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Contaminant Effects on Chesapeake Bay Finfishes

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CONTAINMENT PROBLEMS AND MANAGEMENT OF LIVING CHESAPEAKE BAY RESOURCES

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Chapter Fifteen

CONTAMINANT EFFECTS ON CHESAPEAKE BAY FINFISHES

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ABSTRACT

Habitat deterioration is consistent with perceived population declines for several resident and anadromous finfish species in Chesapeake Bay that are subjected to different levels of fishing pressure (e.g., striped bass versus blueback herring). Diminution of habitat quality has natural and anthropogenic roots that are difficult to separate. Recent contaminant effects studies focused on Chesapeake Bay fishes can be grouped as follows: (a) mathematical and statistical modeling studies aimed at elucidating contaminant and stock trend relationships using extant data and theoretical insights, (b) biological and chemical field surveys in selected areas to demonstrate spatio-temporal associations between levels of toxic organic and inorganic chemicals and absence or reduction of sensitive species, (c) measurements of condition factors and tissue residues of chemical contaminants in juvenile and older fishes, (d) laboratory studies of life stage and species sensitivities to an array of toxic contaminants, and (e) in-situ field studies designed to measure the effects of habitat quality on specific life stages of selected species. Contaminant-related research has focused primarily on striped bass, American shad, and river herrings. Two currently intensive areas of investigation are the leaching of tributyltin (TBT)

antifouling paints into marina areas and acidic deposition in freshwater coastal plain tributaries. These recent studies collectively support several tentative conclusions that deserve further study: (a) deterioration of spawning and nursery habitats in Chesapeake Bay, via the influx of toxic contaminants, may be contributing to poor recruitment in some anadromous and resident finfishes, (b) larvae and newly transformed juveniles are more sensitive to most contaminants than embryos and older life stages, (c) recent adverse effects (likely the past 20 years) of contaminants on juvenile production in several finfish species may not be related to historical variations in stock abundance, but could be responsible for keeping several species populations at currently low abundance levels, and (d) adverse contaminant effects on finfishes are likely to be highly variable from year to year, among weeks within a year, from river to river or estuary to estuary, among specific spawning and nursery areas within a river or estuary, from species to species, and among life stages within a species. The current state of the art in fish population models limits the extent to which documented contaminant effects on individuals can be used to precisely predict responses of populations. Finfish management decisions must, at least for the foreseeable future, be based on less than accurate scientific predictions of risks associated with the current contaminant levels in Bay habitats. Future studies should continue to identify those species, life stages, and spawning or nursery areas in Chesapeake Bay that are most sensitive to contaminant effects and would most benefit from stringent controls on contaminant inputs.

INTRODUCTION

Finfish spawning and nursery habitats in Chesapeake Bay are typical of most temperate latitude estuaries—highly fluctuating. Unpredictable, temporally and spatially heterogeneous environmental conditions impose mortalities on the early life stages of anadromous and resident species that collectively exceed 99% during the first year of life.[1,2] Successful species must possess life history traits that form a reproductive strategy for persistence in the fluctuating and uncertain environment[3,4,5] that exposes their fragile early life stages to an array of mortality sources (Figure 1).

Despite unpredictability of high mortality of eggs and larvae and substantial variation in year class success, existing fisheries records show that iteroparous Bay fishes such as the anadromous striped bass (*Morone saxatilis*) and American shad (*Alosa sapidissima*), and the resident yellow perch (*Perca flavescens*) and white perch (*Morone americana*) have until recently been reasonably successful. Since the early to mid 1970's, these four species and other anadromous and resident Bay finfish have experienced a series of relatively poor year classes. Below average reproductive success has been reflected in a steady decline in sport and commercial fisheries landings over the past decade.[6,7,8,9,10] The innate abilities of these species populations to persist in the face of environmental uncertainty, developed over evolutionary time, is apparently being threatened in the latter half of the 20th century by one or more stressors in Chesapeake Bay.

Prior to the late 1940's, there was little concern about pollution and habitat degradation, except in localized situations. For example, Galtsoff[11] studied the effects of sulfate pulp mill wastes on oysters in the York River, Virginia, and Davis[12] investigated the effects of copper pollution in the Patapsco River, Maryland. Massman et al. wrote in 1952 that chemical pollution had temporarily affected some fish species in local areas, but had not resulted in long-range losses of economically important species in the Bay as a whole.[13] About a decade later in 1961, Mansueti[14] observed that Chesapeake Bay had been subjected to the

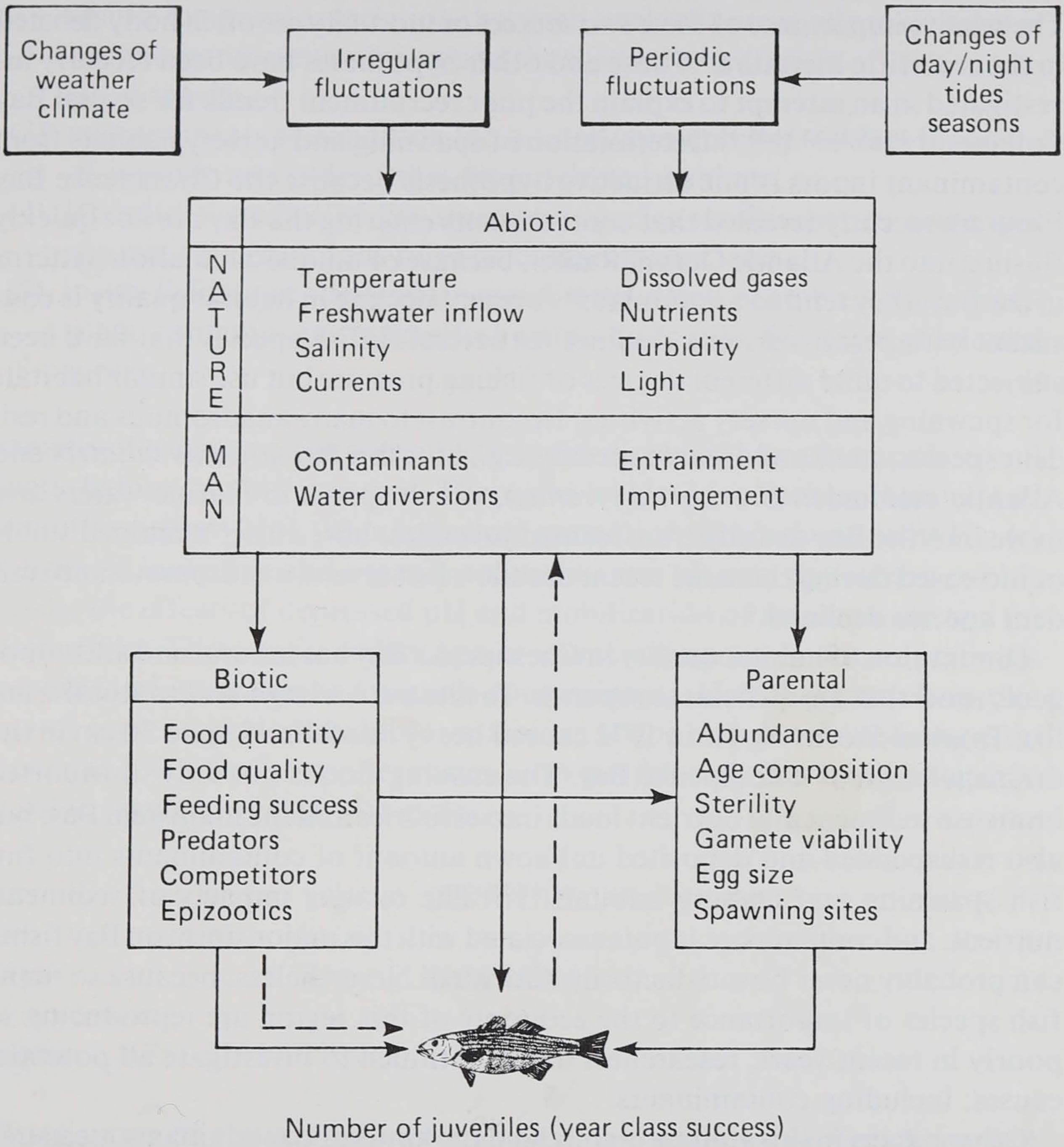

FIGURE 1. Conceptual model of environmental factors which may alter early life stage survival and influence juvenile abundance of Chesapeake Bay finfishes.

effects of civilization. He characterized some effects as catastrophic and others as moderate but sustained.

By 1967, however, L. Eugene Cronin reported that several anadromous and resident Bay finfishes were declining.[15] He suggested that "subtle chemical pollution seems to have high potential for serious and unexpected damage to the estuarine ecosystem", even though chemical contaminants were not reported to be a general problem in the Bay at that time. When the U.S. Environmental Protection Agency initiated the multi-year, $27 million Chesapeake Bay Program (1976-1981), toxic chemical pollution was one of three major study objectives.[16]

Overfishing and deterioration of habitat quality (commonly labelled pollution) are usually the prime causes of dramatic and extended fish stock declines.[17] The relative importance of these two sources of mortality are often hotly debated in the scientific literature.[18] These and other hypotheses have been recently investigated in an attempt to explain the poor recruitment trends for several Bay finfishes.[19,20,21,22,23,24,25,26] Deterioration of spawning and nursery habitats from contaminant inputs is one attractive hypothesis because the Chesapeake Bay Program recently revealed that contaminants entering the Bay are not quickly flushed into the Atlantic Ocean. Rather, because of unique circulation patterns in the Bay, they tend to accumulate.[6] A recent decline in habitat quality is consistent with perceived stock declines for several finfish species that have been subjected to quite different degrees of fishing pressure but use similar habitats for spawning and nursery activities. In contrast to many anadromous and resident species, stocks of oceanic species (e.g., bluefish *Pomatomus saltatrix* and Atlantic menhaden *Brevoortia tyranus*), which spawn in marine waters and move into the Bay as fully transformed juveniles, have either remained stable or increased during the same recent decade when several anadromous and resident species declined.[6]

Diminution of habitat quality in Chesapeake Bay has natural and anthropogenic roots that are difficult to separate. To illustrate, a large-scale natural event like Tropical Storm Agnes in 1972 caused heavy rainfalls of up to 30 cm in the drainage basin of Chesapeake Bay. The ensuing floods not only transported immense sediment and nutrient loads into estuaries and the mainstem Bay, but also resuspended and deposited unknown amount of contaminants into finfish spawning and nursery habitats.[27,28] The relative impacts of sediment, nutrient, and contaminant inputs associated with this major storm on Bay fishes can probably never be satisfactorily allocated. Nevertheless, because so many fish species of importance to the economy of this region are reproducing so poorly in recent years, researchers have continued to investigate all potential causes, including contaminants.

Other papers in this volume highlighted the kinds of contaminants measured in the water column and sediments of Chesapeake Bay. The objective of this paper is to discuss the role that these contaminants may be playing in the declining population status of several finfishes. Review of a series of recent studies will form the basis for our perspectives on the contaminant effects hypothesis. This paper is not intended to provide an exhaustive literature review of toxic chemical effects on Bay fish species. Rather, we intend to summarize a representative sample of current contaminant effects research and establish a milestone for measuring progress to date and for planning future investigations.

Contaminant effects studies on Chesapeake Bay finfishes can be grouped as follows:

(1) mathematical and statistical modeling exercises aimed at elucidating contaminant versus stock trend relationships using extant data and theoretical insights,
(2) biological and chemical surveys of selected habitats designed to reveal spatio-temporal associations between levels of potentially toxic organic and inorganic contaminants and the absence or reduction of various sensitive species,
(3) measurements of condition factors and contaminant residues in tissues of juvenile and older fishes from selected habitats,
(4) laboratory studies of life stage and species sensitivities to acute and chronic concentrations of toxic contaminants, and
(5) in-situ field studies designed to measure the effects of ambient water quality and contaminants in specific habitats on specific life stages of selected species.

Examples of recent contaminant effects studies discussed here encompass several aquatic habitat quality concerns. These are: (a) biocides, such as chlorine and organotins (tributyltin); (b) polynuclear aromatic hydrocarbons or PAHs; (c) mixtures of inorganic and organic contaminants; (d) acid deposition, which includes the effects of depressed pH and mobilization of toxic metals; and (e) radionuclides. These topics embrace point source and non-point source pathways for a range of inorganic and organic contaminants. Current research on this array of topics is understandably variable in scope and distributed unevenly among the 100+ finfish species that occupy portions of Chesapeake Bay during some segment of their life cycles.

RESULTS

BIOCIDES

Kepone

Restrictions on the commercial harvest of some species of finfish are still in effect in the James River, Virginia, 10 years after the discovery that the pesticide Kepone (decachlorooctahydro-1, 3, 4-metheno-2H-cyclobuta (cd) pentalen-2-one) had contaminated the estuary. Kepone was produced in the City of Hopewell (at river km 120) by two firms between 1967 and 1975. The pesticide entered the tidal river through a variety of primarily point-source routes which included chemical plant discharges, runoff from contaminated land fills, and sewage effluents. Bender and Huggett[29] reviewed the data available through 1982 on the status of Kepone contamination in the James River estuary. This discussion is based primarily on that review. Loesch et al.[30] surveyed several Virginia tributaries to Chesapeake Bay and detected Kepone above the action level (0.3 μg/g) in tissues from juvenile finfishes collected in the James River and its tributary, the Chickahominy River. No Kepone above the action level was detected in tissue samples of juveniles collected in the Mattaponi, Pamunkey, Rappahannock, or Potomac rivers.

Bottom sediments of the James River are contaminated from the source at Hopewell to near the river mouth. Figure 2 shows the mass of Kepone estimated

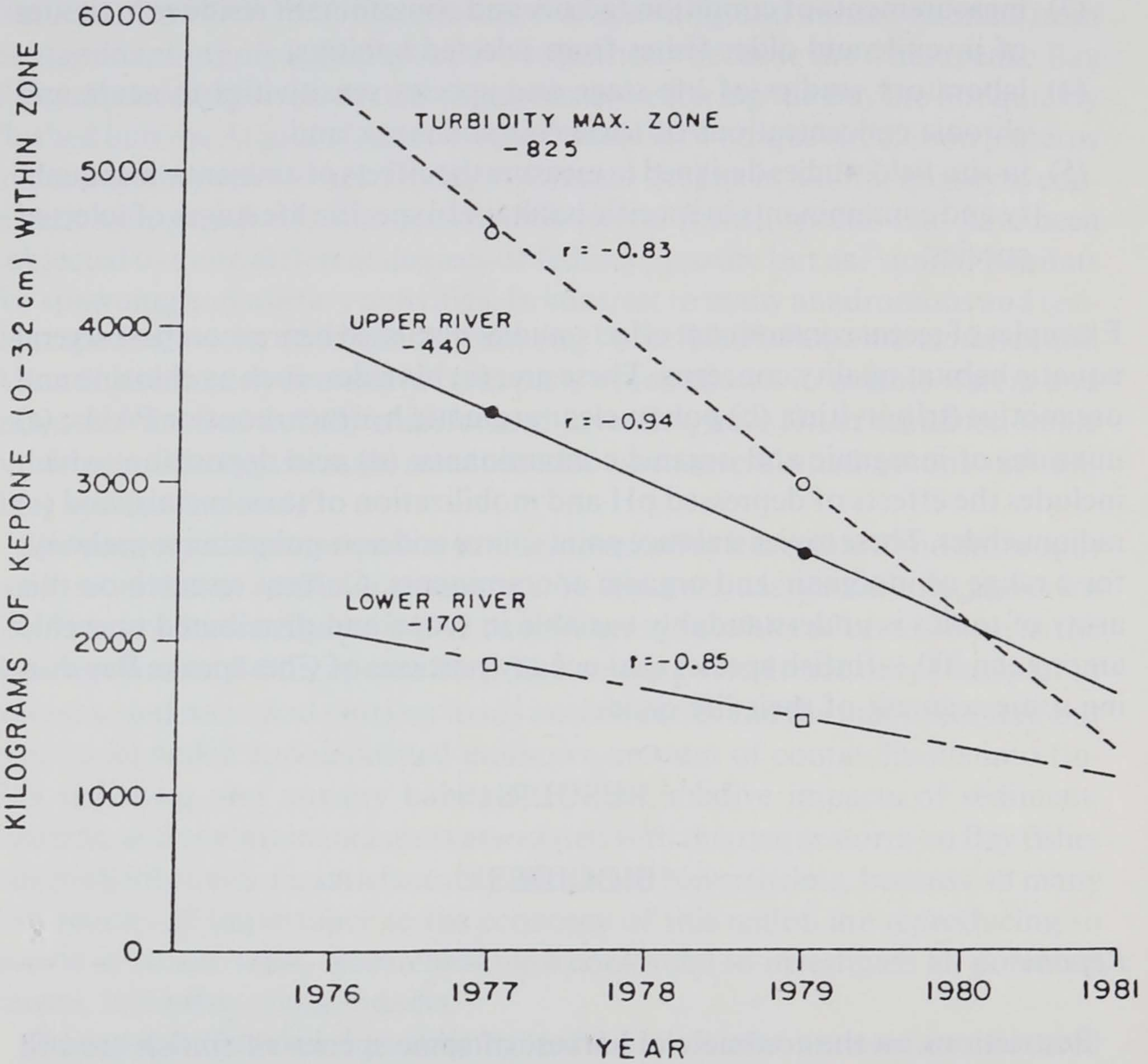

FIGURE 2. Calculated regression lines of Kepone concentrations in the sediments of three zones in the James River.[29]

to be present in the upper 32 cm of river bed sediments as a function of time and location. The rate of burial or dilution (i.e., the slope of the lines in Figure 2) is greatest in the turbidity maximum zone, followed by the upper estuary, and is considerably less in the lower estuary. Since these bed sediments serve as the source of Kepone available to aquatic organisms, the rate at which burial or dilution occurs is extremely important in determining exposure levels.

The relationship between Kepone residues observed in finfish species as a function of the change in Kepone mass in river sediments over time is shown

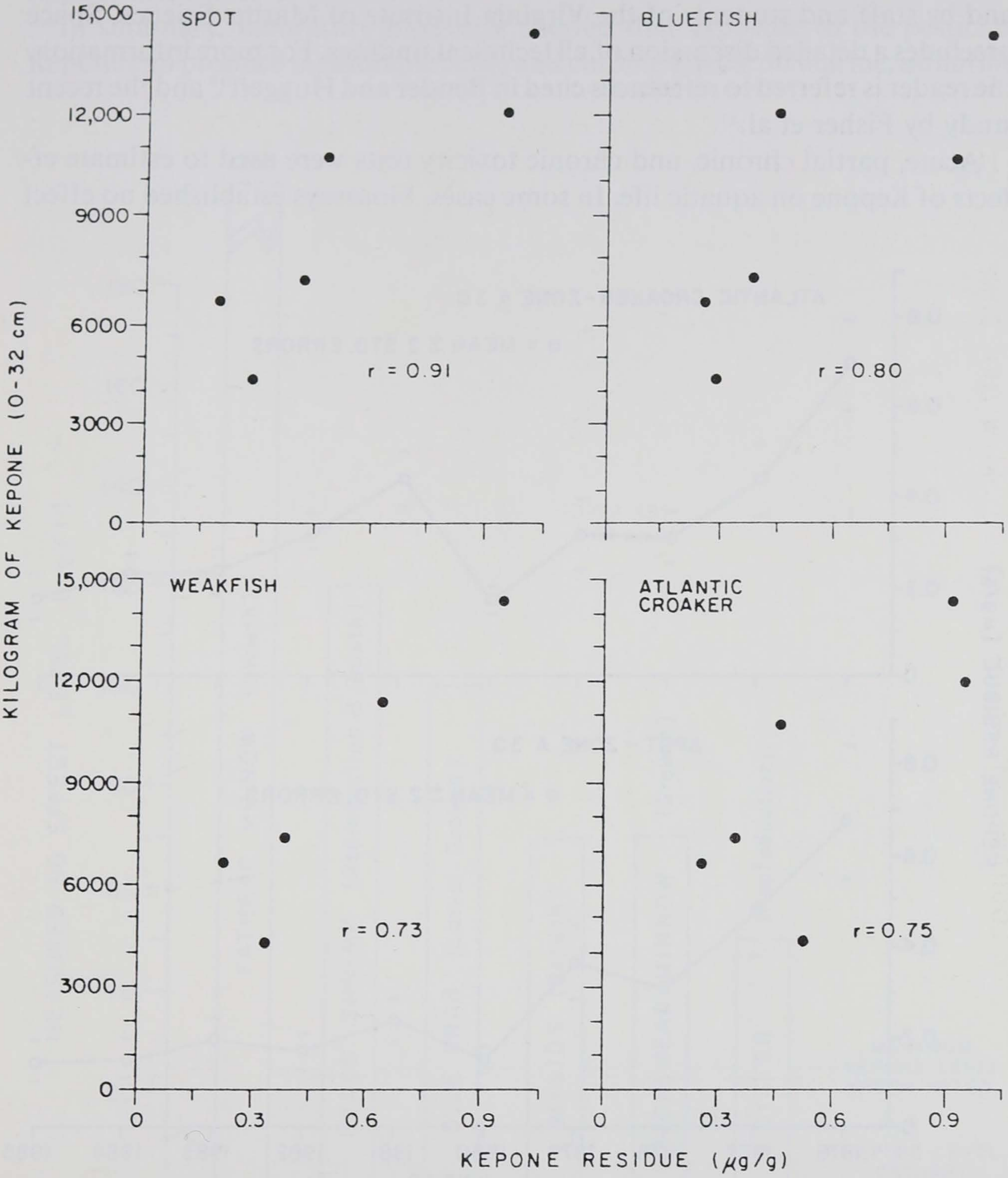

FIGURE 3. Kepone concentrations in James River sediments (0-32cm) from 1976 to 1981 versus Kepone residues in four species.[29]

in Figure 3. Figure 4 depicts the change in third quarter (July-September) residues for Atlantic croaker (*Micropogonias undulatus*) and spot (*Leiostomus xanthurus*) from the lower James River as a function of time (1976-1985). Residues for both species declined through 1980, increased in 1981, and then again declined, but at a much slower rate.

After Kepone contamination of the James River was discovered in 1975, numerous studies were conducted to estimate its impact on aquatic biota. The majority of investigations to establish effects levels were conducted by researchers at the U.S. Environmental Protection Agency laboratory in Gulf Breeze, Florida, and by staff and students of the Virginia Institute of Marine Science. Space precludes a detailed discussion of all technical findings. For more information, the reader is referred to references cited in Bender and Huggett[29] and the recent study by Fisher et al.[31]

Acute, partial chronic, and chronic toxicity tests were used to estimate effects of Kepone on aquatic life. In some cases, bioassays established no effect

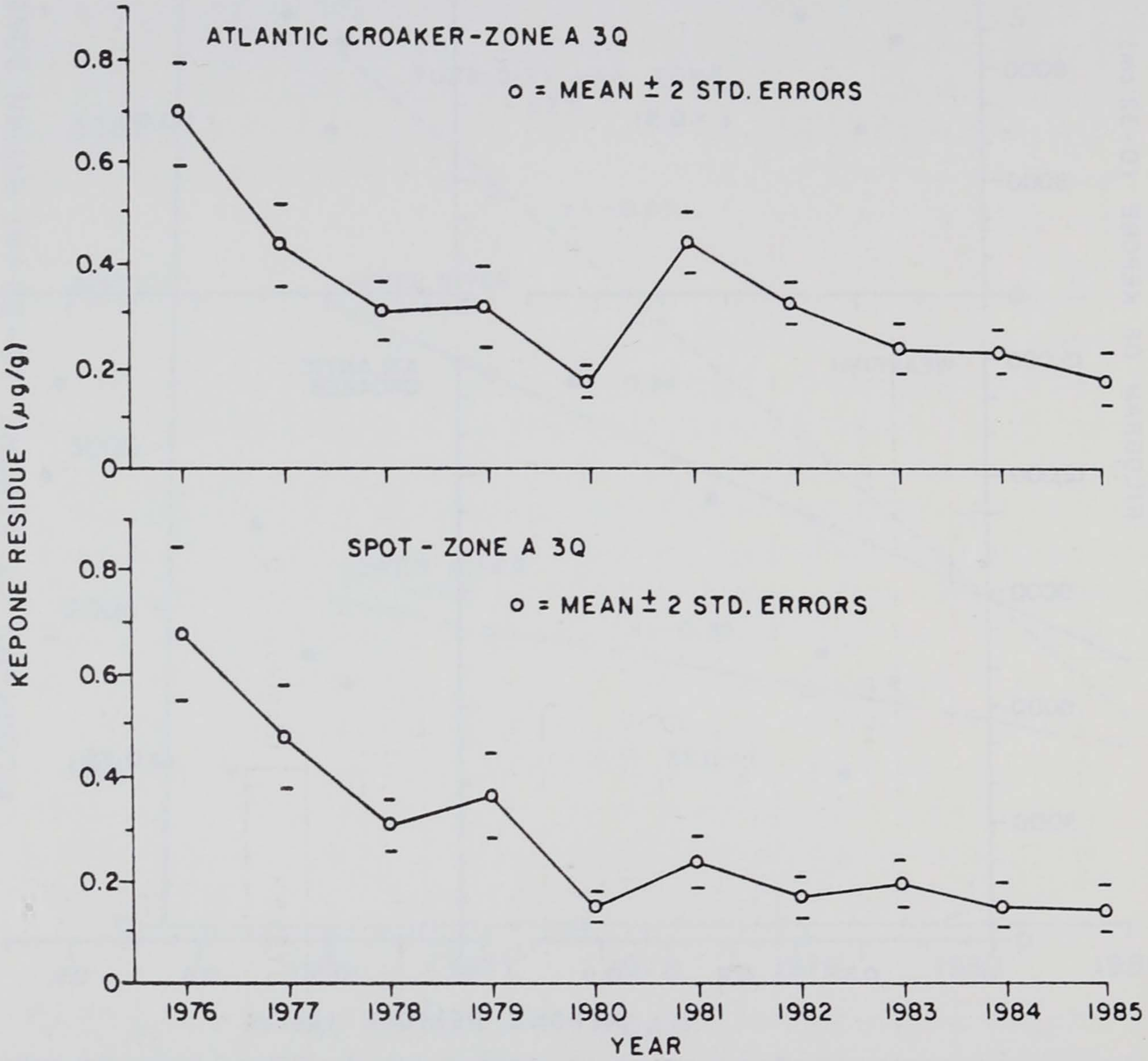

FIGURE 4. Kepone residues in Atlantic croaker and spot, 1976-1985.

levels; i.e., exposure levels at which no significant difference in growth or reproduction were observed compared to the control groups. Other studies estimated the maximum acceptable toxicant concentration (MATC) application factor, defined as the ratio of the chronic no effect level to the 96-h LC50 level (concentration which kills 50% of the test organisms). Figure 5 compares the measured no effect level for several test species to levels of Kepone found in the James River. Exposure levels in the river are well below no effect levels. Figure 6 shows the MATC's for eight finfish species using a very conservative application factor of 0.001.[32]

In summary, laboratory bioassays showed that exposure to the pesticide Kepone can produce measurable acute and chronic effects on marine, estuarine

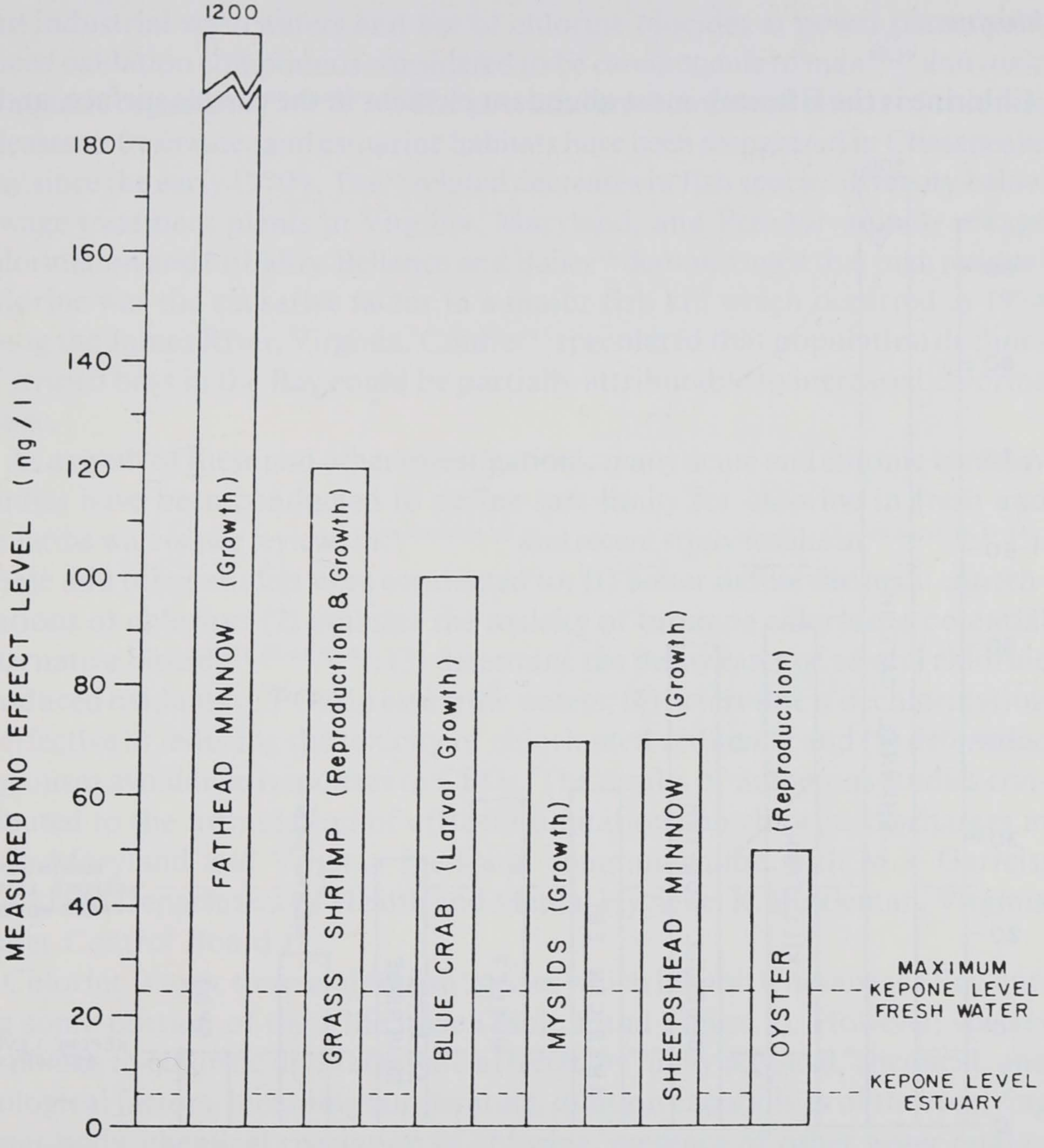

FIGURE 5. Measured no effect levels for Kepone.[29]

and freshwater finfishes that inhabit Chesapeake Bay. However, Kepone concentrations necessary to cause detrimental effects are considerably greater than concentrations measured in the James River. If these conclusions about Kepone effects on finfish are correct, then the major impact of this contaminant in the James River is economic loss due to restrictions on fishing. The James River was closed to all forms of fishing in December 1975 because of Kepone contamination.[30] The ban has since been modified several times. At present, seasonal restrictions limiting commercial fishing for some species are still in effect, and the harvest of striped bass is prohibited throughout the year. Quantitative estimates of economic impacts on the fishery are not available. Many commercial fishermen participated in legal actions against the manufacturing firms to collect damages. All claims were settled out of court.

Chlorine

Chlorine is the fifteenth most abundant element in the earth's surface, and

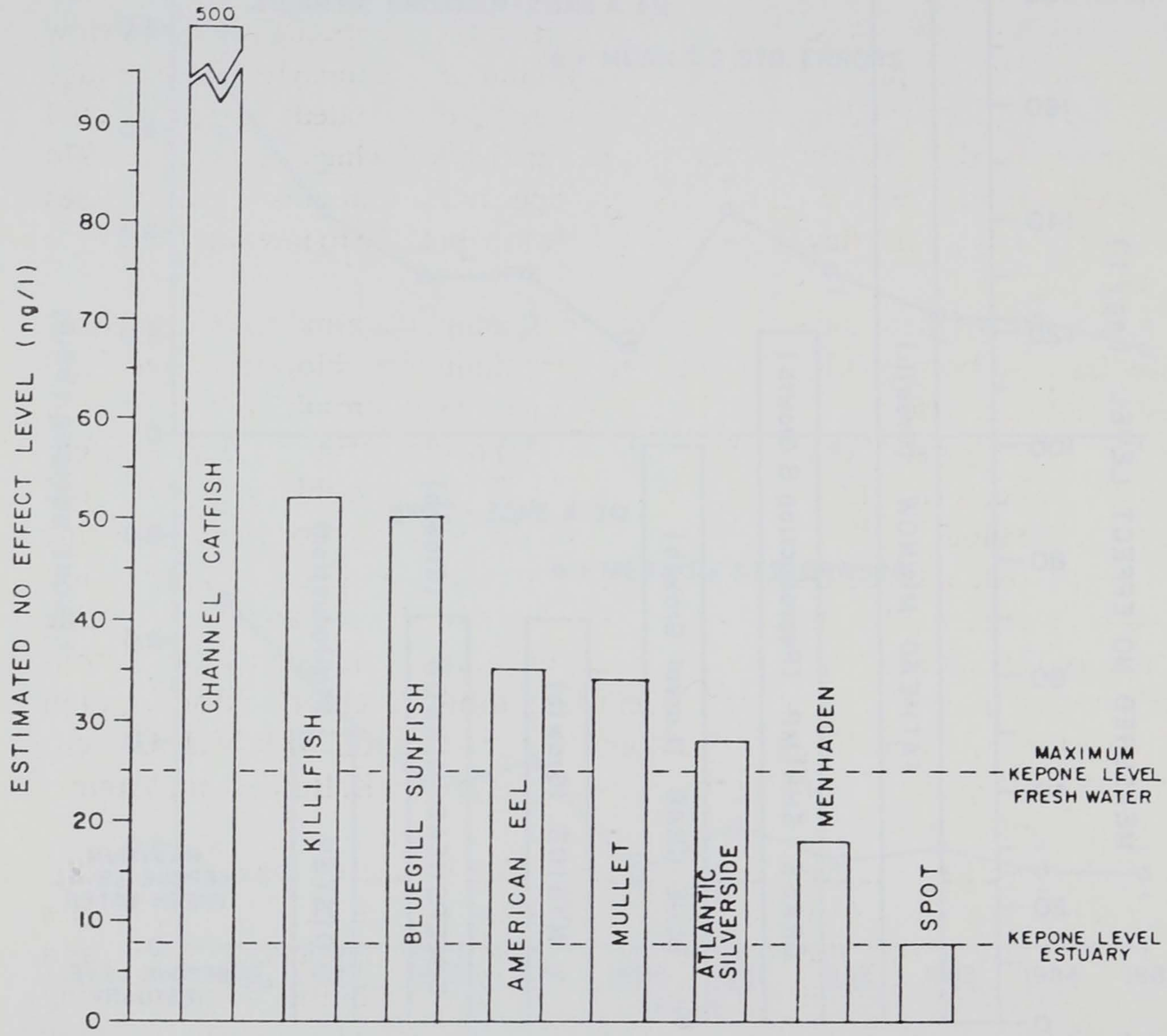

FIGURE 6. Estimated no effect levels for Kepone.[29]

is most commonly present as the Cl^{-1} anion form in estuaries and oceans where concentrations can reach 1.9% by weight.[33] In freshwater habitats, chlorine has an average concentration of about 8 mg/L. Chlorine has been a widely used biocide for disinfection in water and wastewater treatments in both municipal and industrial applications since the late 1800's. Presently, chlorine is the predominant biocide for control of fouling in condenser systems of electrical generating stations (power plants) in the United States.[34,35]

Chlorination of fresh and saline waters may form halogenated organics in water bodies receiving chlorinated discharges. Jolly et al.[36] identified 50 chloro-organic compounds from natural freshwater chlorinated at a power plant. Chlorination of saline waters results in the formation of predominantly brominated rather than chlorinated organics.[37]

During the early 1970's, researchers learned that chlorination of municipal and industrial wastewaters and use of chlorine biocides at power plants produced oxidation compounds considered to be carcinogenic to man[38,39] and toxic to aquatic organisms.[40,41,42,43,44] The potential environmental effects of chlorine releases to freshwater and estuarine habitats have been recognized in Chesapeake Bay since the early 1970's. Tsai[45] related decreases in fish species diversity below sewage treatment plants in Virginia, Maryland, and Pennsylvania to sewage chlorination and turbidity. Bellanca and Bailey[46] demonstrated that high residual chlorine was the causative factor in a major fish kill which occurred in 1974 along the James River, Virginia. Coulter[47] speculated that population declines of striped bass in the Bay could be partially attributable to increased chlorine usage.

As a result of these and other investigations, many acute and chronic bioassay studies have been conducted to define safe limits for chlorine in fresh and estuarine waters (see reviews in[48,49,50,51,52] and recent study results in [53,54,55,56,57,58]). These and other studies were conducted to: (1) better define the toxic concentrations of chlorine; (2) evaluate the toxicity of bromine chloride, a potential alternative biocide[59,60,61,62,63]; (3) determine the decay rates of several chlorine produced oxidants (CPOs) in estuarine waters; (4) determine if dechlorination is effective in reducing the toxicity of chlorinated effluents; and (5) determine organism avoidance responses to CPOs. The results of numerous studies contributed to the formulation of effluent limitations on chlorine discharges in both Maryland and Virginia (personal communication with M.J. Garreis, Maryland Department of Health and Mental Hygiene; K. Buttleman, Virginia Water Control Board.)

Chlorine is toxic to several finfish species which inhabit Chesapeake Bay during some portion of their life cycle (Table 1 and Figure 7). However, species responses to chlorine discharges are affected by many physical, chemical, and biological factors, including temperature, dilution capabilities of the receiving water body, chemical speciation of chlorine, presence of other water quality parameters (e.g., ammonia, dissolved oxygen, pH), and the life stages or age

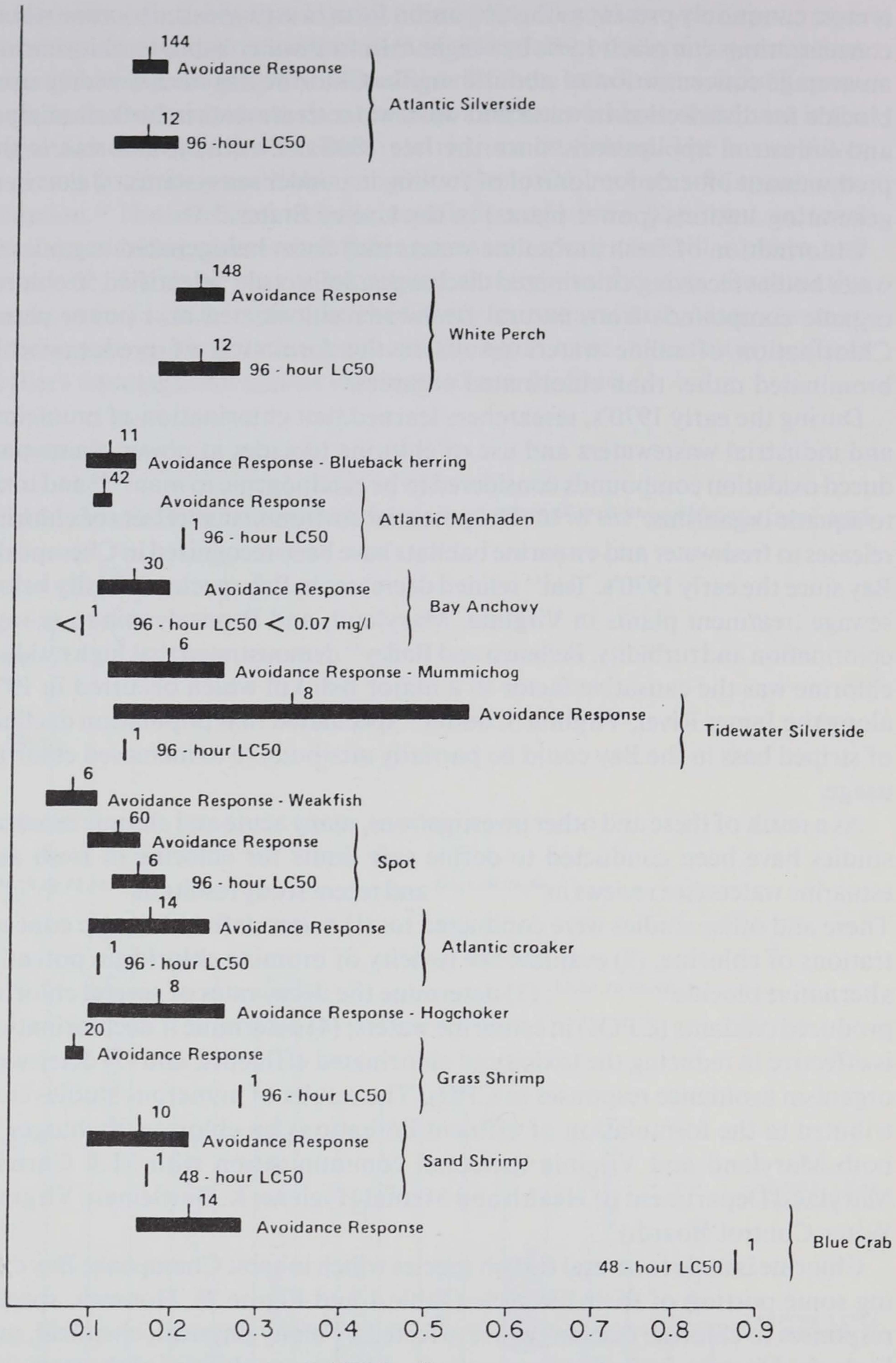

FIGURE 7. Levels of chlorine produced oxidants that are toxic to and avoided by marine and estuarine animals.[68,69]

groups of exposed organisms. Reports of fish kills directly caused by chlorine discharges are rare except where clean water was not accessible to exposed fished.[70] Mobile life stages can detect and avoid potentially toxic chlorinated effluents;[71] however, only a few Bay species have been intensively studied across a wide range of temperatures and salinities.[72] Since the larval stages tend to be more sensitive to chlorine than older juveniles in laboratory tests,[53] larvae are less mobile, possess less avoidance capability, and are therefore more vulnerable than older life stages to chlorine discharges into freshwater tributaries of Chesapeake Bay.

After the 1974 James River fish kill, a task force was formed in Virginia to recommend measures designed to reduce potential impacts from CPOs in estuarine waters. Of particular concern was the oyster resource in the James River, but protection of finfish species was also important. Roberts et al.[73] showed that oyster and clam larvae were extremely sensitive to CPOs with LC50 concentrations in the low ug/L (parts per billion) range. The James River is the major source of seed oysters in Virginia. State officials were concerned that

TABLE 1

Mean acute toxicity of chlorine (in ug/L of total residual chlorine) to finfish species which inhabit the Chesapeake Bay system during all or a portion of their life cycles.

Species	Mean LC50s (24- to 96-h)	Reference
Channel catfish	90	[64]
Ictalurus punctatus		
Yellow perch	205	[64]
Perca flavescens		
Atlantic silverside	37	(64]
Menidia menidia		
Tidewater silverside (juvenile)	54	[64]
Menidia beryllina		
Naked goby (juvenile)	80	[64]
Gobiosoma bosci		
Spot	90	(64]
Leiostomus xanthurus		
Striped bass (*Morone saxatilis)*		[53]
larva (22-d old)	140 (141-147)[a]	
juvenile (60-d old)	190 (178-209)[a]	
juvenile (388-d old)	230 (226-240)[a]	
Atlantic menhaden		
Brevoortia tyranus		
Alewife	129 (30-227)[b]	[66]
Alosa pseudoharengus		
Blueback herring		
A. aestivalis	250	[67]
larva (48-h old)		

[a] 95% confidence interval

[b] Range

chlorine could be responsible in part for decreased spatfalls observed in the river since the early 1960's.[74] Current chlorine discharge limits in the Virginia and Maryland portions of Chesapeake Bay appear to be sufficiently strict to protect finfish spawning and nursery habitats.

In summary, point-source discharge of chlorine and oxidant products into the waters of Chesapeake Bay are not likely to be detrimental to finfish populations except in localized portions of small freshwater tributaries or in the immediate vicinity of major discharges. Areas inhabited by sensitive and relatively non-mobile eggs and larvae are most at risk. Juvenile and older finfish appear capable of avoiding toxic concentrations of CPOs, a behavior that should decrease adverse effects. However, when fish avoid a specific area, spawning activities may be impaired and potential habitat is lost to the population, either temporarily or permanently. The relative importance of lost habitat will influence the ultimate effects of chlorine discharges on Bay fish populations.

Organotins

In recent years, the potential effects of organotin compounds, such as tributyltin (TBT), on Chesapeake Bay biota has become a major environmental issue. Several factors are responsible for the concern: (1) increased use of TBT in antifouling paint on both recreational and commercial watercraft in the Bay; (2) presence of potentially toxic concentrations of TBT in marina areas of the Bay;[75] (3) concentrations of TBT exceeding proposed water quality standards in the United Kingdom (20 ng/L)[76] have been reported in some Chesapeake Bay rivers;[75] (4) a recent proposal by the U.S. Navy to use organotin-based paints on all Naval vessels;[77] and (5) laboratory and field studies in England, France, and the United States which have shown that TBT is highly toxic to several aquatic species.[78,79,80]

The use of organotin paints to prevent growth of fouling organisms on boat hulls has increased since the early 1960's. These paints possess excellent antifouling actions, long lifetimes, and almost no corrosion.[81,82] Tributyltin (TBT), triphenyltin, and tricyclohexyltin compounds are the major biocidal organotins.[83] Presently, there are no effluent guidelines or water quality regulations for organotins in the United States.[83]

Organotin biocides are generally more toxic to aquatic biota than are other major organic contaminants such as polycyclic aromatic hydrocarbons (PAHs), chlorinated pesticides, and polychlorinated biphenyls or PCBs.[83] Organotins can be lethal to fish in the low ug/L concentrations.[84,85] Toxic levels of organotin compounds for several species of Bay finfishes are presented in Table 2. The sheepshead minnow (*Cyprinodon variegatus*) is very sensitive, with a 21-d LC50 for bis (tri-n-butyltin) oxide (TBTO) of 1 ug/L. The limited amount of information on avoidance capabilities of Bay finfishes to organotins (Table 2) suggests that some species (e.g., striped bass) may not avoid low concentrations

TABLE 2

Toxicity of TBTO[a] to finfishes found in Chesapeake Bay.

Species	Concentration (ug/L)	Exposure Time	Type of Test	Test Medium	Type of Response	Life Stage	Reference
Mummichog (*Fundulus heteroclitus)*	24	96 hr	Static	SW	Mortality	Adult	[86]
	1.0 - 13.8	20 min	Flow-through	SW	Avoidance	Adult	[87]
	20.8 - 28.0[b]	96 hr	Flow-through	SW	Mortality	Sub-adult	[91]
	15.2 - 30.4[b]	96 hr	Flow-through	SW	Mortality (LC50)	Larva	[91]
Striped bass (*Morone saxatilis*)	14.7 - 24.9	20 min	Flow-through	FW	Avoidance	Juvenile	[88]
Sheepshead minnow (*Cyprinodon variegatus*)	13 - 17	96 hr	Static	SW	Mortality (LC50)	Juvenile	[89]
	1	21 d	Flow-through	SW	Mortality (LC50)	Juvenile	[85]
	22.8 - 30.1[b]	96 hr	Flow-through	SW	Mortality (LC50)	Sub-adult	[91]
Atlantic menhaden (*Brevoortia tyranus*)	5.5 - 24.9	20 min	Flow-through	SW	Avoidance	Juvenile	[88]
	3.6 - 6.4[b]	96 hr	Flow-through	SW	Mortality (LC50)	Juvenile	[91]
Atlantic silverside (*Menidia menidia)*	6.7 - 11.6[b]	95 hr	Flow-through	SW	Mortality (LC50)	Sub-adult	[91]
Tidewater silverside (*Menidia beryllina*)	2.3 - 4.0[b]	96 hr	Flow-through	SW	Mortality (LC50)	Larva	[91]
Bluegill (*Lepomis macrochirus*)	7.6	96 hr	Static	FW	Mortality	Juvenile	[90]
Channel catfish (*Ictalurus punctatus)*	12	96 hr	Static	FW	Mortality (LC50)	Juvenile	[86]

[a] TBTO = bis (tri-n-butyltin) oxide

SW = salt water

FW = fresh water

[b] Concentrations measured in test tanks

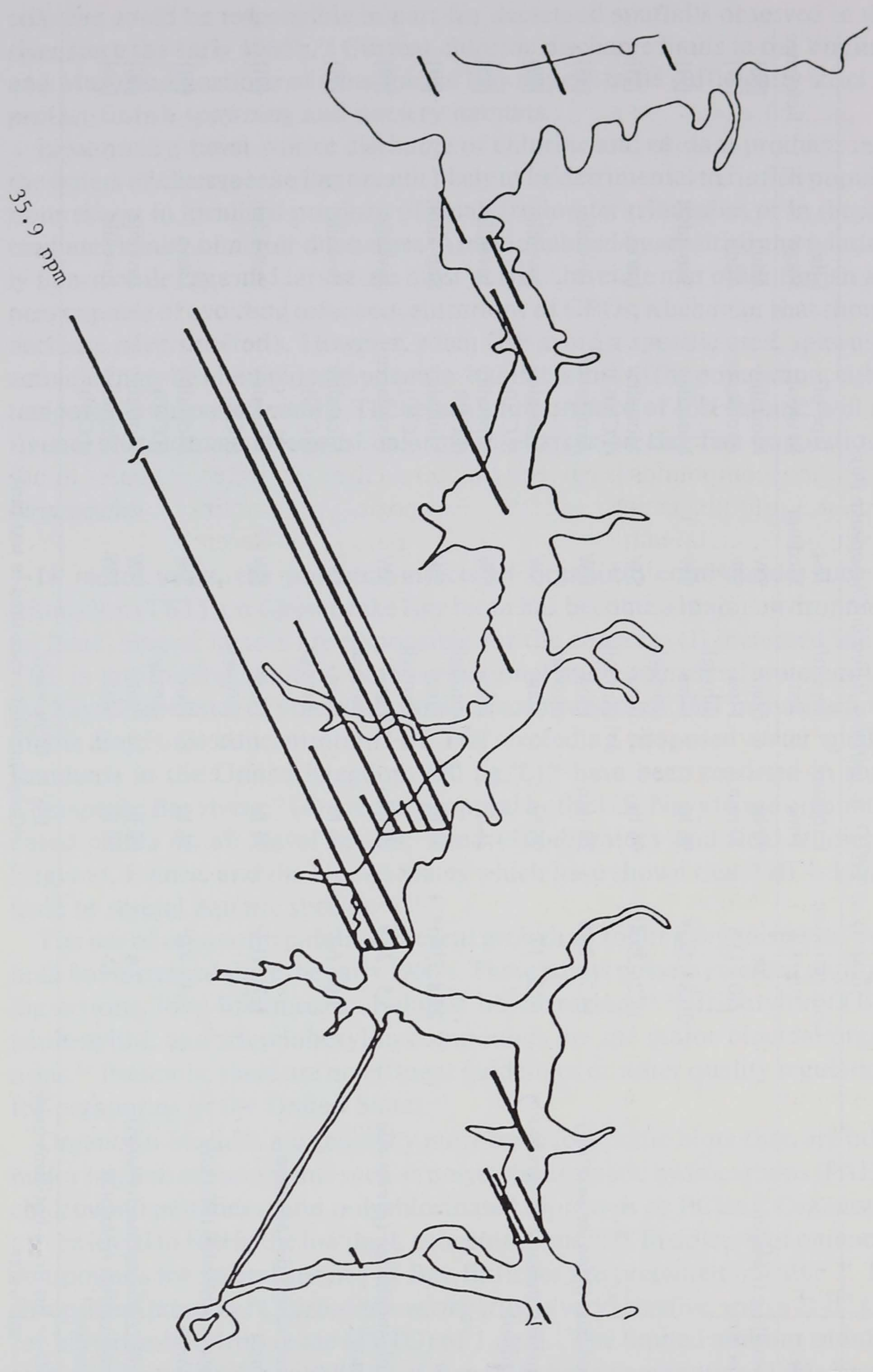

FIGURE 8. Surface sediment concentrations (mg/kg-dry wt) of benzo(a)pyrene along the Elizabeth River, Virginia, in 1985 (0.75 cm = 1 mg/kg or ppm.[94])

in the environment. Adverse effects on such species could occur if their limited avoidance capabilities resulted in extended exposures to TBT-contaminated areas. The sublethal effects of long-term, low-level exposures of organotins to finfishes have not been adequately studied.[83,87] Conversely, the mummichog appears to possess keen avoidance capabilities to low levels of organotins, presumably due to their highly sensitive chemoreceptor system.[87]

In summary, TBT leached into the aquatic environment from antifouling coatings on boat hulls is most concentrated in harbor and marina areas.[83] Therefore, their potential effects on Chesapeake Bay finfishes would presumably be most serious in these localized areas. However, because boats and ships are mobile and organotin compounds bioconcentrate in the food chain, all Bay habitats navigable by TBT-treated boats could be exposed to these contaminants. The effects of TBT on finfish populations in Chesapeake Bay are not yet known. Laboratory toxicity data suggest serious potential problems so research activity on these compounds is currently intense. Given projected increases in use of these antifouling paints[92] and their high toxicity, organotins must be viewed as a major contaminant problem in Chesapeake Bay. Recently, the States of Maryland and Virginia passed legislation to restrict the application of TBT paints on watercraft that use Chesapeake Bay.

Polynuclear Aromatic Hydrocarbons

Polynuclear aromatic hydrocarbons (PAHs) can enter the aquatic environment via several routes, but primarily through the incomplete combustion of

TABLE 3

Percentage of fish showing gross abnormalities from exposure to contaminants in the Elizabeth River, Virginia. Data are means of three samples collected in October, November, and December, 1983.[94]

Abnormality by	Kilometers from River Mouth										
Species	6.5	8.5	10.5	12.5	15.0	17.0	19.0	21.5	23.5	25.5	28.0
Fin Erosion											
Hogchoker[a]	0.7	0	0	0.4	1.4	5.5	4.3	11.2	1.9	0	0.5
Toadfish[b]	0	0	11.0	5.0	0	11.5	30.1	26.3	25.0	0	0
Cataracts											
Spot[c]	0	0	0.1	0	3.0	0.8	9.6	6.0	0.2	0.3	0
Weakfish[d]	0.2	0	0	0.8	1.0	1.8	3.5	14.0	21.0	2.5	7.5
Atlantic Croaker[e]	3.3	1.4	1.5	2.2	4.5	7.9	15.8	15.9	18.1	2.5	5.6

[a] *Trinectes maculatus*
[b] *Opsanus tau*
[c] *Leiostomus xanthurus*
[d] *Cynoscion regalis*
[e] *Micropogonias undulatus*

carbonaceous materials or through industrial processes that convert coal into synthetic fuels.[93] Other sources of PAHs include the manufacture of carbon black, creosote, soot, vehicular emissions (especially diesel), residual oil, and wood smoke. PAHs are of concern to scientists because some can become mutagenic or carcinogenic after being metabolized.

Field observations suggest that fishes in the Elizabeth River, Virginia, are severely stressed because of sediment contamination with PAHs. Figure 8 shows the distribution of one PAH, benzo(a)pyrene, in surface sediments along the Southern Branch of the Elizabeth River.[94] Incidence of abnormalities (e.g., skin lesions, cataracts, fin erosion) in native fishes increased at sampling stations which were heavily contaminated with PAHs (Table 3 and Figure 9).

In laboratory exposures of spot (*Leiostomus xanthurus)* to contaminated sediments from the Elizabeth River, dermal lesions and fin rot similar to those in fish collected from the river were observed.[95] Weeks and Warinner[96] found that the phagocytic efficiency of macrophages from spot and hogchoker (*Trinectes maculatus*) resident in the Elizabeth River was reduced when compared to fish from control stations. The bioavailability of PAHs to oysters in the Elizabeth River was demonstrated using transplant studies.[94]

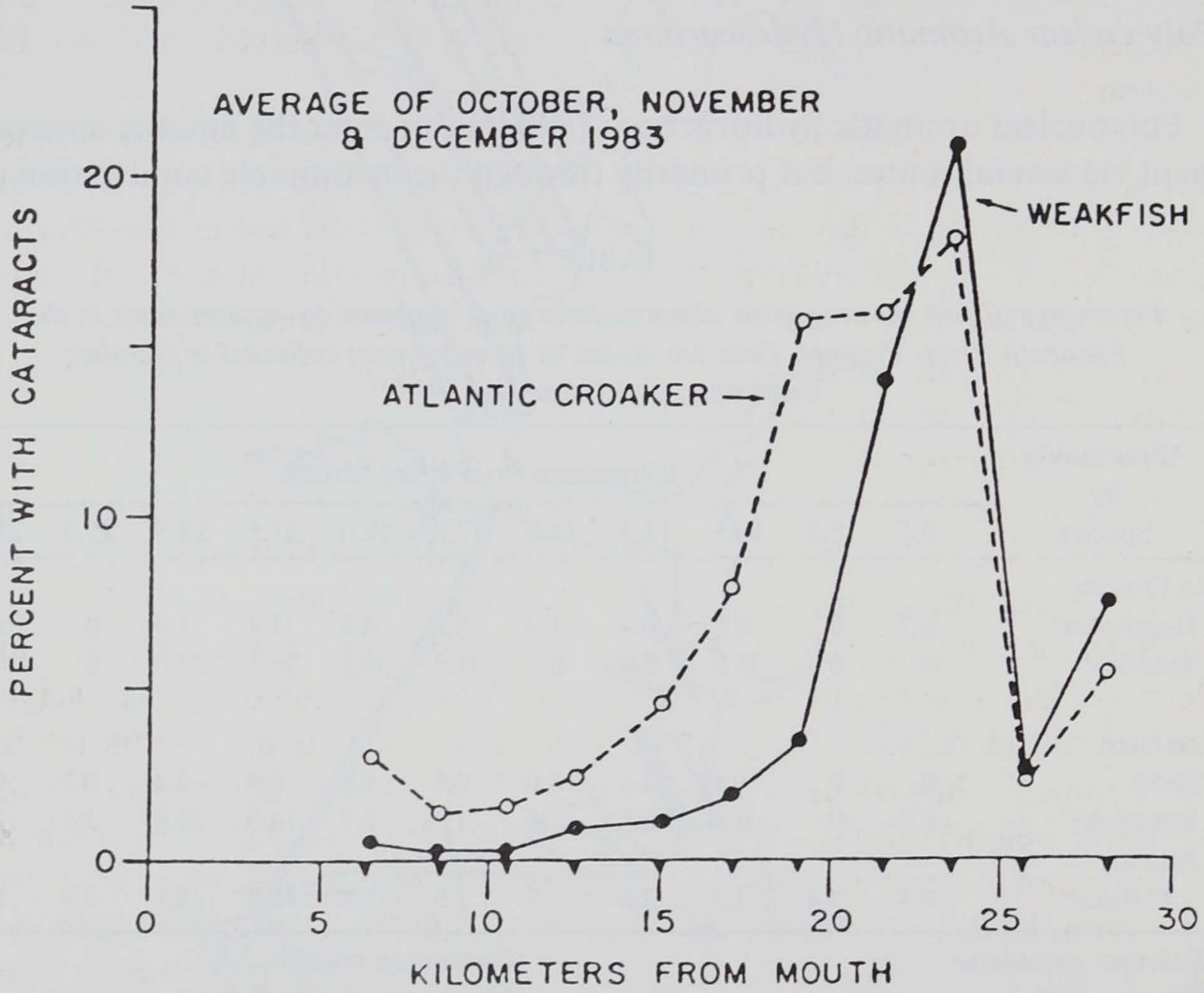

FIGURE 9. Average occurrence of cataracts in Atlantic croaker and weakfish from stations along the Elizabeth River, Virginia.[94]

In summary, PAHs are widespread contaminants in freshwater and estuarine systems and have also been implicated in adverse effects on finfish and shellfish in other areas such as the Niagara River,[97] Oregon Bay,[98] and Puget Sound.[99] Future studies focused on PAH contamination in Chesapeake Bay should include: (1) studies to define the levels of sediment contamination necessary to cause acute and chronic effects on fishes, (2) surveys of the mainstem Bay and tributaries to determine whether increases in the incidence of abnormalities in fishes are related to PAH presence and concentration, and (3) laboratory and field investigations to determine the specific PAH compounds responsible for observed abnormalities.

INORGANIC AND ORGANIC CONTAMINANT MIXTURES

Passage of the Chaffee Amendment to the Anadromous Fisheries Conservation Act in 1980 stimulated an ambitious research program aimed at determining why anadromous stocks of striped bass along the Atlantic coast had declined since the mid-1970's and how these stocks could be restored to former abundance levels.[100] The role that contaminants may have played in this decline was a major study objective led by the Columbia National Fisheries Research Laboratory (CNFRL) of the U.S. Fish and Wildlife Service in Columbia, Missouri.

During the first phase of the CNFRL contaminants program, a comprehensive survey was conducted in several major Atlantic coast spawning rivers (including Chesapeake Bay) and selected hatcheries. This survey analyzed the tissues of striped bass collected in these waters for residues of over 100 organic and inorganic contaminants.[101] The survey was complemented by other recent surveys of inorganic and organic contaminant residues in fish tissue samples collected in Chesapeake Bay[102,103,104]

The most prevalent organic contaminant residues found in the tissues of juvenile striped bass from the Hudson River, New York; the Nanticoke and Potomac Rivers in Chesapeake Bay; and the Edenton Fish Hatchery, North Carolina (control group) by Mehrle et al.[19] were polychlorinated biphenyls or PCBs (Aroclors 1248, 1254, 1260). Chlordane, DDT, DDD, and DDE were also detected, but at concentrations equal to or less than 0.06 ug/g (wet weight), and not considered to be significant residue levels.[19] Total organochlorine residues in Chesapeake Bay striped bass were higher in the Potomac River (0.21 - 0.40 ug/g wet weight) than in the Nanticoke River (0.06 - 0.09 ug/g wet weight).

The major inorganic constituents detected in juvenile striped bass tissues were cadmium, lead, zinc, arsenic, and selenium.[19] Selenium residues tended to be slightly higher in Potomac River juveniles (0.22 - 0.84 ug/g wet weight) compared to fish collected in the Nanticoke River (0.19—0.64 ug/g wet weight). Concentrations of other metal residues were similar in these two Bay populations.

Mehrle et al.[19] correlated tissue residues with vertebral development and suggested that contaminants such as cadmium, lead, and PCBs could decrease survival of striped bass larvae and early juveniles. The risk seemed highest in the Hudson River population where juveniles had the highest concentrations of contaminant residues and lowest structural-integrity indices for their vertebrae. Swimming stamina and condition factor indices were also low in Hudson River juveniles, suggesting that the poor condition symptoms were consistent with the uptake of environmental contaminants.[105] By comparison, vertebral integrity was intermediate for Nanticoke and Potomac river juveniles, and highest in fish of Hudson River origin that had been reared in the relatively uncontaminated waters of the Edenton Hatchery.[19]

Neither study[19,105] concluded that contaminants were not affecting the status of striped bass populations in the Potomac and Nanticoke rivers. Rather, the problem of contaminants appeared to be more critical in the Hudson River, where levels of PCBs and other chemicals constitute a major environmental issue[106,107,108] that resulted in the closure of the fisheries for striped bass and other finfishes in 1976 that continues to this day.[109,110] Interestingly, however, the Hudson River population of striped bass continued to produce average or above average year classes during the 1970's and early 1980's when reproductive success for populations in Chesapeake Bay and other Atlantic coast estuaries was dismal.[5] Although clear evidence is lacking, closure of the fishery for striped bass in the Hudson River may have contributed to the favorable trends in annual juvenile production.

The first phase of the CNFRL studies[19,101,105] detected relatively small quantities of several contaminants (PCBs, organochlorine pesticides, dioxins, dibenzofurans, petroleum hydrocarbons, cadmium, copper, lead, arsenic, selenium) in the tissues of juvenile striped bass collected in several Atlantic coast rivers. However, no single contaminant was found in sufficient concentration or frequency to explain the observed decline in coastal stocks.[101] This conclusion stimulated a series of laboratory toxicity studies which focused on the array of inorganic and organic contaminants measured in juvenile striped bass tissues.[19] These studies departed from traditional single contaminant experiments and evaluated acute and chronic effects of mixtures containing two or more compounds at environmentally-realistic concentrations.[16,111,112]

Several studies exposed the early life stages of striped bass to a complex mixture of contaminants (Table 4) in fresh and saline water.[24,101,113] Palawski et al.[113] also compared the relative acute toxicities of inorganic and organic components of this toxicant mixture and measured individual toxicities of cadmium chloride, copper sulfate, zinc chloride, nickle chloride, arsenic pentoxide, selenium selenite, and sodium chromate on 35 to 80-d old juveniles.

Growth of larvae and early juveniles was unaltered by exposure to the contaminant mixture at 25 to 400% of the environmental concentration (Table 5) in fresh water or 2 and 5 ppt saltwater.[101] Percent fertilization and hatching suc-

TABLE 4

Concentrations of organic and inorganic contaminants included in the contaminant mixture stock solution.[24,101][a] *Organic compounds were dissolved in acetone; inorganics were dissolved in hydrochloric acid.*

Aroclor 1248	10 ng/L
Aroclor 1254	10 ng/L
Aroclor 1260	10 ng/L
DDE	3 ng/L
Toxaphene	3 ng/L
Chlordane	5 ng/L
Kepone (chlordecone)	15 ng/L
Perylene	40 ng/L
Fluorene	40 ng/L
Phenanthrene	40 ng/L
Anthracene	40 ng/L
Fluoranthene	40 ng/L
Perene	40 ng/L
Benzoanthrene	40 ng/L
Chrysene	40 ng/L
Arsenic (as pentoxide)	1 ug/L
Selenium (as selenite)	2 ug/L
Lead (as nitrate)	1 ug/L
Cadmium (as chloride)	3 ug/L
Copper (as sulfate)	1 ug/L

[a] Stock solution of contaminant mixture[113] did not contain Anthracene or Fluoranthene.

cess were not diminished by various dilutions of the contaminant mixture; but a 100% concentration reduced survival of yolk-sac larvae after a 144-h continuous exposure from fertilization.[24]

Juvenile survival decreased during exposure to the contaminant mixture, which was most toxic in moderately soft fresh water (hardness of 40 mg/L as $CaCO_3$) compared to 1 or 5 ppt saltwater.[113] This increased toxicity in freshwater was attributed to differences in speciation of metals associated with water chemistry, especially for cadmium, copper, and zinc. Wright et al.[114] demonstrated that uptake and toxicity of cadmium to larval and juvenile striped bass are inversely related to calcium levels in the test medium. The organic chemical fraction of the contaminant mixture (Table 4) was not toxic to juvenile striped bass at concentrations 51 times greater than environmental levels.[113] This conclusion implies that inorganic rather than organic contaminants in the mixture pose a potentially greater risk to the survival of young striped bass during the late larval and early juvenile stages (35 to 80-d old). The relative toxicity of organic versus inorganic components in the contaminant mixture has not been determined for younger life stages of striped bass or other fish species.

The contaminant mixture tested by[24,101,113] did not contain all organic compounds that may pose a threat to Bay fishes. For example, the mixture did not contain the herbicide atrazine (2-chloro-4-ethylamino-6-isopropyl), that is widely used in the Chesapeake Bay watershed and present at low concentrations (up to 2 ug/L) in the water column.[115] The limited toxicity data for atrazine suggest that current levels in Bay habitats will not adversely affect finfishes.[116] The contaminant mixture also did not contain any organotin compounds, biocides considered to be a major contaminant problem in Chesapeake Bay (discussed above).

Sublethal effects of the CNFRL's contaminant mixture on swimming performance, feeding behavior, and predation avoidance for juvenile striped bass after 20 to 60-d exposures were inconclusive.[101] Whole body residues of inorganic and organic contaminants in juveniles exposed for up to 90 days were relatively low and in the range observed in wild juveniles collected from several Atlantic coast rivers.[9] These results support the premise that the CNFRL series of laboratory studies exposed test organisms to environmentally-realistic concentrations of contaminants.

Striped bass yolk-sac larvae were more sensitive to the contaminant mixture than embryos, older larvae, and juveniles.[24,101,113] This finding corroborates the general pattern of life stage sensitivities in finfishes reported by others.[53,114,117,118,119,120] Overall, striped bass were as sensitive as most salmonid fishes to seven metals and three organic pesticides, but much more sensitive than several cyprinids, ictalurids, and centrarchids (Table 5). Cadmium, copper, and zinc were extremely toxic to young striped bass. Wright et al.[114] observed that 7-d old larvae were very sensitive to cadmium (5 - 10 ug/L) when exposed in a low calcium (8 mg/L) medium. Pathological changes were induced in the visual system of 28-d old larvae after only 24-h exposures to 80-150 ug/L copper in a dose-dependent fashion.[121] By comparison, arsenic, selenium, nickel, and chromium were much less toxic to young striped bass.[113]

Klauda[122] also demonstrated that the acute toxicities of arsenic and selenium to striped bass eggs, larvae, and juveniles are relatively low, either as isolates

TABLE 5

Comparison of the relative sensitivity (96-h median lethal concentrations, ug/L) of four finfish species to seven metals tested in soft fresh water.[113]

Metal	Species			
	Striped Bass	*Rainbow Trout*	*Fathead Minnow*	*Bluegill*
Cadmium (as chloride)	4	1	630	1,940
Copper (as sulfate)	100	17	25	660
Zinc (as chloride)	120	93	780	5,370
Selenium (as selenite)	1,325	1,800	10,000	4,500
Nickel (as chloride)	3,900	15,000	4,580	5,180
Chromium (as chloride)	28,000	59,000	17,600	118,000
Arsenic (as pentoxide)	40,500	28,000	42,000	41,760

or mixtures in a 3-7 ppt salinity medium (Table 6). Klauda[122] tested arsenate (+5) and selenate (+6) because they are generally the dominant inorganic forms available to early life stages of fishes via waterborne pathways in estuarine waters.[123,124] Arsenite (+3) and selenite (+4) are more prevalent in freshwater and also more toxic to fishes than arsenate and selenate.[125] Various forms of arsenic (arsenate, arsenite, methylarsenic acid, dimethylarsenic acid) and selenium (selenate, selenite, elemental selenium, heavy metal selenides, methylated forms) can occur in aquatic environments.[126] About 30% of arsenic and selenium inputs to the environment come from coal combustion,[127] hence these contaminants can be expected to be present in aquatic habitats near coal-fired power plants operating in Chesapeake Bay.[125]

Klauda[122] showed that the joint toxicities of arsenate and selenate in mixtures were additive to striped bass yolk-sac larvae, but subadditive and suggestive of antagonism to post larvae and juveniles (Table 7). Selenium reduces the toxicity of mercury, cadmium, and copper in several aquatic organisms,[128,129,130,131] but antagonism with arsenic had been previously observed only in mammals.[132] Continuous exposure of young striped bass to sublethal levels of selenate (89 to 1,360 ug/L) for 60 days post-hatch was associated with an increased frequency (52%) of lower jaw deformities.[122] Cumulative toxicity during long-term exposures could decrease feeding ability in postlarvae and juveniles and alter survival probabilities.

In summary, these laboratory studies with early life stages of striped bass demonstrated that survival can be diminished by relatively brief encounters with environmentally-documented concentrations of inorganic and organic contaminant mixtures. Such findings suggest that contaminants cannot be ignored as a possible factor contributing to the decline of striped bass stocks in Chesapeake Bay and other Atlantic coast estuaries. Toxic forms of inorganic contaminants (especially cadmium, copper, zinc) appear to pose a major threat to young striped

TABLE 6

LC50 values (ug/L) for early life stages of striped bass exposed to sodium arsenate or sodium selenate for 96 hours in estuarine water (3-7 ppt).[122]

Life Stage	Age[a] (days after hatch)	LC50 (95% Confidence Interval)	
		Arsenate	Selenate
Yolk-sac larva	1	18,690 (16,780-20,590)	9,790 (8,260-11,310)
Post larva	17	7,280 (6,510-8,050)	13,020 (11,560-14,480)
Juvenile	72	18,960 (18,130-19,780)	85,840 (81,650-90,030)

[a] Age at start of 96-h test

bass in freshwater reaches of spawning and nursery areas.

The accuracy of this important conclusion will be influenced by the extent to which continuous exposure laboratory studies can accurately predict metal-induced effects on young striped bass survival in nature. These predictions may be reliable, based on results with other species,[133] unless metal concentrations in natural waters are temporally quite variable[134] or fish avoid potentially lethal levels. Avoidance responses to copper and zinc should be effective at reducing mortality, but fish appear to possess limited abilities to detect and avoid lethal concentrations of cadmium.[71]

As developing young striped bass migrate downstream and reach saline habitats, the toxicity of heavy metals should decrease and pose a less serious threat to their survival. Prior exposure of young striped bass to sublethal doses of metals as larvae or early juveniles could also enhance their chances for survival to maturity. Several studies have shown that pre-exposure of fishes to copper,[135] cadmium,[136] arsenic,[137] zinc,[138] and other toxicants can reduce their deterious affects. For some fish species, metal-binding proteins called metallothioneins, produced primarily in liver tissue, are presumably involved in the development of enhanced tolerance to some heavy metals.[139,140,141,142,143] Exposure of young fishes to sublethal concentrations of inorganic aluminum during acidic episodes may stimulate increased amounts of calmodulin, a calcium binding protein, in gill tissues and reduce the toxic effects of aluminum on ionic fluxes.[144]

ACIDIC DEPOSITION

Deposition of chemical pollutants from the atmosphere is a major environmental issue of international scope.[145] Concern that acidic deposition (often called acid rain) is a contaminant problem that can lead to aquatic habitat acidification and detrimental effects on finfish populations was first documented in southern Scandinavia in the 1950's[146] and about a decade later in eastern North America.[147] Acid precipitation in northeastern United States is 60 to 70% sulfuric acid and 30 to 40% nitric acid[148] and assumed to originate primarily from gaseous industrial emissions of oxides of sulfur and nitrogen produced during fossil fuel combustion and metals smelting. Changes in fish species composition and elimination of sensitive species, due to decreased recruitment of young individuals, have been well documented in Scandinavia, the Netherlands, Scotland, eastern Canada, and northeastern United States.[145,149,150,151,152] However, relationships among acid deposition, surface water acidity, and fish population status are much less definitive in other regions of the United States.[153]

Short-lived acidification events (also called episodes, pulses, spates) associated with snow-melt and intense rain storms can severely stress the early life stages of finfishes.[154] Acidic episodes may be more detrimental to fish populations than long-term, gradual habitat acidification processes. Adverse effects of habitat acidification on finfish have been attributed to increased hydrogen ion concentrations (i.e., more acidic pH), and elevated levels of metals. The metals prob-

TABLE 7

Cumulative percent mortality of striped bass early life stages exposed to sodium arsenate (As) and sodium selenate (Se) for 96 hours in estuarine water (3-6 ppt).[122]

Treatment	Yolk-Sac Larva[a]		Postlarva[b]		Juvenile[c]	
	Mean Concentration (ug/L)	Percent Mortality	Mean Concentration (ug/L)	Percent Mortality	Mean Concentration (ug/L)	Percent Mortality
As Only	0	22	0	6	0	17
	9,300	50	3,400	25	10,300	57
	17,200	60	7,300	60	18,400	37
	28,700	98	12,500	100	19,100	47
Se Only	0	22	0	6	0	17
	4,900	17	8,900	6	48,500	40
	9,800	39	14,300	7	90,100	97
	14,300	40	20,200	35	142,100	100
As/Se Mixture	10,300/ 4,700	60	3,500/7,600	15	9,800/ 40,600	87
	21,300/10,100	88	8,400/14,300	76	18,400/101,000	70
	29,800/14,200	100	11,900/21,000	96	25,200/146,900	80

[a] Age = 1-d old at start of 96-h test

[b] Age = 16-d old at start of 96-h test

[c] Age = 75-d old at start of 96-h test

lem is primarily due to pH-related mobilization or leaching of toxic metals (e.g., aluminum, cadmium, copper, zinc, lead, manganese) from watershed soils and aquatic sediments, and secondarily from acid precipitation itself which can contain several heavy metals, particularly near smelters.[155,156,157,158,159] Elevated hydrogen ion concentrations can also decrease fish tolerances to low dissolved oxygen levels[160] and enhance their sensitivities to an array of inorganic and organic contaminants via changes in compound toxicity or accumulation kinetics.[161,162,163] Other studies have shown that contaminant toxicities to fishes are either unrelated to pH[164,165] or ameliorated in low pH waters.[161,166,167] This problem is complicated by pH-related changes in chemical speciation of metals.

Acidic deposition is not limited to northern climates, but also occurs in the middle Atlantic and southeastern United States.[168,169,170,171,172,173] Recent investigations suggest that habitat acidification may be an important ecological

TABLE 8

Cumulative percent mortality and LT_{50} (time in hours to 50% mortality) for American shad yolk-sac larvae exposed to pH and aluminum during a 55-h continuous exposure experiment in the laboratory.

Nominal Treatment		Cumulative Mortality (%)		Time to 50%
pH	Aluminum (ug/L)	After 24 hours	After 55 hours	Mortality (h)
7.5	0[a]	0	6	—
	50	34	46	—
	100	30	50	—
	200	36	52	60.0
	400	40	80	30.0
6.7	0	6	14	—
	50	32	98	31.8
	100	24	92	37.5
	200	31	100	24.3
	300	100	100	b
6.2	0	29	100	29.7
	50	29	98	24.9
	100	52	100	b
	200	82	100	17.8
	400	100	100	b
5.7	0	23	100	28.0
	50	33	100	23.7
	100	46	100	24.7
	200	92	100	b
	400	100	100	b

[a] Control group

[b] All test organisms were dead within 16 hours.

problem in Chesapeake Bay,[174,175,176] especially in freshwater reaches of higher order streams which drain the Coastal Plain physiographic province.[177,178] This region is underlain by thick layers of unconsolidated sand and gravel, silty sand, clay, marl, and shell beds superimposed upon buried rocks of the Piedmont province.[179] The thickness of Coastal Plain sediments preclude interaction between acid deposition and bedrock, pH and base saturation characteristics of the soils are low, and alkalinity values in smaller tributaries are characteristic of acid-sensitive surface waters.[169,180]

Water chemistry data collected in 23 higher order streams draining inner and middle coastal plain areas of Maryland's eastern and western shores revealed acidic conditions during a relatively wet spring, March and April of 1983 (Figure 10). Several streams exhibited temporary, storm-associated depressions of pH (to 4.5) and alkalinity (to 0.3 mg/L as $CaCO_3$), accompanied by increases in dissolved aluminum levels to 4.0 mg/L.[177] Other recent studies in Maryland detected acidic pulses associated with rainstorms in freshwater sections of the Choptank River,[182] the Nanticoke River,[25] and Granny Finley Branch[183] on the eastern shore; and in Lyons Creek on the western shore.[26] pH depressions in these coastal plain streams are usually short-lived phenomena exhibiting rapid changes in hydrogen ion concentration (Figure 11) accompanied by equally rapid changes in stream stage, turbidity, and dissolved aluminum levels.[26,178,184]

Recent field and laboratory studies demonstrated that the early life stages of striped bass, blueback herring and American shad are very sensitive to

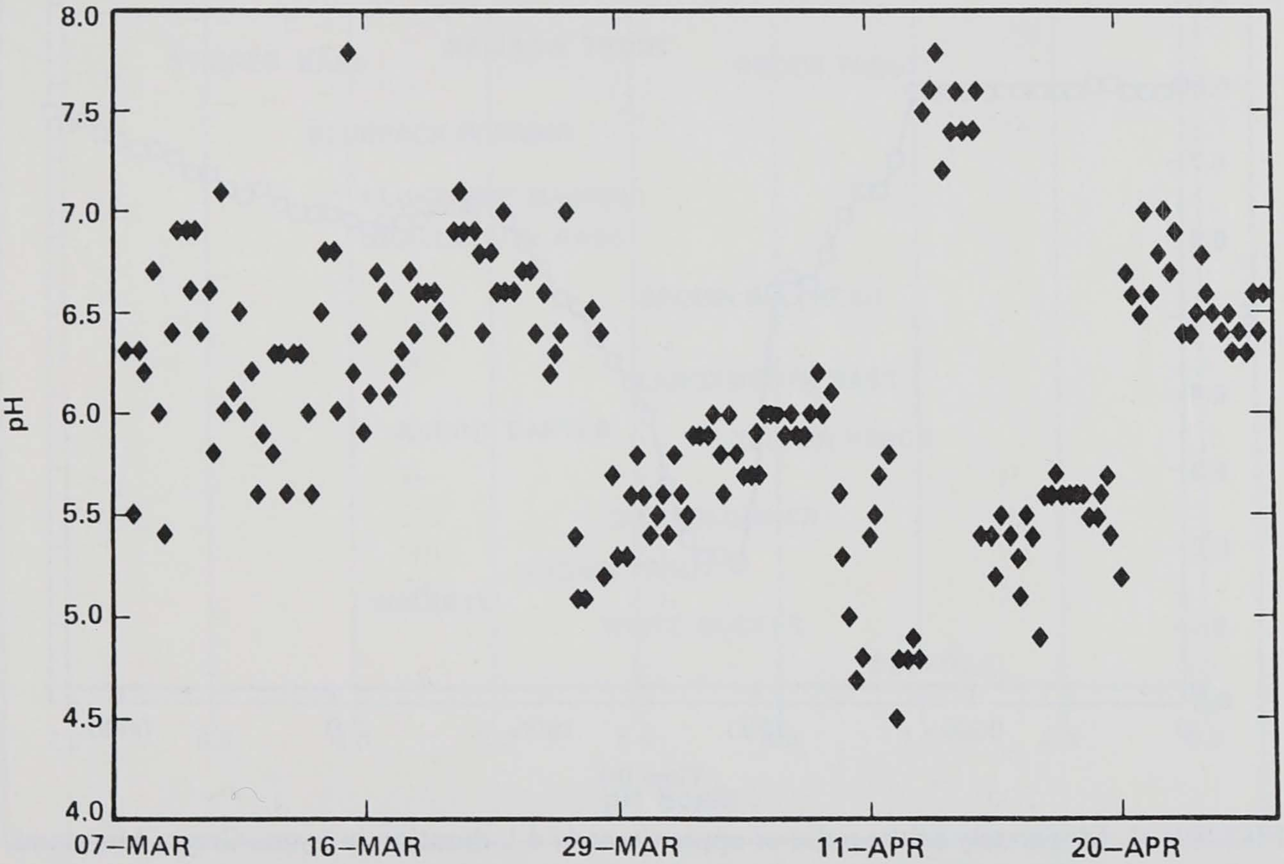

FIGURE 10. pH measurements in 23 higher order tributaries of Chesapeake Bay during spring 1983.[181]

moderate acidity. These three anadromous species are among the most sensitive finfishes in Maryland yet studied (Figure 12 and Table 8). Fertilized alosid eggs were generally more resistant than larvae to pH and dissolved aluminum in the laboratory. Blueback herring[26,117] and American shad (Table 8) larvae tolerated pH 6.5 in the laboratory, but succumbed to pH 5.7 or 6.2. The toxic effect of pH was intensified by simultaneous exposure to dissolved inorganic aluminum, especially for American shad. These laboratory-derived predictions of pH and aluminum toxicity to blueback herring and American shad must still be verified during in-situ field experiments.[26] Survival of striped bass larvae was diminished by exposure to pH and aluminum in the laboratory,[186] supporting field observations in the Nanticoke River.[25] Investigators are beginning to study the effects of acidic pulses on two semi-anadromous Bay species, yellow perch and white perch.[180]

In summary, research into the role of habitat acidification on the population dynamics of finfishes in Chesapeake Bay has just begun, and most studies have focused on Maryland waters.[138] The available data demonstrate that survival of striped bass larvae was diminished by storm-associated changes in pH, dissolved aluminum, and water hardness in one spawning-nursery area, the Nanticoke River, during spring 1984. Laboratory data also indicate that blueback herring and American shad eggs and larvae are very sensitive to pH and

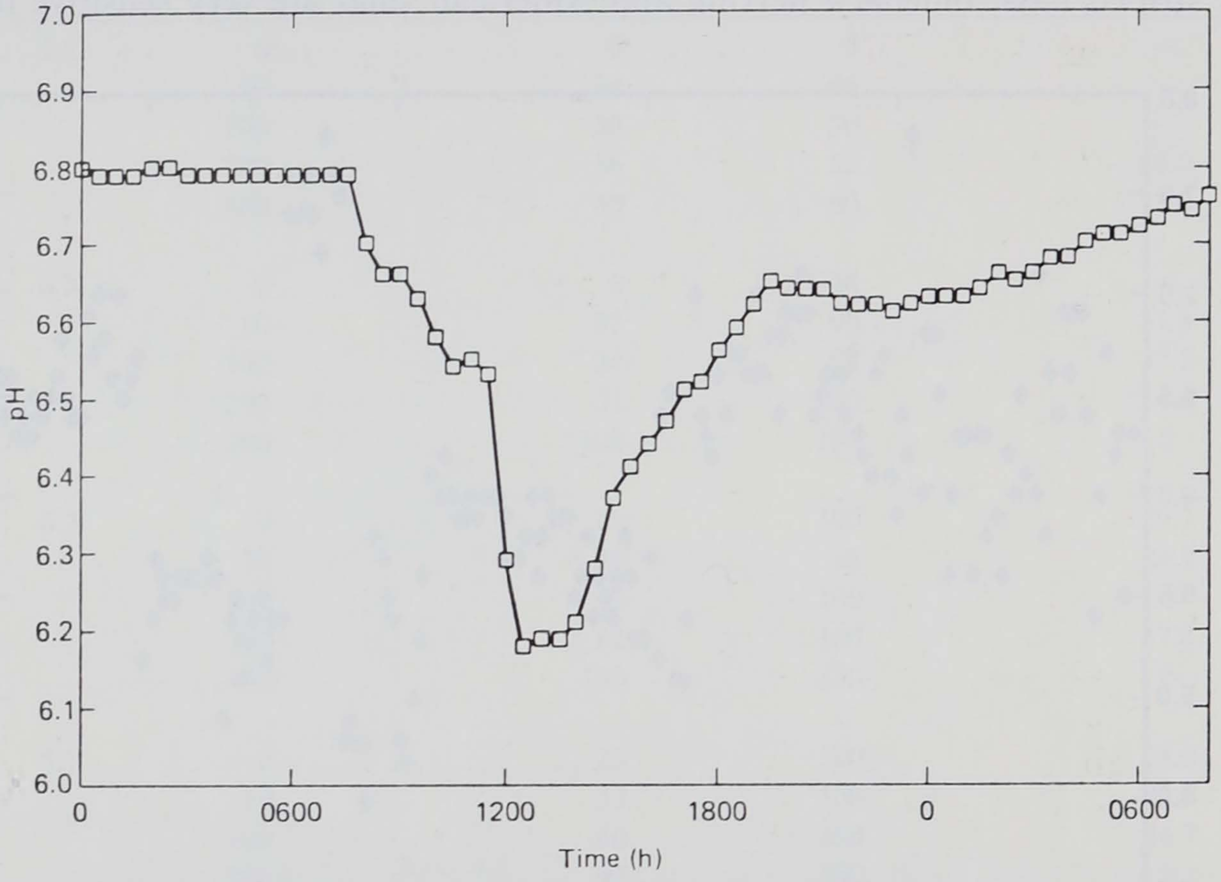

FIGURE 11. Moderately acidic pulse of approximately 4-h duration in Lyons Creek, Maryland, during June 1986 in response to a 2-d rainstorm measuring 0.94 cm. Stream pH was measured with an in-situ monitor, Hydrolab Datasonde model 2030-DS.[26]

aluminum conditions that have been measured in several coastal plain spawning sites. To date, however, no direct link between acidic deposition, habitat acidification, and fish mortality has been established for any Maryland watershed.[138]

RADIONUCLIDES

Nuclear power plants in the United States are licensed and regulated by the Nuclear Regulatory Commission (NRC). Conditions imposed in the operating licenses for each plant allow routine discharges of low levels of radioactivity to the environment. These releases must be within the guidelines of Federal regulations. Within the Chesapeake Bay system are five nuclear power plants: Peach Bottom and Three Mile Island on the Susquehanna River in Pennsylvania, Calvert Cliffs on the western shore of the Bay in Maryland, and the North Anna and Surrey plants on the James River in Virginia.

Radionuclide releases from the Calvert Cliffs Nuclear Power Plant to atmospheric, terrestrial, and aquatic environments are monitored by the utility company (Baltimore Gas and Electric Company) and two Maryland State agencies (Department of Health and Mental Hygiene, DHMH; Power Plant Research

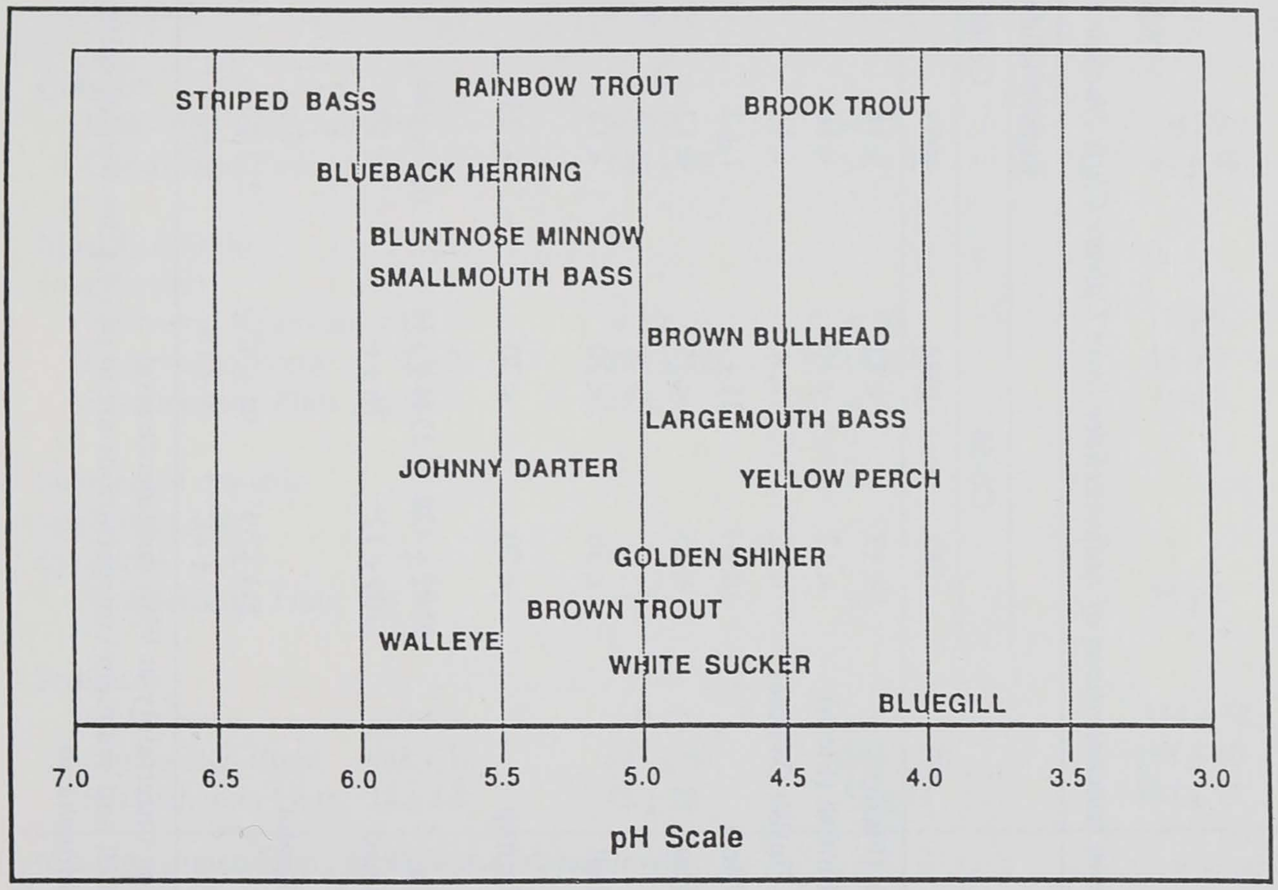

FIGURE 12. Critical pH values causing mortality in some segment of the life cycle for Maryland fishes.[185]

TABLE 9

Maximum concentrations of radionuclides from Calvert Cliffs Nuclear Power Plant in aquatic biota samples from Chesapeake Bay, 1983-1984.[187]

Sample	Radionuclide Concentration[a,b] (pCi/kg wet weight)							
	Co-58		Co-60		Zn-65		Ag-110m	
	1983	1984	1983	1984	1983	1984	1983	1984
Seaduck (Flesh)	< 13	< 12	< 14	< 14	< 28	< 28	< 17	< 17
Edible Finfish (Flesh)	< 12	< 10	< 10	< 8	< 20	< 8	< 12	< 10
Forage Finfish (Whole)	< 12	< 8	< 15	< 8	< 30	< 12	< 20	2 ± 2
Oyster Meat	10 ± 5	21 ± 4	1 ± 2	< 8	67 ± 13	21 ± 8	420 ± 16	250 ± 10
Crab Meat	< 15	< 15	< 12	< 8	< 12	< 12	15 ± 7	10 ± 5 6 ± 5
Crab Shell	< 30	< 20	< 14	< 10	< 40	< 16	< 25	56 ± 19 38 ± 11
Grass Shrimp	< 20	< 20	± 25	< 15	< 30	< 20	8 ± 9	46 ± 18 6 ± 4
Epifauna	2192 ± 428	2344 ± 153	661 ± 244	351 ± 76	± 200	< 100	339 ± 250	307 ± 89
Macroalgae	109 ± 12[b]	16 ± 6[c]	< 10	< 10	< 15	< 15	< 15	31 ± 9[c] 8 ± 5[d]
Bay Sediment								
Clay	233 ± 59	91 ± 47	213 ± 31	173 ± 21	< 30		39 ± 17	58 ± 25
Sand	87 ± 21	70 ± 18	50 ± 10	52 ± 8	± 30		24 ± 37	12 ± 8

[a] Counting uncertainty at 95% confidence level

[b] Crab shell and sediment concentrations are in pCi/kg dry weight; epifauna concentrations are in pCi/kg ash weight.

[c] *Enteromorpha* sp.

[d] *Ulva* sp.

Program, PPRP). Radiological surveillance indicates that this plant is in compliance with operating license guidelines imposed and regulated by the NRC to assure no adverse human health or environmental effects.[187] Low levels of plant-related Co-58 and Co-60 were detected in Bay sediments in the Calvert Cliffs area. Zn-65 and Ag-110m were detected in some aquatic biota, but not

TABLE 10

Maximum concentrations of radionuclides in aquatic biota attributed to Peach Bottom Atomic Power Station, 1983-1984.[187]

	Radionuclide Concentration (pCi/kg, wet weight)[a,b]			
Sample Type	Co-60	Zn-65	Cs-134	Cs-137[c]
Edible Finfish (Flesh)				
Holtwood	<10	<20	< 14	6 ± 6
Conowingo Pond	<10	82 ± 21	94 ± 15	288 ± 20
Conowingo Dam Tailrace	<10	21 ± 11	53 ± 17	81 ± 11
Susquehanna Flats	<10	<60	<27	22 ± 19
Forage Finfish (Whole)				
Holtwood	<10	<20	<10	14 ± 3
Conowingo Pond	19 ± 5	639 ± 200	51 ± 6	76 ± 15
Conowingo Dam Tailrace	<7	59 ± 11	49 ± 5	70 ± 7
Crayfish				
Holtwood Reservoir	<25	<50	<25	<30
Conowingo Pond	7 ± 15	106 ± 66	81 ± 44	94 ± 48
Mussel (*Elliptio complanata*)				
Holtwood Reservoir	<15	<30	<20	2 ± 8
Conowingo Pond	2 ± 2	269 ± 32	10 ± 7	11 ± 7
Susquehanna Flats	<15	13 ± 9	<7	1 ± 2
Submerged Aquatic Vegetation (*M. spicatum*)				
Susquehanna Flats	± 8	<8	<4	35 ± 7
Sediment				
Holtwood	0	0	0	334 ± 12
Conowingo Pond	988 ± 18	837 ± 67	308 ± 20	1163 ± 40
Susquehanna Flats	28 ± 12	45 ± 22	57 ± 11	383 ± 13

[a] Counting uncertainty at 95% confidence level.

[b] Sediment concentrations are pCi/kg wet weight.

[c] Primarily attributable to weapons testing fallout; however where Cs-134 was also present, a power plant produced Cs-137 increment is indicated.

in edible finfish (Table 9). Oysters in the vicinity of the plant discharge contained the highest levels of Zn-65 (67 ± 13 pCi/kg wet weight) and Ag-100m (420 ± 16 pCi/kg wet weight) of edible aquatic biota. These radionuclide concentrations fluctuated over time in response to variations in quantities of radioactivity released by the power plant and by oyster assimilation and depuration rates. If consumed by humans, the maximum concentrations of radionuclides detected in finfish or other aquatic biota could produce radiation doses that are orders of magnitude lower than doses resulting from naturally radioactive sources in the Bay.

Radiological surveillance of the Peach Bottom Atomic Power Station on the Susquehanna River in Pennsylvania, 4.8 km upstream from the Pennsylvania-Maryland border, was conducted by the utility company (Philadelphia Electric Company), DHMH, and PPRP.[187] The data indicate that the plant is in compliance with operating license guidelines. Low levels of plant-related Zn-65, Cs-134, and Cs-137 were detected in sediments and aquatic biota in the Conowingo Pond, the lower Susquehanna River, and the Susquehanna Flats portion of the upper Chesapeake Bay (Table 10). Edible finfish species with detectable concentrations of radionuclides included channel catfish, carp, hybrid (striped x white) bass, walleye, white perch, smallmouth bass, and largemouth bass.

The other nuclear power plant located on the Susquehanna River in Pennsylvania, about 67 km upstream from the Pennsylvania-Maryland border, is the Three Mile Island Nuclear Station. Owned jointly by Metropolitan Edison Company, Pennsylvania Electric Company, and Jersey Central Power and Light Company, the plant has not operated since the accident at Unit 2 in March 1979.[187]

In summary, routine discharges of radionuclides to Chesapeake Bay from nuclear power plants are not causing detectable adverse effects on finfishes, at least in Maryland waters. Due to NRC licensing requirements, these plants are closely monitored by the operating utilities and appropriate State agencies.

DISCUSSION

This review of current contaminant effects studies in Chesapeake Bay provides relatively convincing evidence that contaminants *may have been* or *may be* a factor responsible for the recent decline of several finfishes, or at least *may be* contributing to the continuing series of poor year classes. The growing body of tissue residue data show that many inorganic and organic contaminants present in Bay habitats are accumulated by finfishes. Laboratory studies reveal that exposure to mixtures of several contaminants can decrease survival of larvae and juveniles. Such observations are necessary to pursue the postulate that contaminants actually *have affected* or *are affecting* finfish populations in Chesapeake Bay. However, these observations are not, in themselves, sufficient to reach a definitive conclusion. It is also necessary to rigorously demonstrate that contaminants are adversely affecting finfish survival in nature, and compile evidence that the status of finfish populations has been altered by exposure to environmental contaminants.

In-situ cages, enclosures, or microcosms represent a useful first step toward verification of laboratory-derived predictions of contaminant effects on fish populations or in nature.[188] This approach has recently been used in Chesapeake Bay to assess the response of striped bass and blueback herring to acidic episodes and other habitat quality concerns.[25,26] In-situ studies with striped bass larvae in the Nanticoke River[25] successfully corroborrated laboratory study results on the toxicity of pH and aluminum.[186] In-situ studies with blueback herring are continuing. Although valuable in extrapolating laboratory study results to nature, the in-situ approach is logistically limited to a few species and a few spawning or nursery sites in any given year, unless a massive research effort is funded. Nevertheless, in-situ studies are feasible, underway in Chesapeake Bay, and yielding important results.

Taing the next step to a prediction of species population response to contaminant effects is exceedingly difficult.[189] Translation of a contaminant effect, via direct mortality, indirect mortality (mediated through growth, behavior, physiology), or changes in other community components that alter food availability or predator and competitor numbers, into a change in the size or productivity of a focal species population will necessarily become entangled in the array of inter- and intraspecific processes that regulate population size. The operation of these interacting process may eliminate or exaggerate the ultimate effect of contaminant-induced stress on fish populations, unless catastrophic levels of mortality result that are so obvious they cannot be easily masked. This level of ecological complexity suggests that efforts to evaluate fish population responses to contaminants will require concomittant study of other abiotic and biotic factors that could affect population dynamics.

The fisheries literature offers limited documentation that contaminants in Chesapeake Bay or other habitats have affected the status of finfish populations. Whenever serious decline or complete collapse of a commercial or sports fisheries has been documented, the proposed primary causes are usually an intensification of fishing pressure leading to overexploitation or severe changes in the aquatic environment (e.g., pollution) or both.[17] Identifying the relative importance of these two mortality sources, where both exist, is difficult. Examples of decimated fish species that were exposed to overfishing alone or overfishing and pollution together are numerous in the literature; but historical documentation of a declining fishery exposed to pollution in the absence of overfishing does not appear to exist.[17]

Mathematical models and multivariate statistics are two analytical techniques that have been applied to the study of contaminants and Chesapeake Bay finfish populations. Goodyear[20] addressed this topic for striped bass with a simulation modeling approach. He acknowledged evidence suggesting that environmental contaminants present in the Bay could impair survival of young fish in freshwater portions of spawning and nursery grounds. He also accurately stated that the level of excess mortality imposed on the striped bass population by toxic chemicals is unknown. Based on a Leslie matrix modeling approach, Goodyear concluded that an increase in population fecundity sufficient to offset even severe losses due to contaminant toxicities could be achieved by a reduction in fishing mortality. Such a reduction in fishing mortality could halt or even reverse the current stock decline.[20]

Use of various modeling approaches for assessing the effects of contaminants on fish populations was recently reviewed by Vaughan et al.[190] They compared five current approaches and concluded that a combination of bioenergetics and Leslie matrix approaches offers a powerful tool for estimating long-term impacts of toxic contaminants on fish populations, even though this modeling combination is very data intensive. A Leslie matrix model requires several age-class-specific parameters including: fecundity per mature female, proportion of females that are mature, sex ratios, mortality rates (natural and fishing), and first and last age classes having mature females. A Leslie matrix approach is advantageous because mortality (all age classes), individual growth (via growth rates), and reproduction (via condition factors, egg production, egg viability) can all be altered in the model to reflect specific contaminant effects on these processes.

Bioenergetics models examine the factors affecting growth of an individual fish, but these models can also be used to simulate the growth of representative individuals from each cohort over its lifetime.[190] When a bioenergetics approach is applied to an entire fish population, numbers of fish in each cohort must be obtained. Other data needs include a time series of ambient water temperature in the focal habitats and corresponding estimates of either body size or daily consumption. Additionally, estimates are needed for the physiological parameters describing rates of consumption, respiration, egestion, excretion, and reproductive loss as functions of body size, temperature, and other variables. One major advantage of the bioenergetics approach is that the predicted population response to contaminant stress can reflect the particular mode of action of that stress.

Vaughan et al.[190] concluded that a Leslie matrix-bioenergetics combination approach would allow detailed comparisons of stressed and unstressed fish populations. By comparison, surplus production and stock-recruitment models generally require long time series of data on population parameters that are difficult to estimate. Yield models are also less desirable for addressing contaminant effects on fish populations because any effect of toxicant stress on reproduction is confounded with mortality before the age of recruitment.

Vaughan et al.[190] cautioned that fisheries scientists should not expect too much from currently available fish population models. Many questions that we want to ask are beyond the current state of the art, whether the questions relate to the effects of contaminants or power plants.[191] Vaughan et al.[190] also stressed that modeling approaches should be used to compare stressed to unstressed fish populations rather than attempting the unrealistic goal of precisely predicting absolute population effects.[192]

Schaaf et al.[193] used a Leslie matrix model to simulate fish population changes through time and develop a technique for assessing the effects of acute and chronic pollution on several marine stocks via a comparison of stock vulnerability to pollution. Deterministic, stochastic, density-independent, and density-dependent versions of their simulations were achieved by modifying one element of the matrix, S_0, first year survival. They related various population responses among the fish stocks to V_x, the age distribution of expected egg production, and demonstrated that information on age-specific egg production of a stock can yield a prediction of that species' response to pollution perturbation.

Schaaf et al.[193] acknowledged the limitations of their modeling approach and the inherent difficulties in obtaining reliable estimates of S_o and compensation factors for even the most intensively studied fish stocks. They view their approach as useful for bounding the magnitude and time horizon of contaminant impacts, and thereby provide information useful to resource managers. For one of eight fish species examined, Atlantic menhaden, their modeling approach predicted that heavily exploited stocks are most susceptible to additional pollution stress.[193]

The other approach to assessing the effects of contaminants on finfish populations that has been applied to Bay species involves multivariate statistics. This approach is an extension of the method commonly used to relate commercial landings or juvenile abundance indices to environmental variables,[16,194] in an attempt to understand recruitment variability. Although more empirical than theoretical, a desirable advantage, use of regression statistics for assessing the role of contaminants has several limitations. Of major importance is that relationships identified by step-wise multivariate and time series regression statistics are correlative and not necessarily causal. However, if a large degree of annual variation in commercial landings or juvenile abundancce can be accounted for, statistically, by contaminant levels, one can be reasonably confident that contaminants play an important role in determining numbers of fish harvested or numbers of juveniles produced. Caution must be exercised, however, if contaminant levels are collinearly related to other key parameters that were either excluded or included in the regression analyses.

A multivariate statistical approach is being used by NOAA to study contaminant effects on fishes. The Ocean Assessments Division is funding studies to determine if populations of finfish and shellfish are threatened by contaminants present in estuarine and coastal waters.[195] A major objective of this program is to examine historical data and determine if past trends in stock abundance of important species can be correlated with contaminant inputs. One ambitious goal of this program is to "determine the necessary extent of control on chemical inputs to prevent their affecting fish populations."[195, p. 2).

As part of NOAA's program, Polgar et al.[196] investigated the relationships among pollutant loadings and stock levels in several northeastern United States estuaries, including the Potomac River. Using a reconstructed time series of long-term trends in commercial fisheries abundance, climate, and several indices of pollution loading,[197] Polgar et al.[196] evaluated hypotheses concerning effects of human population changes and dredging on stock trends for striped bass and American shad in the Potomac estuary. Human population history was one surrogate pollution variate. Dredging history, an indicator of habitat alteration, was the other pollution variate included in the analysis.

Climatic factors rather than the two surrogate pollution variates appeared to dominate striped bass dynamics in the Potomac estuary from 1929 through 1976.[196] The effect of human population change on the index of American shad stocks was significant compared to climatic factors. This result suggests that some aspects of anthropogenic pollution in the Potomac estuary watershed (e.g., industrialization, land use, municipal waste treatment, but not dredging) were somehow linked to variability in shad stock size from 1929 through 1976. Summers et al.[198] suggested that the primary pollutant variable was sewage loading in shad spawning habitats.

This review of current studies which have attempted to link variations in abundance of selected Chesapeake Bay finfish populations to anthropogenic factors (including contaminants) presents limited support for the hypothesis that contaminants have been or are playing an important role in the declining status of several species populations. Lack of strong quantitative evidence for adverse contaminant effects is, however, no cause for optimism that habitat quality in the Bay is good. Some fish populations may already be severely affected by contaminants, coupled with stress from other sources of mortality like fishing, but we may not be able to detect it.[199]

The current state of the art in our understanding and ability to model fish population dynamics may prevent us from detecting all but catastrophic effects of contaminant stress. Recognition of the limitations of our science is necessary but hardly reassuring to ecologists, resource managers, and administrators alike. However, as long as potentially toxic contaminants continue to enter Chesapeake Bay and alter habitat quality in important fish spawning and nursery areas, studies of contaminant effects should continue. To effectively manage finfish populations and knowledgeably harvest only surplus production, scientists must strive to understand the role of all mortality sources on these populations, including contaminants.

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