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## OYSTER MORTALITY STUDIES IN VIRGINIA

### IV. MSX IN JAMES RIVER PUBLIC SEED BEDS<sup>1</sup>

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#### ABSTRACT

"MSX," an unnamed pathogen of oysters, caused an epizootic in Chesapeake Bay which removed from production nearly half of Virginia's private oyster-planting acreage between 1959 and 1961. The organism did not appear in James River seed beds until fall of 1960. A tongue-shaped distribution of MSX was apparently related to influx of salt water along the channel. In 1960-61 and 1961-62, infections of MSX appeared at Wreck Shoal in the middle of the seed area in October, and disappeared the following April coincident with lowest salinities. Infection levels were approximately 30 to 35% each year in populations adjacent to the channel. No appreciable cold-season mortality occurred at Wreck Shoal. MSX was nearly absent from Wreck Shoal oysters during the warm season in summer salinities of about 15 ppt, but at Brown Shoals, with salinities 2 or 3 ppt higher, it persisted through spring freshets and caused summer deaths. From observations for three rather wet years, it is concluded that persistence of MSX infections in the James River seed area depends upon importation of infective material from the saltier waters of Lower James River and Hampton Roads. Also, damage to the seed area will probably be reflected in quality of seed rather than direct mortality. Planting infected seed in high-salinity waters leads to serious losses.

#### INTRODUCTION

The 12-mile stretch of James River beginning at the bridge above Hampton Roads exhibits several important characteristics which make it the major seed area for Virginia: (1) Natural reproduction occurs regularly with an intensity that produces seed of excellent quality; (2) growth is slow and fattening is poor, which almost necessitates use of the oysters as seed; (3) predation is negligible and diseases are restricted by low salinities.

Most of the oyster grounds in the seed area are public "rocks" from which wild oysters are harvested. The importance of James River as a source of seed oysters to private planters in Virginia can scarcely be overestimated. The apparent introduction of a new pathogen of

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<sup>1</sup>Contribution No. 155 from the Virginia Institute of Marine Science.

oysters called for special monitoring of the seed area for possible damage. The undescribed pathogen called "MSX," which in Virginia was first observed in 1959, has caused far more damage to private oyster grounds than to public beds. MSX occurs only in areas with summer salinities of about 15 ppt or higher and this is about the level required by other diseases (e.g. Dermocystidium) and predators (oyster drills). Consequently, only planted beds flourish in high-salinity waters and our remaining public beds, with wild populations of oysters unreplenished by man, are limited to low-salinity areas. MSX was not found in the seed area until October 1960 after two summers of heavy losses in high-salinity areas of Chesapeake Bay (Andrews and Wood, in preparation). Delaware Bay, which also has a seed area in relatively low-salinity waters above the planting region, experienced a kill of seed oysters in 1958, the second and peak year of MSX activity in this estuary (Haskin, 1960).

The primary objective of monitoring in James River was to keep informed about distribution and incidence of MSX in order to advise planters of the location of infected seed. By the fall of 1960, when MSX had infested the lower seed area, planting had ceased in the high-salinity waters of Virginia because losses were intolerable. Most of the planting that continued was in waters approximately of the salinity range of the seed area. Fortunately, no serious consequences have ensued from use of limited amounts of infected seed in these low-salinity planting areas.

James River has been valuable for studies of the environmental tolerances of MSX. A dense population of susceptible oysters in the seed area has been continuously exposed to MSX, being close to intensive plantings in an infested area of high salinity (Hampton Roads). Some appreciation of the effectiveness of Hampton Roads as a reservoir of infective material can be obtained from the history of private plantings and losses. Prior to 1960 extensive new beds of James River seed were planted each year. The last seed plantings were made in the spring of 1960. After severe losses most remaining beds were harvested by June 1961. After this date less than 10% of normal oyster populations were left in Hampton Roads and mortalities have continued in these survivors. Therefore, in 1962 most old oysters were dead and new susceptibles were not being planted.

Another advantage of James River for disease studies is the large drainage area and the high runoff which produce a rather steep horizontal salinity gradient from the mouth to Jamestown. The river also has probably the greatest seasonal fluctuation of salinity in the Chesapeake Bay area. Since the "fringe" of the range of MSX falls in the

lower seed area, James River provides a place for assessing salinity tolerances of MSX.

#### PROGRAM OF MONITORING MSX

Three locations were chosen as major stations for tray observations and sampling of native populations (Fig. 1). Brown Shoals at the lower end of the seed area is characterized by rather permanent infestations of Dermocystidium and oyster drills. Wreck Shoal in the middle of the seed area represents optimum conditions for seed production and typically has the greatest spatfall and negligible mortality. Horsehead Rock is near the upper limit of the seed beds and was the source of control groups of oysters for trays. No serious predators or diseases have ever been found at this level of the river. A few supplementary stations are also shown in Fig. 1. The major stations were deliberately located close to the channel. Distribution patterns indicate that diseases and predators tend to follow the deeper, saltier waters of the channel in moving upstream (Andrews and Hewatt, 1957). The circulation of tidal waters provides a mechanism for transport of materials upstream (Pritchard, 1953).

The management of tray stations on oyster beds has been described (Andrews, Wood and Hoese, 1962). For each tray in James River, native oysters were dredged from the vicinity of the station. Trays were examined approximately biweekly. Death rates were obtained from trays and were expressed as number dead per thousand per month (easily converted to per cent) regardless of the lengths of periods between visits. Few gapers were recovered for diagnosis of disease because death rates were low, and to avoid biasing death rates, live oysters were sampled very infrequently from trays. Incidence of disease was obtained from frequent samples of live oysters from natural beds at the three major stations. On each trip, counts of live oysters and boxes were made from dredge hauls as a check on death rates in trays.

#### SALINITY AND TEMPERATURE RANGES IN JAMES RIVER

The winter season of minimal temperatures (about 5 C) lasts from mid-December until mid-March. During this period oysters were inactive. Ciliary motion is very sluggish, no food is processed through the gut, and little water is pumped. Temperatures rise rapidly in April and May. From mid-June to 1 October, warm season temperatures of 25 to 30 C prevail. Spawning and setting of oysters occur during this period. October and November are periods of rapid cooling, although mortalities may persist through these months.

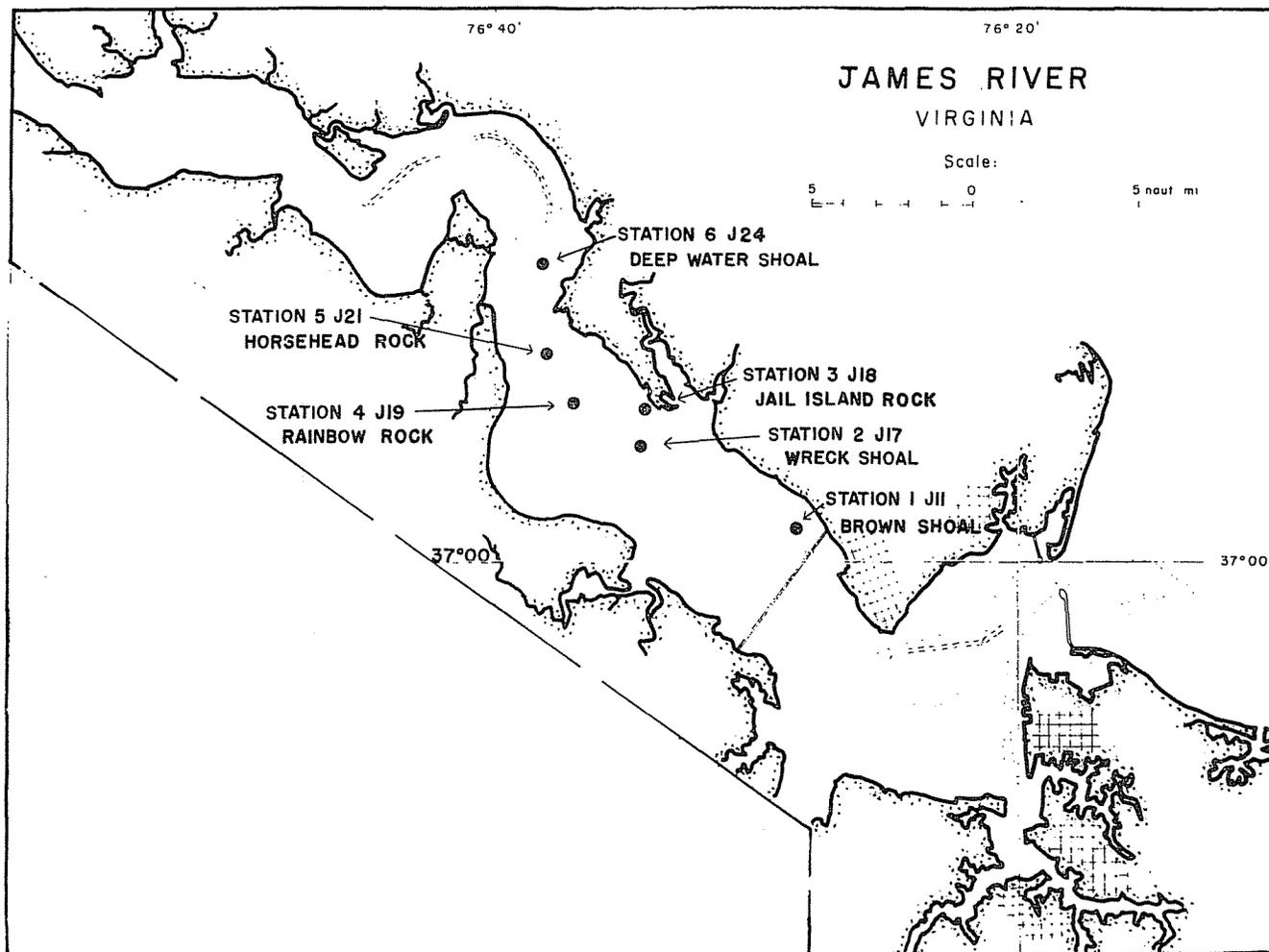


Fig. 1. A map of James River showing Hampton Roads below the bridge and the seed area above it. Only major tray and sampling stations are shown. The J numbers indicate distance up-river from the mouth.

The James River shows marked seasonal changes in salinities. Low salinities occur in winter and spring with minimal levels being reached typically about 1 May. High salinities occur in late summer and fall. Most oyster beds in James River are in shallow water (less than 10 ft), hence little vertical gradient is observed. Hydrographic stations were located in the channel but long experience has shown that surface salinities in the channel approximate those on adjacent oyster bottoms. Surface salinities given in this paper were taken in the channel opposite Wreck Shoal at Nun Buoy "12" (Fig. 2).

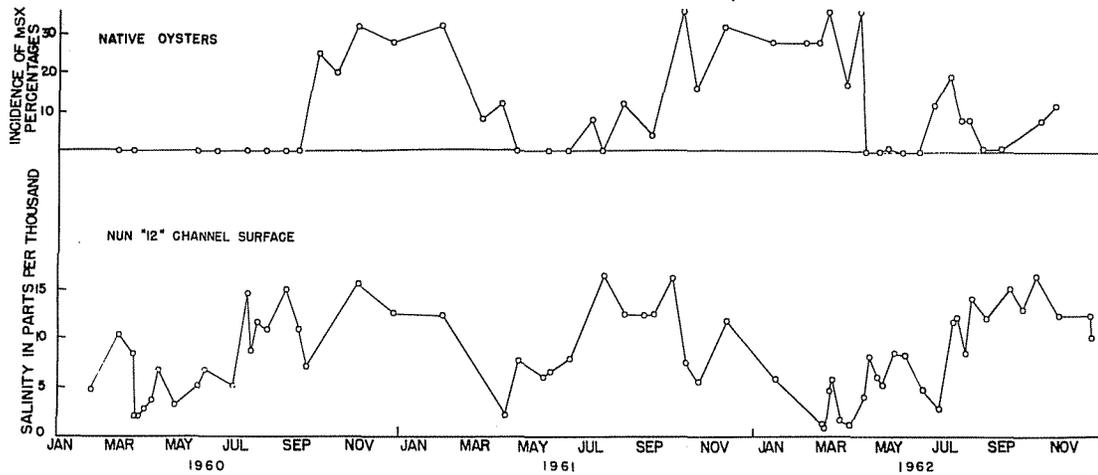


Fig. 2. Salinities of surface waters in channel adjacent to Wreck Shoal offshore station (No. 2); and seasonal incidence of MSX in native oysters dredged at station 2.

In assessing salinity as a limiting factor in the penetration of MSX into the seed area, it is important to know the climatological conditions of the years studied. These are difficult to describe from weather data alone, because the James River drainage area extends into several climatological divisions of Virginia. Special attention should be given to winter and spring weather for this is the period when salinity conditions apparently become intolerable for MSX. The year 1960 was

very wet in Tidewater but dry in other parts of Virginia. However, ~~exceptionally low temperatures in March and exceptionally high ones in April~~ resulted in a heavy runoff from melting snow in the latter month. Consequently salinities were persistently low until July in the seed area. The sudden change from cold to warm weather about 1 April is reflected in the salinity curve (Fig. 2). Precipitation in 1961 was from 4 to 6 inches above normal, yet salinities were not as depressed as in 1960. The winter and spring of 1961-62 was quite wet again and this was reflected in rather strongly depressed salinities in March and April.

It may be concluded that all three years covered in this study had above average rainfall and that spring salinities were below average in the seed area. Both intensity and duration of low salinities, as well as temperature level, are probably involved in controlling diseases and predators. The salinity data were obtained at various stages of tide in irregularly-timed trips. However, Fig. 2 does show seasonal trends and the levels obtained in spring and summer, when MSX activities are important. Further data on the range of salinities and the maximums and minimums found at three levels of the seed area are given in Table 8 of Andrews and Hewatt (1957). Additional hydrographic information may be obtained from Hewatt and Andrews (1954), Andrews, Haven and Quayle (1959), and Chesapeake Bay Institute Data Report No. 7.

## MSX INFECTIONS AT WRECK SHOAL

### Seasonality

Wreck Shoal appears to be the key station for interpreting the effects of environment on MSX activities. Seasonal incidence of MSX is shown in Fig. 2. Each point on the graph represents a sample of 25 oysters from the same locality on Wreck Shoal adjacent to the channel. No infections were found throughout the warm season of 1960 (March through September), when the heaviest losses of the MSX epidemic were being experienced in lower Chesapeake Bay. Beginning in October 1960 and extending through February, about one-fourth to one-third of the oysters in all samples showed MSX infections. In March and April incidence declined without appreciable mortality having occurred and by 1 May 1961 all infections had disappeared from Wreck Shoal oysters.

In the summer of 1961 occasional infections were found at Wreck Shoal in July and August. These may have been new infections initiated in June, or they may have been old infections persisting from the previous summer at a low level of intensity, which built up again with the advent of favorable summer salinities and temperatures.

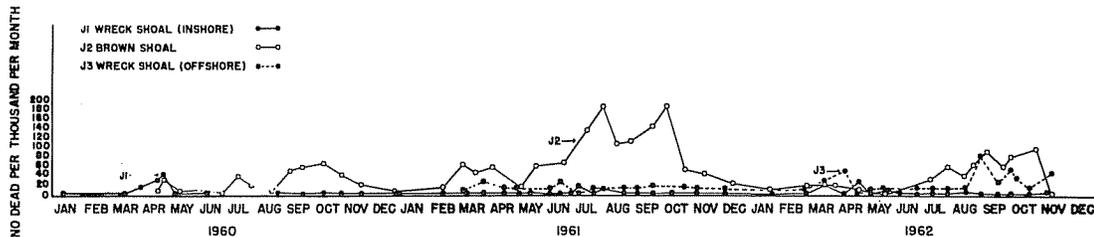


Fig. 3. Death rates of native oysters in trays from 1960 to 1962.

and no diseases. Slight increases in death rates occurred in late winter of 1960 (not related to diseases) and again in early spring of 1962 (probably related to MSX).

The chief conclusion from mortality data is that no appreciable loss of oysters was associated with the cold season prevalence of MSX at Wreck Shoal. Therefore, incidence of MSX was not appreciably changed by mortalities. During the warm season, incidence was low and mortality remained low.

#### MSX AT BROWN SHOAL

The Brown Shoal station at the lower edge of the seed area provides an interesting environment for oyster diseases, intermediate between low-salinity seed beds where diseases are absent and high-salinity planting beds where oysters are now decimated by MSX. Salinities average about 3 ppt higher at Brown Shoal than at Wreck Shoal with summer levels of 18 to 20 ppt (Andrews and Hewatt, 1957). The kill on Brown Shoal has never exceeded 50% and a substantial population of oysters has always been present for disease-producing organisms to persist in. Some recruitment of new year-classes has occurred.

Mortalities at Brown Shoal are shown in Fig. 3 and Table 1. Deaths from MSX began in the fall of 1960 as indicated by a 13 to 18% count of boxes through the winter. Oysters dredged from the vicinity of the tray station were placed in Tray J2 on 1 April 1960. One year later 30% were dead. Both Dermocystidium and MSX infections were

found in gapers and it is difficult to determine the proportion of deaths caused by each. However, only 4 of 8 gapers were infected with Dermocystidium in the late summer and fall, when the fungus is known to cause deaths in Chesapeake Bay. Another 17 gapers were obtained in the late winter and spring, when Dermocystidium is rarely found in gapers, but not all of these gapers were diagnosed for MSX.

From April 1961 to April 1962, box counts from natural beds never exceeded 31%, yet 58% of the oysters in Tray J2 died. In 12 gapers collected from the tray, two had serious Dermocystidium infections (one of these also had MSX) and 4 of 6 examined for MSX were infected. From April 1962 to December 1962, mortality at Brown Shoal in Tray J2 was 30% and on the natural bed it was considerably lower (Table 1).

Incidence of MSX in live oysters at Brown Shoal is given in Table 2. Infections appeared earlier in the late summer and fall of 1960 than at Wreck Shoal and resulted in some deaths from MSX. Deaths were not numerous enough to depress incidence below the level of prevalence found at Wreck Shoal in the fall and winter of 1960-61. Furthermore, there is no evidence that MSX prevalence was changed by low spring salinities as it was at Wreck Shoal. Death rates from MSX were high in the early summer of 1961 before any Dermocystidium appeared.

MSX infections continued through the summer of 1961, and in the fall and winter of 1961-62 about 20 to 25% of natural bed oysters were infected. This is lower than the level of incidence at Wreck Shoal, but previous selection plus a few fall deaths could account for the difference. By 1 April 1962, nearly all infections had disappeared at Brown Shoal and a substantial increase in box counts suggests that infected oysters died in late winter. Since 1 April 1962, MSX has been present at Brown Shoal only in a very small percentage of oysters. Low incidence and no substantial death rate indicate that very few new infections of MSX occurred at Brown Shoal in 1962.

Incidence of Dermocystidium in live oysters at James River stations is given in Table 3. Only very small losses could be expected on natural beds at Brown Shoal from weighted incidences of 0.50 or less (Andrews and Hewatt, 1957), but Tray J2 reached an incidence level (1.24) in 1961 which could be expected to produce a significant kill. The excess losses in Tray J2 over the death rates on Brown Shoal beds can probably be assigned to Dermocystidium.

Table 2. Incidence of MSX at Brown Shoals, James River. Sample size of 25 oysters.<sup>a</sup>

<u>1960</u>		<u>1961</u>		<u>1962</u>	
Date	Per cent infected	Date	Per cent infected	Date	Per cent infected
13 Oct 1959 (5)	0	15 Feb	60	22 Jan (24)	12
		7 April	32	27 Feb	16
1 April (21)	0	22 May	40	30 Mar	4
13 May (24)	0	14 July	16	25 April	0
27 June	4	1 Aug	24	22 May	0
28 July	0	14 Aug	16	18 June	0
18 Aug	12	11 Sept	20	9 July	4
7 Sept	4			27 July	0
20 Sept	16			14 Aug	4
12 Oct	16			23 Aug	0
31 Oct	12			7 Sept	4
21 Nov	32	14 Nov	12	24 Sept	4
27 Dec	36	14 Dec	24	29 Oct	4
				19 Dec	12

<sup>a</sup>Except as noted by numbers in parentheses behind date.

Table 3. Incidence of Dermocystidium marinum in live oysters, James River, Virginia. Sample size was 25 oysters.

Year	Station	Date	Intensities of infection by percentages				Weighted Incidence <sup>a</sup>
			H	M	L	N	
1960	Brown Shoal	13 July				100	0.00
		20 Sept	4	8	8	80	0.52
		31 Oct		12	12	76	0.48
	Tray J2	27 Dec			4	96	0.04
	Wreck Shoal	21 Nov				100	0.00
1961	Brown Shoal	28 Sept			8	92	0.08
		14 Nov			8	92	0.08
	Tray J2	6 Oct		32	24	44	1.24
1962	Brown Shoal	27 July		4		96	0.12
		14 Aug				100	0.00
		7 Sept				100	0.00
		24 Sept		8		92	0.24
		29 Oct			4	96	0.04
	Tray J3	8 Oct		12	8	80	0.44

<sup>a</sup> Weighted incidence was obtained by assigning values of 5 for heavy infections (H); 3 for moderate (M); 1 for light cases (L); and 0 for negatives (N).

## MSX ACTIVITY IN LOW- AND HIGH - SALINITY AREAS

Two populations of seed oysters which became infected with MSX in James River in early summer of 1961 provide an interesting comparison of the effects of salinity level. One was the population on Wreck Shoal which has been described. The other population consists of lower James River seed moved to Plot 14-16 in Mobjack Bay in August 1961. Summer salinities are about 25 ppt in Mobjack Bay, which is some 10 parts higher than at Wreck Shoal. Both groups were of similar age and history.

Incidence of MSX in these two populations from August 1961 through 1962 is shown in Fig. 4. Although infections appeared somewhat earlier in Mobjack Bay than at Wreck Shoal, the level of infection through the late fall and winter was similar. In April and May, incidence in Mobjack oysters increased to well over 50% from late summer infections but MSX almost disappeared from Wreck Shoal oysters. By the first of May 1962 over 60% of Mobjack tray oysters were dead (Andrews, in preparation), whereas less than 10% of Wreck Shoal oysters had died. Low incidence of MSX at Wreck Shoal during the warm season from May to October 1962 resulted in less than 10% total annual losses whereas the Mobjack oysters suffered 80% mortality in one year from the time the first deaths from MSX were observed in August 1961.

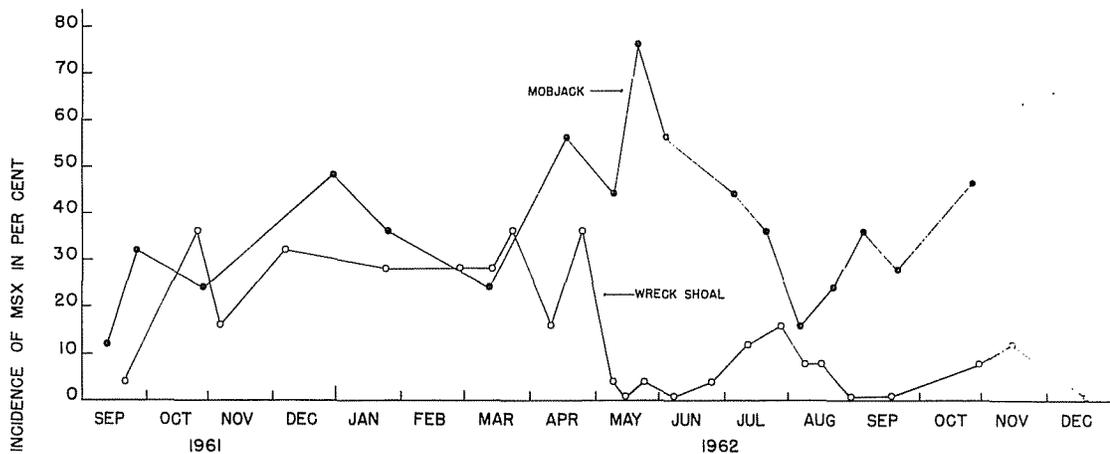


Fig. 4. Comparison of seasonal incidence of MSX in low- and high -salinity areas.

Since both lots were James River seed stock, infected while still in the seed area, the striking differences in mortalities and persistence of infections appear to be related to salinity levels. It is probable, however, that the planting in Mobjack Bay was exposed to increased dosages of infective particles in the fall of 1961, which may have accelerated and increased mortalities.

## DISCUSSION

The epidemiology and theory of disease of MSX has been discussed (Andrews, in preparation) but further elaboration is possible with the James River data. Nothing is known of dosage of infective material and the source is only indirectly suggested by field studies. My theory of disease rests upon the presumption that MSX is transmitted directly from oyster to oyster via water currents.

The timing of infections is important in understanding MSX activities in James River. It was shown (Andrews, in preparation) that disease-free oysters imported to high-salinity areas after 1 August probably became infected in late summer but did not show infections until the following spring. If infections do not develop in high salinities it is assumed that they are not likely to develop in lower salinities. Hence, the Wreck Shoal infections which appeared in October must have been initiated before 1 August. Furthermore, oysters imported to high-salinity areas from Wreck Shoal in August 1960 promptly showed MSX infections—over a month earlier than they appeared in the population on Wreck Shoal. This means that Wreck Shoal oysters had undetectable infections when moved. It appears, therefore, that early summer infections occurred at Wreck Shoal but that they did not become evident until October. Furthermore, the usual late summer kill was omitted at Wreck Shoal. It is presumed that the prevailing summer salinities did not prevent infections but inhibited development of MSX. Late appearance and high incidence in late fall was also observed in Pocomoke Sound which appears to be a fringe area for MSX. Late fall and winter incidences were relatively high at Wreck Shoal (30%) and often exceeded the levels in high-salinity areas where fall mortalities had occurred.

The source of infective material is presumed to be Hampton Roads and Brown Shoals in lower James River. Oysters in the vicinity of Wreck Shoal could not have provided a source since sick and dying oysters were absent in summer when infection occurred. More important is the probability that distribution of MSX in James River is related to circulation. MSX is present on deeper beds near the channel but absent from inshore shallow areas at the same level of the river. This tongue-shaped pattern of distribution has been observed previously for Dermocystidium in James

River (Andrews and Hewatt, 1957). The data supporting this apparent distribution of MSX are scattered by season and location, making presentation difficult and interpretation somewhat subjective. At the level in James River just above the bridge, MSX has been found in inshore shallow waters on both sides of the channel but less often and with lower incidence than at the Brown Shoal station near the channel. MSX has never been found at the inshore tray station opposite Wreck Shoal.

The apparent decline of MSX in the seed area in 1962 easily fits into my theory of the source of infective particles. Since no new susceptibles had been planted in Hampton Roads, a very small scattered population of oysters, mostly on public beds, remained in 1962. Even Brown Shoals, which never had more than 50% kill, showed relatively few deaths in 1962. In the absence of information on changes in virulence of the pathogen, I suggest that infective particles have become scarce. If MSX were being supported at epizootic levels by infective particles originating from another host or from the immediate locality of Wreck Shoal, there is no evident reason why a decline in activity should have occurred in 1962. An abundant supply of thickly-populated susceptibles still exists in the seed area. There is evidence of continued virulence in York River (Andrews, in preparation).

The disappearance of MSX from oysters at Wreck Shoal in April, during the lowest salinities of the year, seems to be quite strong circumstantial evidence that salinity is the primary controlling agent. No clear case of reduced incidence in high-salinity waters has occurred in Virginia without accompanying mortality. In high salinity, death of infected oysters reduces their number so that it is frequently difficult to reconcile excessive mortality rates with moderate levels of incidence found in live oysters. The impression is gained that few if any oysters recover in high-salinity areas once MSX infections are patent.

If oysters contribute to the expulsion of MSX on Wreck Shoal, it is peculiar that it can only happen in April and that all oysters are capable of disposing of infections during the same short period. Wreck Shoal oysters moved to Gloucester Point in early April retained their infections and exhibited a substantial death rate in spring and summer. Hence MSX remained viable all winter and infections were well established physiologically in early April, yet all were gone by 1 May.

It is a little dismaying to inspect Fig. 2 for the level of salinity during the period (June and July) when initiation of infections was believed to have occurred. In 1960 and 1961, salinities apparently never rose above 10 ppt during June, an important month for new infections, and only in late July were summer levels reached at Wreck Shoal.

Summer salinities of about 15 ppt apparently delayed the development of infections at Wreck Shoal, whereas about 20 ppt at Gloucester Point permitted deaths from early summer infections to begin in August. There is some evidence that first-summer deaths from MSX begin even earlier in the high-salinity waters of Mobjack Bay. It is possible that dosage of infective particles is the important factor in delaying plasmodial infections, but again close timing and group response suggest that physical factors are involved, and the quick appearance in oysters transplanted to high-salinity waters implies that infections are inhibited at Wreck Shoal. Sprague (1961) reported that MSX persisted and killed oysters at a salinity range of 14 to 16 ppt in aquaria.

The distribution of many estuarine organisms is apparently regulated by salinity, hence it is not surprising to find that MSX has limits too. It may well be that April purging of MSX requires a salinity as low as 5 ppt, for in both 1961 and 1962 salinity reached this level. It is interesting to note in comparison that some Dermocystidium survived one winter and spring at the upper end of the seed area and exposure to fresh water during that sojourn. Virginia winter temperatures do not appear to affect the survival of MSX.

The ecological technique of searching along the gradient of a factor for its effect on distribution and tolerance of an organism has been applied in regard to salinity and MSX. A rather steep salinity gradient exists from Hampton Bar in lower James River to Horsehead in the upper seed area. At the low-salinity station at Horsehead, no MSX has ever been found and no disease-caused mortalities have been established. At Wreck Shoal, infections of MSX occur, apparently from downriver imports of infective particles, but development is slow with no late summer and fall deaths. In April infections disappear, apparently from exposure to low salinities, and no appreciable death rate occurs because Wreck Shoal oysters are essentially free of evident infections during the warm season. At Brown Shoals also, oysters apparently acquire infections from downriver imports of infective material. MSX did not persist in a substantial population of Brown Shoal oysters after the source of infective particles was removed by decimation of Hampton Roads beds. At Brown Shoals infections developed earlier and a few deaths occurred in the fall and winter following early summer infections. Infections were not removed by spring low salinities. Mortalities were less than 50% per year with both MSX and Dermocystidium as agents of death. The decimation of oyster beds in fully epizootic areas, such as Hampton Bar, is described in another paper (Andrews, in preparation).

MSX poses no great threat to the James River seed area in terms of mortality unless exceptionally dry seasons are coordinated with

intensive epizootics in Hampton Roads as a source of infective material. Virginia is perhaps fortunate that the current epizootic occurred during rather wet years. Whenever infections are present in James River, the quality of seed oysters is seriously compromised for planting in high-salinity waters. This is fully exemplified by the plot 14-16 oysters in Mobjack Bay (Andrews, in preparation) in which oysters began dying within a month after transplanting and nearly 80% were dead within a year. It is concluded that the presence of MSX in the James River seed area depends upon repeated invasions from saltier waters. Oyster setting may also be dependent upon the tidal transport system to carry larvae upstream from Hampton Roads. The decimation of Hampton Roads oyster beds was followed by a nearly complete failure of spatfall in the seed area of James River in 1961 and 1962.

#### ACKNOWLEDGMENTS

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