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ANAEROBIC MORTALITIES OF OYSTERS IN VIRGINIA CAUSED BY LOW SALINITIES

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ABSTRACT Oysters on natural beds in the upper seed area of the James River died anaerobically in the winter and early spring of 1979–80 during prolonged exposure to fresh water and low salinities (< 5 ppt). Heavy rains in the fall of 1979 combined with the usual winter-spring runoff to produce low salinities. Oysters in trays were transplanted in late March and early April to six high-salinity areas where mortalities were found a month later. The oysters died slowly within closed shells because they were unable to feed and respire in the nearly fresh water. This produced a strong, malodorus stench and blackened shell margins that are characteristic of anaerobiotic decay. Similar phenomena occurred previously in the Rappahannock River about 1 May during several wet years during the past three decades. At depths of 5 to 6 m, dissolved oxygen was depleted and everything on the bottom became black with iron and other heavy metal sulfides. Dead oysters were not discovered until June after waters had become aerobic again.

INTRODUCTION

For 21 consecutive years, disease-free oysters from low-salinity waters of the James River were transplanted to high-salinity waters in several rivers of Virginia to monitor the incidence of "Delaware Bay Disease" (or MSX) which is caused by the haplosporidan *Minchinia nelsoni* Haskin, Stauber, and Mackin (1966). This pathogen requires water salinities of ⩾ 15 ppt to attack oysters effectively. Horsehead Rock in the upper seed area of the James River (Figure 1) has annual salinity maxima of 12 to 15 ppt in late summer; its oysters are usually free of diseases and parasites including *M. nelsoni.* Because salinities are low in the James River seed area, little selection by MSX has occurred since its introduction to Chesapeake Bay in 1959 (Andrews and Wood 1967). These oysters are, therefore, relatively susceptible to the disease and are used for comparisons of annual intensities of MSX infections and mortalities in Chesapeake and Delaware bays (Haskin and Ford 1982).

During late winter and spring, Horsehead Rock oysters are routinely exposed to low-salinity and even fresh water which causes suspension of normal feeding activities, and aerobic respiration is interrupted for months. Oysters usually withstand this low-salinity stress during cold water temperatures by closing their valves and becoming dormant or narcotized (Andrews et al. 1959). In years of heavy rainfall and runoff, this state of anaerobiosis may extend to 1 May without serious oyster mortalities, provided that the dormancy is not interrupted by a period of normal feeding activities. Oysters begin to filter feed when water salinity and temperature approach 5 ppt and 10°C, respectively.

Fall and winter of 1979–80 were wet in Virginia because of record rainfalls in September and November 1979. Oyster spatfall in the James River in September 1979 was light in intensity, but useful in the post-MSX years after 1959 when only light or insignificant spatfalls occurred. Most small spat (< 5 mm) located from Wreck Shoal to Horsehead Rock were killed in late fall by low-salinity water. Larger and older oysters including yearlings survived during the fall and winter except at Deep Water Shoal, the upper river seed bed exposed to the most fresh water. Few boxes were found when Horsehead Rock oysters were dredged in March for experimental tray studies of MSX. Approximately one third of the oysters at Horsehead Rock died in the spring of 1980 as low salinities persisted, but all oysters died at Deep Water Shoal.

MATERIALS AND METHODS

Experimental oysters were usually collected in March before their dormancy period ended to avoid inclusion of dying oysters (gapers) and hidden boxes (empty shells). This prevented occurrence of dead oysters in trays for a month or two after transplantation into high-salinity waters. Apparently, some oysters died with tightly closed shells in March 1980, before transplantation, and slow anaerobic decomposition was initiated in cold waters (< 5°C). The oysters were held at the Virginia Institute of Marine Science (VIMS) pier for short periods while being sorted and counted. This allowed them to adjust to moderate salinities before transplantation to higher salinities. The trays of oysters were then distributed to stations in three rivers along the Western Shore of Chesapeake Bay and on the seaside of Eastern Shore for disease monitoring. Shells of dead oysters did not open and deaths were not discovered during handling operations.

Trays of oysters were placed on natural beds to monitor diseases and mortalities without the influence of predation and adverse siltation (Andrews et al. 1962). Each tray held 0.036 m^3 (1 bu) of oysters, was completely enclosed with 2.5-cm (1-in.) galvanized mesh hardware cloth, and was raised 0.3 m above the bottom on legs. Monthly examinations were made to determine the number of live and dead oysters, to collect gapers for disease testing, and to remove

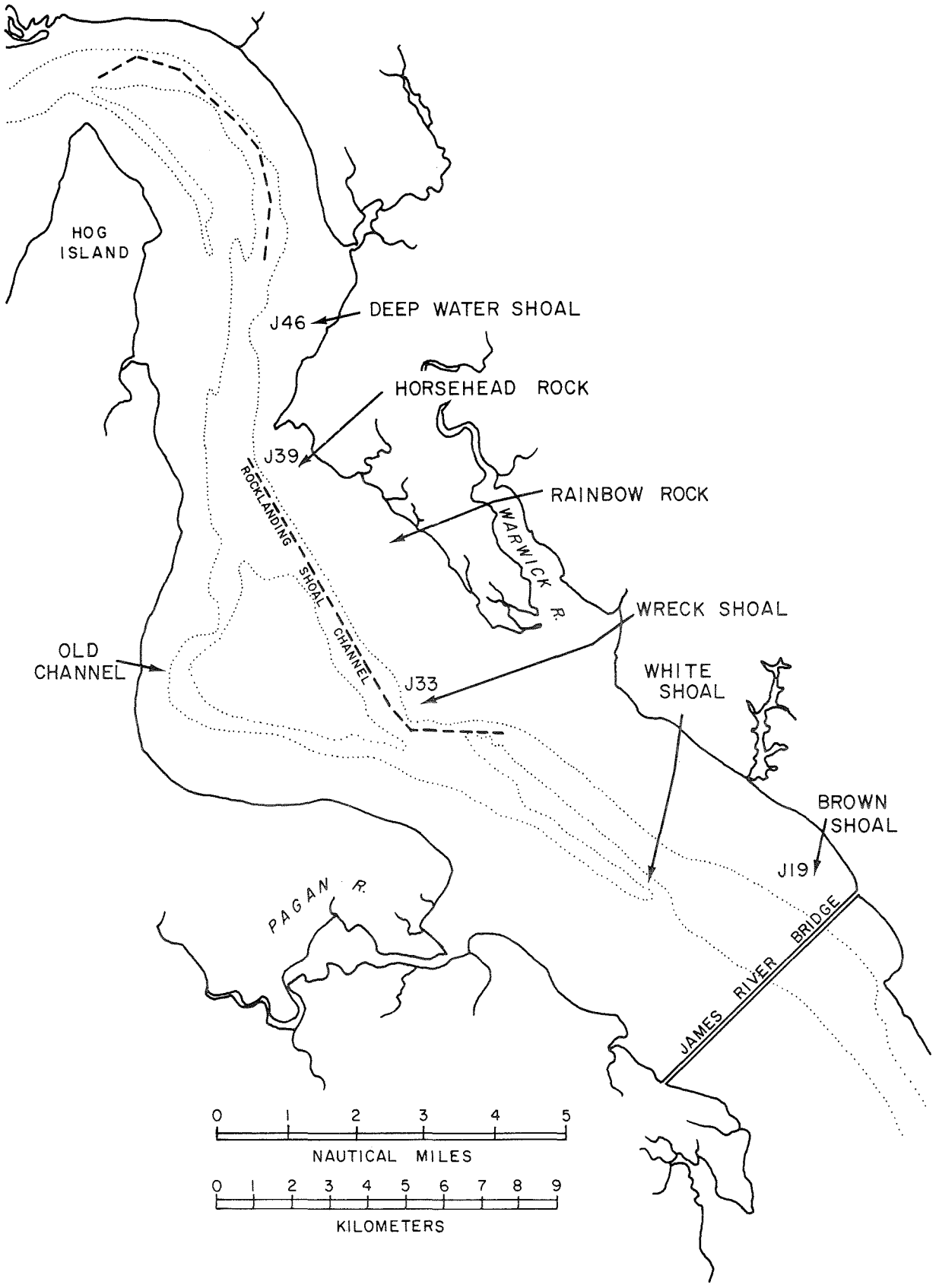

Figure 1. Map of seed-oyster area in James River, Virginia. Natural oyster beds extend from the bridge (J19) to Deep Water Shoal (J46). (Distances are expressed in kilometers from the river mouth.)

fouling organisms. This method of monitoring disease has been used for 25 years in Virginia, particularly in areas where planting of oysters has ceased because of MSX mortalities. Oysters were handled individually and randomized in the trays during examination.

RESULTS

The first lots of oysters were dredged from Horsehead Rock and transplanted to the VIMS pier in the York River on 20 March 1980. No evidence of dead or dying oysters was observed at that time. On 26 March, the oysters were sorted again for boxes, and three trays with 500 oysters each were moved to a Gloucester Point station above the York River Bridge for monitoring disease prevalences and mortalities. These tray oysters were re-examined on 23 April and the mortality was < 1% for the 28-day period (8 of 1,500 dead). The next examination on 13 May revealed many boxes and dead oysters with slimy meats. The deaths were unusual because the shells remained closed after the tissues became soupy. The oysters did not exhibit the usual hollow sound characteristic of "cluckers" or empty shells. Dead oysters were recognized by black anaerobic streaks along the shell margin or bill, and they exuded an extremely malodorus and sulfurous stench that is typical of anaerobic decomposition.

By 6 June 1980, most dead oysters had been detected and the survivors had new shell growth. The mortality was almost identical in the three trays at Gloucester Point for a mean of 27.5% (412 of 1,500 dead) (Figure 2). Oysters in trays that were moved to James River, Rappahannock River, and Mobjack Bay showed similar timing and extent of mortalities (Table 1). Boxes and gapers began appearing about one month after transplantation to high-salinity waters. Occurrence of boxes ceased after about 1 June at Gloucester Point (Figure 2) although some trays in other rivers were not examined until later. Surviving oysters appeared healthy with sharp new shell margins. Typical MSX mortalities began about 1 August 1980.

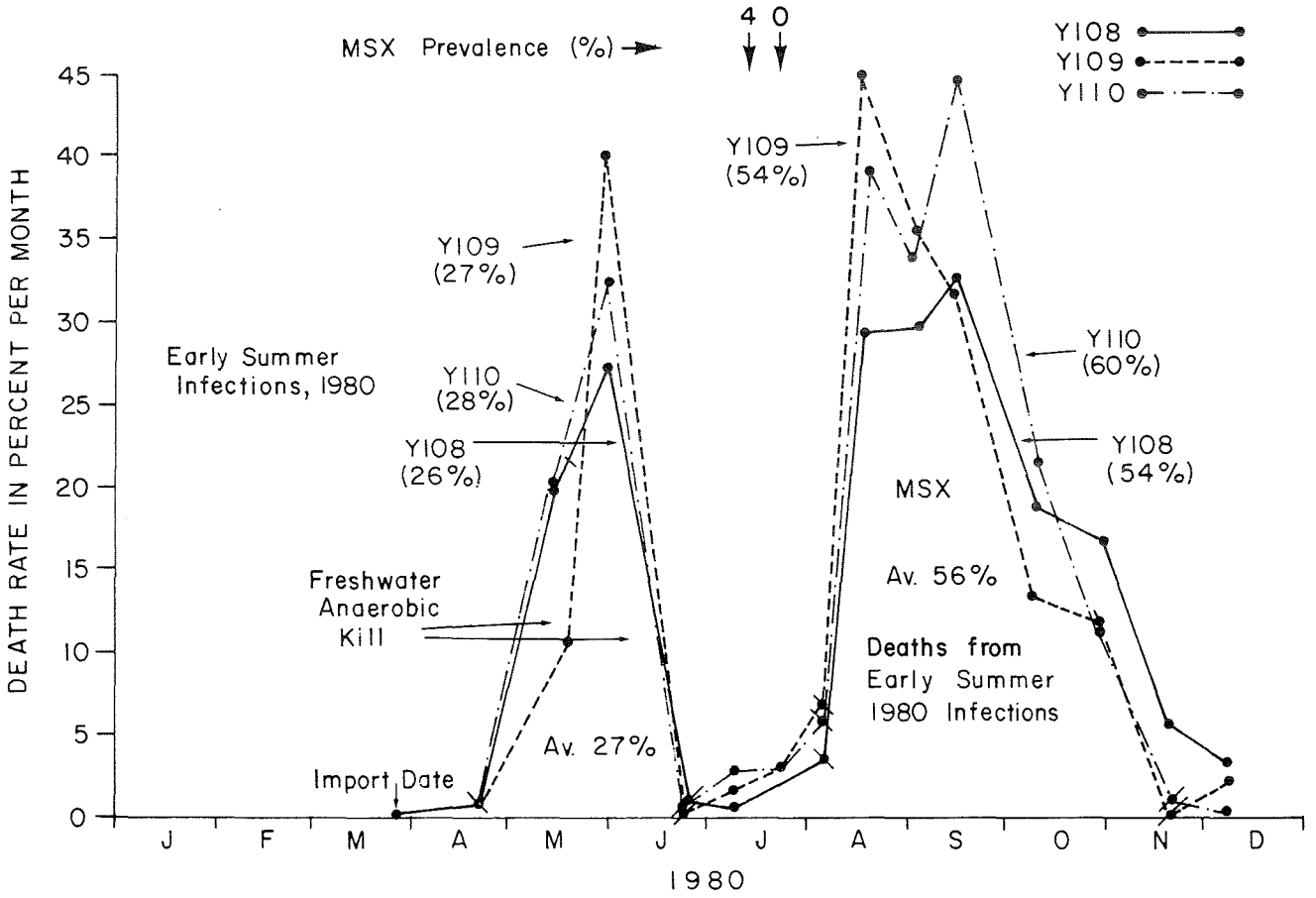

Figure 2. Mortalities from anaerobic deaths in May and from *Minchinia nelsoni* (MSX) in late summer and fall are shown for three replicate lots of oysters held in trays at Gloucester Point, Viginia. Two samples of 25 live oysters each were taken in July, too early to show intensity of MSX infections; 30 of 37 gapers (81%) had the disease after 1 August. Almost a month elapsed before anaerobic deaths became apparent. No further deaths occurred after 29 May when peak mortality rates occurred in all trays.

TABLE 1.

Anaerobic mortalities of Horsehead Rock oysters transplanted in early spring 1980 to higher salinity waters.

Location	Tray	Transplant Date	Mortalities Dates[1] Begun	Ended	Total (%)
York River	Y108	20 Mar	23 Apr	29 May	26
(Gloucester	Y109	20 Mar	22 Apr	29 May	27
Point)	Y110	20 Mar	24 Apr	29 May	28
Mobjack Bay	MJ31	20 Mar	25 Apr	16 Jun	37
Piankatank River	PK16	2 Apr	5 May	7 Jul	30
Rappahannock River					
Parrotts Rock	R42	2 Apr	5 May	7 Jul	31
Balls Point	R43	2 Apr	5 May	7 Jul	32
James River					
Hampton Bar	J52	8 Apr	29 Apr	28 May	43
Brown Shoal	J53	8 Apr	29 Apr	19 Jun	45
Wreck Shoal	J54	8 Apr	29 Apr	19 Jun	43
Seaside of Eastern Shore Chicoteague Bay	S126	20 Mar	30 Apr	2 Jun	33
Swash Bay	S129	20 Mar	1 May	3 Jun	33

[1] Refinement of periods of mortality was limited by dates of examination of trays which were usually 3 to 5 weeks apart. The Gloucester Point data were best defined with counts on 13 May, 20 May, and 6 June, in addition to dates listed in table.

DISCUSSION

Oysters that were transplanted as late as 8 April 1980 showed no external signs of anaerobic mortality. The discovery of deaths depended on timing of examinations; therefore, dates in Table 1 are not precise for the duration of anaerobiosis or the time of death. Late transplantations, however, resulted in higher mortality rates. This implies that the deaths were still occurring at Horsehead Rock although they were undetected because the shells were tightly closed.

The observed mortalities resulted from unusual weather and salinity regimes. The oysters were under low-salinity stress throughout 1979 from > 150 cm of rainfall over Virginia (compared to a 112-cm annual mean). The heavy rainfall and runoff were exceptional for the fall, and salinities were unusually low throughout the James River seed area (Tables 2 and 3). Additional data on mean salinities in the seed area for 12 years are given in Andrews and Hewatt (1957). Oysters on shallow (< 3 m) seed beds in the James River live in salinities close to those found at the surface in adjacent channels. An alert was issued by VIMS to warn of possible oyster mortalities from the low-salinity exposure. No mortality of adult oysters was observed in the fall and winter of 1979, and oystermen avoided the upriver seed beds in the spring of 1980. Adult oysters, however, had very poor meats with Condition Indices of ≤ 4.0 (range = 4 to 15) (Hopkins, 1949).

$$CI = \frac{\text{dry weight of meat (g)}}{\text{volume shell cavity (m}\ell\text{)}} \times 100$$

TABLE 2.

Typical surface salinities (ppt) in James River by seasons (means, 1952–1961).

Season	River Distance (km) J0 (Mouth)	J33 (Wreck Shoal)	J46 (Deep Water Shoal)
Winter	19	8	0
Spring	16	11	0
Summer	20	16	10
Fall	22	16	5

TABLE 3.

Surface and bottom salinities in upper half of James River seed area from October 1979 to June 1980[1].

Date	Salinities (ppt) at Channel Stations, All Tides Wreck Shoal (Nun "12" [J33])		Horsehead Rock (Nun "22" [J39])	
20 Sep 79	10.7	[14.9]	5.0	[5.2]
26 Sep 79	1.2		0.1	
2 Oct 79	1.9	[7.2]	--	
10 Oct 79	1.5	[2.0]	--	
15 Oct 79	1.7	[3.5]	1.4	[3.3]
22 Oct 79	6.0	--	--	
24 Oct 79	9.2	[10.4]	3.4	[4.4]
30 Oct 79	7.4	[8.8]	3.4	[5.0]
10 Dec 79	1.9	[11.5]	1.4	[1.7]
17 Jan 80	10.6	[12.2]	6.2	[6.3]
28 Jan 80	4.7	[4.8]	0.9	[1.8]
14 Feb 80	13.2	[14.0]	8.4	[10.4]
19 Mar 80	7.9	[7.9]	0.9	[1.1]
10 Apr 80	3.3	[10.9]	0.5	[0.5]
23 Apr 80	1.5	[7.1]	--	
2 May 80	6.3	[10.3]	1.9	[2.3]
9 Jun 80	6.9	[13.5]	6.0	[7.9]

[1] Bottom (12-m) salinities in brackets.

Oysters in the upper James River routinely enter winter dormancy about 15 December each year as water temperatures of > 5°C prevail. If salinities drop appreciably below 5 ppt, anaerobic dormancy occurs and water pumping for feeding and respiration is precluded. Closed oysters, even at warm temperatures, exhibit greatly reduced rates of heart beat and ciliary action (Stauber 1940); those oysters that are forced into anaerobic metabolism during winter exhibit no muscular or ciliary activity (Andrews et al. 1959). Winter-dormant oysters that were held at 20°C in fresh, well

water remained tightly closed, and internal salinity levels gradually declined over several weeks to months before they died (Andrews et al. 1959). The oysters at Horsehead Rock may have experienced intermittent closings and openings caused by the fluctuating salinities in late fall and winter of 1979–80. Anaerobic respiration is extremely wasteful of glycogen reserves in warm water, but metabolism is suppressed by low water temperatures in winter. Because rainfall continued to depress salinities through the winter, Horsehead Rock oysters were probably in a continuous state of anaerobic closure from about December 1979 through March 1980.

Oysters in winter dormancy normally respond quickly with increased cardiac and respiratory activity (heart beat and ciliary movement, respectively) when they are exposed to water of suitable salinity and temperature. In 1958, oysters that were opened in the laboratory, after months of winter dormancy in fresh water, began ciliary and cardiac activities in < 5 minutes after exposure to air (Andrews et al. 1959). When an oyster dies, the compressed hinge ligament usually opens the shell. When oysters died in the spring of 1980, their shells were held closed by the catch muscles, and the tissues decayed anaerobically. Presumably, those oysters were dead or moribund when transplanted to VIMS; they had lost their capacity to pump water by ciliary action of the gills. The consistent mortality rates in all 12 of the trays of transplanted Horsehead Rock oysters suggested that death or survival had already been determined before transplantation. On 8 April 1980, oysters for the last three trays were collected from Horsehead Rock and brought to Gloucester Point for acclimation to higher salinities. They were transplanted to stations in the James River on 29 April. Mortality was somewhat higher in those lots (43+%) suggesting that more oysters had reached the point of death during the extra 20 days in low-salinity water at Horsehead Rock.

The anaerobic deaths of oysters from prolonged exposure to fresh water caused abnormal sequences of death, with closed shells and a delay of about one month before their discovery. Similar occurrences of anaerobic deaths and delayed discovery were observed on deep water oyster beds in the Rappahannock River during periods of high freshwater flow. Those events, characterized as "black-bottom" phenomena, always occurred in late April or early May of wet years when spring freshwater discharge was highest. Dead oysters sometimes appeared later, after anaerobic conditions on the bottom had disappeared.

The black-bottom condition was observed in the Rappahannock River in at least four years (1949, 1953, 1958, and 1980). Freshwater flow rates and water temperatures at the Fredericksburg gauging station were well above normal during those wet winter and spring seasons (1 October to 1 May). In 1949, for example, the accumulative total for seven months of mean monthly flow rates for the period 1 October 1948 to 1 May 1949 was 684 m^3/sec (23,150 cfs), whereas the 55-year mean was 381 m^3/sec (13,449 cfs). In addition, 10 cm of rain fell during the first 10 days of May over the watershed.

In 1949, the only year that mortalities were known to occur in the Rappahannock River, wide areas of public oyster grounds in 4 to 7 m of water became anaerobic in early May. Everything on the bottom, including oysters and mud, was blackened by iron and other heavy metal sulfides. The black color disappeared soon after the shells were exposed to air. Dredged materials had a strong, hydrogen sulfide odor. The oxygen deficiency was caused by a combination of the following factors which depleted the supply and/or prevented replenishment: (1) large accumulations of organic matter from heavy freshwater runoff; (2) intensive density stratification caused by warm fresh waters overlaying cooler, saline waters; (3) high oxygen demand created by rapidly increasing water temperatures in May; and (4) intensive phytoplankton blooms stimulated by high nutrient levels in the runoff. Because all of these factors existed, high oxygen demand near the bottom occurred when poor vertical mixing limited resupply. Mortalities were confined to deep oyster beds adjacent to the channel, whereas shallow beds had neither black bottoms nor oyster mortalities.

Earlier laboratory experiments documented anaerobic mortalities in oysters exposed to fresh water. Andrews et al. (1959) demonstrated that winter-dormant oysters maintained in fresh well water at 20°C died slowly with their shells closed and released malodorus sulfide gases.

Most oyster mortalities that result from exposure to low salinities or fresh water occur during warm seasons (Andrews 1955). Hurricane deluges in late summer and sudden ice thaws during wet springs are the usual causes of low salinities. Low-salinity mortalities depend primarily on temperature levels and duration and continuity of exposure. At winter temperatures of < 5°C, oysters are quite tolerant of low-salinity conditions, either by dormancy or, if salinities are > 5 ppt, by opening their shells for water exchange for respiration. Oysters are also tolerant of low-oxygen levels of down to about 1 mℓ/L during warm seasons. When waters become anaerobic during warm seasons, stress is rapidly increased and duration of survival is greatly decreased. Above 15°C, survival is limited to a few days; however, if oysters are slowly acclimated to winter dormancy, their shells remain tightly closed during freshwater exposure, and death and decomposition are slow and prolonged. The byproducts of anaerobic decomposition of meats must also affect the resiliency of the hinge ligament which causes a long delay in detecting death by open valves.

Low oxygen conditions occur in channel depths of $\geqslant 8$ m every summer in the Potomac and Rappahannock rivers, but total oxygen deficiencies which cause black bottoms seldom penetrate up to the 4- to 5-m depths on oyster beds adjacent to the channels. Watermen that fish crab pots in the summer at 7- to 8-m depths near channels frequently find dead crabs in their pots that are apparently

caused by low-oxygen levels. The black bottoms on oyster beds have been observed only in the first half of May, and not in summer when oxygen deficiencies occur regularly in deeper channels.

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