1.00	10	20	1.32E-04	5.00E-04	2.63E-01
Copper	3180.00	0.5	1.00	10	20	8.71E-03	3.70E-02	2.35E-01
Lead	1330.00	0.5	1.00	10	20	3.64E-03	1.40E-04	2.60E+01
							TOTAL	3.75E+01

TABLE C-91d BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS; AREA II; CHILD

c-121

31-May-89

New Bedford Harbor
Ingestion of Sediments: Area II; Child
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	21.00	0.5	1.00	10	20	5.75E-05	1.00E-04	5.75E-01
Cadmium	7.60	0.5	1.00	10	20	2.08E-05	5.00E-04	4.16E-02
Copper	570.00	0.5	1.00	10	20	1.56E-03	3.70E-02	4.22E-02
Lead	160.00	0.5	1.00	10	20	4.38E-04	1.40E-04	3.13E+00
							TOTAL	3.79E+00
REALISTIC WORST CASE								
PCBs	125.00	0.5	1.00	10	100	1.71E-03	1.00E-04	1.71E+01
Cadmium	14.00	0.5	1.00	10	100	1.92E-04	5.00E-04	3.84E-01
Copper	2790.00	0.5	1.00	10	100	3.82E-02	3.70E-02	1.03E+00
Lead	559.00	0.5	1.00	10	100	7.66E-03	1.40E-04	5.47E+01
							TOTAL	7.32E+01

TABLE C-119. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

c-119

31-May-89

New Bedford Harbor
Ingestion of Sediments: Area II (Popes Island); Child
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Ingested (g/event)		Ingestion TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	11.00		0.5		1.00	10	20	3.01E-05		1.00E-04	3.01E-01
Cadmium	0.00		0.5		1.00	10	20	0.00E+00		5.00E-04	0.00E+00
Copper	492.00		0.5		1.00	10	20	1.35E-03		3.70E-02	3.64E-02
Lead	156.00		0.5		1.00	10	20	4.27E-04		1.40E-04	3.05E+00
										TOTAL	3.39E+00
REALISTIC WORST CASE											
PCBs	34.00		0.5		1.00	10	100	4.66E-04		1.00E-04	4.66E+00
Cadmium	0.00		0.5		1.00	10	100	0.00E+00		5.00E-04	0.00E+00
Copper	771.00		0.5		1.00	10	100	1.06E-02		3.70E-02	2.85E-01
Lead	272.00		0.5		1.00	10	100	3.73E-03		1.40E-04	2.66E+01
										TOTAL	3.16E+01

Note: TK - Toxicokinetic Factor

TABLE C-11F. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

c-118

31-May-89

New Bedford Harbor
Ingestion of Sediments: Area II (Palmer Island); Child
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Ingested (g/event)		Ingestion TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	3.00		0.5		1.00	10	20	8.22E-06		1.00E-04	8.22E-02
Cadmium	0.00		0.5		1.00	10	20	0.00E+00		5.00E-04	0.00E+00
Copper	310.00		0.5		1.00	10	20	8.49E-04		3.70E-02	2.30E-02
Lead	139.00		0.5		1.00	10	20	3.81E-04		1.40E-04	2.72E+00
										TOTAL	2.83E+00
REALISTIC WORST CASE											
PCBs	11.00		0.5		1.00	10	100	1.51E-04		1.00E-04	1.51E+00
Cadmium	0.00		0.5		1.00	10	100	0.00E+00		5.00E-04	0.00E+00
Copper	310.00		0.5		1.00	10	100	4.25E-03		3.70E-02	1.15E-01
Lead	139.00		0.5		1.00	10	100	1.90E-03		1.40E-04	1.36E+01
										TOTAL	1.52E+01

Note: TK - Toxicokinetic Factor

TABLE C-~~120~~ 916. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

c-120

31-May-89

New Bedford Harbor
Ingestion of Sediments: Area II (Marsh Island); Child
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\underset{\text{(mg/kg/day)}}{\text{Body Dose}} = \underset{\text{(ug/g)}}{\text{Sediment Concentration}} \times \left[\left(\underset{\text{(g/event)}}{\text{Amount Contacted}} \times \text{Dermal TK Factor}\right) + \left(\underset{\text{(g/event)}}{\text{Amount Ingested}} \times \text{Ingestion TK Factor}\right)\right] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	8.00	0.5	1.00	10	20	2.19E-05	1.00E-04	2.19E-01
Cadmium	0.00	0.5	1.00	10	20	0.00E+00	5.00E-04	0.00E+00
Copper	300.00	0.5	1.00	10	20	8.22E-04	3.70E-02	2.22E-02
Lead	191.00	0.5	1.00	10	20	5.23E-04	1.40E-04	3.74E+00
							TOTAL	3.98E+00
REALISTIC WORST CASE								
PCBs	22.00	0.5	1.00	10	100	3.01E-04	1.00E-04	3.01E+00
Cadmium	0.00	0.5	1.00	10	100	0.00E+00	5.00E-04	0.00E+00
Copper	300.00	0.5	1.00	10	100	4.11E-03	3.70E-02	1.11E-01
Lead	323.00	0.5	1.00	10	100	4.42E-03	1.40E-04	3.16E+01
							TOTAL	3.47E+01

TABLE C-92. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

31-May-89

New Bedford Harbor
Ingestion of Sediments: Area III; Child
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
PCBs	4.00	0.5	1.00	10	20	1.10E-05	1.00E-04	1.10E-01
Cadmium	0.00	0.5	1.00	10	20	0.00E+00	5.00E-04	0.00E+00
Copper	94.00	0.5	1.00	10	20	2.58E-04	3.70E-02	6.96E-03
Lead	55.00	0.5	1.00	10	20	1.51E-04	1.40E-04	1.08E+00
							TOTAL	1.19E+00
REALISTIC WORST CASE								
PCBs	29.00	0.5	1.00	10	100	3.97E-04	1.00E-04	3.97E+00
Cadmium	0.00	0.5	1.00	10	100	0.00E+00	5.00E-04	0.00E+00
Copper	154.00	0.5	1.00	10	100	2.11E-03	3.70E-02	5.70E-02
Lead	106.00	0.5	1.00	10	100	1.45E-03	1.40E-04	1.04E+01
							TOTAL	1.44E+01

Note: TK - Toxicokinetic Factor

92a

TABLE C-~~122~~. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

c-122

31-May-89

New Bedford Harbor
Ingestion of Sediments: Area III (Fort Rodman): Child
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Ingested (g/event)		Ingestion TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	2.00		0.5		1.00	10	20	5.48E-06		1.00E-04	5.48E-02
Cadmium	0.00		0.5		1.00	10	20	0.00E+00		5.00E-04	0.00E+00
Copper	0.00		0.5		1.00	10	20	0.00E+00		3.70E-02	0.00E+00
Lead	0.00		0.5		1.00	10	20	0.00E+00		1.40E-04	0.00E+00
										TOTAL	5.48E-02
REALISTIC WORST CASE											
PCBs	7.00		0.5		1.00	10	100	9.59E-05		1.00E-04	9.59E-01
Cadmium	0.00		0.5		1.00	10	100	0.00E+00		5.00E-04	0.00E+00
Copper	0.00		0.5		1.00	10	100	0.00E+00		3.70E-02	0.00E+00
Lead	0.00		0.5		1.00	10	100	0.00E+00		1.40E-04	0.00E+00
										TOTAL	9.59E-01

Note: TK - Toxicokinetic Factor

TABLE C-126b. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS;

31-May-87

New Bedford Harbor
Ingestion of Sediments: Area III (Fort Phoenix); Child
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Ingested (g/event)		Ingestion TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
PCBs	0.59		0.5		1.00	10	20	1.62E-06		1.00E-04	1.62E-02
Cadmium	0.00		0.5		1.00	10	20	0.00E+00		5.00E-04	0.00E+00
Copper	0.00		0.5		1.00	10	20	0.00E+00		3.70E-02	0.00E+00
Lead	0.00		0.5		1.00	10	20	0.00E+00		1.40E-04	0.00E+00
										TOTAL	1.62E-02
REALISTIC WORST CASE											
PCBs	0.75		0.5		1.00	10	100	1.03E-05		1.00E-04	1.03E-01
Cadmium	0.00		0.5		1.00	10	100	0.00E+00		5.00E-04	0.00E+00
Copper	0.00		0.5		1.00	10	100	0.00E+00		3.70E-02	0.00E+00
Lead	0.00		0.5		1.00	10	100	0.00E+00		1.40E-04	0.00E+00
										TOTAL	1.03E-01

Note: TK - Toxicokinetic Factor

TABLE C-93. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS; ACUTE EXPOSURE; AREA I; CHILD

15-Jun-89

New Bedford Harbor
Ingestion of Sediments: Acute Exposure
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Ingested (g/event)		Ingestion TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
Area I											
PCBs	378.00		0.5		1.00	10	1	1.89E-02		1.00E-02	1.89E+00
Cadmium	19.20		0.5		1.00	10	1	9.60E-04		4.00E-03	2.40E-01
Copper	591.00		0.5		1.00	10	1	2.95E-02		NA	
Lead	384.00		0.5		1.00	10	1	1.92E-02		NA	
REALISTIC WORST CASE										TOTAL	2.13E+00
Area I											
PCBs	6393.00		0.5		1.00	10	1	3.20E-01		1.00E-02	3.20E+01
Cadmium	69.00		0.5		1.00	10	1	3.45E-03		4.00E-03	8.62E-01
Copper	3180.00		0.5		1.00	10	1	1.59E-01		NA	
Lead	1680.00		0.5		1.00	10	1	8.40E-02		NA	
										TOTAL	3.28E+01

Note: TK = Toxicokinetic Factor
Criteria used to evaluate risk are the 1 day and 10 day, RfD for Cd and PCBs.
No appropriate standards or criteria are available to assess acute exposure to copper or lead.

TABLE C-93A. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS; ACUTE EXPOSURE; AREA I; OLDER CHILD

15-Jun-89

New Bedford Harbor
Ingestion of Sediments: Acute Exposure
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)		Amount of Soil Ingested (g/event)		Ingestion TK Factor	Body Weight (kg)	No. of Events per year			Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE											
Area I											
PCBs	378.00		0.5		1.00	40	1	4.73E-03		1.00E-02	4.72E-01
Cadmium	19.20		0.5		1.00	40	1	2.40E-04		4.00E-03	6.00E-02
Copper	591.00		0.5		1.00	40	1	7.39E-03		NA	
Lead	384.00		0.5		1.00	40	1	4.80E-03		NA	
REALISTIC WORST CASE										TOTAL	5.32E-01
Area I											
PCBs	6393.00		0.5		1.00	40	1	7.99E-02		1.00E-02	7.99E+00
Cadmium	69.00		0.5		1.00	40	1	8.62E-04		4.00E-03	2.16E-01
Copper	3180.00		0.5		1.00	40	1	3.98E-02		NA	
Lead	1680.00		0.5		1.00	40	1	2.10E-02		NA	
										TOTAL	8.21E+00

Note: TK - Toxicokinetic Factor

Criteria used to evaluate risk are the 1 day and 10 day HA for Cd and PCBs.

No appropriate standards or criteria are available to assess acute exposure to copper or lead.

TABLE C-93B. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS; ACUTE EXPOSURE; AREA I; ADULT

C-93B

15-Jun-89

New Bedford Harbor
Ingestion of Sediments: Acute Exposure
Noncarcinogenic Effects

This table calculates estimated body doses and harzard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times \{(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})\} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
MOST PROBABLE CASE								
Area I								
PCBs	378.00	0.5	1.00	70	1	2.70E-03	1.00E-02	2.70E-01
Cadmium	19.20	0.5	1.00	70	1	1.37E-04	4.00E-03	3.43E-02
Copper	591.00	0.5	1.00	70	1	4.22E-03	NA	
Lead	384.00	0.5	1.00	70	1	2.74E-03	NA	
							TOTAL	3.04E-01
REALISTIC WORST CASE								
Area I								
PCBs	6393.00	0.5	1.00	70	1	4.57E-02	1.00E-02	4.57E+00
Cadmium	69.00	0.5	1.00	70	1	4.93E-04	4.00E-03	1.23E-01
Copper	3180.00	0.5	1.00	70	1	2.27E-02	NA	
Lead	1680.00	0.5	1.00	70	1	1.20E-02	NA	
							TOTAL	4.69E+00

Note: TK - Toxicokinetic Factor
Criteria used to evaluate risk are the 1 da, and 10 day HA for Cd and PCBs.
No appropriate standards or criteria are available to assess acute exposure to copper or lead.

TABLE C-94. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS; ACUTE EXPOSURE; AREA II AND III; CHILD

New Bedford Harbor

C-94 Ingestion of Sediments: Acute Exposure

15-Jun-89 Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\underset{(mg/kg/day)}{\text{Body Dose}} = \underset{(ug/g)}{\text{Sediment Concentration}} \times \left[\left(\underset{(g/event)}{\text{Amount Contacted}} \times \text{Dermal TK Factor}\right) + \left(\underset{(g/event)}{\text{Amount Ingested}} \times \text{Ingestion TK Factor}\right)\right] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Maximum Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
Area II								
PCBs	125.00	0.5	1.00	10	1	6.25E-03	1.00E-02	6.25E-01
Cadmium	14.00	0.5	1.00	10	1	7.00E-04	4.00E-03	1.75E-01
Copper	2790.00	0.5	1.00	10	1	1.40E-01	NA	
Lead	559.00	0.5	1.00	10	1	2.79E-02	NA	
							TOTAL	8.00E-01
Area III								
PCBs	29.00	0.5	1.00	10	1	1.45E-03	1.00E-02	1.45E-01
Cadmium	ND	0.5	1.00	10	1	0.00E+00	4.00E-03	0.00E+00
Copper	154.00	0.5	1.00	10	1	7.70E-03	NA	
Lead	106.00	0.5	1.00	10	1	5.30E-03	NA	
							TOTAL	1.45E-01

Note: TK = Toxicokinetic Factor

Criteria used to evaluate risk are the [illegible] and [illegible] day HA for [illegible] PCBs.

No appropriate standards or criteria are available to [illegible] acute exposure to compounds [illegible]

TABLE C-94a. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS; ACUTE EXPOSURE; AREA II AND III; OLDER CHILD

C-94A

15-Jun-89

New Bedford Harbor
Ingestion of Sediments: Acute Exposure
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Maximum Concentration (ug/g)	Amount of Soil Ingested (g/event)	Ingestion TK Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
Area II								
PCBs	125.00	0.5	1.00	40	1	1.56E-03	1.00E-02	1.56E-01
Cadmium	14.00	0.5	1.00	40	1	1.75E-04	4.00E-03	4.37E-02
Copper	2790.00	0.5	1.00	40	1	3.48E-02	NA	
Lead	559.00	0.5	1.00	40	1	6.98E-03	NA	
							TOTAL	2.00E-01
Area III								
PCBs	29.00	0.5	1.00	40	1	3.62E-04	1.00E-02	3.62E-02
Cadmium	ND	0.5	1.00	40	1	0.00E+00	4.00E-03	0.00E+00
Copper	154.00	0.5	1.00	40	1	1.93E-03	NA	
Lead	106.00	0.5	1.00	40	1	1.33E-03	NA	
							TOTAL	3.62E-02

Note: TK - Toxicokinetic Factor
Criteria used to evaluate risk are the 1 day and 10 day DWA [illegible] and Form.
No appropriate standards or criteria are available to assess acute exposure to copper [illegible].

TABLE C-94B. BODY DOSE CALCULATIONS FOR NONCARCINOGENIC RISKS; INGESTION OF SEDIMENTS; ACUTE EXPOSURE; AREA II AND III; ADULT

C-94B

15-Jun-89

New Bedford Harbor
Ingestion of Sediments: Acute Exposure
Noncarcinogenic Effects

This table calculates estimated body doses and hazard indices.

The equations to calculate body dose level and hazard indices are:

$$\text{Body Dose (mg/kg/day)} = \text{Sediment Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal Tk Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion Tk Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{No. Events}}{\text{year}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{1\ \text{yr}}{365\ \text{days}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Maximum Concentration (ug/g)		Amount of Soil Ingested (g/event)		Ingestion Tk Factor	Body Weight (kg)	No. of Events per year		Standard/ Guideline (mg/kg/day)	Hazard Index
Area II										
PCBs	125.00		0.5		1.00	70	1	8.93E-04	1.00E-02	8.93E-02
Cadmium	14.00		0.5		1.00	70	1	1.00E-04	4.00E-03	2.50E-02
Copper	2790.00		0.5		1.00	70	1	1.99E-02	NA	
Lead	559.00		0.5		1.00	70	1	3.99E-03	NA	
									TOTAL	1.14E-01
Area III										
PCBs	29.00		0.5		1.00	70	1	2.07E-04	1.00E-02	2.07E-02
Cadmium	ND		0.5		1.00	70	1	0.00E+00	4.00E-03	0.00E+00
Copper	154.00		0.5		1.00	70	1	1.10E-03	NA	
Lead	106.00		0.5		1.00	70	1	7.57E-04	NA	
									TOTAL	2.07E-02

Note: Tk - Toxicokinetic Factor
Criteria used to evaluate risk are the 1 day and 10 day HA for Cd and PCBs.
No appropriate standards or criteria are available to assess acute exposure to copper or lead.

TABLE C-98

SITE: New Bedford
EXPOSURE: Ingestion of Sediment
Carcinogenic Effects
Cove Area (Area 1)
Child

CONTAMINANTS OF CONCERN:	CONCENTRATION IN SEDIMENT (ug/g) WORST CASE ONE	WORST CASE TWO	FRACTION ABSORBED (%) WORST CASE ONE	WORST CASE TWO	CAG POTENCY ESTIMATE (mg/kg/day)-1
PCBs	20.9100	124.5[illegible]	1.00	1.00	7.70E+00

DATE: 16-Jun-89

DIRECTORY:
DISK: 2d5
FILE: INGPI

Most Probable Case
FREQUENCY OF CONTACT (days) — NUMBER OF EXPOSURES — DAYS
SUB-CHRONIC DOSE: 1 — 365
LIFETIME DOSE(70 years):
- No. of years exposure — 5 (years)

AMOUNT OF SEDIMENT CONSUMED (g/exp) — WORST CASE ONE — CHILD: 0.5
BODY WEIGHT (kg) — CHILD: 10

Realistic Worst Case
FREQUENCY OF CONTACT (days) — NUMBER OF EXPOSURES — DAYS
SUB-CHRONIC DOSE: 20 — 365
LIFETIME DOSE(70 years):
- No. of years exposure — 5 (years)

WORST CASE TWO — CHILD: 0.5

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCBs	286.0000	0.5	1.00	10.0	1.43E-01	NA	3.9E-05	3.9E-05	7.70E+00	NA	4.31E-06	2.15E-05

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCBs	359.[illegible]	[illegible]	1.[illegible]	[illegible]	[illegible]	NA	1.1E[illegible]	1.1E-03	7.70E+00	NA	1.20E-04	6.01E-04

TABLE C-95

SITE: New Bedford
EXPOSURE: Ingestion of Sediment
Carcinogenic Effects
Popes Island (Area II)
Child

DATE: 16-Jun-89
DIRECTORY:
DISK: 205
FILE: INGFOPE

CONTAMINANTS OF CONCERN:	CONCENTRATION IN SEDIMENT (ug/g) WORST CASE ONE	WORST CASE TWO	FRACTION ABSORBED (%) WORST CASE ONE	WORST CASE TWO	CAG POTENCY ESTIMATE (mg/kg/day)-1
PCB's	20.9100	124.5700	1.00	1.00	7.70E+00

Most Probable Case

	NUMBER OF EXPOSURES	DAYS	AMOUNT OF SEDIMENT CONSUMED (g/exp) WORST CASE ONE	BODY WEIGHT (kg)
FREQUENCY OF CONTACT (days)				
SUB-CHRONIC DOSE:	20	365	CHILD: 0.5	CHILD: 10
LIFETIME DOSE(70 years):				
- No. of years exposure		5 (years)		

Realistic Worst Case

	NUMBER OF EXPOSURES	DAYS	WORST CASE TWO
FREQUENCY OF CONTACT (days)			
SUB-CHRONIC DOSE/	100	365	CHILD: 0.5
LIFETIME DOSE(70 years):			
- No. of years exposure		5 (years)	

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	11.0000	0.5	1.00	10.0	5.50E-03	NA	3.0E-05	3.0E-05	7.70E+00	NA	3.32E-06	1.66E-05

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/fish meal)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	34.0000	0.5	1.00	10.0	1.70E-02	NA	4.3E-05	4.3E-04	7.70E+00	NA	5.13E-05	2.56E-04

TABLE C-96

		CONTAMINANTS OF CONCERN:	CONCENTRATION IN SEDIMENT (ug/g) WORST CASE ONE	WORST CASE TWO	FRACTION ABSORBED (%) WORST CASE ONE	WORST CASE TWO	CAG POTENCY ESTIMATE (mg/kg/day)-1
SITE:	New Bedford						
EXPOSURE:	Ingestion of Sediment						
	Carcinogenic Effects	PCBs	20.7100	124.5900	1.00	1.00	7.70E+00
	Falmer Island (Area II)						
	Child						

DATE: 16 Jun-89

DIRECTORY:
DISK: 205
FILE: INGFI

	Most Probable Case FREQUENCY OF CONTACT (days)	NUMBER OF EXPOSURES	DAYS	AMOUNT OF SEDIMENT CONSUMED (g/exp) WORST CASE ONE	BODY WEIGHT (kg)
	SUB-CHRONIC DOSE(	20	365	CHILD: 0.5	CHILD: 10
	LIFETIME DOSE(70 years):				
	- No. of years exposure		5 (years)		
				WORST CASE TWO	
	Realistic Worst Case FREQUENCY OF CONTACT (days)	NUMBER OF EXPOSURES	DAYS	CHILD: 0.5	
	SUB-CHRONIC DOSE	100	365		
	LIFETIME DOSE(70 years):				
	- No. of years exposure		5 (years)		

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)			y)	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCBs	3.0000	0.5	1.00	10.0	1.50E-03	NA	8.2E-06	8.2E-06	7.70E+00	NA	9.04E-07	4.52E-06

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub chronic	Risk Estimate Chronic
PCBs	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	7.7[illegible]	NA	1.66E-05	8.29E-05

TABLE C-97

		CONCENTRATION IN SEDIMENT (ug/g)		FRACTION ABSORBED (%)		CAG POTENCY
SITE: New Bedford	CONTAMINANTS	WORST CASE	WORST CASE	WORST CASE	WORST CASE	ESTIMATE
EXPOSURE: Ingestion of Sediment	OF CONCERN:	ONE	TWO	ONE	TWO	(mg/kg/day)-1
Carcinogenic Effects	PCB's	20.9100	124.5900	1.00	1.00	7.70E+00
Marsh Island (Area II)						
Child						

DATE: 16-Jun-89

DIRECTORY:
DISK: 205
FILE: INGMI

Most Probable Case	NUMBER OF				
FREQUENCY OF CONTACT (days)	EXPOSURES	DAYS	AMOUNT OF SEDIMENT CONSUMED (g/exp)		BODY WEIGHT (kg)
			WORST CASE ONE		
SUB-CHRONIC DOSE	20	365	CHILD:	0.5	CHILD: 10
LIFETIME DOSE(70 years):					
- No. of years exposure		5 (years)			
			WORST CASE TWO		
Realistic Worst Case	NUMBER OF		CHILD:	0.5	
FREQUENCY OF CONTACT (days)	EXPOSURES	DAYS			
SUB-CHRONIC DOSE:	100	365			
LIFETIME DOSE(70 years):					
- No. of years exposure		5 (years)			

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Most Probable Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	8.0000	0.5	1.00	10.0	4.00E-03	NA	2.2E-05	2.2E-05	7.70E+00	NA	2.41E-06	1.21E-05

New Bedford
Ingestion of Sediment
Carcinogenic Effects
Realistic Worst Case
Child

Compound	Concentration (ug/g)	Amount of Sed Consumed (g/exposure)	Fraction Absorbed (%)	Body Weight (kg)	Amount of Contaminant Absorbed (mg/exposure)				CAG Potency Estimate (mg/kg/day)-1	Risk Estimate Short-term	Risk Estimate Sub-chronic	Risk Estimate Chronic
PCB's	22.0[illegible]	0.5	1.00	10.0	[illegible]	NA	[illegible]	2.0E-04	7.[illegible]E+00	NA	3.32E-05	1.66E-04

APPENDIX D

TOXICOLOGICAL EVALUATIONS

D.0 INTRODUCTION

This appendix contains the toxicological evaluations for the four contaminants of concern at New Bedford Harbor: polychlorinated biphenyls (PCBs), cadmium, copper, and lead. Each evaluation includes a discussion of the contaminant's pharmacokinetic properties, its acute and chronic toxicity (from humans and animals studies) including, if applicable, carcinogenicity, mutagenicity, teratogenicity, and reproductive toxicity, interactive effects, and high risk subpopulations.

D.1 TOXICOLOGICAL EVALUATION FOR POLYCHLORINATED BIPHENYLS (PCBs)

The purpose of this section is to describe the toxicological properties of polychlorinated biphenyls (PCBs), particularly with respect to risk to public health from contamination at New Bedford Harbor. The Toxicological Evaluation is not intended to be a comprehensive evaluation of primary scientific studies. Rather, it is meant to be an overview of information gleaned from review articles and summary documents regarding the nature and extent of the toxicity of PCBs. This chapter has an emphasis on the potential health effects that could result from exposure by the anticipated exposure routes for the New Bedford Harbor population. Therefore, the Toxicological Evaluation will emphasize routes of exposure (oral, dermal, and inhalation) in presenting toxicity information. Furthermore, information on the actual health effects previously observed in humans will be presented whenever available. The Toxicological Evaluation will include the following sections: (1) Background Information, (2) Toxicokinetics, (3) Overview of Health Effects Observed in Humans, (4) Toxicity, (5) Interactive Effects, (6) High Risk Subpopulations, and (7) Summary.

D.1.1 Background

Polychlorinated biphenyls (PCBs) are man-made chemical compounds with the general chemical structure as is shown in Figure 2-6. They are mixtures of chlorinated biphenyl isomers. Commercial production of PCBs dates from 1930 (Cordle, 1982). Their properties of thermal stability, nonflammability, and dielectric capability resulted in their use as a liquid insulating material in electrical capacitors, transformers and other electrical equipment. Beginning in 1971, PCBs were sold in the United States only for use in closed systems. In 1977, PCB production ceased entirely (EPA, 1982c, d). Release of PCBs still occurs, however, during maintenance and disposal of electrical equipment, particularly transformers and capacitors.

Monsanto, the U.S. manufacturer of PCBs, used the tradename "Aroclor" followed by four digits. The first two digits refer to the 12 carbon atoms of the biphenyl and the second two digits

indicate the approximate percent by weight of chlorine in the mixture. For example, Aroclor 1254 is a mixture of biphenyls having an average chlorine content of 54%. A PCB mixture with average chlorine content of 54% is also referred to as "54% PCB."

PCBs are manufactured in other countries under tradenames such as Kaneclors (Japan), Clophens (Germany) and Phenoclors (France). Table D-1 gives the percent composition of various commercial PCB mixtures. One mixture, Aroclor 1016, is an exception to the standard nomenclature.

Some PCB mixtures have been reported to be contaminated with polychlorinated dibenzofurans, a highly toxic class of compounds which causes health effects similar to those seen with PCBs. As shown in Table D-2, the potential for contamination does not compromise the large literature on PCB toxicity. Toxicological studies with PCB mixtures uncontaminated with polychlorinated dibenzofurans have reported the same health effects (liver damage, including tumors, and dermal lesions) as studies with reportedly contaminated mixtures. For instance, studies of Aroclor 1260 "free" of chlorinated dibenzofurans (limit of detection: 1 ppm) have reported toxic effects in the liver and skin of rabbits given dermal applications of this uncontaminated Aroclor (Vos and Totenboom-Ram, 1972).

PCBs do not occur naturally; their presence in the environment is associated with human activities. PCBs are inert chemicals which are relatively resistant to degradation. Because of their stability, they have a long half-life and thus accumulate in the environment. They were first reported in environmental samples in 1966 in Baltic Sea fish (Jensen, 1966, EPA, 1977a) and are now widely distributed in the environment. Background levels of PCBs can be found in outdoor air (0.02-30 ng/m^3) (Eisenbreich et al., 1981), on soil surfaces (2.3-444 mg/kg) (Creaser and Fernandes, 1986), and in water (0.04-3 ng/l) (Tanabe et al., 1983, 1984; Boon and Duinker, 1986; Giam et al., 1978). In addition, fish in certain waters are widely contaminated with PCBs. Consumption of contaminated fish is a major source of PCB exposure to humans.

D.1.2 Toxicokinetics

Toxicokinetics is the study of the uptake, distribution, biotransformation and elimination of foreign compounds, such as toxic contaminants. The purpose of this section is to discuss toxicokinetic issues regarding exposure to PCBs. Toxicokinetic parameters govern the rate at which a contaminant enters and is eliminated from the body and, therefore, the contaminant concentration in the body or at a particular target organ. When the rate of absorption exceeds the rate of elimination, some

TABLE D-1

PERCENT COMPOSITION OF AROCLORS AND KANECHLORS[a]

Empirical Formula	Aroclor Number							Kanechlor Number		
	1221	1232	1016	1242	1248	1254	1260[b]	300	300	500
$C_{12}H_{10}$	11	<0.1	<0.1	<0.1	ND	<0.1	ND	NR	NR	NR
$C_{12}H_9Cl$	51	31	1	1	ND	<0.1	ND	NR	NR	NR
$C_{12}H_8Cl_2$	32	24	20	16	2	0.5	ND	16.6	3.0	NR
$C_{12}H_7Cl_3$	4	28	57	49	18	1	ND	59.8	32.8	5.0
$C_{12}H_6Cl_4$	2	12	21	25	40	21	1	23.0	43.8	26.5
$C_{12}H_5Cl_5$	<0.5	4	1	8	36	48	12	0.6	15.8	55.0
$C_{12}H_4Cl_6$	ND	<0.1	<0.1	1	4	23	38	NR	4.6	12.8
$C_{12}H_3Cl_7$	ND	ND	ND	<0.1	ND	6	41	NR	NR	NR
$C_{12}H_2Cl_8$	ND	ND	ND	ND	ND	ND	8	NR	NR	NR
$C_{12}HCl_9$	ND	ND	ND	ND	ND	ND	ND	NR	NR	NR
Percent chlorine	20.5-21.5	31.4-32.5	41	42	48	54	60	NA	49	54

[a] Source: Callahan et al., 1979; IARC, 1974; Masuda et al., 1974

[b] Kanechlor 600 (Japanese), Phenoclor DP6 (French) and Clophen A60 (German); all contain ~60% Cl (Hasuda et al., 1974; Fishbein, 1974).

ND = None Detected
NR = Not Reported

TABLE D-2

POLYCHLORINATED DIBENZOFURAN CONCENTRATION
IN COMMERCIAL PCBS*

PCB	Concentration (ug/g) 4-C1	5-C1	6-CL	Total
Aroclor 1016 (1972)	<0.001	<0.001	<0.001	<0.003
Aroclor 1248 (1969)	0.5	1.2	0.3	2.0
Aroclor 1254 (1969)	0.1	0.2	1.4	1.7
Aroclor 1254 (1970)	0.2	0.4	0.9	1.5
Aroclor 1260 (1969)	0.1	0.4	0.5	1.01
Aroclor 1260 (lot AK3)	0.2	0.3	0.3	0.8
Clophen A-60	1.4	5.0	2.2	8.4
Phenoclor DP-6	0.7	10.0	2.9	13.6

*Source: Adapted from Bowes et al., 1975

contaminants may accumulate to concentrations capable of eliciting toxic effects (Loomis, 1983). For other contaminants the rate of elimination has little effect on toxic response because their potency is so high. Many chemicals also undergo extensive biotransformation by metabolic enzyme systems that may render the chemical more or less toxic depending on its particular characteristics. Thus, these pharmacokinetic parameters often govern the severity, mode and site of toxic action.

D.1.2.1 Absorption

The absorption potential of a chemical is a critical factor in determining the toxic effects elicited. The efficiency of absorption of pure PCBs can differ for the oral, dermal and inhalation routes of exposure. In addition, matrix effects can significantly alter absorption behavior. For instance, PCBs can be administered as contaminants of soil or food constituents, as solutions in corn oil or organic solvents, or as trace contaminants in water. Interpretation of toxicity data for use in risk assessment must carefully consider the available absorption information.

Because the toxicological studies that will be employed for risk characterization are all based on administered doses, one cannot compare the estimated absorbed dose from the risk assessment to the administered dose from the laboratory experiment. Instead, one must compare two administered doses and make any necessary adjustments if the efficiency of absorption is known or expected to differ because of physiological effects and/or matrix or vehicle effects. The toxicokinetic factor (TKF) is used for this purpose. The TKF is defined as the ratio of the estimated absorption factor for the route of interest and for the contaminated medium of interest to the absorption factor for the laboratory toxicology study from which the cancer potency factor or reference dose was derived. Most commonly, this will be a study where the test compound was administered orally. For PCBs, the EPA cancer potency factor was derived from a laboratory study in which PCBs were given to rats in the diet.

Oral Exposure--The gastrointestinal absorption factor for the study from which the EPA cancer potency factor was derived (Norback and Weltman, 1985) is estimated as 80%. This study is a chronic feed study using Sprague-Dawley rats. PCBs (Aroclor 1260) were administered in the diet. Aroclor 1260 was mixed in corn oil and then added to Purina Rat Chow. No information on the efficiency of gastrointestinal absorption was available from the study. To estimate the efficiency of gastrointestinal absorption, the toxicological literature was searched for appropriate studies on PCBs. Six studies were identified that contained relevant absorption information.

Allen et al. (1975) gave single oral doses of 2,5,2',5'-tetrachlorobiphenyl (18 mg/kg bw) to four adult rhesus monkeys by gastric intubation. PCBs were given in 2.5 mL of corn oil on an empty stomach. Unmetabolized PCBs were analyzed in the feces by gas chromatography. Minimum gastrointestinal absorption was found to be 88%. PCBs found in the feces over a specified post-dosing time were presumed to be unabsorbed material. Because PCB metabolites are known to be eliminated in the bile, the possibility exists that some of the PCBs present in the feces were absorbed and then eliminated. As such, only minimum absorption efficiencies can be determined from this and similar studies.

Allen et al. (1974) gave single oral doses of PCBs (Aroclor 1248) (1.5 or 3.0 g/kg bw) to two adult rhesus monkeys by gastric intubation. The vehicle was not specified but is presumed to be corn oil. Dosing was done on an empty stomach. Unmetabolized PCBs were analyzed for in feces by gas chromatography. Recovery was reported to be high. Minimum gastrointestinal absorption was reported to be 94%.

Norback et al. (1978) gave single oral doses of 2,4,5,2',4',5' hexachlorobiphenyl (1g/kg bw) to two adult rhesus monkeys by nasogastric intubation. Corn oil was the vehicle. No information was available concerning the animals' stomach contents at the time of dosing. Total radioactivity was measured in the feces and the bile (bile duct cannulated). Minimum absorption was 13% in one animal and 41% in the other (average = 27%).

Because these investigators were measuring both parent and metabolized species in the feces (total radioactivity), the degree of absorption may be underestimated if metabolites were present in the feces during the first week. In addition, according to the limited experimental details available in this abstract, bile was returned from the cannulated bile duct to the duodenum. If so, not all of the radioactivity in the feces may be due to unabsorbed material. Thus, the absorption reported in this study is a minimum gastrointestinal absorption.

Albro and Fishbein (1972) gave single oral doses of 20 different PCB congeners (5-100 mg/kg bw) and the unabsorbed marker compound, squalene, to CD rats. The mixture was given by stomach tube to fed animals who were allowed food and water ad libitum. No vehicle was specified. Although this was not a diet study, per se, it is possible that dietary components were present in the stomach at the same time as were the test compounds. Minimum absorption was reported to be 90% for all congeners.

Tanabe et al. (1981) gave repeated oral doses of Kanechlors (300, 400, 500, 600) (c. 30 mg/kg bw/day x 5 days) to Wistar rats. The dose was given in corn oil. Commercial diet was given ad libitum. No information on the animals' stomach contents was reported. Parent compounds were analyzed in the feces by GC/MS analysis. Minimal gastrointestinal absorption was reported to be 85% for total PCBs. Cl_5 to Cl_7 congeners had 75-90% absorption.

Berlin et al. (1974) gave a single oral dose of 2,4,5,2',5' pentachlorobiphenyl (7 mg/kg bw) to three CBA mice. The PCBs were given as an aqueous emulsion. No information on the animals' stomach contents was given. Minimum gastrointestinal absorption was reported to be 93%.

These studies, which involve both rodents and primates and various PCB mixtures and purified congeners, all show that PCBs are very efficiently absorbed from the gastrointestinal tract. It is possible that absorption of PCBs that are thoroughly mixed in the diet is lower than absorption from these studies in which PCBs are dissolved in corn oil and given by gastric intubation. In the chronic feeding study of Norback and Weltman (1985), however, the PCBs were added to the diet as a corn oil solution. It has been determined that the above studies do yield reasonable estimates of the degree of absorption expected in the Norback and Weltman study. The six absorption factors were averaged to yield the estimate of 80%.

Absorption by the gastrointestinal route has also been reported in humans. Increased consumption is associated with increased PCB blood levels in several studies. Humphrey (1976) reported blood levels of PCBs in people who consumed contaminated sport fish from Lake Michigan. Annual consumption of $\geq$ 24 pounds resulted in a mean blood level that was 4 times the blood level of PCBs in persons who ate $\leq$ 6 pounds per year, Schwartz et al. (1983) also found elevated levels of PCBs in blood serum and breast milk of women who ate PCB-contaminated fish. These investigators described a quantitative relationship between PCB consumption and serum and blood PCB levels. In addition, Drotman et al. (1983) found a positive correlation between the PCB concentration in human breast milk and the number of PCB-contaminated eggs consumed by lactating women. Finally, numerous adverse health effects have been well-documented in a Japanese population who accidentally ingested rice oil that was heavily contaminated with PCBs.

Dermal Exposure--Extensive absorption also occurs with dermal application of PCBs. For use in the New Bedford Harbor risk assessment, PCB toxicokinetic factors (TKFs) have been developed for two types of contaminated sediment: heavily contaminated sediments in which the concentration of PCBs exceeds 1% and less

contaminated sediments ([PCBs]<1%). It is expected that the former factors would only be used for assessing the risks to PCBs from exposure at certain "hot spot" areas. The two approaches are required because free PCBs are present in samples that are contaminated in the % range. It is inappropriate to consider the effects of matrix components in reducing the bioavailability of PCBs unless the ratio of sediment to PCBs is large, such as when PCBs are present at ppm levels or lower.

The dermal absorption factor for pure PCBs is estimated as 41%. This estimate is based on the result of several studies which have investigated the efficiency of dermal absorption of pure PCBs or PCBs given in aqueous solution.

Shah et al. (1981) placed 2,4,5,2',4',5'-hexachlorobiphenyl on the shaved backs of Duplin ICR mice for various times. The PCB was administered in 100 mL of acetone, which was quickly evaporated. Total radioactivity was determined in specific tissues, organs, excretory products and the carcass. Radioactivity at the application site was analyzed to determine the quantity of unabsorbed chemical. 45% of the administered dose was systemically absorbed in 30 minutes (n=30), and 55% was absorbed in 1 hour (n=3). After evaporation, the quantity of PCB on the skin surface was a film of thickness 0.0005 mm (assuming that the density is 1 g/cm^3).

Wester et al. (1983) placed 42% PCB (4.1 and 19.3 ug/cm^2) on the shaved abdomens of four rhesus monkeys for 24 hours. The PCB was administered in 50 uL of hexane/benzene (1:1) which evaporated quickly. The efficiency of dermal absorption was determined by comparing the total urinary excretion of radioactivity following topical administration to that following parenteral administration. From 15-34% of the administered dose was systemically absorbed. The average absorption for the four animals was 21.5 $\pm$ 8.5%. After evaporation, the dose of PCB corresponded to a thin film of 4-19 x 10^{-5} mm thickness (assuming that the density is 1 g/cm^3).

Guinea pigs were dosed with 42% PCB (4.6 ug/cm^2) or 54% PCB (5.2 ug/cm^2) on the skin on the back of the ear for 24 hours. Dermal absorption was 33.2 $\pm$ 6.3% (n=3) for 42% PCB and 55.6 $\pm$ 2.6% (n=3) for 54% PCB. These values indicate the dermal absorption that was observed after 24 hours. No earlier time points were determined. Shah et al. (1981), however, found that dermal absorption of PCBs in mice was not linear over time. Instead, it plateaued after only a short period of time (approx. 1 hour). Thus, the absorption observed by Wester et al. (1983) over 24 hours was probably virtually complete after 1-2 hours.

Wester et al., (1987) found that 96% of the 54% PCB in a dilute aqueous solution (1.6 ug/mL) bound to powdered human skin

(stratum corneum). In this experiment 1.5 mL of aqueous solution was mixed with 1.5 mg of powdered skin. The fraction of chemical bound was determined by measuring the amount of radioactivity on the skin and in the supernatant. In another in vitro experiment, 12% of the PCBs in the same aqueous solution were bound to and absorbed through a section of fresh human skin obtained from surgical reduction. The administered dose corresponds to a thin film of aqueous solution 1.6 mm in depth above the skin surface.

For purposes of estimating the dermal absorption of pure PCBs, the results of the in vitro powdered skin experiment were excluded. The absorption may have been abnormally high due to the very high surface area of the skin. The other four results for mice, guinea pigs, rhesus monkeys, and humans were averaged to yield 41.3 $\pm$ 16.8% absorption for pure PCBs. The dermal absorption factor for PCBs in sediments contaminated with PCBs at levels below 1% is estimated as 5.4%.

No experiments were located in the available literature in which the dermal absorption for PCB-contaminated soils or sediments was measured. In one study, however, the absorption of structurally similar TCDD was compared for a TCDD solution in methanol and for TCDD-contaminated soil. Poiger and Schlatter (1980) dosed hairless rats (Naked ex Back-Cross and Holzman strain) with radiolabelled TCDD. The percent of the administered dose in the liver after 24 hours was compared for two situations: 26 mg TCDD in 50 uL per 3 cm^2 of methanol; and 350 or 1300 mg TCDD in a soil/water paste of 75 mg/3-4 cm^2. The percent of the dose in the liver after administration of the soil paste was the same for the two dose levels. The values were averaged and compared to the percent of the dose in the liver following administration of pure PCB from a methanol solution:

$$\frac{\text{dermal absorption, soil}}{\text{dermal absorption, solvent}} = \frac{1.95}{14.8} = 13\% \ (0.13)$$

It is assumed that PCBs are adsorbed to soil and retarded in their dermal absorption to the same degree as is TCDD. Thus, the dermal absorption factor for PCBs in contaminated sediments is derived by multiplying the dermal absorption factor for pure PCBs by the expected ratio of the dermal absorption from sediments to the dermal absorption of pure PCBs.

Dermal absorption factor for PCBs in contaminated sediment = 0.13 x 0.413 = 0.054 (5.4%). This value agrees well with the dermal absorption factor used by EPA for PCB-contaminated soil in contact with human skin (5%) (EPA, 1986b).

Dermal exposure to PCBs has not been well-documented in humans. However, many epidemiological researchers have postulated that occupational exposures to PCBs are probably due both to

inhalation and direct dermal contact with PCBs. For instance, in a study of occupational exposure of electrical workers to PCBs (Pyralen 3010 and Apirolio, 42% chlorine), Maroni et al. (1981) concluded that absorption of PCBs occurred mainly through the skin as compared to respiratory absorption.

Inhalation Exposure--Airborne PCBs are assumed to be rapidly absorbed by humans based on the results of numerous occupational studies in which PCB exposure was related to adverse health effects. However, little experimental information quantitating the efficiency of inhalation uptake was found in the available literature.

PCBs were rapidly absorbed by inhalation when male Wistar rats were exposed to a commercial PCB mixture containing 42% chlorine (Pydranil A200) (Benthe et al., 1972). After 15 minutes exposure to 30.4 g/m3, significant liver concentrations of PCBs were reported.

The PCB concentration used in this experiment is significantly higher than the concentrations that have been reported historically in workplace environments. Absorption factors were not quantified in the experiment.

D.1.2.2 Distribution

The toxicity of a chemical is influenced by its distribution and storage in the body. Distribution of ingested or injected PCBs follows a two-phase pattern. During the first phase, distribution from the blood muscle and liver occurs quite rapidly. During the second phase, redistribution to fat, skin and fat-containing organs occurs more slowly (Matthews and Anderson, 1975).

Distribution following inhalation exposure shows a similar pattern. Initial distribution is to the liver. Within the first 24 hours, rapid increases in brain and fat levels is seen. At the end of 2 days, liver and brain levels of PCBs fall, and levels in adipose tissue attain a maximum (Benthe et al., 1972).

PCBs can be detected in human blood, body fat, and breast milk. Blood PCB levels are the best indicators of recent exposure to PCBs, and levels in fat are the best indicators of long-term exposure. Tables D-3, D-4, and D-5 show the following blood serum PCB concentrations: 1) in United States populations without occupational exposure to PCBs (range: 0-60 ng/mL); 2) subpopulations consuming fish from PCB-contaminated waters (range: < 3-366 ng/mL); and 3) populations with occupational exposure (range: 1-3330 ng/mL). Other tissues that tend to accumulate PCBs include gonads, adrenals and kidneys (Kurachi and Mio, 1983b).

TABLE D-3

SERUM PCB CONCENTRATIONS IN UNITED STATES POPULATIONS WITHOUT OCCUPATIONAL EXPOSURE TO PCBs[1]

Area and Sampling Method	Number of Subjects	Year	PCB Level, ng/mℓ: Arithmetic Mean	Geometric Mean, Median[2]	Arithmetic Standard Deviation	95% Confidence Interval	Range	Reference
Populations Without Occupational Exposures, 1968-1983								
Charleston County, SC Volunteers	616	1968	4.9	--	--	--	0-29	Finkles et. al., 1972
Lake Michigan Random Nonfisheaters	29	1973	17.3	15[2]	--	--	<5-41	Humphrey, 1983a
Bloomington, IN Volunteers and Controls	110	1977	18.8	--	10.8	17-21	6-79	Baker et. al., 1980
Michigan PBB Cohert	1631	1978-9	7.7	6.4	--	--	<1-57	Kreiss et. al., 1982
Billings, MT Random Packinghouse Workers	17	1979	7.5	5.8	6.8	4-11	2-30	Drotman et. al., 1981
Franklin, ID Volunteers	105	1979	--	--	--	--	<5	Drotman et. al., 1981
Random Unexposed Workers	19	1979	12	--	--	--	10-27	Chase et. al., 1982
Newton, KS Volunteers	7	1979	4.9	4.2	3.1	2-8	2-11	Vernon et. al., 1981
Lake Michigan Random Nonfisheaters	418	1980	--	6.6[2]	--	--	<3-60	Humphrey, 1983a
Canton, MA Volunteers	10	1980	7.1	5.2	5.2	3-11	1-18	Condon, 1983

3.88.80
0045.0.0

TABLE D-3 (cont.)

SERUM PCB CONCENTRATIONS IN UNITED STATES POPULATIONS WITHOUT OCCUPATIONAL EXPOSURE TO PCBs AND IN SUBPOPULATIONS CONSUMING FISH FROM PCB-CONTAMINATED WATERS[1]

Area and Sampling Method	Number of Subjects	Year	PCB Level, ng/mℓ: Arithmetic Mean	Geometric Mean, Median[2]	Arithmetic Standard Deviation	95% Confidence Interval	Range	Reference
Old Forge, PA Volunteers	138	1981	3.6	--	--	--	<3-43	Reid and Fox, 1982
Jefferson, OH Volunteers	59	1983	5.8	4.4	6.5	4-8	1-45	Welty, 1983
Fairmont, WV Volunteers	40	1983	6.7	5.0	5.3	5-8	1-23	Welty, 1983
Norwood, MA Volunteers	990	1983	4.9	4.2	3.5	4-6	2-30	Condon, 1983

[1] Source: Kreiss, 1985
[2] Median

TABLE D-4

SERUM PCB CONCENTRATIONS IN UNITED STATES POPULATIONS WITHOUT OCCUPATIONAL EXPOSURE TO PCBs AND IN SUBPOPULATIONS CONSUMING FISH FROM PCB-CONTAMINATED WATERS[1]

Area and Sampling Method	Number of Subjects	Year	PCB Level, ng/mℓ: Arithmetic Mean	Geometric Mean, Median[2]	Arithmetic Standard Deviation	95% Confidence Interval	Range	Reference
Lake Michigan Volunteer Sportfishers	90	1973	72.7	56[2]	--	--	25-366	Humphrey, 1983b
Triana, AL Volunteers	458	1979	22.2	17.2	22.3	20-24	3-158	Kriess et. al., 1981
Lake Michigan Volunteer Sportfishers	572	1980	--	21.4[2]	--	--	<3-203	Humphrey, 1983b
New Bedford, MA Volunteers	11	1981	31.4	23.6	29.3	13-50	5-101	Condon, 1983

[1] Source: Kreiss, 1985
[2] Median

TABLE D-5

SERUM PCB CONCENTRATIONS IN POPULATIONS WITH OCCUPATIONAL EXPOSURE[1]

Facility	Number of Subjects	PCB Levels, mg/mℓ Arithmetic Mean	Geometric Mean, Median[2]	95% Confidence Interval	Range	Reference
Railway Car Maintenance	86	33.4	--	--	10-312	Chase et. al., 1982
Capacitor Plant	34	394[2]	--	234-554	trace-1700	Ouw et. al., 1976
Capacitor Plants	290	124[3] 48[4]	67[3] 21[4]	98-150[4] 38-58[4]	6-2530[3] 1-546[3]	Fischbein et. al., 1979 Wolff et. al., 1982a
Capacitor Plants	80	342[2]	--	--	41-1319	Maroni et. al., 1981a
Capacitor Plant	221	--	119[3] 25.3[4]	-- --	1-3330[3] 1-250[4]	Smith et. al., 1982
Public Utility	14	--	24[3] 24[4]	15-39[3] 16-35[4]	5-52[3] 7-24[4]	Smith et. al, 1982
Private Utility	25	--	22[3] 29[4]	17-25[3] 20-43[4]	9-48[3] 7-250[4]	Smith et. al., 1982

[1] Source: Kreiss, 1985
[2] Blood Level
[3] Lower PCB Homologs
[4] Higher PCB Homologs

The biological half-life of PCBs in humans is relatively long, and humans appear to retain PCBs in their tissues more effectively than any other mammal (EPA, 1977a). In 1971, the National Human Monitoring Program reported PCBs in measurable amounts in 31.6% of 637 human adipose tissue samples (Kutz, 1976). PCB residues most frequently found in human tissues closely resemble Aroclors 1254 or 1260 (Highland, 1976).

PCBs cross the placenta, and tissue levels in the fetus may exceed those in dams in the final stage of gestation. PCBs are also transferred through milk fat. Tissue levels in 1-month-old weaned rats exceeded those of dams (Mizunoya et al., 1974). This same phenomenon was observed in fetal and nursing infant rhesus monkeys (Allen and Barsotti, 1976). Masuda et al. (1979) noted that the lesser chlorinated isomers, being more rapidly metabolized, did not transfer as readily through milk fat as those isomers which tend to accumulate in fat tissue.

PCBs have also been reported in the breast milk of women throughout the world (Miller, 1977; Kuwabara et al., 1979, Hofvander et al., 1981; Rogan et al., 1980; Wickizer et al., 1981) with reported levels ranging from 10 ppb in Norway to 100 ppb in Germany (Wasserman, 1979). As noted earlier, elevated levels of PCBs in breast milk have been reported in women who consumed PCB-contaminated fish (Schwartz et al., 1983).

D.1.2.3 Metabolism and Excretion

The toxicity of a chemical can be increased or decreased through metabolism. The metabolic processes and their rates can also determine the elimination of a chemical. PCBs and their metabolites are excreted primarily in the feces due to biliary excretion. Excretion in the milk, hair, and urine have also been observed. Urinary excretion has been found to be only a minor route of excretion in all species tested. Excretion of PCB metabolites occurs almost exclusively in conjugated form. Glucuronide, sulfate, and glutathione conjugates have all been observed. Excretion of PCBs in an unmetabolized form from the stomach wall and into the feces has also been reported in rats (Yoshimura and Yamamoto, 1975).

All mammals tested metabolize PCBs in a similar fashion. Differences in metabolism of PCBs among species of mammals are primarily quantitative (Allen and Norbeck, 1976; Safe et al., 1975). Hydroxylated products are the major PCB metabolites although methyl sulfones, methyl ether derivatives, and other metabolites have also been observed. The degree of chlorination and the position of the chlorine substituents profoundly affects the rate and extent of metabolism. Although the effects of chlorine substitution have not been systematically studies,

several conclusions appear to be generally true. The presence of at least two adjacent, unsubstituted hydrogen atoms is required for rapid metabolism of PCB isomers. All of the mono-, di-, and trichlorinated isomers and all tetrachlorinated isomers, except one, satisfy this requirement. Hence, metabolism of the lesser chlorinated isomers occurs more rapidly than metabolism of the more heavily chlorinated isomers. Gage and Holm (1976) noted that the isomers which translocate to adipose tissue to a greater extent are the isomers which are the least metabolized and excreted, i.e. the more highly chlorinated isomers. This same finding has been reported in humans (Chen, 1982).

Metabolism seems to preferentially involve hydroxylation at the 4- or 3-, 4- positions (Safe, 1980). Hydroxylation may be directly through hydroxylating enzyme systems, or through formation of arene oxide intermediates. This latter process is of particular concern because arene oxides are known to be reactive electrophilic agents that form reaction products with important cellular macromolecules such as RNA and DNA. The processes of mutagenesis and carcinogenesis are often associated with the in vivo formation of such reaction products. Van Miller et al. (1975) and Norback et al. (1976) postulated an arene oxide intermediate as a likely intermediate of PCB metabolism. The observation of trans-3,4-dihydro-3,4-dihydroxy-metabolites in monkeys (Norback et al., 1976; Hsu et al., 1975a,b) and rabbits (Gardner et al., 1973) was interpreted as strong evidence for arene oxide formation. Arene oxides characteristically hydrate in vivo to yield dihydrodials.

D.1.3 Overview of Health Effects Observed in Humans

Information of human health effects due to PCB exposures is available from studies of occupational populations and the general population. Many important parameters, such as route, duration, and magnitude of exposure and the demography of the exposed group, differ in these two classes of scientific studies. Occupational exposure occurs from both inhalation and dermal routes of exposure. Exposure to the general population is thought to be primarily from ingestion of contaminated foods, mainly fish.

Studies of occupational exposure to PCBs have shown varying degrees of dermatologic and hepatic effects. Possible liver cell damage has been demonstrated only in occupationally exposed groups with higher ranges of PCB levels (Kreiss, 1985). Workers with occupational exposure to PCBs (Table D-5) have also been evaluated for various subclinical effects, such as changes in serum levels and other parameters. Drill et al. (1981) concluded that serum levels of liver-associated enzymes appear to be the most sensitive indicators of PCB exposure in humans, and that changes in liver enzymes may occur at levels below those at which chloracne occur.

There is some evidence that PCBs cause cancer in humans based on studies of people exposed to PCBs in the workplace and on studies of people who accidentally consumed PCB-contaminated rice oil in Japan. Although the results of these studies are suggestive of a cause and effect relationship between PCB exposure and cancer, the EPA considers the data at this time to be inadequate to conclude that PCBs are human carcinogens.

Studies of the toxic effects of PCBs have also been conducted on the general population, particularly on groups who consumed contaminated fish. Exposure levels were assumed to be lower than in occupational settings. Kreiss et al. (1981) found a direct correlation between increasing serum PCB levels and elevated blood pressure in 458 volunteers 12 years of age from Triana, Alabama, who had consumed contaminated fish. Serum PCB levels were also positively correlated with consumption. Humphrey (1983) studied farm families exposed to PCBs from eating home-raised milk and beef from animals exposed to PCB-contaminated feed. Follow-up studies indicate that PCB circulating serum blood levels decrease only slowly or not at all.

Accidental human ingestion of PCB-contaminated rice oil in Japan and Taiwan resulted in effects similar to those seen following occupational exposure. The observed effects include acneform eruptions of the skin and abnormal hepatic function. (Kuratsune et al., 1969, 1972; Goto and Higuchi, 1969; Hirayama et al., 1974; Chang et al., 1980a,b; Chen et al., 1980, 1981). This syndrome was called Yusho. Outbreaks of sensory neuropathy were reported in both the Japanese and the Taiwanese populations (Chen et al., 1985; Murai and Kuroiwa, 1971). A negative correlation was noted between PCB blood levels and nerve cell functioning. Adverse developmental effects were also observed following the poisoning. Infants born to Japanese woman who had Yusho during pregnancy were generally small for their gestational age and exhibited several adverse effects, including abnormal brown pigmentation on the skin, eruption of teeth at birth, and facial edema. (Yamashita, 1977; Yamaguchi et al., 1971; Taki et al., 1969; Funatsu et al., 1971; Kikuchi et al., 1969).

The average total amount of PCBs consumed by each person was estimated to be ~2g, which was much higher than the estimated doses in studies of contaminated fish ingestion. The contaminated rice oil also contained polychlorinated dibenzofurans, which have health effects similar to PCBs. Thus, the specific role of PCBs in the causation of the above effects cannot be stated with certainty.

In a study of New Bedford residents, 1.3% of 840 participants had serum PCB levels < 30 ppb. The Center for Disease Control (CDC)

estimates that 99 percent of unexposed persons in the United States have PCB levels less than 30 ppb and 95 percent have levels below 20 ppb (MDPH, 1987). Mean serum PCB levels for persons without occupational exposure were usually between 4 and 8 ppb (ATSDR, 1987). For the New Bedford residents, those individuals who ate locally caught seafood tended to have higher PCB serum concentrations than individuals who did not consume local seafood; however, levels in both groups were generally low (i.e., >30 ppb). Blood pressure levels were not correlated with serum PCB levels in this study. (MDPH, 1987).

D.1.4 Toxicity

The toxicity of PCBs has been well-studied in animals, particularly chronic toxicity by the oral route. In addition, there are numerous studies of humans exposed to PCBs in an occupational setting or due to accidental ingestion of PCBs. This section discusses the toxicity of PCBs in humans and laboratory animals with respect to several effects: (1) lethality and decreased longevity, (2) systemic and target organ toxicity, (3) developmental toxicity, (4) reproductive toxicity, (5) genotoxicity, and (6) carcinogenicity. Toxicity information is discussed separately for the three routes of exposure that are relevant to exposures to PCBs at the New Bedford Harbor site.

D.1.4.1 Lethality and Decreased Longevity

PCBs have a low order of acute lethality. Tests of acute lethality of PCBs in laboratory animals, with the exception of the guinea pig, suggest that PCB products have similar toxicity regardless of species or age of the animal.

Inhalation Exposure--Pertinent data on the acute toxicity of PCBs were not located in the available literature for either humans or animals.

Oral Exposure--

HUMAN. Pertinent data were not located in the available literature.

ANIMAL. PCBs have a low order of acute lethality in most species tested. The single dose oral LD50 value in rats, rabbits, mice, and mink ranged from 0.5-20 g/kg body weight (Grant and Phillips, 1974; Bruckner et al., 1973; Kimbrough et al., 1978; Fishbein, 1974; Garthoff et al., 1982; Aulerich and Ringer, 1977). There is evidence that the guinea pig may be more sensitive to the acute toxicity of PCBs than other species (Miller, 1944; McConnell and Kinney, 1978). The LD50 in the latter study was

0.5 mg/kg body weight. No test result for monkeys, which have been shown to be more sensitive than rodents to other of PCBs' toxic effects, have been found in the available literature.

Dermal Exposure--

HUMAN. Pertinent data were not located in the available literature.

AMIMAL. Median lethal doses for single application of Aroclors to the skin of rabbits ranged from > 1269 mg/kg for Aroclors 1242 and 1248 in 50% corn oil to < 3169 mg/kg for undiluted Aroclor 1221 as reported by Nelson et al. (1972) and summarized by Fishbein (1974). LD50 values for other species have not been located in the available literature.

D.1.4.2 Systemic Target Organ Toxicity

Liver Effects--The liver appears to be the organ most sensitive to PCB exposure and is the organ that is most frequently affected by PCB exposure in toxicity studies. Enlargement of the liver is an effect that has been repeatedly observed in humans and laboratory animals. Alterations in blood serum levels of liver-associated enzymes have also commonly been observed after PCB exposure. Fat deposition and necrosis are also often observed. The most significant effect on the liver caused by PCB exposure is the induction of liver tumors and other proliferative lesions. This effect is discussed fully in Section E.6 - Carcinogenicity.

Inhalation Exposure--

HUMAN. Epidemiological studies and clinical surveys indicate that occupational exposure to Aroclors can produce alterations in liver enzymes that may be indicative of subclinical liver cell damage (Ouw et al., 1976; Alvares et al, 1977; Fishbein et al. 1979, 1985; Baker et al., 1980; Smith et al., 1981a,b,c; Brown and Jones, 1981; Fishbein, 1985; Emmett, 1985; Lawton et al., 1985; Drill et al., 1981; Kriess, 1985). The subjects of these studies were primarily involved in the manufacture and repair of electrical equipment (e.g., capacitors, transformers), and many had measurable and often high serum levels of PCBs.

The significance of the enzyme effects is unclear, because the effects were not associated with clinically detectable liver damage in most cases. However, significant correlations between plasma PCB levels and various serum parameters have been reported. Drill et al. (1981) concluded that levels of serum glutamic-oxaloacetic transaminase (SGOT) and serum glutamic-pyruvic transaminase (SGPT) appear to be the most sensitive indicators of PCB exposure in humans. In addition,

changes in serum levels of liver enzymes may occur at levels below those at which chloracne occur.

Clinically evident liver disease has also been observed in some occupational studies. Drinker (1937) described a fatal case from PCB exposure. The worker developed chloracne followed by jaundice. At autopsy, cirrhosis of the liver with superimposed acute yellow atrophy was found. Alvares et al. (1977) reported impaired liver function in the absence of overt toxic effects. Liver disease has also been shown to occur independently from chloracne (Kimbrough, 1972). Maroni et al. (1981) observed liver failure in several electrical workers exposed to PCBs.

The methodological limitations of all the above occupational studies should be noted. In no cases were airborne PCB exposure concentrations known with certainty. In addition, although the exposures are generally presumed to be by the inhalation route, there is a growing body of evidence that dermal exposures may contribute significantly to total PCB exposure in workplace settings.

No information was found in the available literature regarding the effects of PCB inhalation by members of the general population.

ANIMAL. Degeneration of liver tissues was observed in rats, mice, rabbits, cats, and guinea pigs that were exposed to Aroclor 1254 vapors (1.5 mg/m^3) for prolonged periods. The noted effects were reversible after the 213 day dosing was completed. Similar effects were not seen with Aroclor 1242 (Treon et al., 1956).

Oral Exposure--

HUMAN. Kriess et al. (1981) found a direct correlation between increasing serum PCB levels, selected serum enzyme parameters and elevated blood pressure in 458 volunteers 12 years of age from Triana, Alabama, who had consumed contaminated fish. Serum PCB levels were positively correlated with consumption. In a study of residents of Bloomington, Indiana, hepatic function indices were not adversely affected in individuals having PCBs in their serum (Baker et al., 1980).

Accidental human ingestion of PCB-contaminated rice oil in Japan and Taiwan resulted in effects similar to those seen following occupational exposure including abnormal hepatic function (Kuratsune et al., 1969, 1972; Goto and Higuchi, 1969; Hirayama et al., 1974; Chang et al., 1980a,b; Chen et al., 1980, 1981). The average total amount of PCBs consumed was estimated to be ~2g, much higher than the estimated doses in studies of contaminated fish ingestion. The contaminated rice oil also

contained polychlorinated dibenzofurans, which have similar health effects to PCBs, so the contribution of the PCBs alone to the observed liver toxicity cannot be determined.

ANIMAL. Numerous experimental studies in which PCBs were fed or administered by gavage to animals have demonstrated adverse effects on the liver. Increases in liver-to-body weight ratio appear to be one of the sensitive indicators of PCB exposure. Carter (1983) observed enlargement of the liver in rats ingesting diets containing as little as 20 ppm Aroclor 1254 (1 mg/kg bw/day) for 14 days.

PCBs have been widely used to induce the increased synthesis of hepatic enzymes, often in studies with other chemicals. Since large doses have been given to obtain a maximal response, few studies identify threshold oral doses. Chu et al. (1977) and Garthoff et al. (1977) reported that 5-50 mg Aroclor 1254/kg diet for 2 weeks resulted in the induction of many hepatic enzyme systems.

Fatty degeneration and other clinically evident pathohistological effects of the liver have also been observed in rats exposed to $\geq$ 20 ppm Aroclor 1254 or 1260 for 28 days (Chu et al., 1977), rats exposed to $\geq$ 20 ppm Aroclor 1254 or 1260 for 8 months (Kimbrough et al., 1972), and mice exposed to 37.5 ppm but not 3.75 ppm Aroclor 1254 for the same duration of 6 months (Koller, 1977).

Adverse effects in the liver have also been observed in monkeys fed PCBs in the diet. Barsotti et al. (1976) observed hepatic effects that included focal areas of necrosis, enlarged hepatocytes, and lipid droplets in two rhesus monkeys fed Aroclor 1248 at 2.5 or 5.0 ppm for greater than 173 days.

Although only one animal per dose was examined, these effects must be regarded as treatment-related because of the characteristic nature of the hepatic response. Also, similar effects on the liver were observed in an earlier study by Allen (1975) in which the animals received Aroclor 1248 in the diet at levels of 100 and 300 ppm for 2 or 3 months.

As described in Section E.6-Carcinogenicity, chronic dietary studies in rodents reveal that the formation of proliferative lesions of the liver constitute a major effect of oral PCB exposure (hyperplasia, nodule formation, benign tumor formation, and malignant tumor formation).

Dermal Exposure--

HUMAN. Pertinent data were not located in the available literature. It has been recently suggested, however, that occupational exposures to PCBs formerly attributed to the

inhalation of PCB-contaminated air may have been due in significant part to the dermal exposure route.

ANIMAL. Although fewer toxicological studies have been conducted by the dermal exposure route compared to the oral, similar effects are seen. In addition to the direct effect on the skin, dermal application of PCB mixtures to mice, guinea pigs, and rabbits causes systemic lesions of the liver, including atrophy and fatty infiltration (Von Wedel, 1942). For instance, numerous histologically apparent liver lesions were observed in rabbits given subchronic dermal doses of Aroclor 1260 and other similar mixtures. The PCB mixtures were given daily as an isopropanol solution at doses of 118 mg/day for 38 days (Vos and Beems, 1971) or 120 mg/day for 28 days (Vos and Notenboom-Ram, 1972). Adverse skin and kidney effects were also reported. The Aroclors in this experiment were reported to be free of polychlorinated dibenzofuran contamination.

Skin Effects--

Inhalation Exposure--

HUMAN. Adverse skin effects are among the health effects that were reported in workers shortly after the manufacture of PCBs began. Jones and Alden (1936) reported that 23 of 24 workers in a nonenclosed PCB manufacturing process developed chloracne. Chloracne is a skin condition characterized by the outbreak of numerous pimple-like eruptions and associated rashes. It has been repeatedly observed in chemical workers exposed to certain chlorinated solvents. Schwartz (1936) reported burning of the eyes, among other effects, in men working with PCBs.

Chloracne, skin rashes, burning eyes, and burning skin have been repeatedly reported in studies of individuals occupationally exposed to various PCB mixtures (Jones and Alden, 1936; Drinker et al., 1937; Schwartz, 1943; Meigs et al., 1954; Fishbein et al., 1979; Warshaw et al., 1979; Maroni et al., 1981; Ouw et al., 1976; Alvares et al., 1977; Zauder, 1979; ATSDR, 1988). These effects have been generally attributed by the authors to inhalation exposures because PCB contamination of the workplace air was reported. Because of inadequate monitoring data, however, inhalation exposure levels cannot be estimated with confidence. Furthermore, the contribution of direct dermal contact to PCBs in these workplace settings cannot be determined. It is unclear whether the skin effects observed in workers were due to inhalation exposure, direct contact exposure, or both.

ANIMAL. Pertinent data were not located in the available literature.

Oral Exposure--

HUMAN. No information was located in the available literature regarding the effects on the skin and cutaneous tissues due to oral exposure to pure PCBs. Although skin disorders have been reported in Japanese and Taiwanese patients who accidentally consumed PCB-contaminated rice oil, the potent toxicants polychlorinated dibenzofurans (PCDFs) were also consumed. Thus, the effects cannot be solely attributed to the PCBs themselves. The levels of contaminating PCDFs in these PCB mixtures greatly exceeded the levels observed in the PCB mixtures manufactured and used in the United States.

ANIMAL. Skin effects following oral exposure to PCBs have been well-characterized in rats, mice, and monkeys. Therefore, direct contact of the skin with PCBs is not necessary for the development of adverse effects to skin tissues. Rats exposed to Aroclor 1254 in the diet (50 ppm) developed hair loss, facial edema, and exophthalamos (protrusion of the eyeballs) after 72 weeks. These effects did not occur after 104 weeks of exposure to dietary levels of 25 ppm (NCI, 1978). Skin effects were also observed in mice exposed to Aroclor 1254 in the diet (200 ppm) for 23 weeks (Bill, 1983).

Monkeys exhibit the same signs of PCB-induced toxicity as other species, including humans. Skin lesions, such as edema of the face and eyelids are an early sign of toxicity induced by Aroclors (Allen et al., 1974a; Allen, 1975). These signs progress to facial hair loss, the development of chloracne, and generalized edema. The monkey is highly sensitive to the toxic effects of PCBs in chronic bioassays. The most common observations in monkeys exposed to Aroclor 1248 in the diet for a period of 8 to 39 months were skin lesions similar to the chloracne observed in humans (Barsotti and Allen, 1975; Allen and Barsotti, 1976; Allen et al., 1980; Becker et al., 1979). These effects were observed at the lowest doses tested, ranging from 2.5 to 3 ppm in the diet (0.095 - 0.126 mg/kg bw/day).

Dermal Exposure--

HUMAN. Pertinent data were not located in the available literature. As discussed in the section on occupational exposure, however, adverse skin effects observed in occupationally exposed people may be due in part to direct contact exposure in addition to inhalation exposure.

ANIMAL. Skin effects have been observed in laboratory animals dosed with PCBs dermally. When given 118 mg/day of Aroclor 1260 and similar mixtures (free of PCDF) for 28-38 days, rabbits were observed to develop skin irritation, acneform lesions, and skin

thickening (Vos and Beems, 1971; Vos and Notenboom-Ram 1972). The observed skin effects are similar to those observed in other species when dosed orally with PCB mixtures.

Immunological Effects--PCBs have been shown to have immosuppressive effects in several laboratory animals. The effects of PCB exposure on the normal functioning of the immune system has not, however, been adequately evaluated.

Inhalation Exposure--

HUMAN. Pertinent data were not located in the available literature.

ANIMAL. Pertinent data were not located in the available literature.

Oral Exposure--

HUMAN. Pertinent data were not located in the available literature.

ANIMAL. PCBs, when ingested, have been shown to cause immunosuppression as measured by functional impairment of immunological responsiveness, decreases in normal immune parameters, and histological evidence of immune tissue damage. Adverse effects have been observed in rats, mice, guinea pigs, and rhesus monkeys.

PCBs have been shown to cause splenic, thymic and lymph node atrophy in rats (Allen et al., 1975; Allen and Abrahamson, 1973; Parkinson et al., 1983). The results of alterations in these organs have been pronounced immunosuppression (Allen et al., 1975).

Mice have been used as a model to demonstrate the suppressive effects of PCBs on the immune system. Increased mortality following exposure in the diet (167 ppm of Aroclor 1016 or 1242 for 6 weeks) was observed in mice injected with a bacterial endotoxin or live malarial organisms (Loose et al., 1978a,b). Loose et al. (1978 a,b) concluded that depressed immune function resulted before histologically discernible damage had occurred to thymus, spleen or lymph mode and was therefore a sensitive indicator of PCB toxicity. Thomas and Hinsdill (1978) observed an enhanced sensitivity to bacterial infection in mice fed Aroclor 1248 at 100 ppm in the diet for 3 to 5 weeks.

Immunosuppression has also been observed in guinea pigs that were fed 50 ppm Aroclor 1260 and related PCB mixtures for six weeks. Treated animals had significantly lowered tetanus autotoxin levels, lowered levels of circulating immune cells (leukocytes

and lymphocytes), and atrophied thymuses (Vos and Van Gendersen, 1973). Splenic atrophy was observed in guinea pigs exposed to 10 ppm of dietary Aroclor 1260 for eight weeks (Vos and de Roij, 1972).

Evidence of immunosuppression has also been reported in rhesus monkeys fed 2.5 or 5.0 ppm of Aroclor 1248 in the diet for 7 months. Monkeys developed shigellosis during after treatment, indicating an increased susceptibility to infection.

Dermal Exposure--

HUMAN. Pertinent data were not located in the available literature.

ANIMAL. In rabbits, adverse effects on the immune system have been reported following 20-27 dermal applications of Aroclor 1260 (free of PCDF contamination) at 118 mg/day (Vos and Notenboom-Ram, 1972; Vos and Beems, 1971). Atrophy of the thymus and histopathological changes in spleen and lymph nodes were the reported effects.

Thyroid Effects--The effects of PCB exposures on thyroid functioning have been investigated only in animals exposed orally. The thyroid is a target organ for PCB toxicity in rats. The observed effects of PCB exposure on thyroxin secretion may explain some of the well-characterized effects of PCBs on reproduction, growth, and development.

Oral Exposure--

HUMAN. Pertinent data were not located in the available literature.

ANIMAL. Rats exposed to low levels of PCBs in the diet for several months have developed adverse thyroid effects that progress from enlargement to degeneration. Reduced serum thyroxin levels accompanied the tissue changes (Collins, et al. 1977; Collins and Capen, 1980 a,b,c; Kasza et al., 1978). The morphological effects have been observed in rats fed 5 ppm of Aroclor 1254 (0.25 mg/kg bw/day) for 4 weeks (Collins and Capan, 1980b) and rats fed 5 ppm of PCBs for 5 weeks (Kasza, et al., 1978).

Stomach Effects--Effects of PCB exposure on the stomach have only been studied in animals exposed orally.

Oral Exposure --

HUMAN. Pertinent data were not located in the available literature.

ANIMAL. Adverse effects of PCBs on the stomach have been observed in monkeys dosed with Aroclor 1242 or 1248 either by gavage or in the diet (Allen and Norback, 1973; Allen et al., 1974a,b; Allen, 1975; Barsotti and Allen, 1975, and Becker et al., 1979). Gastritis that progressed to hypertrophy and hyperplasia of the gastric mucosa was observed at doses as low as a single gavage dose of Aroclor 1248 (1.5 g/kg) (Allen et al., 1974a); 25 ppm of Aroclor 1248 in the diet for 1 year (Barsotti and Allen, 1975) and 3 ppm of Aroclor 1242 in the diet for 71 days (Becker et al., 1979).

D.1.4.3 Developmental Toxicity

Although there is evidence that PCBs have caused teratogenic effects in mice following oral exposure during gestation, there is no evidence of such effects in numerous studies with rats, rabbits, and monkeys. Furthermore, considerable maternal toxicity was observed in mice at dose levels causing structural abnormalities in the newborn animals. Fetal toxicity, on the other hand, has been observed in several species tested. In humans, there are reports of reduced birth weight and gestational age in infants of mothers who had occupational or environmental exposure to Aroclors. These studies do not conclusively demonstrate that PCBs cause fetal toxicity in humans because the role of confounding exposures to other toxic contaminants cannot be defined. However, these effects observed in humans are consistent with the animal data.

Inhalation Exposure --

HUMAN. Taylor et al. (1984) examined pregnancy outcome in female capacitor workers who had a history of high exposures to Aroclors 1016, 1242, and/or 1254. Compared to infants born to women who worked in low exposure areas, the study infants had lower birth weights and lower gestational ages. The differences between groups were small, however, and the decreased birth weight could be explained by the reduction in gestation period.

ANIMAL. Pertinent data were not located in the available literature.

Oral Exposure --

HUMAN. A study of 242 newborn infants whose mothers consumed moderate quantities of PCB-contaminated lake fish showed lower birth weight and smaller head circumference compared to 71 infants whose mothers did not eat contaminated fish (Fein et al., 1984). These effects could not be attributed to confounding variables such as socioeconomic status, maternal age, smoking during pregnancy and exposure to polybrominated biphenyls

(PBBs). Both maternal consumption of fish and levels of PCBs in umbilical cord serum were positively correlated with lower birth weight, smaller head size, and shorter gestation. In another study with 930 children, Rogan et al. (1986) found no correlation between birth weight or head circumference with PCB levels in placenta, maternal serum or cord serum.

Several investigators have also reported behavioral changes in children of women exposed to PCBs. In a study of the same infants whose mothers consumed contaminated fish as reported by Fein et al. (1984), Jacobson et al (1984b) reported that contaminated fish consumption was positively correlated with impaired autonomic maturity, increased numbers of abnormal reflexes, and a decreased range of neurological responsiveness. Rogan et al. (1986) found that the levels at birth of PCBs in milk fat of 3.5 to > 4 ppm were significantly correlated with decreased muscle tone, decreased activity levels, and abnormal reflexes in the infants. Jacobson et al. (1985) also found that intrauterine exposure to PCBs, as determined by fish consumption rates and PCB levels in cord serum, were significantly correlated with poor visual recognition memory in the infants.

Infants born to Japanese women who exhibited symptoms of acute PCB toxicity during pregnancy were generally small for their gestational age and had birth defects including dark brown pigmentation on the skin, eruption of teeth at birth, skeletal malformations of the head, facial edema and exophthalmic eyes (Yamashita, 1977; Yamaguchi et al., 1971; Taki et al., 1969; Funatsu et al., 1971; Kikuchi et al., 1969). As has been discussed in Section E.3.a, the PCBs to which the above women were exposed were contaminated with PCDFs. Therefore, the role of PCB compounds, if any, cannot be determined.

ANIMAL. PCBs have been documented as being fetotoxic in rats, mice, rabbits, and monkeys. There is some evidence that PCBs can cause teratogenic effects, but this has been reported only in mice and at doses that also caused significant maternal toxicity. In rats, rabbits, and monkeys, Aroclors did not cause structural malformations when administered via the oral route during critical periods of organogensis even at doses that caused fetotoxicity and/or maternal toxicity.

PCBs have been shown to cause fetal toxicity. In monkeys fed PCDF-free Aroclor 1016 in the diet (1 ppm) prior to mating and during pregnancy, Barsotti and Van Miller (1984) observed significantly reduced mean birth weight compared to controls.

In another study, Allen et al. (1979) gave Aroclor 1248 (0.5 or 1.0 ppm) to monkeys in their diet before and during pregnancy. This exposure produced fetal loss, reduced infant birth weights and areas of hyperpigmentation of infant skin.

Rabbits and rats have also been shown to be sensitive to the fetotoxic effects of PCBs. In two experiments increased abortions, stillbirths and accompanying maternal deaths were observed in rabbits given > 12.5 mg/kg/day of Aroclor 1254 orally during pregnancy (Villenewe et al., 1971). On rats, 100 mg/kg/day exposure to Aroclor 1254 during pregnancy caused reduced average pup weights and reduced pup survival (Villenewe et al., 1971; Spencer, 1982). Collins and Capen (1980) also observed a reduced litter size from rats fed 500 ppm of Aroclor 1254 on the diet during pregnancy and lactation. In addition, lesions in the thyroid tissues and reduction in serum levels of thyroid hormones were observed in neonatal and weaning rats whose mothers received 2.5 and 25 mg/kg/day.

In mice treated orally with PCB mixtures, structural abnormalities have been reported in addition to fetotoxicity. Marks et al. (1981) orally administered 0.1 to 16.0 mg/kg bw/day of a specific hexachlorinated PCB isomer to mice from day 6 to day 15 of gestation. Fetotoxicity in the 8 and 16 mg/kg/day dose groups was demonstrated as measured by a decrease in the number of live fetuses per dam, a decrease in the number of implants, and an increase in the number of fetal resorptions. Teratogenic effects were also discovered. A significant dose-related increase in the incidence of cleft palate occurred in groups of mice dosed at or above 2 mg/kg/day. Other evidence of teratogenic effects was reported by Watanabe and Suggahara (1981). In both cases considerable maternal toxicity was observed at the dose levels causing teratogenic effects.

Other investigators have observed early abortions, resorptions, still births and/or reduced birth weight, as well as lengthened menstrual cycles and lowered serum progesterone levels in monkeys exposed to 2.5 or 5.0 ppm Aroclor 1248 in the diet (Allen and Barsotti, 1976; Allen et al., 1979, 1980). The purity of the PCB mixtures was not reported in the later experiments.

Dermal Exposure--

Pertinent data were not located in the available literature.

D.1.4.4 Reproductive Effects

In addition to the adverse effects on the developing fetus, PCBs have been shown to adversely interfere with the normal reproductive systems of the animals tested.

Inhalation Exposure--

Pertinent data were not found in the available literature.

Oral Exposure --

HUMAN. Pertinent data were not found in the available literature.

ANIMAL. PCBs have been demonstrated to cause adverse reproductive effects in mice, rats, minks, and monkeys. A series of reproductive effects has been reported including disruption of the estrus cycle, decreased sexual receptivity, infertility, and fetal loss.

Monkeys were exposed to Aroclor 1248 in the diet for 18 months at dose levels of 2.5 and 5.0 ppm. In addition to alterations in menstrual cycles, there was an almost total inhibition of reproduction at the high dose level. At the low dose level there were early abortions and fetal resorptions at a significantly higher rate than observed in controls. The contribution to the observed toxicity caused by contaminating PCDFs could not be determined (Allen et al., 1979, 1980; Barsotti et al., 1976).

Mink appear to be particularly susceptible to PCBs. Levels of 5 ppm Aroclor 1242 in diet completely halted reproduction, and levels of 20 ppm of Aroclor 1016 significantly reduced reproduction (Bleavins et al., 1980). Reduced reproductive efficiency was also observed by Aulerich and Ringer (1977) in minks fed 5 ppm of Aroclor 1254 in the diet for 4 months prior to mating. Total inhibition of reproduction occurred in the animals fed 15 ppm of the PCBs.

Rats appear to be less sensitive to the effects of PCBs on reproduction. Complete losses of fertility were seen in male and female Wister rats caged together for 9 weeks while ingesting 6.4 mg/kg bw/day of Aroclor 1254 in their drinking water (Baker et al., 1977). Males regained normal fertility after removal from treatment for 2 weeks.

When Aroclor 1254 was administered to lactating Holtzman rats at 32 mg/kg bw/day on days 3, 5 and 7 of lactation, the future mating capability of nursing male pups was adversely affected (Sager, 1983). Testicular hyperplasia and subnormal development of accessory sex glands suggested that PCB exposure interfered with circulating levels of androgens.

Sprague-Dawley rats were infertile after receiving 15 mg/kg bw/day on days 6 to 15 of gestation (Spencer, 1982). Adverse reproductive effects due to oral exposure to PCBs prior to or during gestation have also been observed in rats (Brezner et al., 1984), mice (Welsch, 1985; Orberg and Kowstrom, 1973), and rats (Linder et al., 1976).

Dermal Exposure--

Pertinent data were not found in the available literature.

D.1.4.5 Genotoxic Effects

HUMAN. Pertinent data were not located in the available literature.

NONHUMAN. PCBs have not been shown to be strongly genotoxic in any test system. In short-term *in vitro* assays for gene mutations in bacterial cells and mammalian cells in culture and in assays for chromosomal aberrations in cultured mammalian cells, results have been generally negative (ATSDR, 1987). Only one investigation, Wyndham et al. (1976), reported positive results. In this study, Aroclor 1221 was reported to be mutagenic to bacteria. This result has not been given a high degree of confidence, however, because the authors have failed to reproduce their findings.

In tests for chromosomal aberrations and heritable genetic mutations with live animals, results have also been generally negative. Negative results were obtained with fruit flies, chickens, mice and rats. A weakly positive result in a chromosomal aberration assay in ring dove embryos fed Aroclor 1254 at 10 ppm in the diet suggested to the investigators that PCBs might cause chromosomal breakage (Peakall et al., 1972).

D.1.4.6 Carcinogenic Effects

PCBS have been demonstrated to cause cancer in three strains of rats and two strains of mice. The EPA has thus classified PCBs as probable human carcinogens. There is some evidence that PCBs cause cancer in humans based on studies of people exposed to PCBs in the workplace and on studies of people who accidentally consumed PCB-contaminated rice oil in Japan. Although the results of these studies are suggestive of a cause and effect relationship between PCB exposure and cancer, the EPA considers the inhalation exposure data to be inadequate to conclude that PCBs are known human carcinogens at this time.

HUMAN. The results of a mortality study on populations occupationally exposed to PCBs are suggestive that PCBs are carcinogens in humans (Brown and Jones, 1981; Brown, 1987). Brown and Jones (1981) reported a retrospective cohort mortality study on 2566 workers who had worked at least 3 months at one of two capacitor manufacturing plants. Over 50% of the population had exposure for only 2 years or less. Exposure levels were 24-393 mg/m^3 at the first plant and 318-1260 mg/m^3 at the second (NIOSH, 1977). Deaths from liver cancer, cirrhosis of the liver and rectal cancer were slightly higher than expected, but the results were not statistically significant.

The increased rate of cancer of the rectum was statistically significant only among females at one of the plants. The results of this study cannot be interpreted as providing evidence that PCBs do not cause cancer in humans, however, because the statistical power of the study was limited due to the short follow-up period (only 15 years). After an additional 7 years of follow-up, a statistically significant excess risk of liver and biliary cancer was reported, with four of five liver cancers in females of plant 2, the plant in New Bedford, Massachusetts (Brown, 1987). As evidence to support a causal relationship, the authors point out that personal inhalation exposures to PCBs were higher at plant 2 than at plant 1. All of the liver cancers occurred after the fifteenth year of follow-up from the beginning date of employment. All of the women contracting liver cancer also began working at a time when levels of exposure were likely to be highest. The results of this follow-up study had not yet been reported when EPA classified PCBs as a probable human carcinogen (Classification B2).

The results of other epidemiological studies neither support nor refute the above results because of various methodological difficulties. Bahn et al. (1976, 1977) reported a significantly increased incidence of malignant melanoma and pancreatic cancer in petrochemical workers exposed to PCBs and other unidentified chemicals. Quantitative PCB exposure data were not reported; thus, the role, if any, of PCBs cannot be determined from this study.

Bertazzi et al. (1981) reviewed mortality of 290 males and 1020 females who had worked for 6 months or more in capacitor production. Males had a statistically significant increased number of deaths from all neoplasms compared to controls. Neoplasms of the digestive system, the peritoneum, and the lymphatic and hematopoietic tissues were also elevated compared to controls. Among females, all causes of death were significantly elevated. Because of uncontrolled confounding exposures, the role of PCBs cannot be demonstrated by this study. Gustavsson et al. (1986) performed a cohort study of 142 male Swedish capacitor manufacturing workers who had been exposed to PCBs for an average of 6.5 years. Although no excess mortality or cancer incidence compared to national statistics was observed among PCB workers, a carcinogenic effect of PCBs could not be ruled out because of the small cohort and the relatively short follow-up period.

ANIMAL. No pertinent data were located in the available literature.

Oral Exposure --

HUMAN. After a follow-up of over 16 years, a statistically significant increase in liver cancer incidence has been reported in Yusho patients who ingested PCB-contaminated rice oil. The findings are considered to be tentative, however, because the excess of liver cancer occurred in only one geographical area. In addition, the PCBs were known to have been contaminated with PCDFs, which very possibly contributed to the causation of liver cancer. Although the findings are suggestive of a causal relation between oral exposure to high levels of PCBs and excess liver cancer risk, the EPA considers the present data inadequate to conclude that PCBs are known human carcinogens (EPA, 1988a).

ANIMAL. Liver cancer has been reported in three strains of rats and two strains of mice. Norback and Weltman (1985) fed Aroclor 1260 at 100 ppm to 70 male and 70 female Sprague-Dawley rats for 16 months, followed by a 50 ppm diet for an additional 8 months and then by a control diet for 5 months. Female rats had a 95% incidence of hepatocellular carcinomas, with males exhibiting only a 4% incidence. The tumors, although malignant, were relatively unaggressive. PCB treatment did not affect mortality, probably because of the late appearance and slow growth of the tumors. Concurrent morphologic studies showed the sequential progression of liver lesions from cell alteration to neoplastic modules to hepatocellular carcinomas. EPA (1988a) used this study as the basis for its cancer potency value for PCBs using combined incidences of neoplastic nodules and hepatocellar carcinomas.

Malignant liver cancers were also observed in Sherman rats. Exposure of 200 female Sherman rats to 100 ppm Aroclor 1260 in the diet for 630 days resulted in 144 of 184 surviving animals having neoplastic nodules of the liver (Kimbrough et al., 1975). Hepatocellular carcinomas were found in 26 animals compared to only one hepatocellular carcinoma in the 173 control group survivors. The National Cancer Institute (NCI, 1978) reported a similar result in 24 male and 24 female Fischer 344 rats treated with Aroclor 1254 at 25, 50 and 100 ppm for 104-105 weeks. A dose-related increased incidence of nodular hyperplasia in both sexes was reported, as well as three cases of hepatocellular carcinomas among treated males. Morgan et al. (1981) found gastric adenocarcinomas in 6 of the 144 above rats that were exposed to 100 g Aroclor 1254/kg diet for 104-105 weeks.

Other studies support the conclusion that PCBs are animal carcinogens. Carcinogenicity has been reported in animals exposed to furan-free PCB mixtures. Schaeffer et al. (1984) fed male Wistar rats reportedly furan-free diets containing 100 ppm Clophen A 30 (30% chlorine by weight) or Clophen A 60 (60% chlorine by weight) for 800 days. Hepatocellular carcinomas

developed in 61% of the rats fed Clophen A 60. In the Clophen A 30 treated rats, 3% developed hepatocellular carcinomas, while 89% had preneoplastic (pretumorous) lesions. This result strongly suggests that the observed carcinogenic effects of PCBs are not due to contaminating PCDFs.

Mice have also been shown to be sensitive to PCBs' carcinogenic effects. Ito et al. (1973) reported malignant liver tumors in 5 of 12 male mice exposed to 500 ppm Kanechlor-500 for 32 weeks. Kimbrough and Linder (1975) administered Aroclor 1254 in diet to 50 male BALB/CJ mice for 11 months at 330 ppm followed by a 5-month recovery period. Benign liver tumors were observed to 40.1% of the animals.

Dermal Exposure --

HUMAN. The occupational exposures attributed to the inhalation route may have been due, in part, to direct dermal contact with PCBs.

ANIMAL. Because PCBs have not been conclusively shown to be mutagenic in numerous studies, many researchers have postulated that they are tumor promoters and not initiators. At this time there is no experimental support for this hypothesis. Aroclor 1254 was reported to be a weak tumor initiator (DiGiovanni et al., 1978) when tested in the two-stage mouse skin system with a phorbol ester (TPA) as a promoting agent. The purity of the Aroclor was not reported, however. Thus the role of PCBs as tumor initiators cannot be clearly demonstrated by this experiment. When tested as a tumor promoter in mouse skin, Aroclor 1254 proved negative. Berry et al. (1978) initiated mouse skin with the potent initiator dimethylbenzanthracene and repeatedly applied the PCB mixture to the affected skin. Negative results were obtained. In this same experiment Aroclor 1254 did not produce skin tumors when applied (1 mg) twice weekly for the duration of the experiment.

D.1.5 Interactive Effects

Many of the interactive effects between PCBs and other compounds are thought to be due to the capability of PCBs to induce the synthesis of oxidative enzymes in the liver. Oxidative metabolism of drugs or other foreign compounds sometimes leads to detoxification of the compound and other times leads to toxification. The effects of metabolizing enzymes, such as the cytochromes P-450, are thus compound-specific. Because of this, the interactive effects of co-exposure to PCBs are also compound-specific. The following effects have been reported.

PCB pretreatment of rats resulted in increased rate of metabolism and excretion of pentobarbital, and in reduced

pentobarbital-induced sleeping times (Chu et al., 1977; Villeneuve et al., 1972). Hepatotoxicity induced by vinylidene flouride (Connolly et al., 1979) and halothane anesthesia (Sipes et al., 1978) was increased in rats by pretreatment with PCBs. In addition, the mutagenicity of benzo(a)pyrene was reported to be increased by simultaneous exposure to PCBs (Hutton et al., 1979).

As would be expected, PCBs have had both stimulatory and antagonistic effects on tumor development caused by exposures to other chemical compounds. For instance, Aroclor 1254 pretreatment protected mice from the development of lung tumors, but increased the number of mice with liver tumors 18 months after administration of N-nitrosodimethylamine (Anderson et al. 1983). Makiur et al. (1974) reported that Kaneclor 500 inhibited the liver carcinogenic effects caused by 3'-methyl-4-dimethylaminoazobenzene, N-2-fluoreylacetamide, and N-nitrosodiethylamine when administered orally to rats. On the other hand, Nagasaki et al (1975) found that Kanechlor 400 and 500 enhanced the effects of 2-benzene hexachloride in causing liver tumors in mice. Because of the differing effects that oxidative metabolism has on the metabolic disposition of different chemicals, there are no readily apparent trends in the effects that PCBs may have on the toxicity of other chemicals to which exposure occurs.

D.1.6 High Risk Subpopulations

High risk subpopulations include occupationally-exposed workers, pregnant women and fetuses, and nursing infants. Fetuses and neonates lack the hepatic microsomal enzyme systems that facilitate detoxification and excretion of PCBs (Calabrese and Sorenson, 1977; Gillette, 1967; Nyhan, 1961). Breast-fed infants have additional risk caused by a steroid excreted in human breast milk, but not cow's milk, that inhibits glucuronyl transferase activity and thus glucuronidation of PCBs (Calabrese and Sorenson, 1977; Gartner and Arias, 1966). Conjugation of hydroxylated PCB metabolites with glucuronic acid results in metabolites that are much more water soluble than PCBs themselves. This metabolic pathway is known to enhance excretion of PCBs.

Other high risk subpopulations include individuals unable to effect glucuronide formation because of enzyme deficiencies, such as persons with Gilbert's syndrome or Crigler and Najjar syndrome; children simultaneously exposed to the antibiotic novobiocin, which inhibits the glucuronidation enzymes; and persons with hepatic infections, such as infectious hepatitis (Calabrese and Sorenson, 1977; Gillette, 1967; Nyhan, 1961; Gartner and Arias, 1966; Lokeitz et al., 1963; Lester and Shmid, 1964).

D.1.7 Summary

PCBs or polychlorinated biphenyls are inert chemicals which are relatively resistant to degradation and hence accumulate in the environment. They are well absorbed by all routes of exposures (oral, dermal and inhalation) regardless of degree of chlorination. Inhalation and dermal exposure are the routes of occupational exposure. The general population is exposed to PCBs primarily by the oral route through food, particularly fish.

Metabolism is similar among mammalian species, and the degree of chlorination profoundly affects the rate and extent of metabolism. The more highly chlorinated PCBs are resistant to degradation and tend to accumulate in fat. PCBs also tend to accumulate in the gonads, adrenals and kidneys. The biological half-life of PCBs in humans is relatively long, and humans appear to retain PCBs in their tissues more effectively than any other mammal. Blood PCB levels are the best indicator of recent exposure to PCBs, and levels in fat are the best indicators of long-term exposure. PCBs cross the placenta and are excreted in milk fat.

PCBs show similar health effects regardless of route of exposure. PCBs appear to have a low order of acute lethality, and tests of acute lethality suggest that PCB products have similar toxicity regardless of route of administration, species, or age of the animal. The signs of chronic toxicity from PCBs closely resemble the signs of acute toxicity. Liver and cutaneous (skin) tissue are the major target organs for PCBs. Thyroid alterations, systemic lesions of the kidney, immunosuppressive effects, porphyria and changes in blood and urine chemistries have all been reported in animals. Reproductive effects including loss of fertility, increased resorptions (miscarriages), and decreased litter size have been reported in animals.

In humans, studies of occupationally exposed populations have shown varying degrees of dermatologic and hepatic effects. A statistically significant excess risk of liver and biliary cancer has also been reported among women employed at a PCB capacitor manufacturing plant. Serum PCB levels are positively correlated with consumption of contaminated fish in the general population. High serum PCB levels among pregnant women have been associated with increased abortions, premature deliveries, and decreased birth weight. Behavioral changes and a reduction of the length of gestation period have been associated with higher PCB intake. These studies are not conclusive because of various methodological problems, but the results are consistent with the results of laboratory animal studies. More serious health effects, including neurological effects, have been observed in

humans who accidentally consumed rice oil contaminated with PCBs and polychlorinated dibenzodifurans.

The EPA has classified PCBs as a probable human carcinogen (B2 classification) based on the induction of malignant liver tumors in five studies of rodents and suggestive evidence of excess risk of liver cancer in humans by ingestion and inhalation and/or dermal contact.

D.2 Cadmium Toxicity Profile

The purpose of this section is to describe the toxic properties of cadmium, particularly with respect to risk to public health from contamination at New Bedford Harbor.

Cadmium is a transitional metal element which occurs in lead or zinc ores. Trace amounts of cadmium are widely distributed naturally in the environment. Among the sedimentary rocks, the carbonaceous shales, formed under reducing conditions, contain the most cadmium. The most often-quoted average concentration of cadmium in the earth's crust is 0.15-0.20 ppm (Fleischer et al, 1974). In noncontaminated, noncultivated soils, cadmium concentration is largely governed by the amount of cadmium in the parent material. Based on the cadmium concentrations reported for common rocks, one can expect that, on the average, soils derived from igneous rocks would contain the lowest cadmium (<0.1-0.3 ppm), soils derived from metamorphic rocks would be intermediate (0.1-1.0 ppm), and soils derived from sedimentary rocks would contain the largest amount of cadmium (0.3-11 ppm) (Page and Bingham, 1973). Cadmium also enters the environment as the result of manufacture, use and disposal of cadmium-containing products.

D.2.1 Pharmacokinetics

Cadmium is absorbed by ingestion, by inhalation, and, to a very limited degree, by the dermal route of exposure. For the gastrointestinal route of exposure, about 5-6% of the cadmium ingested is absorbed in humans (Rahola et al., 1973; McLellan et al., 1978). This may be altered by a variety of dietary parameters (Bremner, 1974). Washko and Cousins (1976) found that diets low in calcium result in a significant increase in cadmium absorption. Diets deficient in vitamin D, protein, zinc, iron, and copper also increase the extent of cadmium absorption (Worker and Migicovsky, 1961; Suzuki et al., 1963).

Absorption of cadmium from the respiratory tract appears to be much more extensive than absorption from the gastrointestinal tract. For inhalation, absorption depends on particle size and solubility. Absorption of cadmium particles deposited in the respiratory tract of humans is estimated to be 20-25% of the deposited fraction.

Minimal dermal absorption has been demonstrated in animals (Skog and Wahlberg, 1964; Wahlberg, 1965; Kimura and Otaki, 1972). When 300-500 g of radiolabelled cadmium chloride in aqueous solution was applied to the back skin of guinea pigs of both sexes, a maximum of 1.8% was absorbed in 5 hours (Skog and Wahlberg, 1964).

About half of the cadmium distributed throughout the organs is found in the kidney and liver, with the kidney accounting for 30% of the total body burden (Frieberg et al., 1974; Nordberg, 1972). Other organs that accumulate cadmium include the testes, lungs, pancreas, spleen, and various endocrine organs.

D.2.2 Toxicity in Animals

Adverse health effects in animals exposed to high levels of cadmium include renal damage, testicular necrosis (cell death), pulmonary tract damage, endocrine system disruption, hypertension (high blood pressure), anemia, heptatotoxicity and reduced immunological responses. These effects have been most consistently demonstrated to occur after single intraperitoneal or subcutaneous injection of various cadmium salts in solution and at dose levels exceeding 0.5 mg cadmium/kg body weight.

The major effects of long-term exposure to cadmium are similar following both oral and inhalation exposure. The most typical and severe effect of chronic low level cadmium exposure is renal dysfunction. Signs of renal damage have been observed in rats and rabbits with prolonged oral exposure to cadmium over periods of weeks and months, starting at exposure levels of 2 ppm or 10 ppm in drinking water. Aughey et al. (1984) reported that treatment of male Wistar rats for only 24 weeks with 50 mg/L cadmium in drinking water produced clear signs of kidney inflammation.

Significant immune suppression effects have also been observed with oral exposures of mice and rabbits to cadmium chloride in drinking water at levels of 10 ppm for periods of 5-10 weeks. Such immune suppression effects have been observed in the absence of other more classic signs of cadmium toxicity (e.g., renal damage) and may represent unrecognized "subclinical" effects of chronic cadmium exposure.

Reproductive effects have been observed in animals with long-term exposure to cadmium. Significant reductions in birth weights have been reported for offspring born to female rats exposed via inhalation to 3 mg/m^3 cadmium sulfate for 7 months or to 200 ppm cadmium chloride in the diet. The reduction in birth weights appear to result from cadmium effects on maternal and fetal nutritional status and likely occur secondarily to reductions in fetal copper, iron, and zinc levels.

D.2.3 Human Health Effects

Studies of health effects in humans from cadmium exposure include studies of workers occupationally exposed and studies of general populations in areas of high cadmium pollution. Acute effects of cadmium exposure generally result from occupational exposure via inhalation exposure. The chief effect is pulmonary congestion and edema.

The major effect of long-term exposure to cadmium in humans is renal dysfunction. This results in disturbances in mineral metabolism and eventually in formation of kidney stones or osteomalacia (softening of the bones). Pulmonary dysfunction is observed following inhalation exposure of greater magnitude. Cadmium exposure has also been correlated with hypertension in humans.

Renal effects from cadmium exposure are well documented. The prevalence of proteinuria, generally the first symptom to occur, increases with the duration of cadmium exposure. Once renal dysfunction has proceeded to the point of pronounced proteinuria, it is essentially irreversible.

Disturbances in kidney function also exert their effects on bone and mineral metabolism, via effects on calcium and phosphorus metabolism. Extreme examples of this are osteoporosis (loss of bone tissue) and osteomalacia (softening of the bones). An additional secondary effect of renal dysfunction is the formation of kidney stones. Iron-deficiency anemia has also been noted in humans under conditions of occupational exposure to cadmium.

Findings in humans of adverse developmental effects are consistent with animal studies which show reductions in birth weight from oral exposure of pregnant animals. Cadmium has not, however, been reported to cause birth defects in humans.

D.2.4 Carcinogenicity and Mutagenicity

Cadmium compounds have been shown to be carcinogenic in rats and mice by two routes of exposure. Exposure of Wistar rats to cadmium as cadmium chloride at concentrations of 12.5, 25. and 50 ug/m^3 for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors (Takenaka et al., 1983). Intratracheal instillation of cadmium oxide caused mammary tumors in female Fischer 344 rats and tumors at multiple sites in males, although lung tumors were not produced (Sanders and Mahaffey, 1984). Injection site tumors and distant site tumors (e.g., testicular) have been reported by a number of authors as a result of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, oxide, and

sulfide compounds of cadmium to rats and mice (EPA, 1985b). The results of these studies have limited relevance, however, to assessing the risks to human health posed by oral or dermal exposure to cadmium compounds. Seven studies in rats and mice where cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of a carcinogenic response.

Results of mutagenicity tests in bacteria and yeast have been inconclusive. Positive responses have been obtained in mutation assays in Chinese hamster cells and in mouse lymphoma cells (Casto, 1976; Ochi and Ohsawa, 1983, Oberly et al., 1982). Conflicting results have been obtained in assays of chromosomal aberrations in human lymphocytes treated in vitro or obtained from exposed workers. Cadmium treatment in vivo or in vitro appears to interfere with cell replication, which results in abnormal chromosome numbers in germ cells of exposed mice and hamsters (Shimada et al., 1976; Watanabe et al., 1979; Gilliavod and Leonard, 1975).

An increased risk of lung cancer and cancer of the prostate has been reported in workers exposed to cadmium via inhalation. A 2-fold excess risk of lung cancer was observed in cadmium smelter workers. (Thun et al., 1985). The cohort consisted of 602 white males who had been employed in production work for 6 months or more during the years 1940-1969. The population was followed to the end of 1978. Urine cadmium data available for 261 workers employed beyond 1960 suggested a highly exposed population. The increased risk was shown not to be due to the presence of arsenic or to smoking. Four studies of workers exposed to cadmium dust or fumes provided further evidence of a statistically significant positive association with prostrate cancer (Kipling and Waterhouse, 1967; Lemen et al., 1976; Holden, 1980; Sorahan and Waterhouse, 1983). An excessive lung cancer risk was also observed in three other epidemiologic studies (Varner, 1983; Sorahan and Waterhouse, 1983; Armstrong and Kazantzis, 1983), but these studies were compromised by co-exposure to other carcinogens (arsenic, cigarette smoke) or by a small study population.

Based on limited evidence from epidemiologic studies and sufficient evidence of carcinogenicity in rats and mice by two routes of exposure, the U.S. EPA Carcinogen Assessment Group (CAG) has classified cadmium as a Probable Human Carcinogen (B1) by inhalation. Studies of human ingestion of cadmium are inadequate to assess carcinogenicity.

D.2.5 Summary

Trace amounts of cadmium are widely distributed naturally in the environment. Higher amounts enter the environment from the

manufacture, use, and disposal of cadmium-containing products. Cadmium is absorbed by ingestion, by inhalation, and, to a limited degree, by dermal absorption. The major long-term effects of exposure to cadmium are similar for both the oral and inhalation routes. Similar effects are seen in both animals and humans. For humans, the major effect is renal dysfunction. This results in disturbances in mineral metabolism and eventually in formation of kidney stones or osteomalacia. Pulmonary dysfunction has been observed following inhalation exposures of greater magnitude. Cadmium exposure has been correlated with hypertension in humans. In addition to these effects, reduced immune system function, testicular effects, reduction in birth weight and birth defects have been seen in animals. Cadmium has been classified as a Probable Human Carcinogen (B1) by inhalation based on limited evidence from epidemiologic studies and sufficient evidence of carcinogenicity in rats and mice by two routes. Studies of cadmium exposure by the oral route in both animals and humans have been inadequate to assess carcinogenicity.

D.3 Copper Toxicity Profile

The purpose of this section is to describe the toxic properties of copper, particularly with respect to risk to public health from contamination at New Bedford Harbor. Information on copper toxicity was obtained from the document entitled: "Summary Review of the Health Effects Associated with Copper" (EPA, 1987).

Copper is a metallic element with great industrial uses. Estimates for the average crustal abundance of copper range from 24 to 55 ppm (Cox, 1979). Recent reported average value for total copper content of world soils is 30 ppm, with values ranging from 2 to 250 ppm. For normal agricultural soils, total copper is expected to range from 1 to about 50 ppm (Gilbert, 1952).

Copper is essential to human life and health, but, like all heavy metals, it is potentially toxic also. In response to this duality, biochemical mechanisms have evolved that control the absorption and excretion of copper. These mechanisms operate to offset the effects of temporary deficiency or excess of the metal in the diet.

D.3.1 Pharmacokinetics

Copper is an element essential for proper nutrition. Absorption of copper and copper compounds can occur by oral and inhalation routes of exposure, with highest uptake rates occurring in the gastrointestinal tract followed by respiratory absorption. Very little dermal absorption occurs.

Although estimates of the percentage of orally absorbed copper in humans vary widely, recent studies have reported that absorption

ranges from 32% to 70% for adults (Strickland et al., 1972; King et al., 1978; Davies and Bennett, 1983) and from 42% to 85% for children 3-6 years of age (CRP, 1975). Only 20% of inhaled copper mists, fumes or dusts is estimated to be absorbed by the lungs. Inhaled copper may also be ingested and absorbed through the gastrointestinal tract. This occurs from coughing and swallowing which leads to ingestion of copper. Fecal excretion is the primary route of copper elimination.

Copper can also cross the placental barrier and is taken up by the fetus. Fetal copper content and distribution differs from that observed in adults in that total body content and liver concentration of copper are much higher in the fetus.

The pharmacokinetic properties of copper in animals and humans demonstrate that sophisticated homeostatic mechanisms have evolved to cope with copper intake deficiencies and excesses. Despite fluctuations in copper intake, proper copper balance in the body can be maintained by varying the amount of copper absorbed and excreted and by altering copper's distribution to various tissues.

D.3.2 Toxicity in Animals

Oral Exposure--Liver damage, hemolytic anemia, renal damage and gastrointestinal irritation are effects of acute copper poisoning that have been observed to occur in laboratory animals and man (Bremner, 1979; Owen, 1981).

Ingestion of 100 mg copper/kg/day by rats for 1 week resulted in no observable adverse effects (e.g., no liver accumulation and no adverse renal or hepatic morphology). Administration of this dose for up to 6 weeks caused severe renal or hepatic effects in rats. Further administration of copper at this dose to rats for up to 15 weeks resulted in no further damage; rather, regeneration of hepatic and renal tissues was observed (Haywood, 1980). Support for these observations was reported by Rana and Kumar (1980) who observed liver and kidney necrosis in rats fed 25.4 mg copper/kg/day for 20 days.

Increased liver copper concentrations were observed in rats fed 16.7 mg copper/kg/day for 27 days (Boyden et al., 1938) and 11.0 mg copper/kg/day for 21 days (Miranda et al., 1981). Administration of higher levels of dietary copper resulted in elevated liver and spleen copper levels, growth reduction and reduced dietary intake resulting in death (Boyden et al., 1938).

Inhalation Exposure--Limited inhalation studies with copper compounds generally resulted in minor, transient effects in experimental animals; however, guinea pigs exposed to a fungicide

containing copper sulfate, exhibited various lung lesions. (Pimental and Marques, 1969).

Teratogenic Effects--

Copper compounds have been observed to elicit a teratogenic response at ~2 mg copper/kg when injected into female hamsters on the 8th day of pregnancy (DiCarlo, 1980; Ferm and Hanlon, 1974). Lecyk (1980) reported a teratogenic response of orally-administered copper sulfate in mice for the 30-day dietary exposures at 103.5 mg copper/kg/day. Copper deficiency has also been shown to produce responses.

D.3.3 Human Health Effects

Information on the toxicity of copper in humans is provided from 1) studies of occupational exposure and 2) case reports and epidemiologic studies of the general population. Because of the well-developed homeostatic mechanisms, episodes of toxicity from excess copper exposure in humans are relatively rare.

D.3.3.1 Occupational Studies--Despite the fact that occupational exposure to copper and copper products is common, cases of copper intoxication are rare (Cohen, 1974; Williams, 1982). A condition known as "metal fume fever" characterized by influenza-like symptoms (stuffiness of the head, sensations of chills or warmth, and general aches and pains) has been reported in workers exposed to fine copper dusts (~0.1 mg copper/m^3) (Gleason, 1968), copper fumes (Armstrong et al., 1983) and copper oxide and copper acetate dusts (Stokinger, 1981; Cohen, 1974). Vineyard workers exposed to a fungicide containing copper sulfate reportedly had various histological lesions in the lungs and liver (Pimental and Marques, 1969). These lesions were similar to those observed in guinea pigs exposed to the same mixture.

Chronic effects observed from industrial exposure to copper include contact dermatitis (Stokinger, 1981; Cohen, 1974; Williams, 1982), mild anemia (0.6-1 mg copper/m^3) (Finelli et al., 1984) and leukocytosis (Armstrong et al., 1983). A study of 14,562 white male workers from the copper and zinc smelting industries did not show any overall mortality excesses as compared with the mortality of the total U.S. population (Enterline et al., 1986).

D.3.3.2 General Population Studies--Daily intakes of copper ranging from 2-32 mg due to contaminated drinking water has reportedly caused general gastric irritation characterized by nausea, vomiting, abdominal cramps and diarrhea (Spitalny et al., 1984; Nicholas and Brist, 1968; Semple et al., 1960; Wyllie, 1957).

A disease known as Indian Childhood Cirrhosis has frequently been associated with high intakes of copper in children ranging in age from 6 months to 5 years (Bhandari and Sharda, 1982; Bhargave, 1982; Chaudhary, 1983; Pandit, 1982; Pandit and Bhave, 1983; Sharda, 1984; Tanner et al., 1983). This disorder is characterized by widespread hepatic necrosis and very high hepatic copper content (Pandit, 1982; Pandit and Bhave, 1983). It is generally believed that milk and water stored in brass and copper containers leads to increased dietary copper in children, which is at least partly responsible for the pathogenesis of the disease.

Other epidemiology studies have failed to establish a definite link between chronic exposure to copper and cancer or other diseases. Increased mortality from hypertension or hypertensive heart disease and elevated rates of cancer of the trachea, lung, bronchus, liver and biliary passages have been observed in counties containing primary copper smelters (PEDCo, 1978); however, copper ore concentrates also contained arsenic, lead, nickel, antimony and selenium and the authors cautioned against overemphasizing the association between copper and increased disease rates.

The population most sensitive to elevated environmental copper levels is that afflicted with Wilson's disease, an inborn error in copper metabolism (~1 in 200,000 individuals). These individuals have endogenous chronic copper toxicity. Copper levels in all tissues, especially in the liver, are markedly increased, thus causing these people to be extremely vulnerable to minor variations in copper intake. Newborns and young children are more sensitive to excess copper than are normal adults. Increased copper concentrations in the body and underdeveloped homeostatic mechanisms probably contribute to this susceptibility.

D.3.4 Carcinogenicity--Equivocal results have been obtained from experiments designed to evaluate the carcinogenicity and mutagenicity of copper compounds. Administration of copper compounds to mice by subcutaneous injection has been reported to induce tumor formation (BRL, 1968; Haddow and Horning, 1960). The only tumorigenicity studies for orally-administered copper were negative (BRL, 1968).

Microbial mutation assays using copper compounds have generally provided negative results. Some mutagenic activity by copper compounds at high concentrations has been observed in cell culture assays. Copper sulfate was observed to increase the frequency of recessive lethal mutations in fruit flies at high concentrations (Law, 1938).

Schrauzer et al. (1977) reported that there is a direct proportionality between blood copper concentrations and mortality due to cancer of the intestine in males and females and cancer of the lung, breast and thyroid in females. The authors contended that excess copper in the diet may be the cause of the elevated blood copper levels. Morton et al. (1976) observed a significant negative association between copper concentrations in tap water samples and central nervous system malformations (e.g., anencephalus, neural tube malformations) in South Wales. These associations, however, can only be critically evaluated when additional epidemiologic information becomes available.

D.3.5 Summary

In summary, human homeostatic mechanisms act to control copper balance by regulating the absorption, storage, distribution, utilization and excretion of the metal. Deficiencies or excesses in copper by oral exposure rarely result in copper toxicosis. In cases where homeostatic mechanisms are genetically disrupted as in Wilson's disease, or underdeveloped as in the fetus and young children, excess copper intake is not regulated. The symptoms of copper toxicosis can occur in such individuals when copper intakes are high.

In its rating of carcinogenic properties, U.S. EPA classifies copper as a Group D substance (not classifiable) because of insufficient data to determine the carcinogenic potential.

D.4 Lead Toxicity Profile

The purpose of this section is to describe the toxic properties of lead, particularly with respect to risk to public health from contamination at New Bedford Harbor.

Lead is a naturally occurring metallic element. Because of its easy isolation and low melting point, it was among the first metals to be extensively utilized by man. It is widely used in industry because of its softness, resistance to corrosion and radiation, and high density. It is used in pigments for paints and ceramics, in electric batteries, as a lining for tanks and pipes, as an ingredient in solder, as a shielding material for X-rays and atomic radiation and in plastics. Lead has been distributed throughout the biosphere by the industrial activities of man and may be found in water, soil, plants, animals and humans.

D.4.1 Pharmacokinetics

Lead absorption occurs from ingestion and inhalation and, to a much less extent, from dermal absorption. Absorption of lead

from the gastrointestinal tract is estimated at 10-15% for adult humans and 40-50% for children. Gastrointestinal absorption of lead is influenced by numerous dietary factors. Absorption after a 6-hour fast was increased up to 8-fold as compared to lead consumed with food (Wetherill et al, 1974). Absorption is enhanced by low dietary Ca or Fe, high dietary fat, or low or high dietary protein (Sobel et al, 1938; Six and Goyer, 1970, 1972; Baltrop and Khoo, 1975). Absorption is decreased in animals receiving high mineral diets (Barltrop and Khoo, 1975). Gastrointestinal absorption in infant rats was considerably greater than in adults (Forbes and Reina, 1974; Kostial et al., 1971). Similar results have been reported in humans (Alexander et al., 1973; Ziegler et al., 1978).

Absorption also occurs via inhalation. Nozaki (1966) reported inverse relationships between respiration rate, particle size and lung deposition. For adult humans, the deposition rate of particulate airborne lead is ~ 30-50%. It also appears that essentially all of the lead deposited in the lower respiratory tract is absorbed (EPA, 1983) (HEA). Respiratory uptake by children appears to be greater on a body weight basis. One report estimated that a 10-year-old child has a deposition rate 1.6- to 2.7-fold higher than an adult on a weight basis. (EPA, 1983).

Absorption of inorganic lead compounds through the skin is of much less significance than absorption through respiratory and gastrointestinal routes. Dermal absorption of lead in cosmetic preparations has been observed to occur in humans with a maximal efficiency of 0.3% (Moore et al., 1980). For lead alkyls however, absorption through skin is far more significant than other routes of exposure. Transplacental transfer of lead has also been documented and lead uptake by the fetus occurs readily. Lead is stored in bone, kidney and liver where it may remain for long periods of time. Lead is excreted by the kidneys in urine and lesser amounts are eliminated by biliary excretion.

D.4.2 Toxicity in Humans and Experimental Animals

Exposure to lead causes adverse effects in the blood cell forming system, the nervous system, the kidneys and the reproductive system. The hemoglobin forming system is the most sensitive system to lead exposure. Erythrocyte protoprophyrin elevation, an indicator of decreased hemoglobin synthesis, has been reported in women and children at levels of 15-20 ug/dl blood (EPA, 1977d, 1980b, 1983). The threshold for decreased hemoglobin levels is ~40 ug/dl blood in children (Betts et al., 1973; Pueschel et al., 1972) and 50 ug/dl blood in adults (Tola et al., 1973).

Two types of neurotoxic effects are associated with exposure to lead. Levels of lead in blood of over 80 ug/dl in children and

over 100 ug/dl in sensitive adults can cause severe, irreversible brain damage, encephalopathy, and possibly death. In children, permanent learning disabilities are seen at these levels, even if there are no overt signs of lead poisoning. The World Health Organization (1977) estimated that the threshold for noticeable brain dysfunction in children is 50 ug/dl blood. Other adverse effects are associated with exposure to low levels of lead. Slower nerve conduction in peripheral nerves was seen in adults at 30-40 ug/dl blood.

Numerous studies of occupationally-exposed workers have provided evidence that lead-induced chronic kidney effects are associated with blood lead levels as low as 40 ug/dl. Experimental animal studies have shown similar kidney effects.

Reproductive effects have also been reported. Postnatal developmental delays have been reported in pups of rats that received 50-250 mg lead/L drinking water throughout gestation (Kimmel et al., 1976; Reiter et al., 1975). Schroeder and Mitchener (1971) reported marked reproductive effects in a 3-generation study with 25 ppm lead in drinking water. In rats, results included a delay in birth of the first litter, runting and excessive mortality among the offspring before weaning, a decrease in the male/female ratio of the F1 generation, and a decrease in pregnancies and litter size by the third generation. In mice, the effects were similar but more severe.

In humans, lead compounds have been used to induce abortions (Tanssig, 1936). Oliver (191911) reported that the miscarriage rate was elevated among British women occupationally exposed to lead. Other investigators have related lead exposure, both before and during pregnancy, with increases in spontaneous abortions, premature delivery and early membrane rupture (Lane, 1949; Nozaki, 1958; Fahim et al., 1975; Rom, 1976). Altered testicular function was observed in men with blood lead levels as low as 40-50 ug/dl.

Pregnant women, preschool age children and fetuses are high risk populations for exposure to lead.

D.4.3 Carcinogenicity

Several animal studies have associated specific lead salts with tumor formation. Dietary lead acetate was demonstrated to cause renal tumors in Wistar rats (Zawirska and Medras, 1968, 1972; Azar et al., 1973; Boyland et al., 1962). Lead subacetate caused renal tumors in Swiss mice (Van Esch and Kroes, 1969) and in several strains of rats (Van Esch et al., 1962; Oyasu et al., 1970; Mao and Molnar, 1967; Shakerin and Paloucek, 1965; Hass et al., 1967; Ito et al., 1971; Ito, 1973). Nerve cell tumors were also observed in many of these studies.

In humans, no association between lead exposure and cancer mortality was found in two epidemiological studies (Dingwall-Fordyce and Lane, 1973; Nelson et al., 1973). A third study (Cooper and Gaffey, 1975) reported a statistically significant elevation in deaths due to "all malignant neoplasms" and cancer of "other sites" (sites other than major organs). Using different statistical tests, Kang et al. (1980) reanalyzed these data and calculated a statistically significant increase in deaths due to cancer of the digestive organs and cancer of the respiratory system for both lead smelter workers and battery plant workers. Deaths due to all malignant neoplasms were increased among smelter workers only.

Based on inadequate evidence from human studies and sufficient evidence from animal studies, the EPA Carcinogen Assessment Group (CAG) has classified the lead salts, lead acetate, lead phosphate and lead subacetate, as Probable Human Carcinogens (Group B2). Since humans are not environmentally exposed to the particular lead salts that are associated with tumors in animals, lead and lead compounds as a class are classified only as Possible Human Carcinogens (Group C).

Interaction

A number of dietary factors alter the gastrointestinal absorption of lead. High mineral diets inhibit the absorption of lead (Barltrop and Khoo, 1976), and diets low in calcium or iron enhance absorption (Sobel et al., 1938; Six and Goyer, 1970, 1972).

Summary

Lead is a naturally occurring element which has been widely distributed through the biosphere by the industrial activities of man. It is absorbed by ingestion and inhalation. It is also absorbed less significantly by the dermal route. Lead is stored in the bone, kidney and liver. Studies in both animals and humans have documented adverse effects from exposure to lead to the hematopoietic system, the nervous system, the kidneys and the reproductive system. Various lead salts have caused cancer in animals, and these lead salts are classified as Probably Human Carcinogens (Group B2). Since humans are not environmentally exposed to these particular salts, lead and lead compounds as a class are classified as Possible Human Carcinogens (Group C).

APPENDIX E

SUPPORTING DOCUMENTATION

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

NOV 9

OFFICE OF
RESEARCH AND DEVELOPMENT

MEMORANDUM

SUBJECT: Congener-specific PCB analyses on New Bedford Harbor Seafood

TO: Frank Ciavattieri, Remedial Project Manager
Region I

FROM: Susan Braen Norton, Environmental Scientist
Exposure Assessment Group SBN

Staff of the Exposure Assessment Group and the Narragansett Laboratory have completed the analysis of the congener-specific PCB analyses on lobsters and flounders collected from New Bedford Harbor. The objective of the analysis was to provide insight into how best to characterize the risk associated with ingesting seafood from New Bedford Harbor. Specifically, the issue is whether there is sufficient evidence to support using a cancer potency factor other than the most recent PCB potency factor that is based on Aroclor 1260. The analyses, results, and conclusions of our effort are described in this memo.

Staff of the Narragansett Laboratory and Science Applications International Corporation (SAIC) chemically analyzed five flounders, five lobsters, and samples of the commercial mixtures Aroclor 1016, 1242, 1254, and 1260. The five flounders were collected by the Narragansett laboratory from inside the hurricane barrier, and the five lobsters were collected from Area 3 by the Massachusetts Division of Marine Fisheries. Flounder flesh, lobster flesh, and lobster hepatopancreas were included in the study. After extracting the tissues with acetone, PCB congeners were separated and quantified using capillary gas chromatography and an electron capture detector. By using three columns, the Narragansett Laboratory was able to quantify a suite of 51 separate congeners.

The data were statistically analyzed using principal components analysis (via the SIMCA software package). Principal components analysis is a very useful technique that simplifies large matrices of data (51 congeners by 21 samples in this case) to a series of independent "principal components" that best explain the variation in the data. By plotting the principal components, similarities in the patterns of congeners in the samples can be more easily identified. In addition, class models (e.g., for lobster samples) can be constructed and used to test

how close other samples (e.g., Aroclor 1260) are to the class. Finally, by examining how each congener influences the principal components, we can begin to understand how PCB patterns change in the environment.

The analyses of the New Bedford Harbor data consisted of three steps; construction of class models, plotting of principal components, and analysis of specific congeners. First, I constructed class models (using the PARC subroutine in the SIMCA statistical package) for the lobster flesh, lobster hepatopancreas and flounder. Then I tested how close the commercial mixtures came to fitting the models. The results of the classification are shown in Table 1. As can be seen, Aroclor 1254 had the lowest deviation from the class models, with Aroclor 1260 running a close second. Even these two mixtures, however, were greater than 5 standard deviations away from any of the classes.

In the second step, I took another look at the relationship between the samples and Aroclors 1260 and 1254 by plotting principal components. Figures 1, 2, and 3 show the principal components plots for each type of seafood plus Aroclors 1254 and 1260. As expected from the classification step, the biota samples aren't very close to either Aroclor. However, the principal components plot shows that patterns of PCBs in the seafood samples lie roughly in between the two commercial mixtures and are just about equidistant from both.

To gain some insight into how the seafood differs from the commercial mixture, I investigated which specific congeners were present in greater amounts in the seafood than in 1254 and 1260. I divided the fractions of each congener in the seafood by the corresponding fractions in 1254 and 1260 to obtain "enrichment factors". Some of the larger enrichment factors are shown in Figures 4 and 5. Of particular interest is that fractions of some non and mono-ortho substituted congeners (particularly 3,4,5,3',4'-PCB and 3,4,3',4'-TCB) are consistently higher in the seafood compared with the two commercial mixtures. The non-ortho substituted congeners have been shown to induce enzymes in a manner similar to 3-methylcholanthrene and dioxin. Although induction of this enzyme system has not yet been correlated with carcinogenic potency, it has been correlated with other toxic effects, such as thymic atrophy, in experimental animals.

The conclusions of the analyses can be summarized as follows. The PCB mixture in the seafood from New Bedford Harbor cannot be classified as any commercial mixture, although the pattern of PCBs in the seafood appears to lie roughly between Aroclors 1254 and 1260. That the non-ortho substituted congeners are not depleted but are actually enriched in New Bedford Harbor seafood lends some support for taking a conservative approach to assessing risks from seafood ingestion.

TABLE 1

CLASS MODEL ANALYSES

CLASS	Standard Deviations Away From Model			
	A1016	A1242	A1254	A1260
Lobster Flesh	11000	3300	47	67
Lobster Hepato.	22000	6500	37	54
Flounder	1500	500	5.4	9.8

FIGURE 1

PRINCIPAL COMPONENTS PLOT
LOBSTER FLESH

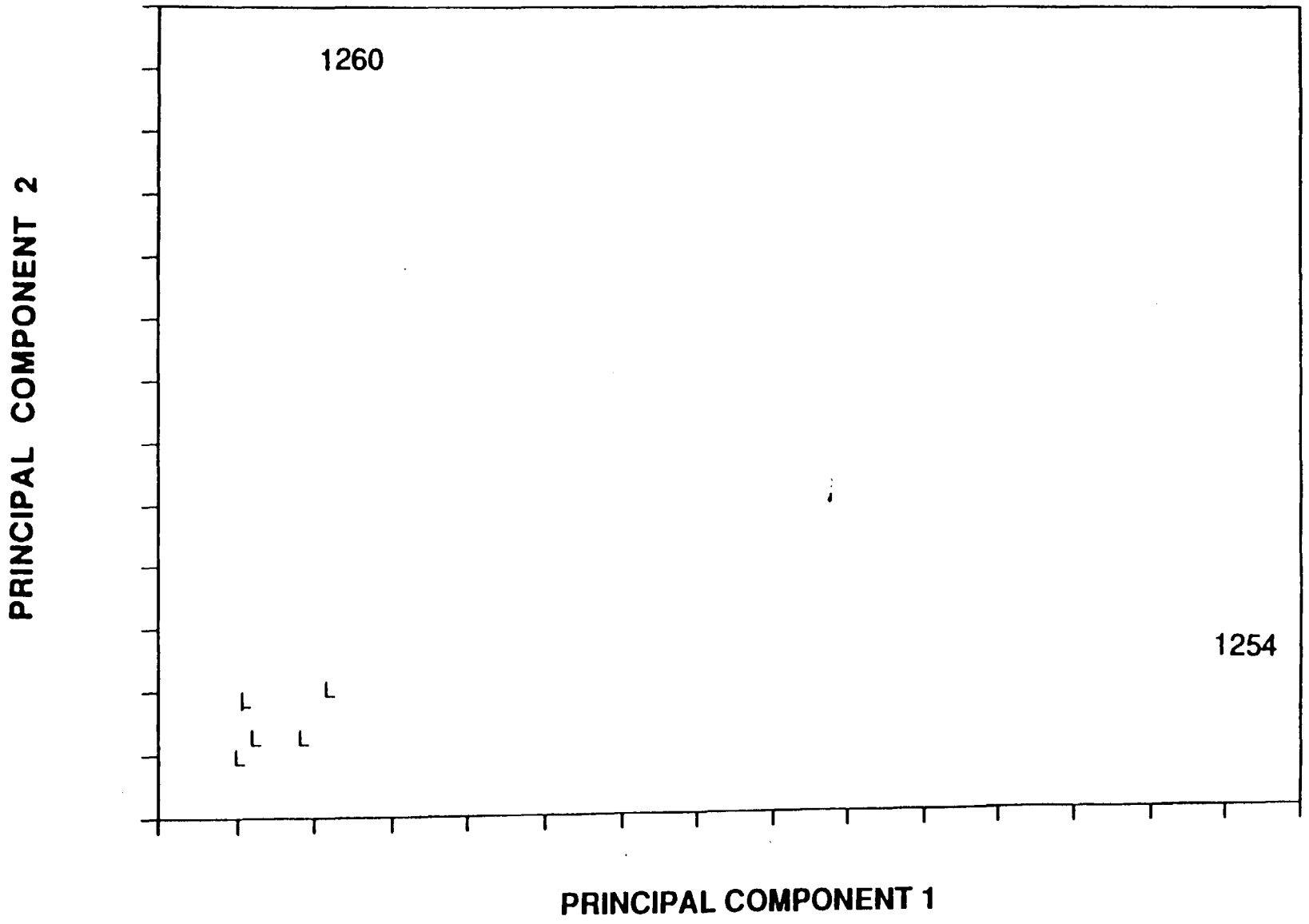


FIGURE 2

PRINCIPAL COMPONENTS PLOT
LOBSTER HEPATOPANCREAS

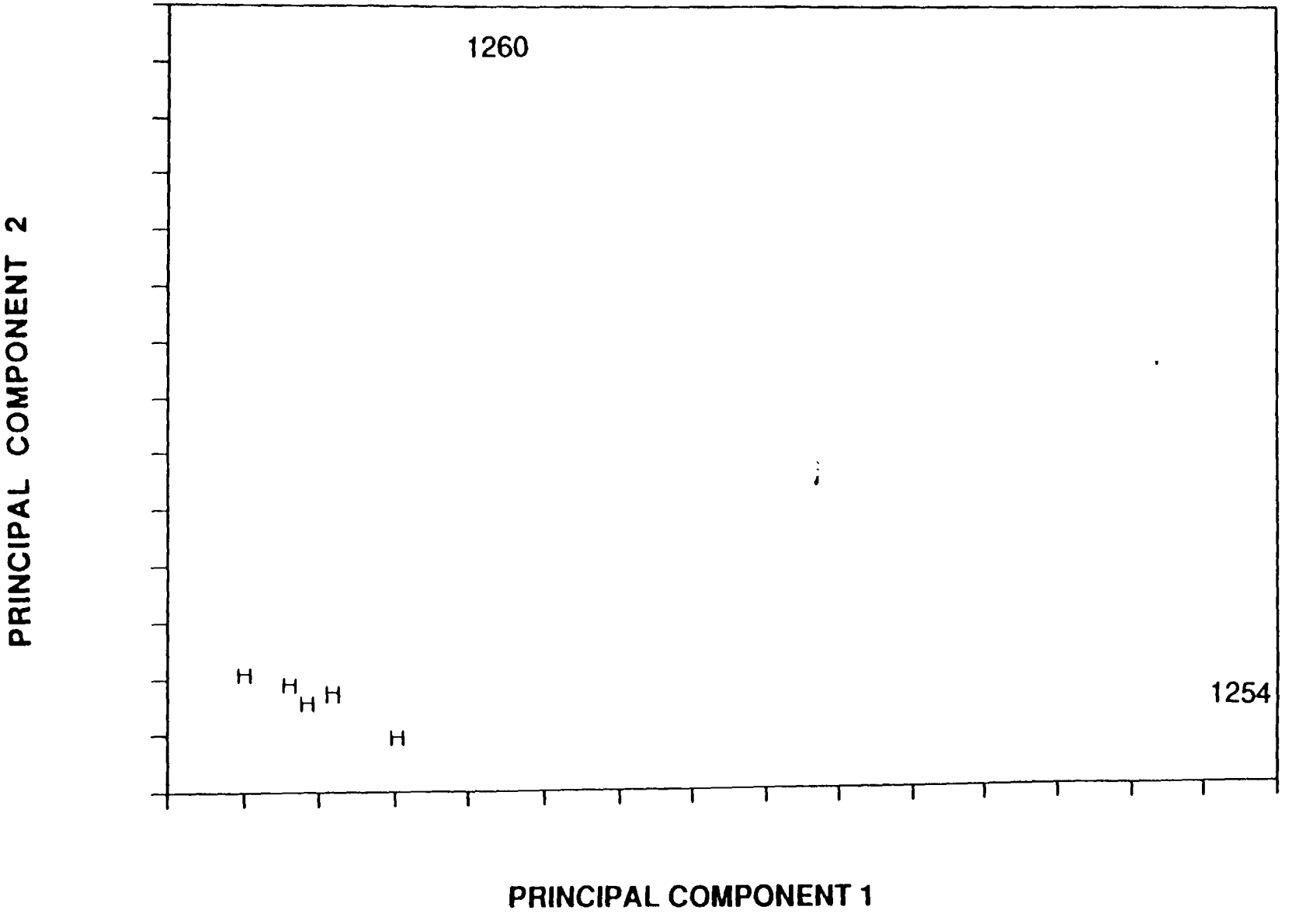


FIGURE 3

PRINCIPAL COMPONENTS PLOT
FLOUNDER FLESH

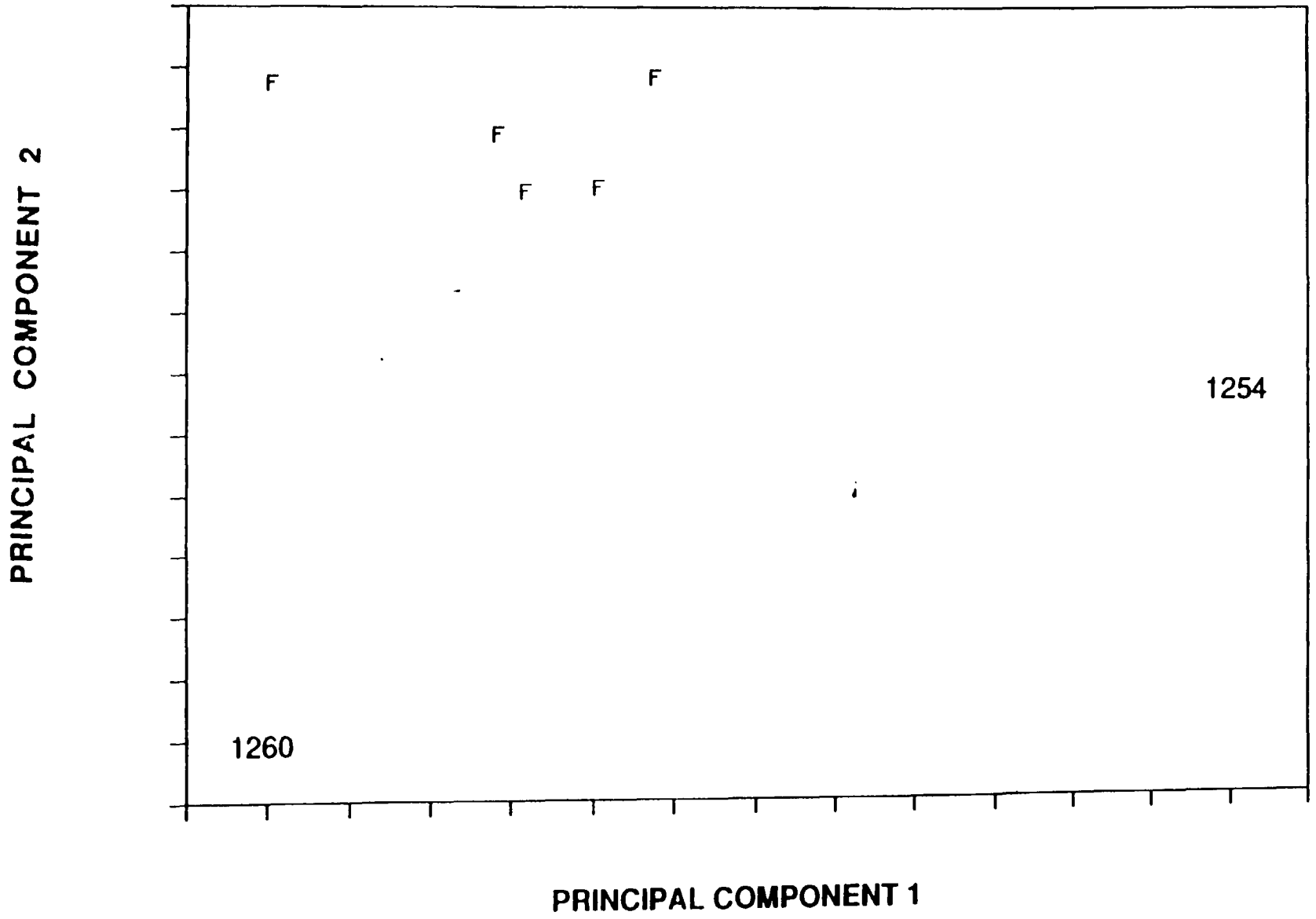


FIGURE 4

ENRICHMENT OF SELECTED CONGENERS

RELATIVE TO AROCLOR 1254

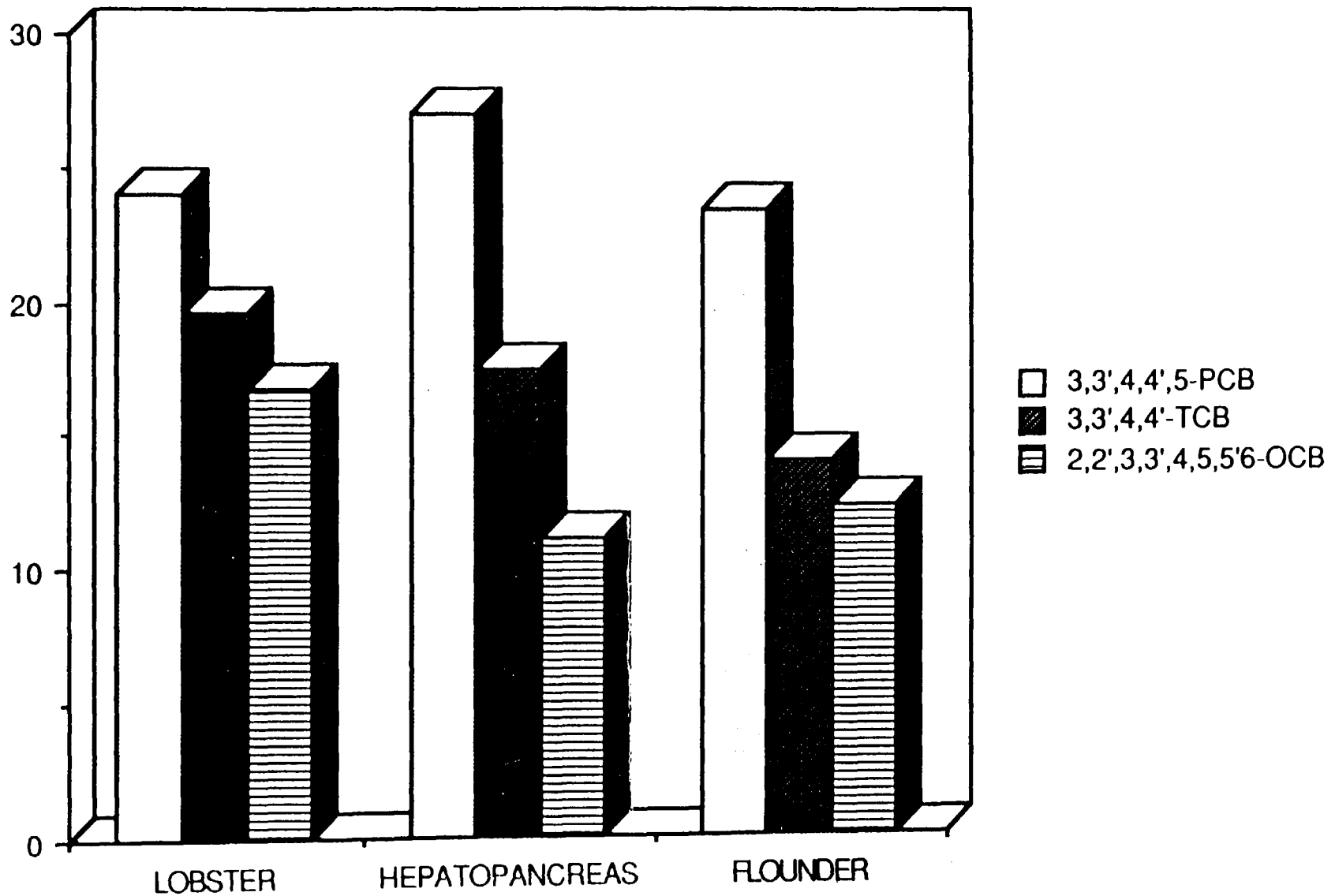
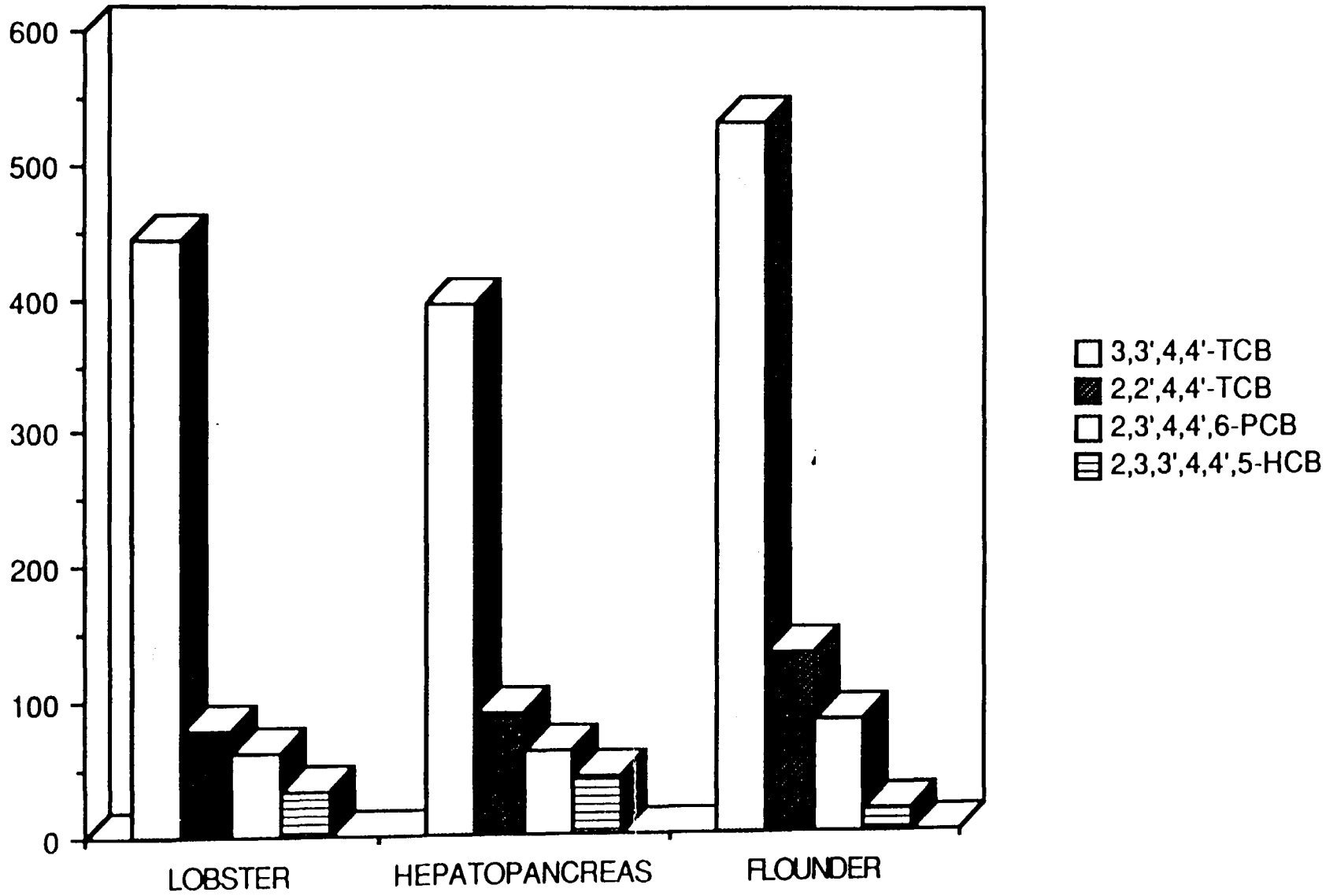


FIGURE 5
ENRICHMENT OF SELECTED CONGENERS
RELATIVE TO AROCLOR 1260



It also should be noted that the non-ortho substituted congeners have not normally been quantified in the past because of difficulties in analytical techniques. The progress that the Narragansett Laboratory has made in separating and quantifying the non-ortho substituted congeners contributed significantly to the findings of the current effort.

I discussed the results of the analysis with Jim Cogliano of the Carcinogen Assessment Statistics and Epidemiology Branch (formerly CAG). CAG's current cancer potency factor for PCBs (7.7 $(mg/kg/d)^{-1}$) is based on the Norback and Weltman (1985) bioassay using rats. This bioassay has a stronger experimental design than either the 1975 Aroclor 1260 bioassay or the NCI Aroclor 1254 bioassay. Hence, the 1985 bioassay currently provides the best estimate of cancer potency for any PCB mixture. Because, as described above, the PCB mixture in seafood does not closely resemble any commercial PCB product, there are uncertainties in using any commercial product to estimate the potency of the mixture in seafood. Since the potency factor for Aroclor 1260 of 7.7 $(mg/kg/d)^{-1}$ is the most scientifically defensible estimate of PCB potency currently available, I would recommend that it be used to assess risks from ingesting seafood from New Bedford Harbor.

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PCB Congeners in American Lobster, *Homarus americanus*, and Winter Flounder, *Pseudopleuronectes americanus*, from New Bedford Harbor, Massachusetts

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Introduction

PCBs are a group of 209 different compounds. The physical, chemical, and toxicological properties vary depending on the amount and position of chlorine substitution on the biphenyl molecule[1]. PCBs were produced and sold as complex mixtures of congeners which in the United States were marketed under the trademark of Aroclors (Monsanto Co.). However, once these commercial mixtures of PCBs are released to the environment their compositions can change due to the differential behaviors of the compounds.

Since the properties of PCB congeners differ depending on the chlorine substitution, environmental alterations may influence the toxicological significance of PCB mixtures. In order to better define the potential significance of the PCBs found in edible marine tissues from New Bedford Harbor, we have applied methods adapted from Stalling et al. (1979) which allowed us to better define the PCB mixtures in these samples. These techniques specifically focus on PCB congeners known to produce biological effects in some species. This report presents the results of analyses conducted on the edible tissues of lobster and flounder collected from New Bedford Harbor.

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[1] Certain PCB congeners have been shown to induce enzyme systems (e.g., Aryl Hydrocarbon Hydroxylase or AHH) in a manner similar to 3-methylcholanthrene and 2,3,7,8-tetrachlorodibenzo-p-dioxin. These congeners are of particular interest since the enzyme induction has been correlated with some toxic effects such as thymic atrophy (see Safe, 1987 and Duinker, 1988 for more detail). In this report, these congeners, which include 77, 81, 105, 114, 118, 126, 156, 157, 167, 169 and 189 are referred to as non- and mono-ortho substituted congeners or AHH inducers.

Materials and Methods

Sample collection

Lobsters (legal size) were collected with lobster pots by the Cat Cove Marine Laboratory of the Massachusetts Division of Marine Fisheries. The five lobsters that were analyzed were taken from Closure Area 3 (Figure 1). Station locations and collection dates for these samples are listed in Table 1. Also listed in this table are the weights of the whole lobsters and the amounts of muscle and hepatopancreas tissues found in each lobster. The flounder samples were collected by otter trawl on January 9, 1987 from just inside of the New Bedford Harbor Hurricane Barrier. All of the samples were frozen until analyzed.

Sample analysis

A flow chart of the analysis procedures is shown in Figure 2 and the techniques that were utilized are described in detail below. Tissue samples were homogenized using a Polytron (Brinkman Corp.) and then 2 to 5 grams of each homogenate was weighed into an acetone rinsed 100 ml centrifuge tube. Octachloronaphthalene (OCN) and PCB congener 100 (Ballschmiter and Zell, 1980) were added as internal standards along with 25 ml of acetone and the mixture was agitated using a Polytron for 20 seconds. The sample was centrifuged for 15 minutes at 25000 rpm at 4°C and then the supernatant poured into a separatory funnel containing 150 ml of water. This procedure was repeated two more times and the extracts combined in the separatory

funnel. The acetone/water mixture in the separatory funnel was then extracted three times with 25 ml of freon each time. These were combined, treated with sodium sulfate and volume reduced using a heating mantle and a Kuderna-Danish evaporator with a 3-ball Snyder column. The extract was volume reduced and solvent exchanged to 10 ml of hexane. One ml was removed for a lipid weight determination and the remaining 9 ml was treated with concentrated sulfuric acid. This involved adding 5 ml of acid to the conatiner, shaking gently and allowing the mixture to sit for two hours.

After the acid treatment, the extract was volume reduced and then analyzed for PCBs as Aroclors. Sample extracts were analyzed as Aroclor 1242 and 1254 using capillary gas chromatography (GC). For these analyses, 1 ul of each sample was injected splitless into a Hewlett Packard 5890 gas chromatograph equipped with a 30 m DB-5 fused silica capillary column (J + W Scientific) and an electron capture detector. Helium was used as the carrier gas at a flow rate of about 1.5 ml/min and the flow of a 95:5 mixture of argon:methane to the detector was 35 ml/min. The oven temperature was held at 60°C for 1 min and then programmed from 60 to 315°C at 10°C/min. The injector temperature was 270°C and the detector was maintained at 325°C.

Aroclor determinations were made by comparing the summed heights of two peaks in each Aroclor with the heights of the same peaks in the samples. The peaks used for Aroclor 1242 were mixed peaks while congeners 138 and 180 (Ballschmiter and Zell,

1980) were measured for the quantification of Aroclor 1254. OCN was used as an internal standard for these determinations.

After the PCB concentrations were measured as Aroclors, the extracts were split and each portion was separated into fractions using carbon/silica columns. Individual PCB congeners were quantified in the fractions from these columns. These separations allowed the quantification of congeners that could not be separated by GC using the chromatographic conditions that we employed. Two columns were required because it was not possible to make the required separations in one step. The carbon/silica columns consisted of 2 cm of a carbon/silica gel mixture in a disposable pipette. This mixture was composed of 5 % Super-A Activated Carbon AX-21 with silica gel (Bio-Sil A, Bio-Rad Laboratories) that had been activated at 150°C for 24 hours.

Both carbon columns were rinsed with 15 ml of toluene, 15 ml of methylene chloride and 10 ml of hexane before the extracts were added. The elution schemes used are shown in Figure 2. The first column was eluted with 15 ml of hexane which was collected as the F1 fraction. The column was inverted and then eluted with 15 ml of toluene which was collected as the F2 fraction. Congeners containing 2, 3 or 4 ortho substituted chlorines elute in the F1 fraction. These compounds were quantified against the congener 100 internal standard. Several congeners containing 1 ortho substituted chlorine were quantified in the F2 fraction. The concentrations of these compounds were calculated using the OCN internal standard which

eluted into this fraction.

The second column was first eluted with 15 ml of methylene chloride which was discarded. The column was inverted and then eluted with 5 ml of toluene which was collected as the F3 fraction. Three congeners containing 0 ortho chlorines (77, 126 and 169) were quantified against OCN in this fraction.

Standards were obtained for all of the congeners that were quantified. These were purchased from Ultra Scientific and the National Research Council of Canada. Congener analyses were done on a Hewlett Packard 5890 GC as described earlier; except, a 60 m DB-5 fused silica column was used. The oven temperature was held at 150°C for 1 min and then programmed from 150 to 315°C at 1°C/min.

Since not all 209 PCB congeners could be obtained as standards it is possible that some of the congeners that were quantified occur as mixed peaks. This is more likely to be a problem for congeners quantified in the fraction containing compounds with 2,3 and 4 ortho chlorines. Fewer compounds are found in the fractions containing compounds with 0 and 1 ortho chlorines and most of these compounds were obtained as authentic standards. Therefore, the identification and quantification of these compounds are more definitive.

Quality Assurance

A spike and recovery study was conducted as a measure of the accuracy of the analytical procedure. Thirteen PCB congeners representing compounds containing 4-10 chlorines and OCN were spiked into three aliquots of a homogenate of mussel (Mytilus edulis) tissue. The recoveries ranged from 78 to 84 % for the 13 PCB congeners and the recovery of OCN averaged 77 %.

Triplicate analyses for individual congeners were conducted on Aroclor 1242 as a measure of the precision of the method. The results of these analyses are presented in Table 5. The relative standard deviations for most of the congeners were less than 1 %. Blank analyses were conducted with each sample set which generally consisted of 5 samples. No significant interferences were found in the blanks. Values of 0.00 in the tables indicate levels below the analytical detection limits.

Results and Discussion

Total PCB concentrations (on a dry weight basis) measured as the sum of Aroclors 1242 and Aroclor 1254 are shown in Table 2. The PCB concentrations on a wet weight basis in these samples are shown in Table 3. Also included in Table 3 are PCB concentrations calculated for the combined tissues of lobsters. PCB concentrations and total tissue weights were used for these calculations. These data can be compared with the FDA Tolerance Level of 2 ug/g for PCBs.

The PCB concentrations in the five lobster samples were similar and all were above the 2 ug/g FDA Tolerance Level (3.14 - 8.79 ug/g). The concentrations found in the flounder, however, showed a wide range (0.10 - 7.05). Three of the five flounder contained PCB levels above the FDA Tolerance Level. This wide range of concentrations may indicate that some of these organisms had only been in New Bedford Harbor for a short period of time.

The Massachusetts Division of Marine Fisheries (MDMF) has monitored PCB concentrations in lobster and several other species from New Bedford Harbor since 1977 (Kolek and Ceurvels, 1981; MDMF, unpublished data). Their data for lobsters from Closure Area 3 indicate that the PCB levels remained relatively constant between 1977 and 1985. Considerable variations were observed in the average seasonal values (1.0 - 8.8 ug/g); however, there did not appear to be a major trend in the data. This is demonstrated by the fact that the average level for 1977 was 3.9 ug/g and the Fall 1985 mean concentration was 4.2 ug/g. The present study reported a concentration mean of 4.97 ug/g for lobsters collected during 1987.

The methods used in the present study to quantify PCBs were different from those used by MDMF. However, a recent intercalibration between the EPA - Narragansett and MDMF laboratories on the measurement of PCBs in composite lobster samples indicated that the values generated by the two laboratories differ by less than a factor of 2 (Pruell et al., 1988). EPA reported slightly higher levels which probably

indicates that the total PCB levels measured in the present study are very similar to those that have been previously found in New Bedford Harbor Area 3 lobsters (MDMF, 1981). This indicates that the PCB levels found in lobsters from Closure Area 3 have remained remarkably constant for the past 10 years.

Forty-five PCB congeners were quantified in Aroclors 1016, 1242 (analyzed in triplicate), 1254 and 1260, in 5 lobster muscle and hepatopancreas samples, and 5 flounder muscle samples. The names and numbers of the congeners that were quantified are listed in Table 4. The results of the congener analyses are reported in Tables 5, 6, 7 and 8. The congeners that were quantified were selected for several reasons. These included the potential to induce AHH (Safe, 1987), the relative abundance in environmental samples and the availability of the standards. All of the congeners listed by Duinker et al. (1988) as "toxic congeners" were quantified except congener 123 which was not available to us as a pure standard.

The congener mixtures found in the various Aroclors (Table 5) agree well with what has been previously reported (Albro et al., 1981; Capel et al., 1985). The sums of the congeners measured for each Aroclor indicate that the congeners that we quantified represented about 22 % of Aroclor 1016, 25 % of Aroclor 1242, 50 % of Aroclor 1254 and 63 % of Aroclor 1260.

The patterns of PCBs seen in the lobster samples (Tables 6 and 7) were very similar to those previously reported in lobsters from New Bedford Harbor by Farrington et al. (1986).

Congeners found in the highest concentrations included numbers 105, 118, 128, 138, 153 and 180. These 6 compounds accounted for about 80 % of the mass of the individual congeners that were quantified in both the muscle and hepatopancreas samples. These compounds were found to be the major components of Aroclors 1254 and 1260 (Table 5).

The flounder samples (Table 8) showed a different PCB pattern than those of the lobster samples. In general more congeners were found in significant amounts in the fish samples. The most prominent compounds in flounder included congeners 105, 118, 138 and 153. Maack and Sonzogni (1988) reported that these were also some of the major components of the PCB mixtures seen in several freshwater fish species from Wisconsin. A similar PCB pattern was also seen in the livers of Atlantic cod from the North Sea (de Boer, 1988).

Several non- and mono-ortho congeners were found in the lobster and flounder samples. These included congeners 37, 77, 81, 105, 114, 118, 126, 156, 157, 167, 169 and 189. Considering the concentrations and relative enzyme inducing potencies of the compounds (Safe, 1987), those that may be the most toxicologically important in these samples are congeners 77, 105 and 126.

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Table 1. Sample information and tissue weights (grams) of lobster from Area III of New Bedford Harbor.

Sample #	CC-#*	Station	Date	Whole Lobster	Muscle Tissue	Hepatopancreas
902177	P1503	MMM	12/18/87	545.	152.	14.8
902178	P1529	UU	12/22/87	385.	136.	12.6
902179	P1506	YY	12/18/87	452.	185.	12.9
902180	P1418	UU	07/06/87	612.	179.	12.3
902181	P1426	MMM	07/01/87	405.	128.	19.2

* - Cat Cove Marine Laboratory Sample Number.

Table 2. Total PCB concentrations in lobster and flounder from New Bedford Harbor.

Concentrations as ug/g dry weight

Sample #	Organism	Muscle	Hepatopancreas
902177	Lobster	5.48	148
902178	Lobster	3.61	204
902179	Lobster	3.95	345
902180	Lobster	4.00	251
902181	Lobster	6.93	114

Sample #	Organism	Muscle
399568	Flounder	37.0
399569	Flounder	15.1
399572	Flounder	0.563
399574	Flounder	1.64
399575	Flounder	29.8

Table 3. Total PCB concentrations in lobster and flounder from New Bedford Harbor.

Concentrations as ug/g wet weight

Sample #	Species	Muscle	Hepatopancreas	Combined*
902177	Lobster	0.63	29.0	3.14
902178	Lobster	0.53	51.7	4.89
902179	Lobster	0.77	55.2	4.32
902180	Lobster	0.48	50.9	3.73
902181	Lobster	0.77	62.4	8.79

Sample #	Species	Muscle
399568	Flounder	7.05
399569	Flounder	3.35
399572	Flounder	0.10
399574	Flounder	0.31
399575	Flounder	6.49

* - Calculated using the weights and PCB concentrations of the muscle and hepatopancreas tissues.

Table 4. Numbers and names of the congeners that were quantified (Ballschmiter and Zell, 1980).

CONGENER NUMBER	NUMBER ORTHO CL *	CONGENER NAME
15	0	4,4'- dichlorobiphenyl
18	2	2,2',5 - trichlorobiphenyl
37	0	3,4,4'- trichlorobiphenyl
44	2	2,2',3,5'- tetrachlorobiphenyl
47	2	2,2',4,4'- tetrachlorobiphenyl
49	2	2,2',4,5'- tetrachlorobiphenyl
52	2	2,2',5,5'- tetrachlorobiphenyl
60	1	2,3,4,4'- tetrachlorobiphenyl
72	1	2,3',5,5'- tetrachlorobiphenyl
77	0	3,3',4,4'- tetrachlorobiphenyl
78	0	3,3',4,5 - tetrachlorobiphenyl
79	0	3,3',4,5'- tetrachlorobiphenyl
81	0	3,4,4',5 - tetrachlorobiphenyl
86	2	2,2',3,4,5 - pentachlorobiphenyl
101	2	2,2',4,5,5'- pentachlorobiphenyl
105	1	2,3,3',4,4'- pentachlorobiphenyl
114	1	2,3,4,4',5 - pentachlorobiphenyl
118	1	2,3',4,4',5 - pentachlorobiphenyl
119	2	2,3',4,4',6 - pentachlorobiphenyl
126	0	3,3',4,4',5 - pentachlorobiphenyl
128	2	2,2',3,3',4,4'- hexachlorobiphenyl
137	2	2,2',3,4,4',5 - hexachlorobiphenyl
138	2	2,2',3,4,4',5'- hexachlorobiphenyl
141	2	2,2',3,4,5,5'- hexachlorobiphenyl
143	3	2,2',3,4,5,6'- hexachlorobiphenyl
153	2	2,2',4,4',5,5'- hexachlorobiphenyl
156	1	2,3,3',4,4',5 - hexachlorobiphenyl
157	1	2,3,3',4,4',5'- hexachlorobiphenyl
158	2	2,3,3',4,4',6 - hexachlorobiphenyl
166	2	2,3,4,4',5,6 - hexachlorobiphenyl
167	1	2,3',4,4',5,5'- hexachlorobiphenyl
169	0	3,3',4,4',5,5'- hexachlorobiphenyl
170	2	2,2',3,3',4,4',5 - heptachlorobiphenyl
180	2	2,2',3,4,4',5,5'- heptachlorobiphenyl
183	3	2,2',3,4,4',5',6 - heptachlorobiphenyl
185	3	2,2',3,4,5,5',6 - heptachlorobiphenyl
189	1	2,3,3',4,4',5,5'- heptachlorobiphenyl
191	2	2,3,3',4,4',5',6 - heptachlorobiphenyl
194	2	2,2',3,3',4,4',5,5'- octachlorobiphenyl
201	3	2,2',3,3',4',5,5',6 - octachlorobiphenyl
204	4	2,2',3,4,4',5,6,6'- octachlorobiphenyl
205	2	2,3,3',4,4',5,5',6 - octachlorobiphenyl
206	3	2,2',3,3',4,4',5,5',6 - nonachlorobiphenyl
207	4	2,2',3,3',4,4',5,6,6'- nonachlorobiphenyl
209	4	Decachlorobiphenyl

* - Number of chlorines substituted in ortho positions.

Table 5. PCB congener concentrations in Aroclor mixtures.

Concentrations as mg/g

Congener	Aroclor 1016	1242A	1242B	1242C	1254	1260
15	13.4	16.2	16.6	16.7	0.00	0.00
18	72.1	68.1	70.2	70.0	0.90	1.06
37	13.8	17.2	17.7	17.7	0.24	0.45
44	38.9	31.7	32.5	31.8	29.3	0.49
47	12.0	9.93	10.1	9.93	1.39	0.12
49	30.9	25.9	26.5	26.1	15.9	0.37
52	36.7	31.0	32.0	31.6	52.4	2.03
60	1.10	23.2	22.0	21.7	5.60	3.00
72	0.15	0.10	0.09	0.12	0.00	0.00
77	0.03	2.86	3.00	3.07	0.22	0.01
78	0.00	0.00	0.00	0.00	0.52	0.00
79	0.00	0.00	0.00	0.00	0.00	0.00
81	0.00	0.24	0.27	0.25	0.44	0.00
86	0.11	3.54	3.46	3.53	0.54	0.94
101	0.76	6.20	6.22	6.00	71.1	27.0
105	0.09	4.10	4.27	4.17	54.9	0.28
114	0.00	0.25	0.27	0.27	1.70	0.00
118	0.10	4.22	4.56	4.43	66.4	2.62
119	0.00	0.11	0.11	0.13	0.79	0.05
126	0.00	0.05	0.03	0.02	0.03	0.00
128	0.00	0.19	0.26	0.23	17.2	3.28
137	0.00	0.08	0.08	0.06	6.12	0.25
138	0.07	0.55	0.59	0.53	69.3	76.3
141	0.00	0.12	0.14	0.11	14.8	33.4
143	0.00	0.05	0.04	0.05	5.66	4.03
153	0.17	0.47	0.55	0.46	42.7	85.4
156	0.00	0.06	0.07	0.06	7.92	2.68
157	0.00	0.00	0.00	0.05	2.86	0.14
158	0.00	0.09	0.08	0.07	10.1	6.74
166	0.67	0.84	1.81	1.31	0.61	2.76
167	0.00	0.00	0.03	0.00	2.85	1.13
169	0.00	0.00	0.00	0.00	0.00	0.00
170	0.00	0.02	0.02	0.03	3.54	28.2
180	0.00	0.00	0.00	0.00	10.5	141.
183	0.12	0.14	0.28	0.21	2.77	39.6
185	0.00	0.05	0.13	0.09	0.37	8.83
189	0.00	0.00	0.00	0.00	0.38	1.35
191	0.00	0.00	0.00	0.00	0.25	2.24
194	0.00	0.00	0.00	0.00	0.49	28.9
201	0.00	0.17	0.17	0.00	0.37	43.9
204	0.00	0.08	0.03	0.04	0.00	2.97
205	0.00	0.00	0.00	0.00	0.00	1.46
206	0.00	0.00	0.00	0.00	0.08	6.19
207	0.11	0.00	0.10	0.00	0.00	1.00
209	0.00	0.11	0.14	0.12	0.26	0.15
Sum	222	248	254	251	502	630

Table 6. PCB congener concentrations in the muscle of lobster from New Bedford Harbor.

Concentrations as ng/g dry weight

Congener	Sample # 902177	902178	902179	902180	902181
15	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00
37	4.98	2.23	2.08	2.99	7.09
44	0.00	0.00	0.00	0.00	2.66
47	13.1	8.68	7.80	11.2	24.7
49	0.00	4.80	0.00	0.00	12.7
52	4.32	4.32	0.00	2.9	13.8
60	9.70	10.3	5.92	9.70	27.4
72	0.50	0.50	0.40	0.40	1.00
77	9.52	3.82	3.91	4.99	7.09
78	0.00	0.24	0.00	0.00	0.00
79	0.00	0.00	0.00	0.00	0.00
81	0.53	0.00	0.00	0.00	0.69
86	0.30	1.95	0.20	0.40	6.50
101	14.6	24.6	8.24	13.8	78.7
105	61.5	32.2	31.8	39.4	63.6
114	1.92	1.05	0.95	1.60	2.76
118	237.	124.	120.	146.	220.
119	2.75	3.45	2.57	2.83	11.5
126	1.80	0.42	0.69	0.91	1.13
128	56.3	33.6	39.1	41.7	88.5
137	11.8	6.94	7.15	9.67	19.2
138	186.	127.	142.	127.	271.
141	0.00	3.18	0.00	0.00	6.72
143	0.00	0.00	0.00	0.00	3.46
153	332.	164.	189.	196.	359.
156	15.8	7.93	8.37	9.03	13.7
157	8.97	4.27	4.69	4.89	8.51
158	10.6	8.72	8.62	9.59	17.9
166	0.00	0.00	2.40	0.00	0.00
167	12.9	5.91	6.21	6.75	11.4
169	0.59	0.00	0.27	0.00	0.00
170	11.1	5.09	6.38	6.12	9.84
180	42.4	21.5	24.2	25.8	50.7
183	8.09	7.45	6.89	5.8	15.2
185	0.00	0.00	1.77	0.00	5.15
189	1.15	0.54	0.79	0.77	1.08
191	0.00	0.00	0.20	0.00	3.43
194	3.74	2.98	3.40	0.80	10.6
201	9.60	5.13	5.87	8.18	14.4
204	0.00	0.97	1.10	2.40	2.96
205	0.00	0.00	1.76	0.50	5.43
206	0.00	0.00	0.00	0.00	0.00
207	0.00	2.05	0.00	3.66	5.70
209	0.00	0.00	0.00	0.00	0.00
Sum	1070	630	645	696	1410

Table 7. PCB congener concentrations in the hepatopancreas of lobster from New Bedford Harbor.

Concentrations as ng/g dry weight

Congener	Sample # 902177	902178	902179	902180	902181
15	0.00	0.00	0.00	0.00	64.3
18	0.00	93.8	0.00	0.00	50.5
37	223.	176.	316.	361.	209.
44	0.00	75.8	0.00	0.00	47.1
47	692.	834.	1544	1560	672.
49	55.2	424.	40.4	113.	324.
52	180.	363.	80.8	285.	355.
60	506.	876.	1030	1230	696.
72	22.4	66.7	91.6	70.9	55.1
77	356.	283.	585.	550.	191.
78	0.00	0.00	0.00	0.00	13.9
79	0.00	0.00	0.00	0.00	0.00
81	0.00	17.4	41.0	0.00	13.3
86	0.00	149.	39.8	0.00	142.
101	712.	2250	1460	1700	1780
105	3350	3110	5930	6080	2300
114	114.	96.8	182.	201.	76.9
118	11600	9420	18800	18900	6920
119	134.	301.	455.	308.	254.
126	79.7	63.1	126.	116.	34.9
128	1980	1900	4920	2800	1170
137	586.	534.	1160	783.	304.
138	10900	12000	26100	15400	6810
141	31.0	251.	67.1	92.7	137.
143	0.00	75.1	0.00	0.00	25.1
153	17400	14000	29600	20400	7920
156	830.	760.	1550	1350	505.
157	537.	444.	986.	777.	301.
158	552.	750.	1510	982.	445.
166	84.0	79.7	150.	107.	45.1
167	781.	642.	1360	1110	415.
169	8.24	3.56	7.34	0.00	1.77
170	601.	482.	1300	644.	287.
180	2400	2110	5110	2860	1370
183	428.	643.	1130	652	378.
185	0.00	0.00	0.00	0.00	19.1
189	71.2	54.5	118.	114.	33.0
191	49.8	41.3	111.	123.	24.9
194	242.	213.	447.	340.	128.
201	372.	382.	698.	361.	236.
204	30.6	34.1	63.6	45.4	8.05
205	0.00	22.3	0.00	68.5	0.00
206	105.	84.6	180.	79.4	60.4
207	0.00	0.00	0.00	0.00	0.00
209	0.00	0.00	0.00	0.00	25.2
Sum	56000	54100	107000	80600	34900

Table 8. PCB congener concentrations in the muscle of flounder from New Bedford Harbor.

Concentrations as ng/g dry weight

Congener	Sample # 399568	399569	399572	399574	399575
15	8.42	1.65	0.00	0.00	0.00
18	24.7	31.2	4.10	12.6	40.2
37	53.8	22.0	1.39	2.36	6.96
44	26.5	20.5	1.99	6.90	21.2
47	145.	107.	1.79	6.92	195.
49	151.	237.	5.79	20.4	220.
52	118.	215.	6.45	21.9	115.
60	105.	70.2	2.02	10.2	156.
72	8.69	6.08	0.00	0.30	11.3
77	73.1	36.5	0.73	1.27	44.2
78	0.00	0.00	0.00	0.00	0.00
79	0.00	0.00	0.00	0.00	0.00
81	6.82	4.37	0.00	0.00	5.52
86	1.23	1.04	2.00	4.03	21.1
101	187.	237.	6.76	19.4	255.
105	420.	175.	3.52	12.1	406.
114	19.8	7.06	0.00	0.75	15.1
118	1100	390.	8.05	33.9	1010
119	38.7	15.5	1.12	1.90	37.0
126	6.02	2.52	0.00	0.14	4.46
128	55.8	37.4	2.63	3.57	149.
137	48.7	19.3	0.00	1.83	49.0
138	698.	302.	9.39	32.7	850.
141	51.7	21.1	0.00	2.32	53.0
143	9.92	8.43	0.00	1.14	11.4
153	895.	357.	12.9	45.0	1050
156	70.9	19.9	0.00	2.58	56.4
157	29.3	9.34	0.00	1.57	28.1
158	85.9	34.1	1.08	2.76	83.8
166	4.64	3.58	0.00	0.00	0.00
167	53.7	14.9	0.00	1.98	41.5
169	0.56	0.00	0.18	0.00	0.00
170	30.7	8.90	0.00	1.00	30.5
180	134.	41.4	2.46	6.53	131.
183	45.1	18.4	1.36	2.35	42.7
185	1.99	1.93	0.00	1.31	3.29
189	3.85	1.00	0.00	0.00	3.42
191	3.23	1.58	1.87	1.10	5.58
194	10.5	3.12	0.00	2.71	10.6
201	10.5	4.58	3.23	2.73	13.6
204	2.14	1.58	0.72	1.42	3.68
205	1.15	0.97	0.00	2.30	3.30
206	4.37	3.18	0.00	0.00	6.38
207	1.67	1.17	0.00	1.21	4.45
209	0.94	0.00	0.00	0.00	0.00
Sum	4740	2490	81.5	273.	5190

Figure 1. Map showing the lobster sampling locations.

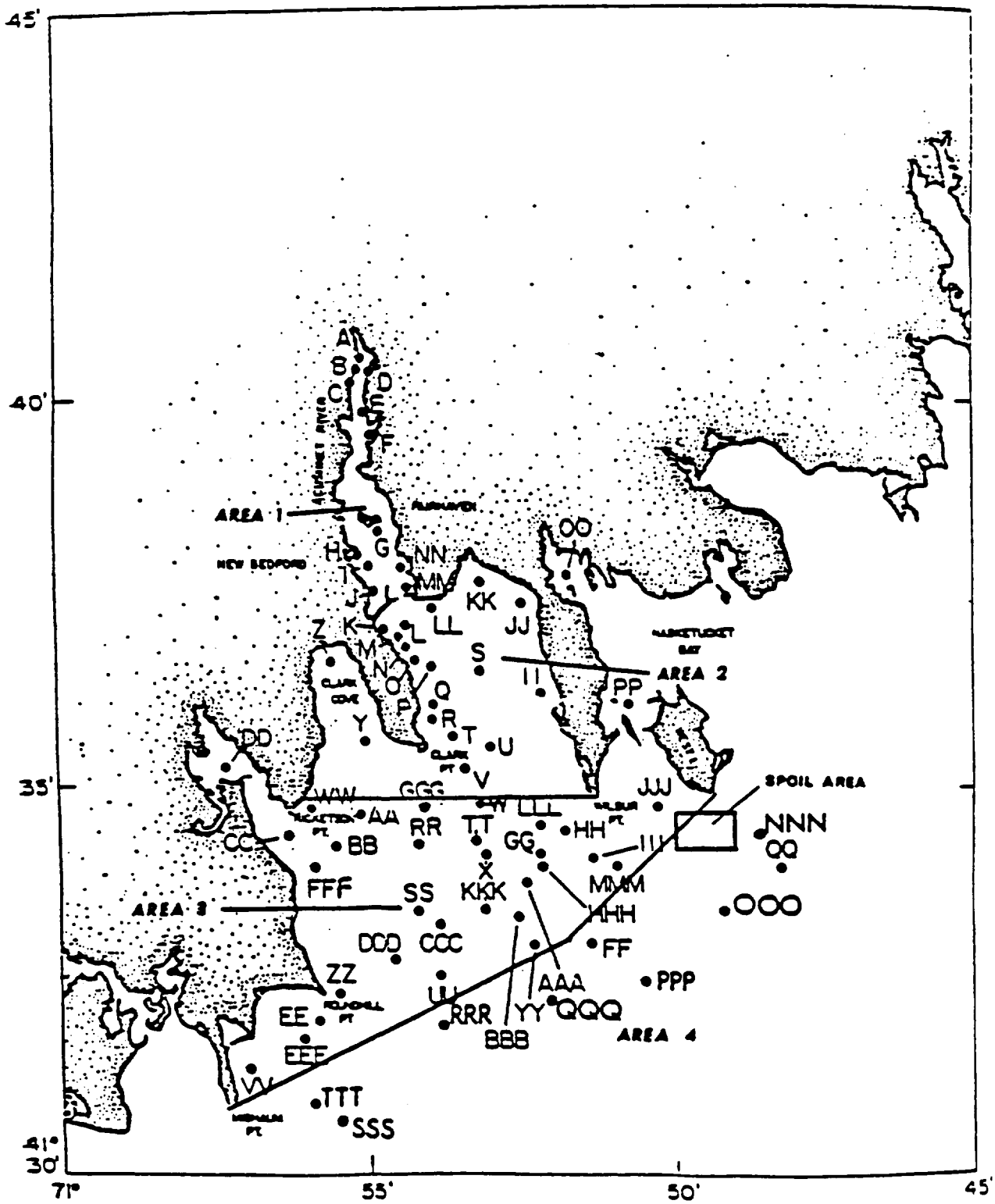
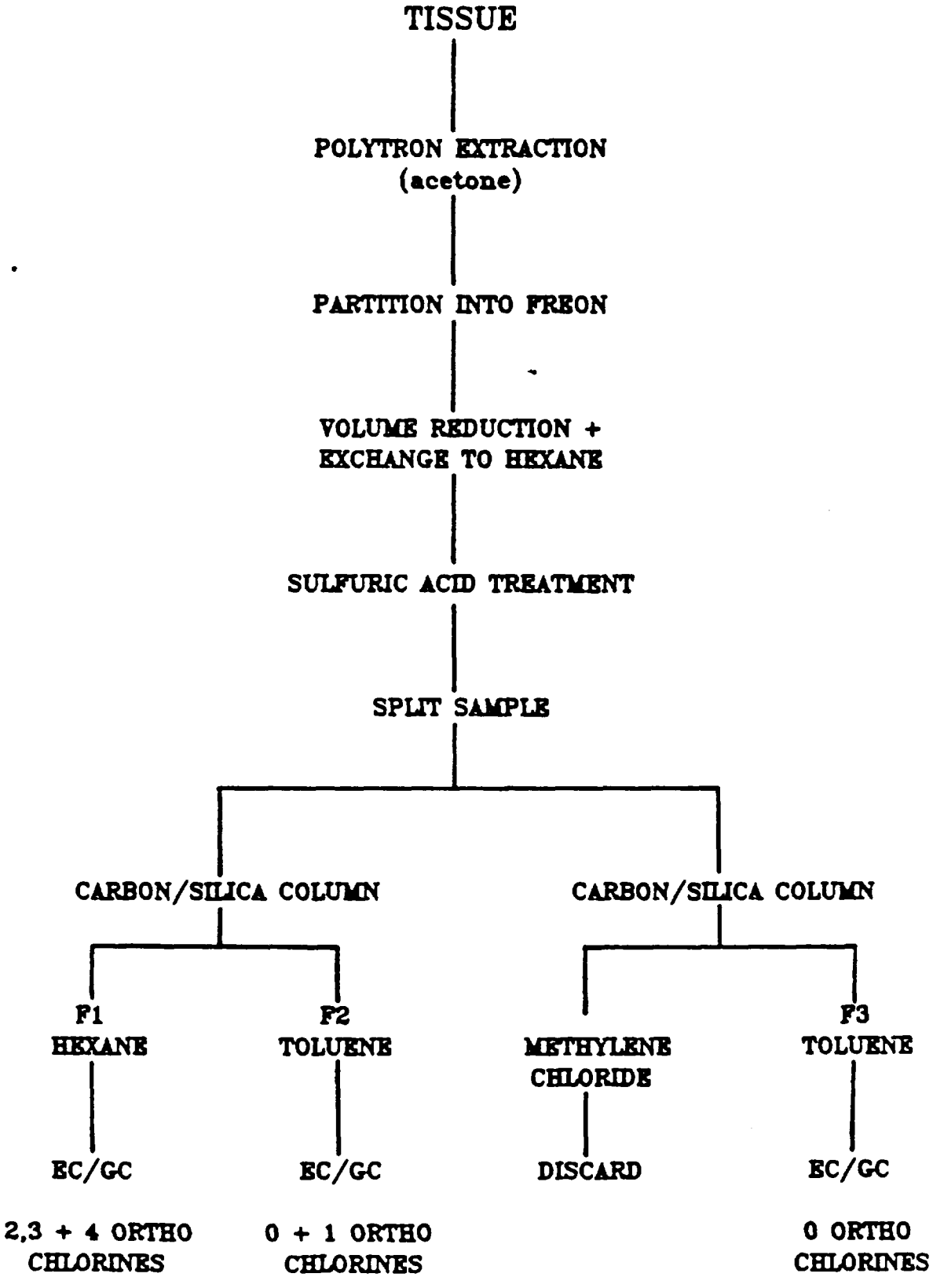
Figure 2. Flow chart of the sample analysis techniques.