1987

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CONTAMINANT PROBLEMS AND MANAGEMENT OF LIVING CHESAPEAKE BAY RESOURCES

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Founded on April 18, 1924

A Publication of
The Pennsylvania Academy of Science

Contaminant Problems and Management of Living Chesapeake Bay Resources. Edited by S. K. Majumdar, L. W. Hall, Jr. and H. M. Austin.

Chapter Sixteen

CONTAMINANT EFFECTS ON CHESAPEAKE BAY SHELLFISH

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ABSTRACT

The paper reviews contaminant effects on Chesapeake Bay shellfish from two avenues (1) adverse biological effects on the organisms and (2) fisheries closures due to bacterial and chemical contamination. The use of shellfish to monitor anthropogenic inputs of chemical contaminants is also discussed. Fisheries closures due to bacterial contamination account for the greatest economic loss due to man's activities. Kepone contamination in the James River, Virginia caused fisheries closures but has not appeared to cause biological damage to the resources. Organotin compounds from antifouling paints appear to pose a threat to Chesapeake Bay shellfish.

INTRODUCTION

Shellfish resources can be damaged by contaminants (chemical and biological) in two ways. The first is most applicable to chemical contamination where concentrations of chemicals in water or sediments may reach levels that cause adverse biological effects on the organisms. This damage may be acute, causing death, or chronic, causing lowered rates of recruitment, growth, etc. The other avenue of impact is economic, an impact brought about by closures of fisheries because of chemical or microbial contamination. In the case of chemical contamination it must be pointed out that concentrations of toxic substances in animals which may cause fisheries closures are not necessarily the same as those which may cause biological effects on the animals. This point is illustrated in Figure 1 which shows the relationship between residues of Kepone in blue crabs,

biological effects levels and closure levels. As can be seen in this figure the residue level at which the fishery is closed is 0.4 ppm while levels at which biological effects, such as carapace thinning do not occur until residues reach – 1.4 ppm. The reverse can be true also, i.e. effects levels expressed in terms of residue levels in the animals may be reached before residues climb to levels which are of public health concern.

Fisheries Closures – Bacteriological

Because certain species of shellfish, e.g. oysters and hard clams, are frequently consumed raw, growing areas are closed to direct marketing of the shellfish resource if they are contaminated by bacteria which can cause human diseases. The areal extent of these closures varies with (1) seasonal patterns of runoff which brings in potential disease causing organisms, (2) operational malfunctions at sewage treatment facilities, and (3) the proximity of the shellfish beds to marinas and other polluted areas. Bacterial monitoring programs to delineate areas of contamination are conducted routinely by the State Health Departments of Virginia and Maryland. Areas are opened and/or closed based on the results of these monitoring programs.

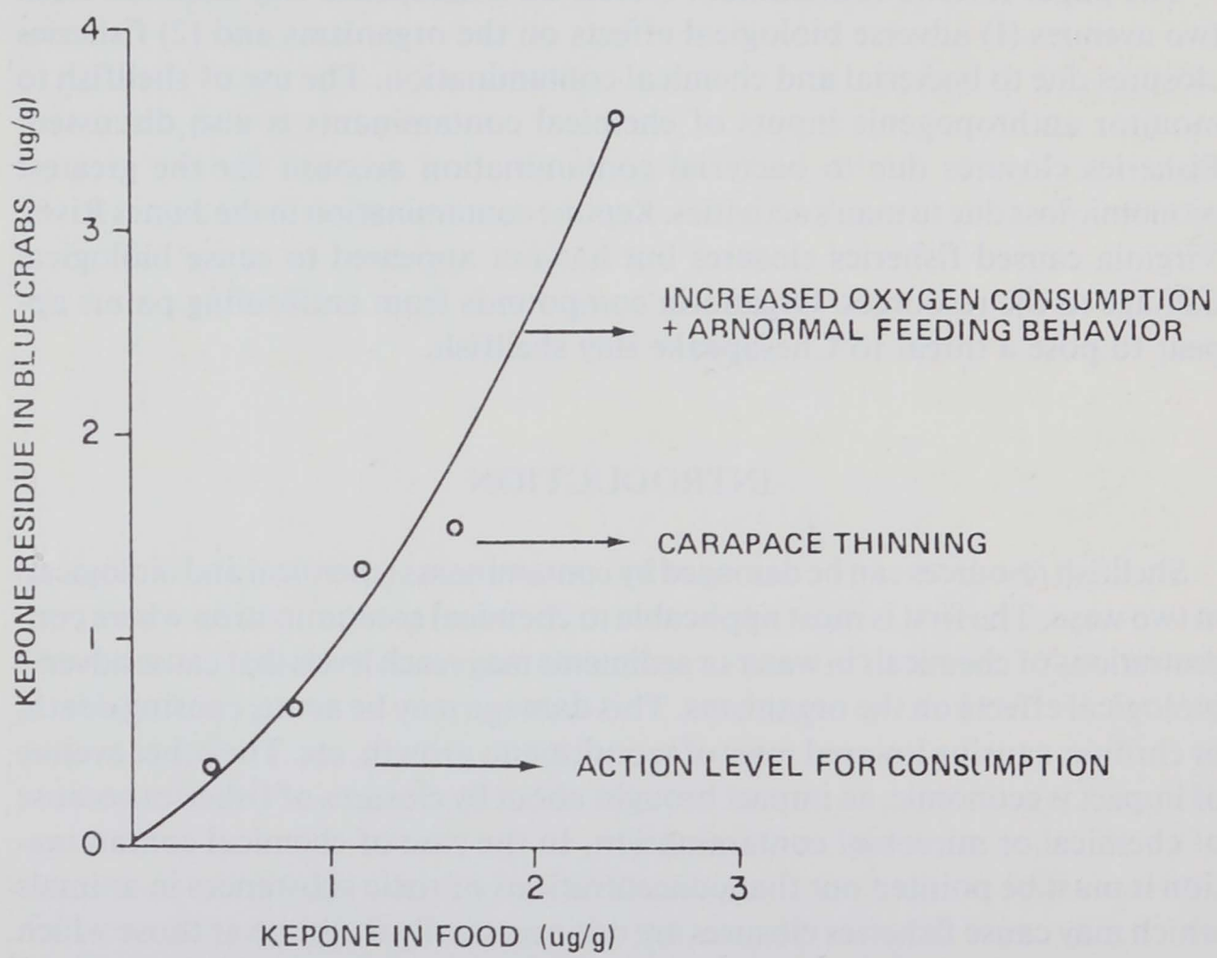

FIGURE 1. Kepone Residues (wet wt) in Blue Crabs vs. Effects Levels, data modified from Fisher, et al.

Table 1 lists the acreages of oyster harvesting areas, public and leased, by basin; these oyster bars are shown in Figure 2. Areas closed to direct marketing because of bacterial contamination are listed in Table 2. The areas condemned during the summer of 1986 represent 35% of the total harvesting areas. Shellfish may be harvested from closed beds and transplanted to "clean" areas where they are allowed to depurate prior to reharvesting and sale. This process is expensive and often results in a loss of 20-30% of the animals which either die or cannot be recovered with normal harvesting techniques. New methods, e.g. the use of cages which are elevated off the bottom, are presently being evaluated for depuration of hard clams in Virginia.[3] This technique, although still in the experimental stage, appears to offer great promise in reducing mortality, increasing reharvesting efficiency and reducing time and expense.

Fisheries Closures – Chemical

Shellfish have the ability to concentrate hydrophobic chemicals orders of

TABLE 1

Acres of Public and Leased Oyster Grounds

Basin	Public Oyster Grounds	Leased Grounds	Total
Chesapeake Bay North	0	21	21
Chesapeake Bay Upper Central	19,038	0	19,038
Chester River	5,547	0	5,547
Eastern Bay	26,979	212	27,191
Choptank River	1,378	454	1,832
Chesapeake Bay Lower Central	29,173	778	29,951
Patuxent River	7,543	1,119	8,662
Honga River	15,475	1	15,476
Fishing Bay	11,811	333	12,144
Nanticoke River	577	190	767
Wicomico River	568	1,268	1,836
Chesapeake Bay South	32,315	0	32,315
Tangier Sound	31,043	889	31,932
Pocomoke Sound	4,899	4,303	9,202
Potomac River	28,523	9,389	37,912
Rappahannock River	44,254	19,022	63,276
Piankatank River	16,000	328	16,328
Chesapeake Bay General	35,566	20,170	55,736
Mobjack Bay	17,061	1,516	18,577
York River	2,381	26,729	29,110
Mattaponi River	0	0	0
Pamunkey River	0	0	0
Chickahominy River	0	0	0
James River	25,152	13,260	38,412
TOTAL	355,283	99,982	455,265

Source[2]

magnitude higher than those found in the aqueous phase. For those chemicals which pose a potential threat to human health, (e.g. chlorinated pesticides, PCBs, and certain dioxins), the Food and Drug Administration and/or the U.S.E.P.A. (United States Environmental Protection Agency) establishes limits above which

FIGURE 2. Chesapeake Bay Oyster Bars shown in black.[2]

interstate transport of the food item is restricted. State health departments frequently adopt these limits and impose closures for commercial and/or recreational harvest of species when necessary. Establishment of limits and subsequent terminology varies with, (1) the geographical extent of the contamination, (2) the type of food items contaminated, (3) the amount of the particular food item consumed by the average citizen and (4) the mammalian toxicity of the compound. Frequently additional warnings on limiting consumption are issued for groups of people considered special risks (e.g. pregnant women and children).

In the lower Chesapeake Bay, shellfish closures due to chemical contamination have been limited to those due to Kepone in the James River. Soon after the 1975 discovery of Kepone contamination in the James River, the oyster and crab fisheries were closed to commercial harvesting. The oyster fishery was reopened in 1976 when it was found that seed oysters, the major oyster resource

TABLE 2

Acres of Condemned Shellfish Areas
Maryland and Virginia

Basin	Acres Condemned[a]
Chesapeake Bay North	0
Chesapeake Bay Upper Central	17,600
Chester River	9,330
Eastern Bay	14,560
Choptank River	5,330
Chesapeake Bay Lower Central	1,000
Patuxent River	14,660
Honga River	350
Fishing Bay	0
Nanticoke River	300
Wicomico River	4,000
Chesapeake Bay South VA	915
Tangier Sound	1,098
Pocomoke Sound	1,485
Potomac River, MD	2,660
Potomac River, VA Tributaries	5,395
Rappahannock River	7,105
Piankatank River	700
Chesapeake Bay General, VA	7,040
Mobjack Bay + Tributaries	827
York River + Tributaries	9,105
Mattaponi River	0
Pamunkey River	0
Chickahominy River	0
James River + Tributaries	53,945[b]
Total	157,405

[a]productive areas only

[b]includes 35,509 acres in Hampton Roads, most of which are too deep to allow oyster harvest

in the river, rapidly depurated Kepone body burdens when transplanted to clean growing areas. The blue crab fishery was affected longer with closures remaining in effect for 4 years. The declining residues in crabs from 1976 through 1985 are presented in Figure 3. Residues in female and male crabs differed dramatically in the early years. Roberts and Leggett[4] concluded the loss of Kepone in the egg masses when female crabs spawn was in part responsible for the differing body burdens in males and females.

Figure 4 depicts the rate decline in Kepone residues for male crabs and oysters in the lower James River. These data are of interest because they show similar rates of decrease with time for these two species, yet it has been shown that crabs obtain most of their residues from food[5] while Kepone appears to be available to oysters from both solution and suspended particles.[6]

Economic losses due to shellfisheries closures in the James during 1976 were estimated at $50,000 for the oyster fishery and $67,000 for the blue crab fishery.[7] Losses due to limiting the harvest of crabs continued for another 3 years; however, the extent of economic loss is difficult to estimate because many fishermen moved their operations to other waters or obtained other employment.

Effects of Chemical Contamination — The potential effects of toxic chemicals

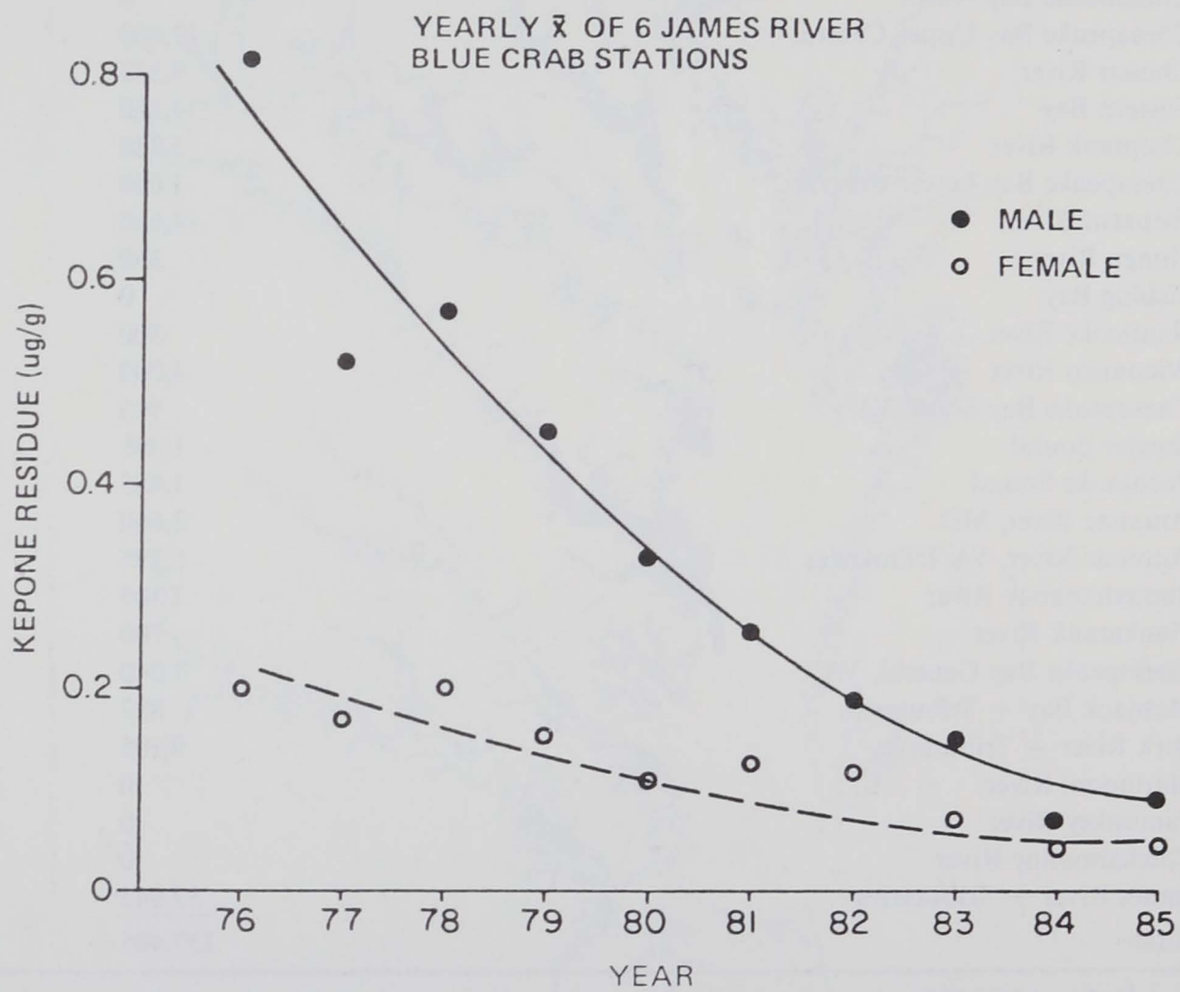

FIGURE 3. Kepone Residues (wet wt) in Male and Female Blue Crabs, vs. Time.

on aquatic animals are usually estimated by conducting laboratory bioassays, the results of which are then related to expected and/or measured environmental concentrations. Acute, partial chronic, and chronic toxicity tests have all been utilized to estimate effects. For shellfish, both molluscs and crustaceans, the larval stages are usually the most sensitive. In this volume, Roberts and Bradley review toxicity data for zooplankton, including larval stages of shellfish. We therefore will limit our discussions to effects on adults, except in the case of Kepone for which, to be complete, we have included the larval data.

KEPONE

After the discovery of Kepone in the James River, numerous studies were conducted to estimate its impact on the biota of the river. The objective of most tests was to estimate those concentrations which would have no deleterious effects on the animals. Once no-effect levels had been determined or estimated,

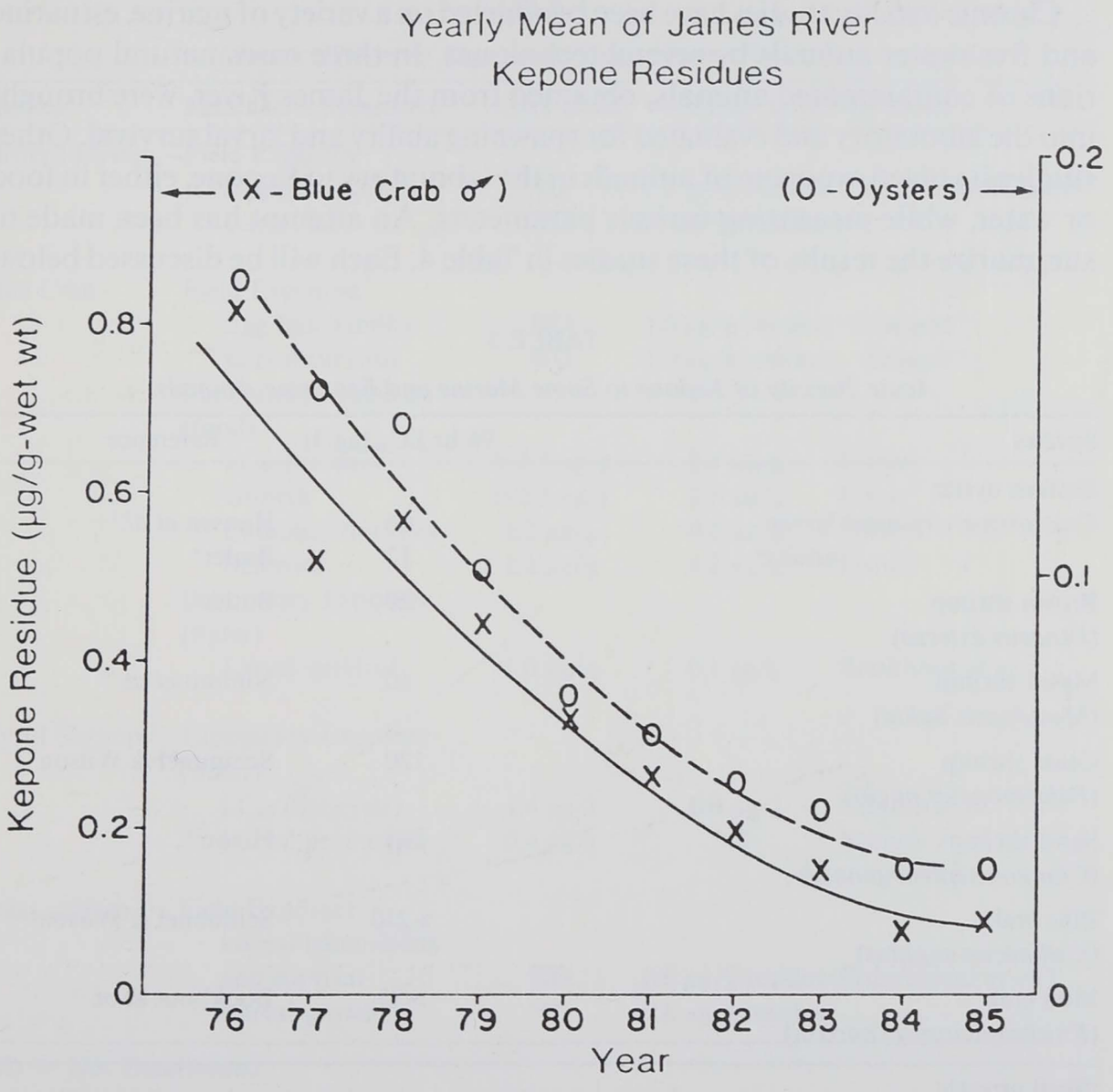

Figure 4. Yearly Mean Kepone Residues (wet wt) in Oysters and Male Blue Crabs vs. Time.

they could be compared with measured levels of Kepone in the river and estimates of the potential for effects could be made.

Acute, partial chronic and chronic toxicity tests have all been utilized to estimate Kepone effects. The ideal procedure to establish safe exposure concentrations involves chronic toxicity testing of the chemical on several different freshwater and/or marine species. Once these tests have been conducted, an application factor can be derived and used to estimate safe chronic exposure concentrations for other organisms from acute toxicity data. The application factor is defined as the ratio of the maximum acceptable toxicant concentration (MATC) to the 96 hour LC_{50}.

Results of acute Kepone toxicity tests conducted on some marine and estuarine animals are summarized in Table 3. Mysid shrimp had an LC_{50} of 10 μg/l while shell growth of oysters was inhibited by 12 μg/l. Dissolved Kepone levels measured in the saline portion of the James since 1979 have ranged between 0.8 and 7 ng/l or 3 orders of magnitude less than those concentrations acutely toxic to the most sensitive shellfish species.

Chronic toxicity studies have been conducted on a variety of marine, estuarine and freshwater animals by several techniques. In three cases natural populations of contaminated animals, obtained from the James River, were brought into the laboratory and evaluated for spawning ability and larval survival. Other studies involved exposure of animals in the laboratory to Kepone, either in food or water, while measuring various parameters. An attempt has been made to summarize the results of these studies in Table 4. Each will be discussed below.

TABLE 3

Acute Toxicity of Kepone to Some Marine and Estuarine Animals.

Species	96 hr LC_{50}(μg/l)	Reference
Eastern oyster		
Crassostrea virginica) larvae	66	Hansen *et al.*[8]
adults[a]	12	Butler[9]
Brown shrimp (*Penaeus aztecus*)	28	Butler[9]
Mysid shrimp (*Mysidopsis bahia*)	10	Nimmo *et al.*[10]
Grass shrimp (*Palaemonetes pugio*)	120	Schimmel & Wilson[5]
Sand shrimp (*Crangon septemspinosa*)	263	Hixon[11]
Blue crab (*Callinectes sapidus*)	>210	Schimmel & Wilson[5]
Mud crab (*Rhithropanopeus harrisii*)	>35	Bookhout *et al.*[12]

[a]Shell growth

In three experiments, oysters, blue crabs and grass shrimp, were collected from locations with different degrees of Kepone contamination and tested for spawning success. A strength of this type of study is that it evaluates animals which have obtained Kepone residues by their natural routes and have, in addition, been subjected to other stresses in the environment. However, since the experimenter has no control of exposure conditions, environmental levels responsible for effects may not be identified.

Oysters were obtained from the James River during July of 1976 and spawned in the laboratory within two days of collection.[13] As measures of the effects of Kepone contamination, egg production, i.e., numbers of eggs produced, larval abnormalities, and setting success were compared to those parameters for control animals from the York River. Kepone contaminated oysters (0.3 μg/g) produced 11 million viable eggs of which 9 million developed successfully to straight hinge larvae and set. Compared to control animals, no increase in larval abnormalities was detected and the development duration was normal. Residue

TABLE 4

Chronic Effects of Kepone on Some Marine Animals.

Species	Parameters Measured	Effect Level	No Effect Level	Reference
Eastern Oyster	Field Exposure			
	Egg production	ND[a]	0.3 μg/g residue	Bender & Huggett[13]
	Larval set	ND	0.3 μg/g residue	
Blue Crab	Field Exposure			
	Egg hatchability	ND	1.0 μg/g residue	Leggett[14]
	Larval survival	ND	1.0 μg/g residue	Leggett[14]
	Laboratory Exposure (food)			
	LC_{50} (65 day)	>2.5 μg/g	2.5 μg/g	Fisher[1]
	Growth	>2.5 μg/g	2.5 μg/g	Fisher[1]
	Carapace thickness	1.2 μg/g	0.8 μg/g	Fisher[1]
	Behavior	2.2 μg/g	1.2 μg/g	Fisher[1]
	Laboratory Exposure (water)			
	Larval survival	1.0 μg/g	0.1 μg/g	Bookhout *et al.*[12]
Mysid Shrimp	Laboratory Exposure (water)			
	LC_{50} (life cycle)	1.4 μg/l	0.4 μg/l	Nimmo *et al.*[10]
	Larval production	0.4 μg/l	ND	Nimmo *et al.*[10]
Grass shrimp	Field Exposure			
	Larval hatchability and survival	ND	0.6 μg/g residue	Provenzano *et al.*[15]
	Larval growth	ND	0.6 μg/g residue	

[a]ND = Not Determined

[b]Egg production greater, so effects not considered significant

levels in James River oysters collected from 10 locations have been monitored by the State Health Department monthly since November of 1975. Of the 120 samples collected in 1976 only 10% exceeded the 0.3 μg/g residue found acceptable in our experiments and in 1977 only 1 of 110 samples exceeded this level. As shown in Table 3, the acutely toxic concentration of Kepone to oysters was 12 μg/l for adults and 66 μg/l for larvae compared to dissolved Kepone levels in the lower saline portion of between 0.8 to 7 ng/l. Based on data from these two experiments it appears highly unlikely that Kepone residues either in the animals or in river water are detrimental to oysters in the James River.

Grass shrimp (*Palaemonetes pugio*), an important member of the food chain in the Chesapeake Bay, were tested in a similar experiment.[15] Shrimp were collected from 6 locations and egg hatchability, larval survival and larval growth were measured as a function of location (degree of Kepone contamination). Larvae hatched from females having Kepone residues of 0.6 μg/g with equal success to those from the control groups. In addition, no effects on development or survival were noted. Acute toxicity of Kepone to this species (Table 3) was estimated to be 120 μg/l.

To investigate the potential effects of Kepone on blue crabs, Leggett[14] studied blue crabs collected from 7 locations, 2 in the lower James and 5 in the lower Bay over a three month period during the summer of 1978. The hatchability and larval of several hundred eggs from each crab was determined and related to degree of Kepone contamination. Over the range of Kepone concentrations in contaminated eggs, i.e., from non-detectable to 1.45 μg/g, no effects of contamination could be demonstrated on embryogenesis, hatchability or larval survival.

Fisher, et al.[1] studied the long-term effects of Kepone exposure to juvenile blue crabs by feeding them with a series of concentrations in naturally contaminated striped bass, *Morone saxatilis,* flesh. Besides mortality, several sublethal effects were measured. Kepone uptake by crabs was linearly related to exposure concentration and reached a maximum of 4.6 μg/g in the first experiment (exposure to 2.5 μg/g). The average number of molt, percent increase in width, mid-body thickness and wet weight per molt did not differ significantly from controls at any Kepone concentration tested in either experiment. At the highest Kepone exposure in each experiment (2.5 and 2.3 μg/g) oxygen consumption was greater than at the other exposure levels and these crabs exhibited "excitable feeding behavior." Also, at high exposure levels (>1.2 μg/g) in the first experiment, crabs which molted had low carapace thickness to width ratios. This effect was not observed in the second experiment. These sublethal effects occurred at tissue levels greater than the average tissue levels found in adult crabs from the James River (Figure 1).

Chronic toxicity of Kepone to mysid shrimp *Mysidopsis bahia,* an estuarine species native to the Gulf states, was studied by Nimmo *et al.*[10] They determined effects by measuring survival, egg production and larval growth after aqueous

exposures. No mortalities were observed among shrimp exposed to Kepone concentrations of 0.4 μg/l, but egg production was reduced compared to that of control populations. Some reduction in growth of young was observed, but the results were erratic. They found growth reductions of 6% at exposure levels of 0.07 μg/l and only 3% at 0.23 μg/l. Although *Mysidopsis bahia* is not native to Chesapeake Bay, a related species, *Noemysis americana,* is resident in the Bay and its tributaries. Roberts *et al.*[16] compared the response of *M. bahia* and *N. americana* to three toxicants (cadmium, sodium lauryl sulfate and Lannate) and found very similar lethal concentrations. Similar sensitivities may hold for Kepone and therefore, we would not predict effects at environmental exposure levels.

ORGANOTINS

In recent years the potential impact of tributyltin (TBT) in Chesapeake Bay has surfaced as a major environmental issue. The following factors are responsible for this concern: (1) the increased use of TBT based paints as antifouling agents on pleasure craft; (2) the recent proposal by the U.S. Navy to utilize TBT on all Navy vessels;[17] and (3) laboratory and field studies in England, France and the U.S., which have implicated TBT in causing abnormalities and/or mortalities in a number of species of shellfish.[18,19,20,21]

Space limitations preclude a complete review of available literature on TBT; however, we have attempted to provide a brief summary of some relevant literature and our assessment of some of the more pressing research needs.

Recent studies in England and France, summarized by Stebbing,[18] have implicated tributyltin in causing decreased spatfall, decreased growth and shell malformations in oysters (*Crassostrea gigas)*). Thain and Waldick[22] showed that a low concentration of tributyltin oxide (TBTO), 0.15 μg/l, inhibited growth of young oysters (*C. gigas*). Thain and Waldock[23] found that the growth of European oyster spat (*Ostrea edulis*) was severely curtailed after 10 days exposure to 0.06 μg/l of TBT. Henderson[24] reported a mortality rate for the American oyster (*Crassostrea virginica*) of 50 percent after 30 days exposure to 2.5 μg/l of TBT. In the same experiment he determined that the oyster's condition index was reduced by exposure to 0.1 μg/l over a period of 57 days.

Stephenson, et al.[21] transplanted oysters (*Crassostrea gigas*), and two species of mussels (*Mytilus edulis*) and *(M. californianus*) in San Diego Bay along a gradient of known seawater TBT concentrations. Reduced shell growth in all three species was found at the stations with the highest levels of TBT. Beaumont and Budd[25] reported about 50% mortality of mussel larvae (*Mytilus edulis*) after 15 days exposure to TBTO concentrations of 0.1 μg/l. For adult mussels of the same species, 96 hr LC_{50} values of 20-60 μg/l have been reported.[26]

Smith[27] found strong evidence that exposure of American mud snails (*Nassarius obsoletus*) to TBT caused a phenomenon known as "imposex" (the

superimposition of male sex characters onto the female). He concluded, however, that for the mud snail such effects produced no significant decrease in reproductive capacity. Gibbs and Bryan[19] described this phenomenon in the dog-whelk (*Nucella lapillus*) and also related its development to TBT exposure. In the case of the dog-whelk, however, they presented convincing evidence that exposures to TBT caused sterility and reproductive failure of the populations. The specific exposure concentrations necessary to induce full imposex development in *N. lapillus* remain to be determined. However, the authors found that exposure of dog-whelks to 0.02 μg/l of TBT for a period of 6 months induced the phenomenon to progress from early to late stages.

The above studies have shown that TBT is quite toxic to a variety of shellfish species. Huggett et al.[28] have shown that potentially toxic concentrations of TBT can exist in marinas in the southern Chesapeake Bay, and Hall et al.[29] found TBT concentrations in marina areas in the upper Chesapeake Bay, which would be toxic to sensitive aquatic animals. However, before the true magnitude of the problem can be determined, we must establish, through long-term exposures, the TBT concentrations which are non-toxic to oysters, clams, and other important shellfish. These studies at a minimum should include an evaluation of the effects of TBT on gametogenesis, larval survival, spat growth and the potential for imposition of "imposex" on certain species. At the present time some of these studies are being conducted.

SHELLFISH AS INDICATORS OF POLLUTION

Animals vary considerably in their ability to accumulate, depurate and metabolize both naturally occurring and xenobiotic chemicals. Chemicals may be taken up from the water across gill membranes, other exposed external body surfaces and/or from contaminated food. The relative importance of the three routes of uptake for aquatic species is often debated and is probably specific for each animal species and class of chemical substance, e.g. metallic ions, polar organics, etc.

Factors which make a given animal species well suited as an indicator of bioavailability of anthropogenic substances in the environment have been identified by various researchers.[30,31,32,33,34] In brief, these factors for oysters and clams are: (1) the pollutants are often accumulated without mortality; (2) the animals are sedentary in life habit; (3) they are often abundant; (4) they are relatively long-lived, (5) they are easily collected; (6) they are adaptable to laboratory studies, so that experimental work can be performed; (7) they usually have a high BCF (bioconcentration factor) for the pollutant of interest; (8) they usually attain a residue which is correlated with the concentration in the environment and (9) they have a limited ability to metabolize the substance.

Many bivalve species have most if not all of the above characteristics. However,

monitoring of contamination by various pollutants in estuaries by the use of bivalves is complicated by the necessity to use different species as one progresses upstream along the salinity gradient. In the lower Chesapeake Bay, most tributary sub-estuaries contain three or four bivalve species, the oyster, (*Crassostrea virginica*), the mussel, (*Mytilus edulis*), the hard clam, (*Mercenaria mercenaria*) and the brackish water clam (*Rangia cuneata*). In this section of the chapter we describe the use of two of these species in detecting anthropogenic

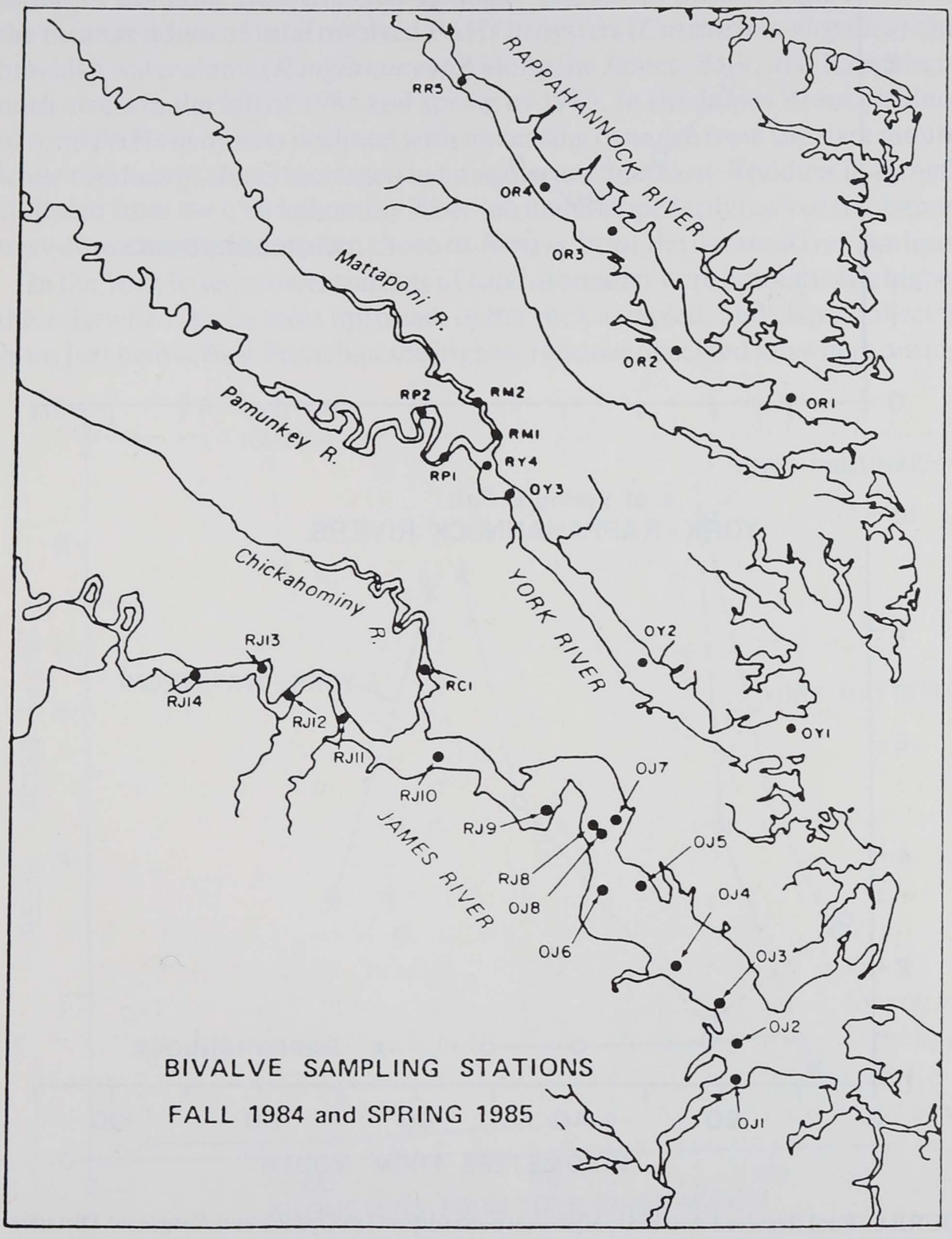

FIGURE 5. Bivalve Sampling Stations for Polynuclear Aromatic Hydrocarbons.

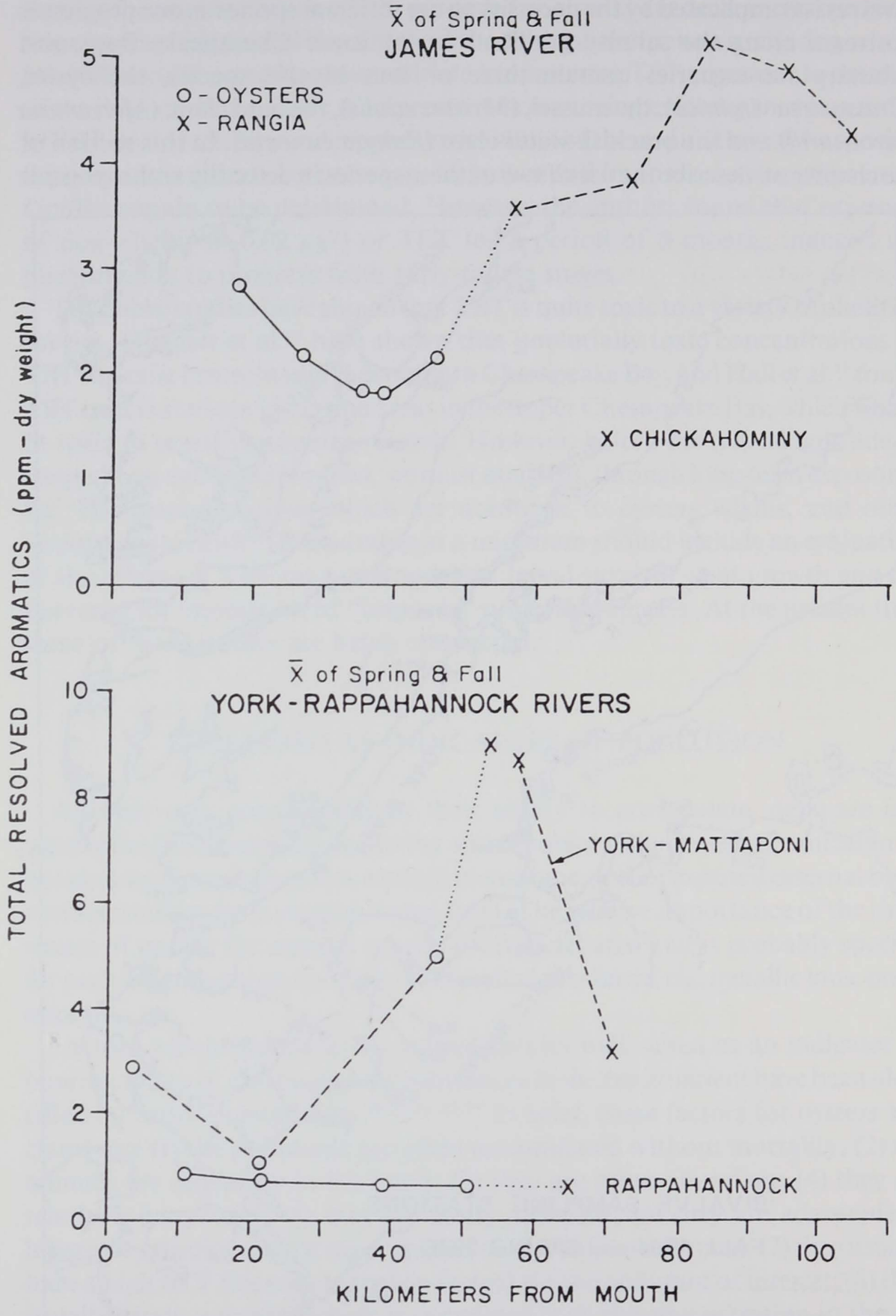

FIGURE 6. Total Resolved Aromatic Hydrocarbons (dry wt) in Oysters and *Rangia* vs. Distance in the James, York and Rappahannock Rivers.

inputs of polynuclear aromatic hydrocarbons (PAHs) in sub-estuaries of lower Chesapeake Bay.

PAHs are widespread contaminants of freshwater and estuarine systems and have been implicated in causing effects on fishes and shellfish in the Niagara River,[35] Oregon Bays[36] and Puget Sound.[36]

Recent surveys of PAH contamination in Virginia's major river systems (see Figure 5 for station locations) indicate high residues in shellfish collected from estuaries draining industrialized or highly populated basins. Figure 6 shows the mean residues of total resolved PAHs in oysters (*Crassostrea virginica*) and brackish water clams (*Rangia cuneata*) along the James, York, and Rappahannock rivers in the fall of 1984 and spring of 1985. In the James River, residues of total PAHs in oysters declined with increasing distance from the river mouth while residues in clams increased in an upstream direction. Residues in *Rangia* collected from the Chickahominy River (an undeveloped tributary of the James) were considerably lower than those in *Rangia* from the James River stations.

In the York River, concentrations of total aromatics were dramatically higher than elsewhere at the most upstream oyster rock sampled, and clams collected from just below West Point had the highest residues observed anywhere during

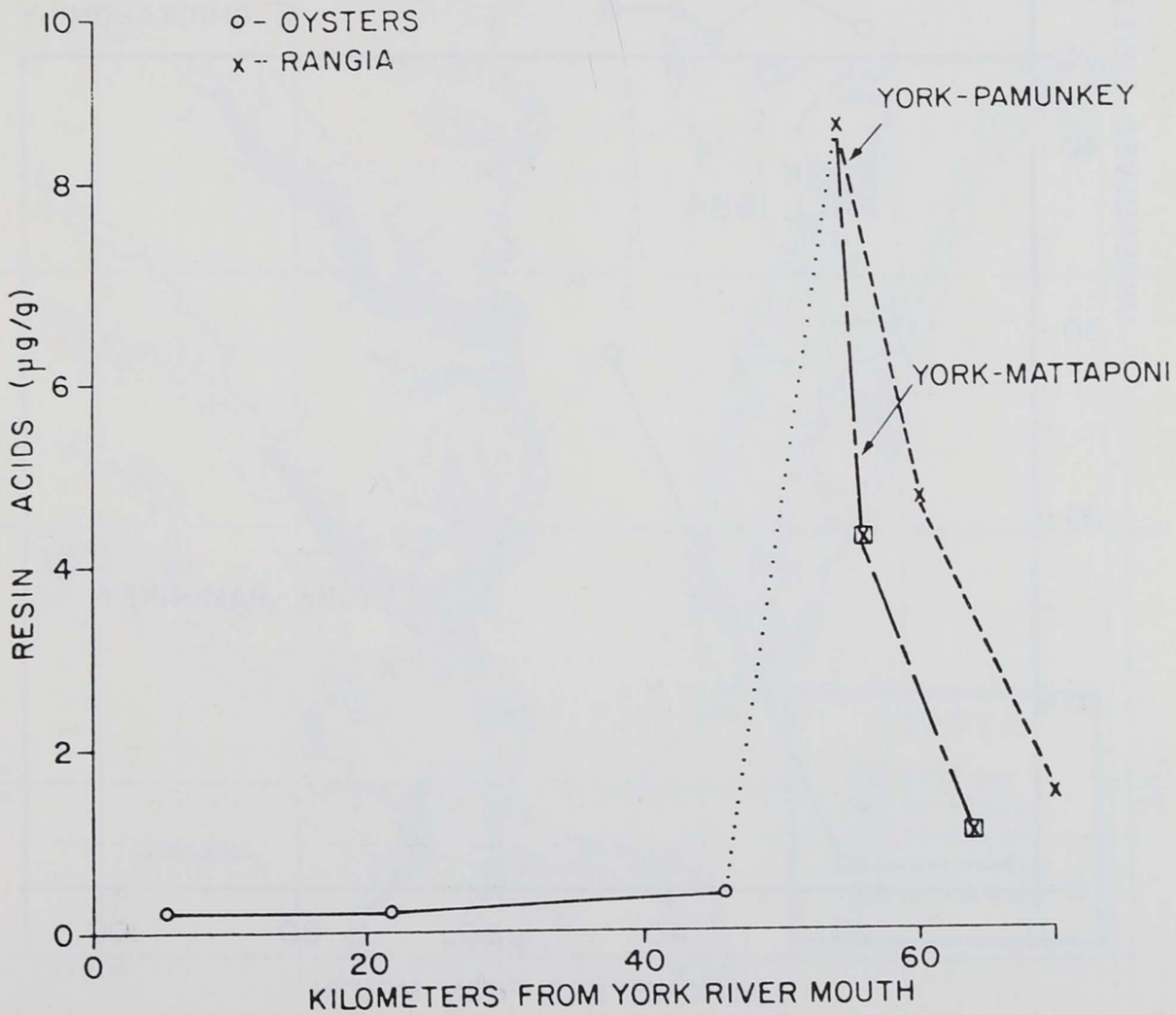

FIGURE 7. Resin Acids (dry wt) in Oysters and *Rangia*.

the survey (Figure 6). A detailed examination of clam samples from the York, Pamunkey and Mattaponi rivers indicated that compounds derived from resin acids of plants accounted for a significant proportion of the resolved aromatics in these samples. The concentrations of the "resin acid derived compounds"

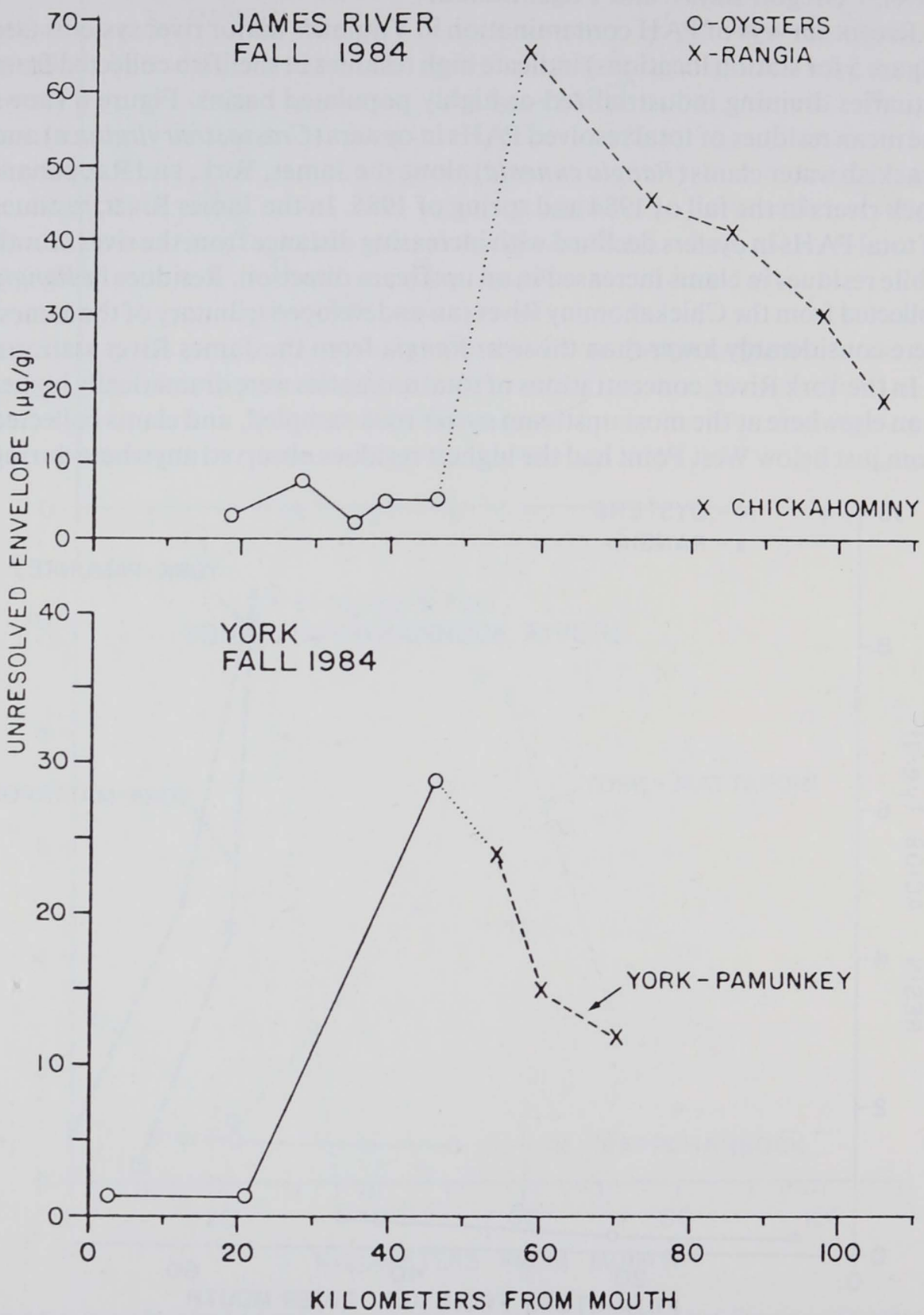

FIGURE 8. Unresolved Envelope (dry wt) in Oysters and *Rangia* in the James and York Rivers.

in the York, Pamunkey and Mattaponi rivers are shown in Figure 7.

Concentrations of hydrocarbons in the unresolved envelopes (mixtures of degraded and undegrated aromatic hydrocarbons) from the fall 1984 samples are shown in Figure 8. Oysters and clams collected from the Rappahannock showed no evidence of unresolved envelopes (UCMs). In both the York and James rivers, substantial increases in the UCM were observed in both oysters and clams collected near the turbidity maximum zone. The lack of a UCM in the Rappahannock samples and the relatively low concentration observed in the Chickahominy samples suggest anthropogenic origins for the envelopes.

At present we have no conclusive evidence to indicate that shellfish populations which show high PAH residues are adversely affected. It should be noted, however, that *Rangia* populations in the upper York and the lower Mattaponi and Pamunkey rivers are very small compared to those in the James and Rappahannock rivers. In addition, clams from these areas generally appear to be in poor condition, i.e. they have lower dry weight to wet weight ratios than clams from other river systems.

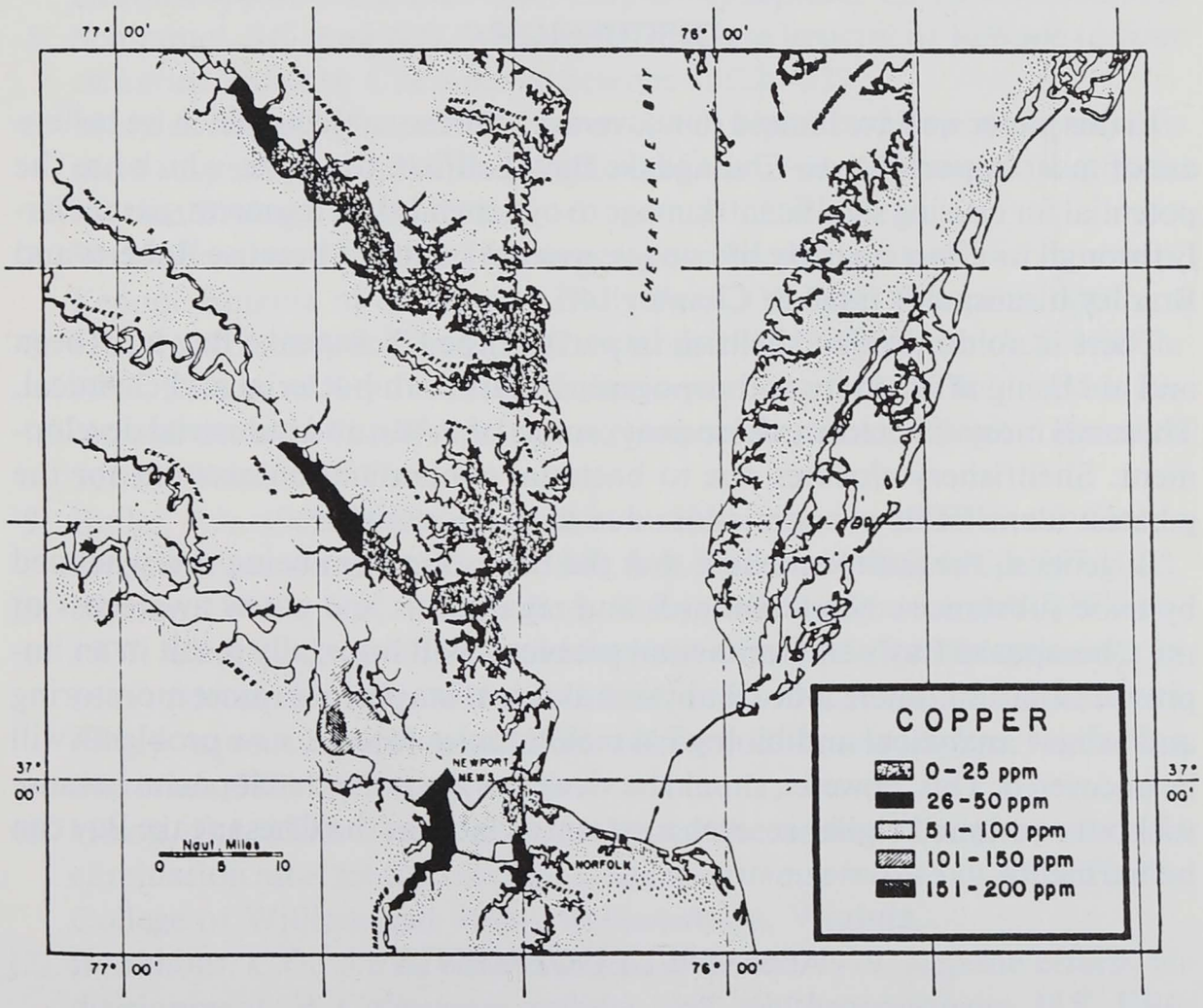

FIGURE 9. Copper Residues in Oysters from Southern Chesapeake Bay from Huggett, et al.[32]

TRACE METALS

Huggett, et al.[33] demonstrated that residues of certain heavy metals (Cd, Cu and Zn) in oysters (*Crassostrea virginica*) were a function of not only source but also the animal's position in the estuary. Figure 9 shows the distribution of copper in oysters from the James, York and Rappahannock rivers. In systems relatively less affected by anthropogenic inputs, e.g. the York and Rappahannock, concentrations are high in the upstream low salinity regimes of those estuaries. Similar distributions were observed for cadmium and zinc. The authors developed a method utilizing ratios of residues between Cu and Zn which allowed for determination of whether the body burdens were derived from natural or man-made sources.

The distribution of the metals Cr, As, Pb, Hg, Zn, Cu, and Cd in oyster tissues from the upper Chesapeake Bay and portions of the lower Bay were summarized from a number of studies in EPA's Chesapeake Bay Program report.[2] They concluded in part, (1) that certain metals, e.g. Cu, Cd, and Zn were high near urbanized areas and (2) that metal contamination levels in shellfish tissue did not violate FDA action levels.

DISCUSSION

In this paper we have limited our coverage to those subjects which we believe are of most importance to Chesapeake Bay shellfish. Chlorine, which has the potential for causing significant damage to oyster and clam resources, particularly through its effect on early life stages, was not included because Roberts and Bradley discuss this issue in Chapter 14.

There is no doubt that shellfish in parts of the Chesapeake Bay have been and are being affected by anthropogenic inputs both bacterial and chemical. The areas most effected are those near centers of urban and industrial development. Shellfishery closures due to bacterial contamination account for the greatest identifiable economic loss due to man's activities.

In general, the authors believe that the Bay is far from being overwhelmed by toxic substances. New standards and regulations and a new awareness of the Chesapeake Bay's environmental problems will hopefully result in an improved situation. There is no doubt that as scientists perform more monitoring and as new analytical and biological technologies emerge, new problems will be uncovered. This, however, should be viewed as a positive development because without continued vigilance, even a system as large as the Chesapeake Bay can be harmed.

ACKNOWLEDGEMENTS

The authors wish to thank Drs. W.J. Hargis and Morris H. Roberts, Jr. for

reviewing the paper and Mrs. S. Sterling for preparing the manuscript. Financial assistance was provided in part by the Virginia State Water Control Board. Mr. Cloyde Wiley provided the data on shellfish closures in Virginia and Mr. Paul DiStefano supplied the information for Maryland.

Figure 9 is reprinted with permission from Water Research, No. 7. R. J. Huggett, M. E. Bender & H. D. Slone, Utilizing metal concentration relationships in the eastern oyster (*Crassostrea virginica*) to detect heavy metal pollution. 1973, Pergamon Press, Ltd.

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