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Narragansett Bay Estuary Program

A REVIEW OF BIOLOGICAL EFFECTS OF TOXIC CONTAMINANTS ON ORGANISMS IN NARRAGANSETT BAY

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FOREWORD

The United States Congress created the National Estuary Program in 1984, citing its concern for the "health and ecological integrity" of the nation's estuaries and estuarine resources. Narragansett Bay was selected for inclusion in the National Estuary Program in 1984 , and the Narragansett Bay Project (NBP) was established in 1985. Narragansett Bay was designated an "estuary of national significance" in 1988. Under the joint sponsorship of the U.S. Environmental Protection Agency and the Rhode Island Department of Environmental Management, the NBP's mandate is to direct a program of research and planning focussed on managing Narragansett Bay and its resources for future generations.

The NBP will develop a draft Comprehensive Conservation and Management Plan (CCMP) by December, 1991, which will recommend actions to improve and protect the Bay and its natural resources.

The NBP has established the following seven priority issues for Narragansett Bay:

- management of fisheries
- nutrients and potential for eutrophication
- impacts of toxic contaminants
- health and abundance of living resources
- health risk to consumers of contaminated seafood
- land-based impacts on water quality
- recreational uses

The NBP is taking an ecosystem/watershed approach to address these problems and has funded research that will help to improve our understanding of various aspects of these priority problems. The Project is also working to expand and coordinate existing programs among federal, state and local agencies, as well as with academic researchers, in order to apply research findings to the practical needs of managing the Bay and improving the environmental quality of its watershed.

This report represents the technical results of an investigation performed for the Narragansett Bay Project. The information in this document has been funded by the State of Rhode Island under the R.I. Clean Water Act Environmental Trust Fund (R.I.G.L. 46-12-24.2 (c)) as part of a Cooperative Agreement between RIDEM and the University of Rhode Island. It has been subject to the Agency's and the Narragansett Bay Project's peer and administrative review and has been accepted for publication as a technical report by the Management Committee of the Narragansett Bay Project. The results and conclusions contained herein are those of the author(s), and do not necessarily represent the views or recommendations of the NBP.

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I Introduction

Understanding biological responses to toxic contaminants depends on the extent to which a suite of measurements can link, in a cause and effect sense, the results of chemical analyses and responses at the different levels of biological organization (Underwood & Peterson, 1988; Bayne *et al.*, 1988). The behavior and concentrations of chemicals in the environment define bioavailability and toxic effects (Capuzzo & Kester, 1987; Cairns & Mount, 1990). Measurement of toxic contaminant effects based on different levels of biological organization - biochemical, cellular, physiological, population and community levels - provide information on different aspects: biochemical and cellular effects give insight into the mechanisms of toxic action (Sheehan, 1984; Underwood & Peterson, 1988) and early warning signs (Sastry & Miller, 1981; Capuzzo & Kester, 1987; Giam *et al.*, 1987) while impact end points at the population and community levels are more meaningful for health and ecological success (Oviatt *et al.*, 1984; Cairns & Mount, 1990).

The evidence of many sources and types of contaminant inputs in Narragansett Bay has led to concern about the significance of the inputs to the bay ecosystem. Several studies have been conducted to measure the effects of contaminants on biological systems using various types of field (Widdows *et al.*, 1981; Black *et al.*, 1988; Pratt *et al.*, 1987), laboratory experiments (Sunda & Huntsman, 1990), and experimental ecosystems (Oviatt *et al.*, 1982, 1984; Frithsen *et al.*, 1985; Sullivan & Ritacco, 1988). However more experimental effort is needed to understand the impacts of toxic contaminants in a cause and response sense. In this report we review the existing data about the impacts of toxic contaminants on organisms in Narraganset Bay. Inputs and sources of contaminants and, general effects of pollution on organisms and populations in the bay are discussed first.

Effects of toxic contaminants on bay organisms are discussed second. The actual or potential effects of toxic contaminants are discussed with respect to organisms in the water column and sediments.

II. CONTAMINANT INPUTS AND SOURCES

Sources and amounts of organic, toxic, and pathogenic contaminants to Narragansett Bay are concentrated in the upper bay (Desbonnet and Lee, 1990). Loadings of BOD and pathogens (measured as fecal coliforms) in the upper bay are about 18,500 metric tons per year and 3.6×10^{15} cells per year, respectively. For toxic contaminants, about 565 metric tons of heavy metals, 1726 metric tons of hydrocarbons are carried into the upper bay and about 26 kilograms of PCBs enter the bay annually.

Most of the contaminant sources are centered around the Providence River at the head of the bay. Point and nonpoint sources of contaminants exist in the lower part of the bay, but they cause adverse conditions on a local basis only (Desbonnet & Lee, 1990). The four principal sources of contaminants to the upper bay are rivers, sewage treatment plants, industrial discharges, and combined sewer overflows (Desbonnet & Lee, 1990). Urban runoff (43.2%), publicly owned treatment works (POTWs) (22.5%), and rivers (18.0%) are major sources of heavy metals loading; runoff (48%) and POTWs (37%) are responsible for most of the hydrocarbon loading. Rivers (69%) and POTWs (31%) comprise the total PCBs loading to the upper bay. Rivers are most important for BOD loading (75%) and CSOs are responsible for most of the pathogens (92.2%).

Sources and inputs of contaminants are reflected in the distribution of contaminants in the bay. In general, the gradient seen from the Providence River to Rhode Island Sound of petroleum hydrocarbons, most metals, nutrients, pathogens and organics are apparent in both the water column and

sediments. Distributions of copper and nickel in surface waters of the bay are shown in figures 1 and 2 as examples of this trend. The general downbay gradient for other toxic contaminants are listed in tables 1 and 2. The concentrations of pollutants are also high in some areas of the mid and lower bay, such as Greenwich Bay, Newport Harbor, and Wickford Harbor. In the future with population increases downbay, the harbors and bays adjacent to the main passage may become more contaminated.

III. EFFECTS OF CONTAMINANTS ON ORGANISMS

III.1 Do organisms in Narragansett Bay show harmful effects from contaminants?

Some reports speculate that the fauna in Narragansett Bay are affected by pollution, although they could not distinguish the effects of individual contaminants (Pratt & Bisagni, 1976; Phelps *et al.*, 1981; Widdows *et al.*, 1981; Phelps *et al.*, 1987). Studies with transplanted mussels (*Mytilus edulis*) provided information about effects of contaminants on the health of mussels, as measured by physiological stress indices: scope for growth, oxygen to nitrogen ratio, and growth efficiency. Oxygen consumption rates by gill respiration were significantly higher in mussels from contaminated upper bay stations (Phelps *et al.*, 1981). Lower growth and survival rates, and failure of reproduction was found in the upper bay (Phelps *et al.*, 1987). These results were reflected in population dynamics of mussels; the long-term population dynamics showed a strong relationship with the contaminant gradient along the bay (Phelps *et al.*, 1987).

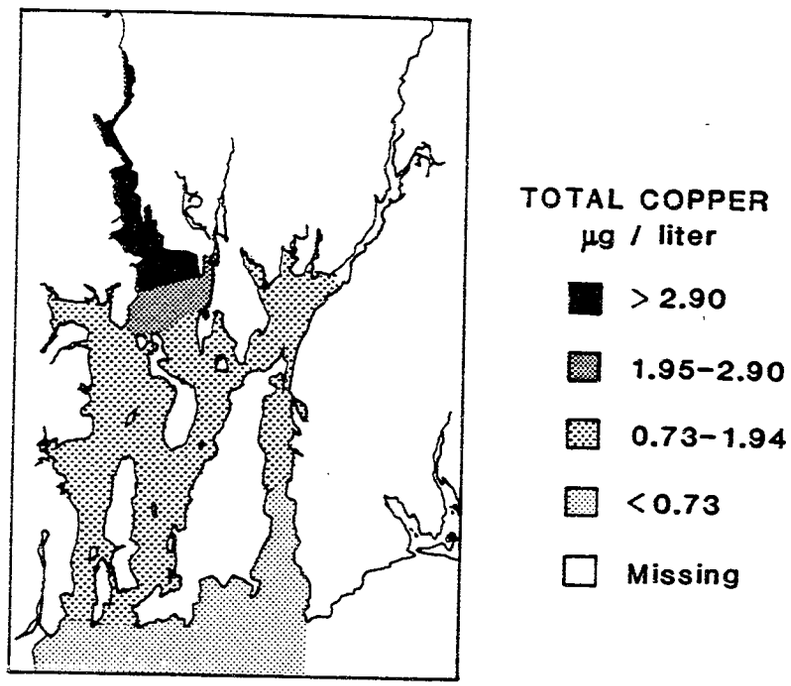

Figure 1. Distribution of copper in the surface water of Narragansett Bay (November, 1985; from Hoffman, 1990).

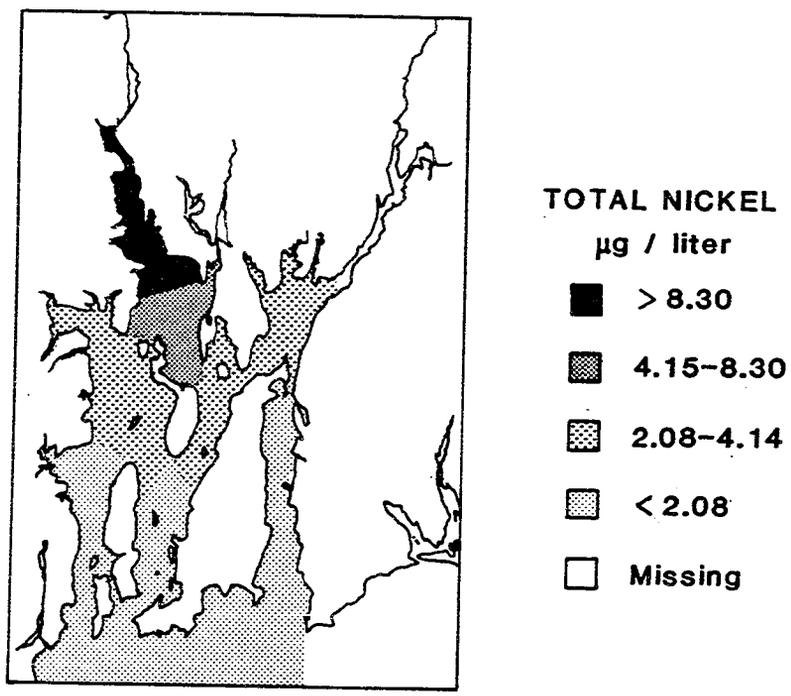

Figure 2. Distribution of nickel in the surface water of Narragansett Bay (October, 1985; from Hoffman, 1990).

Table 1. Concentrations of toxic contaminants in surface sediments from Narragansett Bay (ug/g dry weight).

	Providence River	Upper Bay	Midbay
PCBs (1)	0.6 - 2.3	0.01 - 0.2	0.05 - 0.3
PAHs (2)	1.6 - 19.8	1.0 - 1.4	0.2 - 7.5
PHCs (2)	450 - 9150	454 - 710	112 - 505
Cadmium (3)	1.3	0.9	0.25
Copper (3)	250	211	39
Nickel (3)	38	30	14
Zinc (3)	232	255	87

(1) from Latimer (1989); (2) from Quinn (1989); (3) from Bender *et al.* (1989); Concentrations are shown as a range of the minimum and maximum number observed in the area unless the source provides the average value.

Table 2. Concentrations of dissolved toxic contaminants in surface water from Narragansett Bay (ug/l).

	Providence River	Upper Bay	Midbay
PCBs (1)	0.004	0.003	< 0.001
Cadmium (2)	0.001	< 0.001	< 0.001
Copper (2)	4.6	1.8	1.1
Lead (2)	0.29	0.15	0.02
Nickel (2)	14.8	5.0	2.0
Mercury (3)	0.001 - 0.003	0.001	0.001
Zinc (4)	9.87 - 23.3	0.42 - 3.82	0.25 - 0.36

(1) from Latimer (1989); (2) from Bender *et al.* (1989); (3) from Vandal & Fitzgerald (1988); (4) from Sunda & Huntsman (1990); Concentrations are shown in a range of the minimum and maximum number observed in the area unless the source provides the average value.

In benthic communities, the number of species of macrobenthic fauna was higher at cleaner lower bay stations (Chowder and Marching Society, 1967; Pratt, 1972; Pratt and Bisagni, 1976; Frithsen, 1989). Only 4-5 species of macrofauna were present at the head of the bay (Pratt, 1972). Pratt and Bisagni (1976) concluded that the pollution gradient, and not sediment grain size distribution, was the major factor controlling benthic community distribution. Generally, the species diversity of a community decreases when the ecosystem is under pollution stress (Pearson & Rosenberg, 1978; Carriker *et al.*, 1982; Sheehan, 1984; Weston, 1990) though it may not be a sensitive indicator of pollution in all cases (Gray, 1979; Smith, 1984; Warwick, 1986; Saila *et al.*, 1984). The dominance of opportunistic species, a polychaete, *Streblospio benedicti*, and coot clam, *Mulinia lateralis*, in the upper bay (Frithsen, 1989; Desbonnet & Lee, 1990) also indicated that this area was affected by contaminants. Meroplanktonic benthic larvae were more abundant in the upper bay, as a result of dominance by opportunistic species (Frithsen, 1989). In a histopathological study Kern (1990) found that some stress-induced mortality occurred in quahog populations in Narragansett Bay.

In Narragansett Bay, the communities, with the exception of benthic macrofauna, have not changed over the periods studied (Pratt *et al.*, 1987; Durbin & Durbin, 1988; Hinga *et al.*, 1988), although the pollution level has generally decreased over time. Toxic metals have decreased since the 1950's, especially in the 1980's (Nixon, 1990). PCBs in sediment cores show a maximum between 1950 and the mid 1970's and then a decrease since that time (Latimer, 1989). Hydrocarbon inputs from POTWs in the Providence River have decreased from 226 ± 105 metric tons per year in 1974-1975 to 95 ± 63 metric tons per year in 1985-1986 (Quinn, 1989). In a review of phytoplankton data from Narragansett Bay, Hinga *et al.* (1988) could find no major change in phytoplankton communities over the last 35 years. A survey of zooplankton

community status and trends in the bay showed that it has remained basically the same between 1950 and 1987 (Durbin & Durbin, 1988). Pratt *et al.* (1987) found that the 1985 hard clam distribution pattern in the upper bay was similar to that observed in 1957 and 1965. Abundance of fishes in Narragansett Bay has fluctuated over the past few decades. Decline of fishes in the bay, often suspected to be the result of pollution, showed correlation with temperature change rather than water quality degradation (Jeffries & Terceiro, 1985). Over-fishing was a major culprit in the decline of some resource species.

However in comparing studies on benthic faunal distribution in the bay, Frithsen (1989) found that the dominant macrobenthic species in the midbay, *Nephtys incisa - Nucula annulata*, changed to a *Mediomastus ambiseta - Nucula annulata* dominant community, in the late 1970's. The increase of *Mediomastus* might be an indication of organic enrichment (Frithsen, 1989).

III.2 Toxic contaminant levels in bay organisms

Toxic contaminants carried to the bay are accumulated in organisms by various mechanisms. There have been concerns about the impacts of accumulated toxics on the health of organisms in the bay and the possible risk to human health from consumption of those organisms.

Studies of the levels of toxic contaminants in the organisms in Narragansett Bay have been made on hard clam (*Mercenaria mercenaria*), blue mussel (*Mytilus edulis*), and winter flounder (*Pseudopleuronectes americanus*) (Table 3). The concentrations generally reflect the downbay gradient of contaminant distributions, showing higher levels in organisms from the Providence River. For example, heavy metal and hydrocarbon concentrations in shellfish and PCB concentrations in fish and shellfish were higher in organisms

from the Providence River than in the southern part of the bay.

Levels of contaminants in organisms in the Providence River were below the FDA's alert level for most pollutants. Concentrations of metals, copper, zinc, cadmium and lead, in Narragansett Bay samples were below the proposed FDA alert levels (Bender *et al.*, 1989). Mercury contamination has been a concern in Mount Hope Bay (Desbonnet & Lee, 1990). However, data from Beach *et al.* (1989) indicate its level in hard clams was below the FDA guideline of 1.0 ppm (Table 3). The PCB and mercury concentrations in fish liver were also below the FDA limit (Latimer, 1989). The Narragansett Bay Project proposed alert levels of metals for hard clams based on public consumption rate (Table 4, Hoffman, 1990). The average metal concentrations in hard clams in the bay (Table 3) do not exceed the proposed alert levels. However, occasionally higher individual numbers do occur. Clams from the closed shellfishing area (see Figure 3) showed occasional violations for lead. The worst record of violations occurred for lead in Allens Harbor in the closed area. A few violations of the proposed mercury alert level were detected in Mount Hope Bay. Hoffman (1990) concluded that legally harvested quahogs pose little risk to the clam consuming public. At present there are no guidelines for safe levels of PHCs or PAHs in edible shellfish. So the potential hazards of these substances to human health are still unknown (Quinn, 1989).

Little is known about the actual effects of contaminant levels on organisms, populations, or communities. The concentrations of contaminants in organisms indicate their bioavailability which varies with physical, chemical, and biological conditions of the environment and organisms. However, bioconcentration of contaminants are not necessarily related to toxicity of the contaminants. Behaviors of contaminants in organisms (where, in what chemical forms, and half life in the organism) are as important in contaminant toxicity as bioconcentration. Many organisms have a capacity

Table 3. Levels of toxic contaminants in tissues of blue mussel, hard clam, winter flounder from Narragansett Bay (ug/g dry weight unless noted).

	Mussel	Hard Clam	Winter flounder
Prov. River			
PCBs	0.4-0.95 (1)	0.2-0.6 (2)	0.73* (liver;4)
			0.28* (muscle;4)
PAHs		0.4-0.6 (2)	
PHCs		14-90 (3)	
DDTs		0.02 (3)	
Cadmium		0.96 (6)	0.28* (liver;4)
			0.21* (muscle;4)
Copper		96 (6)	
Lead	6.1 (6)	2.2 (6)	1.0* (liver;4)
			0.66* (muscle;4)
Mercury		0.23 (7)	0.29* (liver;4)
			0.17* (muscle;4)
Nickel	8.7 (6)	8.5 (6)	
Zinc	174 (6)	131 (6)	
Upper Bay			
PCBs	0.4-0.9 (1)	0.2-0.3 (2)	0.8-1.8 (5)
PAHs		0.2 (2)	
DDTs		0.01 (2)	
Cadmium		0.52 (6)	0.7-1.2(5)
Copper		39 (6)	
Lead	4.9 (6)	1.2 (6)	1.0-2.6 (5)
Mercury		0.17 (7)	
Nickel	6.1 (6)	6.5 (6)	
Zinc	150 (6)	102 (6)	
Midbay			
PCBs	0.28-0.48 (1)	0.2-0.3 (2)	
PAHs	0.3-0.4 (9)	0.2-0.4 (2)	
PHCs		2.9-12 (5)	
Cadmium		0.37 (6)	
Copper		14 (6)	
Lead	4.3 (6)	0.6 (6)	
Mercury		0.24 (7)	
Nickel		5.2 (6)	
Zinc	120 (6)	78 (6)	
Mount Hope Bay			
PCBs		0.1-0.3 (2)	
PAHs		0.2-0.4 (2)	
Cadmium		0.7 (7)	
Copper		16.8-20.5 (7)	
Lead		2.5-3.9 (7)	
Mercury		0.4-0.6 (7)	
Nickel		15.3-18.8 (7)	
Zinc		158.6-189.6(7)	

Table 3. Continued.
* indicates wet weight; (1) from Latimer (1989), modified from the original data from Lake *et al.* (1981); (2) from Pruell & Norwood (1989), samples from Nov. 1985 and June, 1986; (3) from Quinn (1989), original data from Pruell *et al.* (1984); (4) from Lee *et al.* (1988), samples from the Warwick Neck; (5) from Oviatt (1989); (6) from Bender *et al.* (1989), values from the sector 4 (midwest passes) were used for midbay; (7) from Beach *et al.* (1989), samples from Nov. 1985 and June, 1986. Averages of the three size groups were used; (8) from NS & T Benthic surveillance Project (1985).

Table 4. Proposed alert levels of metals for quahogs.

Alert level	Cd	Cr	Cu	Hg	Ni	Pb	Zn
Average	1.88*	18.8*	150.5*	0.37*	37.6*	2.26*	752.6*
consumption rate	9.40+	94.0+	752.5+	1.85+	188.0+	11.3+	3763.0+
(18 gm/day)							
High consumption	1.09*	10.9*	87.5*	0.21*	21.8*	1.31*	438.0*
rate (32 gm/day)	5.45+	54.5+	437.5+	1.05+	109.0+	6.55+	2190.0+

* ug/g wet weight (from Hoffman, 1990)
+ ug/g dry weight (dry weight was calculated by multiplying 5 to wet weight).

to keep toxic inorganic metals isolated from cellular sites by forming metalloproteins (Brown *et al.*, 1982; O'Connor & Rachlin, 1982; Jenkins & Brown, 1984). For organic contaminants, many marine organisms can transform xenobiotics to more hydrophilic and excretable metabolites by mixed function oxygenase systems (Payne, 1977; Lee, 1981; Livingstone & Farrar, 1984). Toxic organics may be removed by metabolites and by-products of metabolism of organisms, or by production of particulate products (eggs, molts, and feces) (Capuzzo and Kester, 1987). Therefore, studies of toxic effects should include adverse biological responses to specified levels of bioconcentration based on location, chemical form, and half life (Peddicord, 1984).

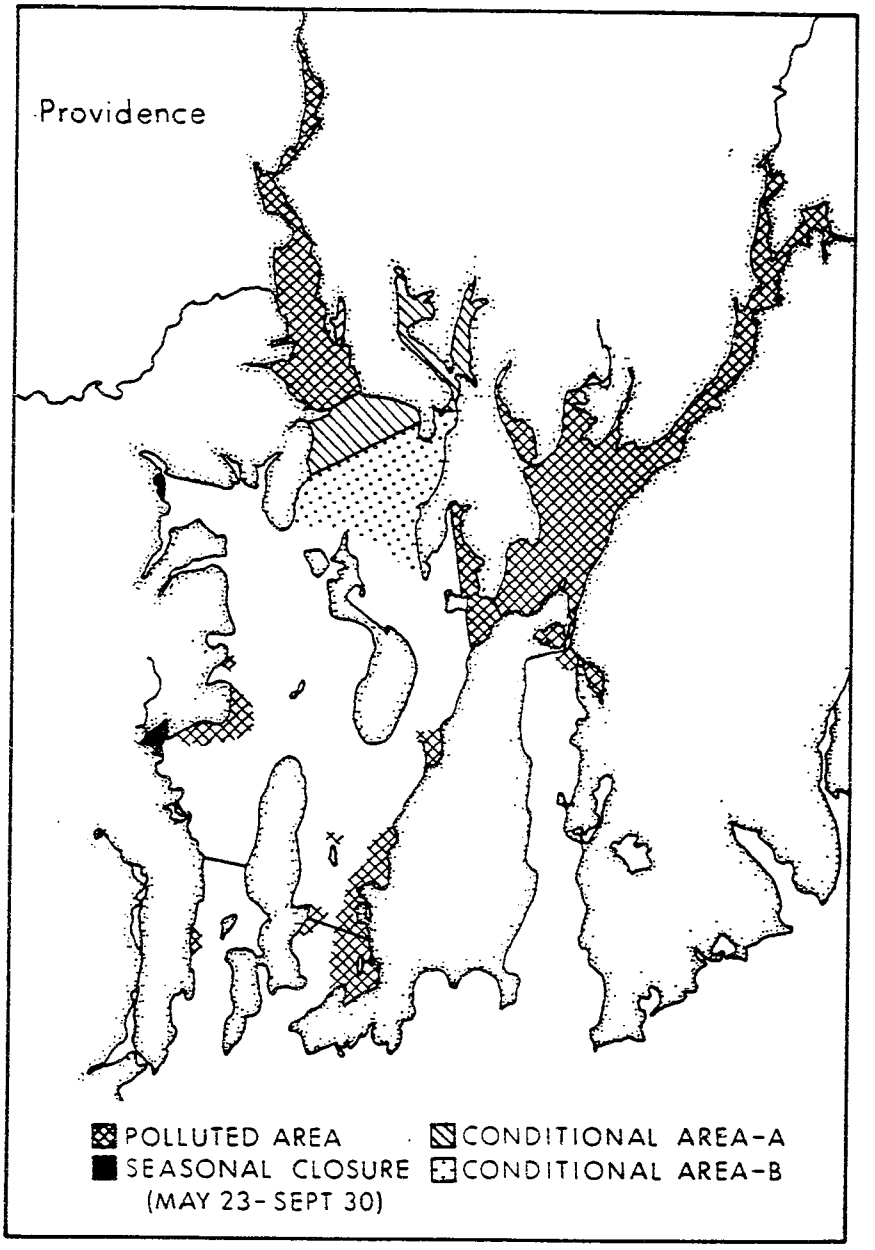

Figure 3. Shellfish closure areas in Narragansett Bay based on fecal coliform concentrations.

Exposures to toxic contaminants seem to reduce the size of larvae of marine fish. Larvae exposed to sulfuric acid, heavy metals, or pesticides, have reduced length (Rosenthal & Alderdice, 1976). In a study on the effect of inherited contaminants in eggs of larval winter flounder, Black *et al.* (1988) found that there were significant correlations between PCB content and larval size. Higher PCB levels in eggs from New Bedford Harbor (39.6 ug/g dry weight) than in the eggs from Fox Island in Narragansett Bay (1.08 ug/g dry weight) were reflected in newly hatched larval size. Larvae from New Bedford Harbor females were 6% smaller in length and 18% smaller in weight than larvae from Fox Island.

Studies of the impacts of contaminant levels in marine organisms have focused on histopathological conditions of organisms, especially on fish (Murchelano & Wolke, 1985; Zdanowicz *et al.*, 1986; Lee *et al.*, 1991). In Narragansett Bay, an incidence of about 3.5% of biliary hyperplasia and 13% of melanin-macrophage center proliferation was found (Zdanowicz *et al.*, 1986). Correlations of liver lesions and contaminant burdens were investigated for winter flounder from three sites of different contaminant levels in Narragansett Bay (Lee *et al.*, 1991). Hepatic macrophage aggregate parameters were significantly increased in fish from Warwick Neck, the site of severest contamination, when compared to fish from other sites. And their occurrence was related with muscle PCB levels and liver cadmium levels. Percent area of hepatic aggregate was also weakly correlated with both liver and muscle PCB burdens. A strong relationship was found between occurrence of hepatic vacuolar cells and muscle PCB levels. Another study conducted in the bay (Murchelano & Wolke, 1985) found that 10% of the winter flounder and windowpane flounder (*Scophthalmus aquosus*) from upper Narragansett Bay and 3.4% of them from New Haven Harbor (in Connecticut) had preneoplastic and neoplastic hepatic lesions in liver. They concluded that more study was needed to establish specific cause-and-effect relationships.

However, they suggested a possible relation of the lesions to toxins by indicating that the morphological changes found were comparable to results from experiments with carcinogens in rodents. In Narragansett Bay, fin lesions, pigment spots or black spot, were observed primarily on fins of the juvenile winter flounder (Buckley & Caldarone, 1988). But the incidence of those fin lesions in the bay do not seem to be related to toxic contamination. These lesions occurred most frequently in fish from the clean mid- and lower bay. Host responses to larval trematode invasion were attributed as the cause of the disease (Buckley & Caldarone, 1988).

Table 5 shows levels of various contaminants in benthic fish livers from some of the study sites for the Benthic Surveillance Project by NOAA (1987). In general, most of the contaminant levels in fish livers from Narragansett Bay, except for lead, PCBs, and zinc, showed moderate levels compared to values from the about 50 U.S. sites examined in the project. Lead level was ordered 6th in the 42 samples in which lead was detected. PCBs and zinc levels ranked 14th and 17th, respectively (Table 5).

As a part of the National Status and Trends Program conducted by NOAA, contaminant levels in mussels (*Mytilus edulis*) were investigated from about 150 sites in the United States. Tables 6 and 7 summarize the data from nine sites of the Mussel Watch Program and from some coastal areas or estuaries of foreign countries. Effects of accumulated toxic contaminants in mussels were investigated in some studies (Martin *et al.*, 1984; Phelps *et al.*, 1987). Physiological condition of mussels from several coastal and bay sites in California showed strong correlation with increased body burdens of six trace elements (chromium, copper, mercury, silver, aluminum, and zinc) and two chlorinated hydrocarbons (chlordanes and dieldrin) (Martin *et al.*, 1984). They also showed that physiological condition of resident *Mytilus californianus* along the California coast was statistically correlated with increased concentrations of petroleum

Table 5. Contaminant levels in benthic fish livers from some coastal areas. Data from Benthic Surveillance Study of the National Status & Trends Program, NOAA (1987); concentrations in ug/g dry weight.

	PCBs	DDTs	Cd	Cr	Cu	Pb	Hg	Ni	Ag	Zn
Narragansett Bay, RI	2.2 (14)*	0.11 (26)*	0.54 (25)*	0.08 (31)*	22.46 (23)*	1.02 (6)*	0.30 (18)*	0.22 (34)*	0.31 (21)*	130.04 (17)*
Boston Harbor, MA	10.5	0.83	0.14	0.26	15.10	0.63	0.12	0.29	0.71	86.08
Buzzards Bay, MA	2.8	0.06	0.54	0.17	15.70	1.16	0.21	0.30	0.35	133.77
Salem Harbor, MA	3.0	0.58	0.08	0.75	10.46	0.53	0.09	0.38	0.51	81.79
Eastern LI Sound, NY	1.6	0.05	2.03	0.11	53.80	0.61	0.42	0.89	0.74	170.80
Western LI Sound, CT	3.2	0.11	1.92	0.43	29.70	1.39	0.51	2.80	0.60	128.20
Chesapeake Bay, VA	1.1	0.14	0.50	0.90	22.23	0.21	0.46	0.86	0.19	145.30
Casco Bay, ME	1.0	0.05	1.14	0.24	68.98	7.40	0.26	0.52	1.58	184.10
Delaware Bay, DE	1.0	0.23	0.15	0.06	17.81	0.19	0.46	1.10	0.22	106.23
Santa Monica Bay, CA	5.9	7.46	2.13	0.30	27.03	0.12	0.29	-	2.07	167.00

*; ranks of the concentrations from Narragansett Bay from about 50 Benthic Surveillance Project sites.

Table 6. Concentrations of contaminants in mussels from some coastal areas. Data from Benthic Surveillance Study of the National Status & Trends Program, NOAA (1987); concentrations in ug/g dry weight.

	PCBs	PAHs	DDTs	Cd	Cu	Pb	Hg	Ni	Ag	Zn
Narragansett Bay	0.2	0.3-0.4	0.01-0.03	1.3-1.9	11.0-14.3	2.9-6.1	0.1	3.4	0.1-0.3	92-113
NY bight	0.7-4.2	1.3-2.6	0.13-0.27	2.1-4.2	0.3-12.7	2.5-6.0	0.2	20.-2.2	0.2-0.7	90-143
Eastern LI Sound	0.4-1.4	0.9-5.3	0.03-0.22	3.0-4.6	11.7-16.7	2.2-7.2	0.1	1.4-2.3	0.1-0.4	106-143
Western LI Sound	0.5-1.0	0.2-0.8	0.04-0.11	2.2-5.7	10.1-20.3	2.5-3.9	0.1	1.2-4.2	0.1-0.5	97-146
Boston Harbor	0.5-1.7	1.5-5.9	0.06-0.13	0.8-1.7	10.3-13.3	4.4-13.0	0.2-0.3	0.7-2.7	1.2-1.7	110-136
Hud/Rar Estuary	1.0-4.3	3.3-20.2	0.17-1.11	3.3-9.3	10.0-16.7	3.2-14.0	0.3-0.5	1.2-4.6	0.5-2.0	84-133
San Diego Bay	2.1	4.4	0.11	6.5	17.0	4.4	0.2	2.5	0.1	273
San Francisco Bay	0.5-0.7	0.6-1.4	0.13-0.32	5.7-7.0	8.0-8.6	0.5-0.8	0.3	3.3-3.9	0.4-0.5	76-77
S. Puget Sound	0.1	2.2	0.01	2.4	8.2-8.7	0.7-0.9	0.1	0.8	0.1	140

Table 7. Concentrations of contaminants in mussels from some coastal and estuarine areas.

	PCBs	PAHs	DDTs	Cd	Cu	Pb	Hg	Ni	Ag	Zn
Medway Estuary, UK (1)	0.07-0.16*									
(2)				0.7-1.2*	1.9-2.3*	0.8-2.2*	0.1-0.3*			31-42*
Liverpool Bay, UK (3)	0.02*		0.02*							
Atlantic coast of Spain & Portugal (4)				1.7-3.6	6.5-14	2-5				190-370
NW Mediterranean Coast (5)	0.23		0.09							
(6)				1.9	18.0	21.5		4.3	0.76	209
Trondheimsfjorden, Norway (7)				1-5	5-88			6-43	1-6	85-359
Beilgian Coast (8)				0.6-2.4	7.9-10.9	2.4-4.5	0.24-0.49			110-175
St Lawrence Estuary, Canada (9)				1.54		1.4				
Coast of New Zealand (10)				0.3-1.6*	1.7-18.0*	0.1-2.0*	0.02-0.48*			3.8-26.0*
Port Davey, Tasmania (11)				0.16-0.22*	0.8-19.6*	0.3-4.1*				22.1-26.7*
SE Australia (12)				0.2-1.1*	1.0-4.9*	0.5-6.5*		1.0-5.4*		12.7-70.6*
Northern Ireland (13)				1.6-3.7	6.1-12.3	3.1-13.2	0.1-2.2	1.9-4.6		66.1-212.5
Coast of Thailand (14)	0.01-0.1		0.02-0.41	0.38	7.3	0.11	0.02			14.2
Gulf of La Spezia, Italy (15)				2.0-0.8	6.9-33.7	15.1-32.4	0.15-0.38	1.3-10.9		203-355

* Wet weight: (1) Wharfe & Van den Broek (1978); (2) Wharfe & Van den Broek (1977); (3) Riley & Wahby (1977); (4) Stenner & Nickless (1975); (5) Marchand *et al.* (1976); (6) Fowler & Oregion (1976); (7) Lande (1977); (8) Meeus-Verdinne *et al.* (1983); (9) Cossa & Bourget (1980); (10) Niesen & Nathan (1975); (11) Thomson (1979); (12) Wootton & Lye (1982); (13) Gault *et al.* (1983); (14) Huschenbeth & Harms (1975); (15) Capelli *et al.* (1975)

hydrocarbons and PCB concentrations in tissues. In Narragansett Bay, increased metallothionein induction and high tissue levels of copper were detected from the mussels transplanted to the upper bay (Phelps *et al.*, 1987).

III.3 Potential effects of toxic contaminant levels in water and sediments of Narragansett Bay

Researchers on toxic contaminants in Narragansett Bay have speculated that the contaminant levels in the bay environment could cause harmful effects on bay organisms (Bender *et al.*, 1989; Latimer, 1989; Quinn, 1989). However, the toxic effects or potential toxic effects of any specific contaminant on bay organisms have not been studied. For levels in the water column, all the measured toxic contaminants except for copper and nickel are below the chronic effect limits of EPA's water quality criteria. Copper and nickel concentrations were above the criteria in the Providence River area (Tables 2 and 8). The average copper concentration (4.67 ug/l) and the highest concentration (6.69 ug/l) observed in the surface water of the Providence River were 61% and 130% higher than the EPA's standard, respectively (Hoffman, 1990). For nickel, the average concentration in the Providence River surface water (12.06 ug/l) was 42% greater than the criterion for nickel, and the highest concentration observed (23.55 ug/l) was 177 greater than the criterion (Hoffman, 1990).

III.3.1 Toxic metals

Toxicity assays on individual organisms have usually been done in laboratory experiments. Figure 4 shows the ranges of metal concentrations that cause adverse effects on marine organisms in laboratory tests and the concentrations of metals in Narragansett Bay. Concentrations of cadmium, lead and mercury in the bay are below concentrations expected

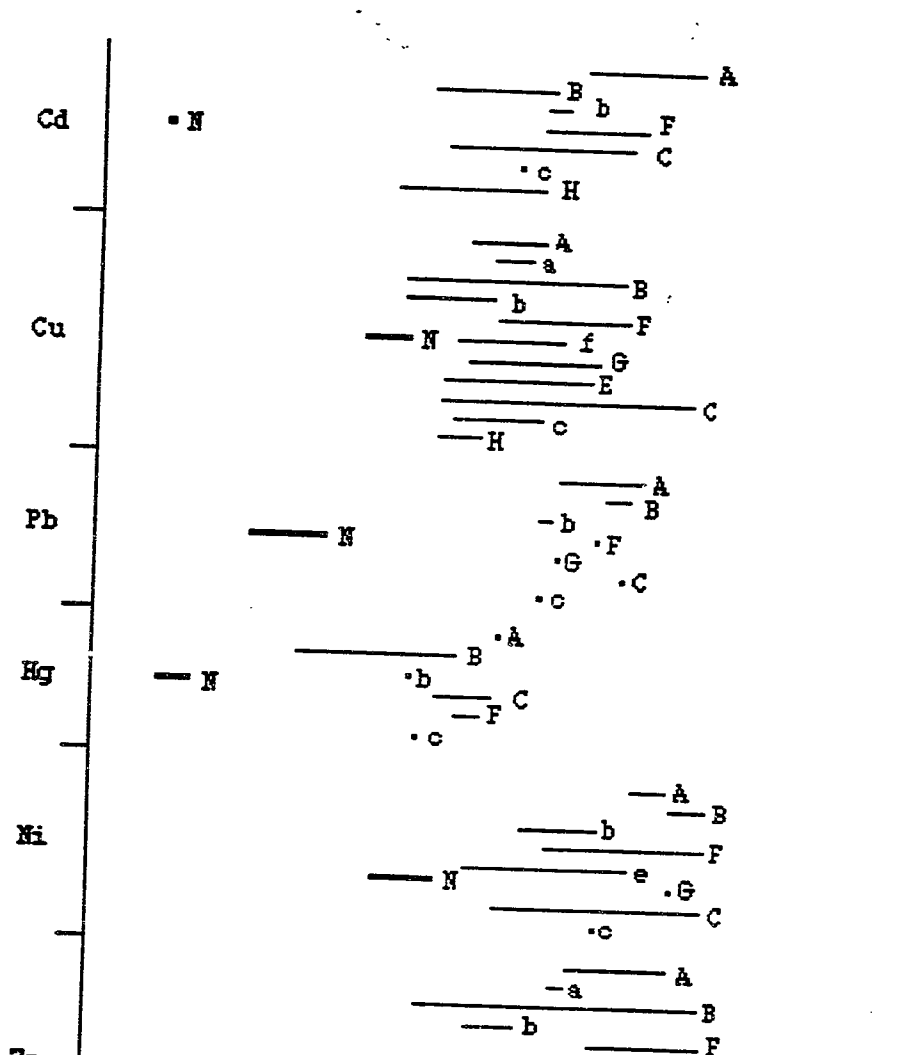

Figure 4. The range of observed adverse effects from laboratory tests for each metal for each main taxonomic group. A=annelids, a=larval annelids, B=bivalve molluscs, b=larval bivalves, C=crustaceans, c=larval crustaceans, E=echinoderms, e= larval echinoderms, F=fish, f=larval fish G=gastropod molluscs, H=hydrozoans. The levels of dissolved metals in surface water of Narragansett Bay are shown N (modified from Mance, 1987).

to cause harmful effects in laboratory tests. Mercury has been known as the most hazardous metal to marine life (Calabrese *et al.*, 1982), but the concentration of mercury in the water of Narragansett Bay is far below the chronic toxicity level of the water quality criterion from EPA (Tables 2 and 8). Concentrations of cadmium and lead in the bay are also well below toxic criterion levels.

Many field and laboratory studies have shown toxic effects of copper on organisms at the concentration of copper found in the Providence River and upper bay area. Recent bioassay experiments with sperm of the purple sea urchin (*Arbacia punctulata)* detected free copper ion toxicity in the Providence River and at Newport Harbor (Sunda & Huntsman, 1990). Reduction of phytoplankton production was detected below 5 ug/l of copper concentration in several studies (Rajendran *et al.*, 1978; Sunda *et al.*, 1981; Breteler *et al.*, 1984). Breteler *et al.* (1984) reported that measurable inhibition of photosynthesis for a phytoplankton community was found at copper concentrations of 2.8 ug/l. At concentrations above 4 ug/l of copper, nanoplankton photosynthesis decreased markedly (Rajendran *et al.*, 1978). Reduced ^{14}C fixation by phytoplankton was detected as low as 0.3 ug/l of copper (Sunda *et al.*, 1981). For a zooplankton community, copper toxicity was found with additions of <5 ug/l (Beers *et al.*, 1977). *Acartia clausi* (hudsonica), one of the dominant zooplankton species in Narragansett Bay, decreased ingestion rate at 1 ug/l of copper (Moraitou-Apostolopoulou & Verriopoulous, 1979). Of the bivalves, blue mussel (*Mytilus edulis*) showed a significant reduction in growth rate at the concentration of 3 ug/l copper (Strömgren, 1982). In long term exposures, the bay scallop died at 5 ug/l (US EPA, 1986). Marine fish are generally known as being less sensitive to heavy metals than other aquatic organisms (Langston, 1990). Growth inhibition of plaice (*Pleuronectes platessa*) was observed at 10 ug/l of copper (Saward *et al.*,

Table 8. Quality criteria for water 1986, (ug/l) (U.S. EPA, 1986).

	PCBs	DDTs	PAHs	Ag	Cd	Cu	Pb	Hg	Ni	Zn
Acute	10	0.13	300	2.3	43	2.9	140	2.1	75	95
Chronic	0.03	0.001	NA	NA	9.3	2.9	5.6	0.025	8.3	86

NA: not available

1975), which is about twice as much as the average concentration observed in the Providence River (Table 2).

Toxic effects other than reduced growth were detected at similar ranges of copper concentrations (Martin *et al.*, 1981; Strömgren, 1982; Bengtsson & Larsson, 1986). A concentration of 5.3 ug/l copper caused 50% abnormal development for pacific oyster (*Crassostrea gigas*) larvae (Martin *et al.*, 1981). For mussel (*Mytilus edulis*), 50% of larvae developed abnormally at 6 ug/l (Martin *et al.*, 1981). Increased vertebral deformities were observed in fourhorn sculpin (*Myoxocephalius quadricornis*) at 0.8 ug/l of copper (Bengtsson & Larsson, 1986). At a copper concentration above 5 ug/l, an apparently permanent closing of the valves was observed in mussel, *M. edulis* (Strömgren, 1982).

Nickel levels in the Providence River area are above the chronic toxicity level (Tables 2 and 8). Phelps and Galloway (1980) found an inverse relationship between scope for growth of the mussels transplanted between sites within the bay and the nickel concentrations in the water column and tissues of mussels. However, a laboratory test with an exposure of 1000 ppb Ni did not show significant physiological stress on the mussel (Phelps *et al.*, 1981). Few studies have been made on marine or estuarine organisms at nickel levels similar to those of Narragansett Bay and possible chronic effects of nickel can not be predicted. A study of heavy metal effects

on filtration rates of the bivalve *Villorita cyprinoides* showed that the rates decreased exponentially as the concentration of nickel increased from 1 ug/l to 5 ug/l (Abraham *et al.*, 1986).

We do not know how these field and laboratory results are actually reflected in ecosystem of Narragansett Bay because effects of contaminants are controlled by relationships among chemical and biological factors such as chemical speciation of contaminants, effects of transfers in food web, toxification and detoxification effects, and evolution of resistance, *etc.* (Klerks & Levinton, 1988). As one of the ways for understanding behavior and effects of toxic substances in the Narraganset Bay ecosystem, experimental enclosure studies have been conducted at the University of Rhode Island (Santschi *et al.*, 1983; Frithsen, 1984; Sullivan & Ritacco, 1988, etc. for metals; Elmgren *et al.*, 1980; Oviatt *et al.*, 1982; Frithsen *et al.*, 1985, *etc.* for petroleum hydrocarbons, for example). For heavy metals, studies have focused on fates in the ecosystem with the exception of a study on copper. Additions of 40 ug/l of $CuSO_4$ to control and nutrient-enriched mesocosms had a toxic effect on the juvenile stages and to a lesser extent, on adult copepods (Sullivan & Ritacco, 1988). Effects of copper toxicity on copepods and recovery of zooplankton populations were related to the degree of nutrient enrichment.

The Apparent Effects Threshold (AET), which is the sediment concentration of a contaminant above which statistically significant biological effects would always be expected, from Puget Sound data was selected as the most appropriate criteria for evaluating the contaminant levels in Narragansett Bay (Hinga, 1990). Based on data on metal distribution in Narragansett Bay, Hinga compared the metal concentrations in the bay with 1988 Puget Sound AET (AET values for Puget Sound data are summarized in the report by Long and Morgan, 1990). Several stations in the Providence River area showed higher metal concentrations (copper,

cadmium, chromium, and zinc) than AET values. Hinga also indicated that metal concentrations in large portions of Narragansett Bay were just above or just below the concentrations where harmful effects on benthic organisms might be expected.

III.3.2 Organotoxic Contaminants

According to Narragansett Bay Project Progress Reports (1985 - 1986), liver damage may be the best indicator of environmental stress in adult winter flounder. Occurrences of hepatic lesions (liver damage) in winter flounder were reported from the Boston Harbor area (Murchelano, 1988). Suspected causes of the lesions were the high concentrations of PCBs and PAHs in sediments of this area. The levels of PCBs and PAHs in Boston Harbor sediments are 70 - 330 ppb (Boehm *et al.*, 1984) and 2.4 - 6.5 ppm (Murchelano, 1988), respectively. The levels of these two toxics in the Providence River area (Table 1) were within the range of those in Boston Harbor, and this suggested that occurrence of hepatic diseases in flounder might have occurred in this area. Liver damage was correlated to contaminant concentrations in winter flounder from Narragansett Bay (Murchelano & Wolke, 1985; Zdanowicz *et al.*, 1986). However, the specific causes for the liver lesions have not been identified. As mentioned (in section III.2), a recent study conducted in the bay showed some correlations between occurrences of liver diseases in winter flounder and cadmium and PCB levels in their tissues (Lee *et al.*, 1991). They collected samples from three sites (Warwick Neck, Whale Rock, and Quonochontaug Pond) which they presumed to represent a pollution gradient of the environment. However, because contaminant levels in the environment were not investigated in the study, it is not known whether the levels of cadmium

and PCBs in sediments and water column were also related to occurrences of diseases in winter flounder.

In a study of neoplastic and non-neoplastic disorders in winter flounder from the New England area, Gardner *et al.* (1989) suggested a causal relationship between degree of sediment chemical contamination and diseases in fish. Specific type of pollutants responsible for the diseases were not revealed. In this study more than 20% of flounder from Fox Island and more than 50% of the flounder from Gaspee Point in Narragansett Bay showed hepatocytic vacuolation. Marlins *et al.* (1984) showed positive correlations between chemical concentrations (hydrocarbons and metals) in sediments and diseases in fish from an urban area in Washington State. The actual causes of the observed diseases were unknown. Buckley and Caldarone (1988) found a parallel between the organic pollution gradient and the incidence of *Glugea* cycts, a pathogen, in winter flounder from Narragansett Bay and recommended further investigation.

Studies on other biological effects of PCBs in sediments showed toxicity of PCBs at concentrations found in Narragansett Bay. Moderate to high toxicity of PCBs to oyster larvae in Commencement Bay were found at 140-368 ppb dry weight (Tetra Tech, 1985). In Puget Sound, toxicity to an amphipod, *Rhepoxynuis abronius*, was found at 259-276 ppb dry weight of PCBs (Dewitt *et al.*, 1988). Chapman *et al.* (1987) set the sediment quality triad minimum (no biological effects) for PCBs to less than 100 ppb dry weight.

For total hydrocarbons toxicity of each hydrocarbon component varies (Neff, 1979; Oviatt *et al.*, 1982; Moore & Farrar, 1985). However harmful effects of total hydrocarbons were revealed in very low concentrations by several studies. In a review of ecological effects of petroleum and petroleum products on marine life, Hyland and Schneider (1976) concluded that sublethal responses, regardless of life stage, indicated that oil would adversely impact certain ecologically and commercially important species in the 1 to

10 ppb water column range. Neff (1979) found that potentially detrimental sublethal responses to PAHs occurred at ambient concentrations substantially lower than those which caused acute toxicity. In a recent study, Payne *et al.* (1988) found effects in flounder at exposures of 1 ug/g dry weight hydrocarbon concentrations in sediment. Liver size and fat content in winter flounder were correlated to concentrations of 1 ppm PAHs in sediments.

Mesocosm experiments were conducted to determine the effects of number 2 fuel oil on the coastal marine environment (Elmgren *et al.*, 1980; Grassle *et al.*, 1981; Vargo, 1981; Elmgren & Frithsen, 1982; Oviatt *et al.*, 1982; Vargo *et al.*, 1982; Frithsen *et al.*, 1985, *etc.*). The results showed severe impacts on benthic fauna and less dramatic effects on the water column community. Chronic, low level additions of No.2 fuel oil (180 ppb in Elmgren *et al.*, 180 and 90 ppb in Oviatt *et al.*, 1982 and Frithsen *et al.*, 1985) significantly reduced benthic macro- and meiofaunal populations while phytoplankton were relatively insensitive to these concentrations. Doses of 190 ppb concentration of no.2 fuel oil affected the zooplankton community as well as benthic fauna (Oviatt *et al.*, 1982). If we extrapolate from these data, then the impoverished benthic communities in the Providence River and the northern part of Narragansett Bay, where chronic water column concentrations of 100 ppb and sediment concentrations of 500 ppm are commonly found, might be the consequence of hydrocarbon inputs (Oviatt *et al.*, 1982).

IV. SUMMARY AND RECOMMENDATIONS

Several studies have found that the condition of organisms, populations, and communities in the Providence River or upper bay might be related to contamination. However, most of them could not attribute the condition to a

specific cause. Higher levels of toxic contaminants in organisms, water column and sediments in this area compared to other areas of the bay, might indicate a relationship between toxic contamination and the condition of the organisms. Toxicity tests conducted on a few contaminants support this idea. However, little is known about the impacts of specific toxic contaminants on the health of marine organisms in a cause and effect manner.

In this report, we compared the toxic contaminant data on bay organisms, water column, and sediments with results from several field and laboratory experiments on toxic effects of contaminants. This may not be the best approach because of the different chemical and biological conditions of those experiments compared to the conditions in Narragansett Bay. Nevertheless, this analysis suggests which contaminants have a higher potential for exerting harmful effects. Copper may be one of the most important candidates for toxic action to organisms in the bay. Toxicity of free copper ion was detected in the Providence River. Many experiments showed toxic effects of copper on phytoplankton, zooplankton, and bivalves below the concentrations observed in this area. Concentrations of PCBs in the Providence River fall within the ranges which showed a relationship to diseases in fish. Correlations between their level in fish tissue and pathological condition in the bay suggest a link between PCBs and the health of fish in the bay. Petroleum hydrocarbons may be important in terms of their chronic effects on community structure of the Providence River and upper bay. Future research on toxic effects of contaminants in Narragansett Bay need to focus on cause and effect relationships between the health of the bay organisms and these three contaminants.

IV.1 Recommendations

1) Toxic contamination is still a problem in the Providence River, upper bay and Mount Hope Bay areas. Efforts to reduce the toxic contaminants by point and nonpoint sources should be continued. Contaminants of particular concern include copper, nickel, cadmium, chromium, zinc, PCBs, petroleum hydrocarbons, and polynuclear aromatic hydrocarbons.

2) Although we do not know the impact of specific toxics on biota in problem areas, the weight of evidences suggests a problem may exist. A practical field monitoring plan might concentrate on hard clam and winter flounder populations which have been well studied. Tissue and sediment concentrations of metals and contaminant organics over time would indicate whether source controls were reducing contaminant loading to the system. Previous mussel studies have indicated that growth, survivorship, fecundity, gill respirometry, scope for growth and population dynamics were useful for stepwise assessment (Phelps *et al.*, 1987).

3) We recommend enclosure studies on whole communities to assess the impacts of contaminants of concern (particularly copper and hydrocarbons). In mesocosms or smaller, shorter term "boxcosms" impacts could be assessed on communities which include various life stages of hard clams and winter flounder so that field and experimental work were coordinated. This approach would sort out the problem of multiple toxic impacts to concentrate on those most likely having adverse impacts. Previous mesocosm work has amply demonstrated the difficulty of applying any kind of single species information whether it is toxicant body burdens, scope for growth or pathologies to response at the population level, never mind community level. By contrast these ecosystem level experiments provide such information directly.

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