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Oyster Mortality Studies in Virginia: III. Epizootiology of a Disease Caused by *Haplosporidium costale* Wood and Andrews[1]

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A short, sharp epizootic disease of oysters on Seaside of Eastern Shore, Virginia, has been associated with a new pathogen, *Haplosporidium costale* Wood and Andrews. Native oysters in trays have shown closely timed May-June losses for three consecutive years. Losses at other seasons were small. May-June losses ranged from 12 to 14 percent in 1959 to 36 to 44 percent in 1960. James River oysters moved to Seaside showed higher losses than natives after a year of acclimation. Oysters in Bayside creeks revealed late summer losses caused by *Dermocystidium marinum* Mackin, Owens, and Collier rather than May-June deaths.

The new pathogen was found in live oysters from March to July, and in a high proportion of gapers in May and June. The epizootiology is well established for these periods but unknown for the rest of the year. Increasing prevalence of another pathogen ("MSX"), causing "Delaware Bay disease," has complicated mortality studies.

Losses are most serious in older oysters which have been held beyond the usual period of culture. Careful timing of planting and early harvesting permit oystermen to avoid serious losses.

Introduction

A devastating mortality of oysters occurred in Delaware Bay in 1957 and 1958 (Haskin, 1958). An infectious and apparently highly contagious disease was suspected as the cause. Shipments of oysters and oyster shells between Delaware Bay and Eastern Shore of Virginia had been extensive in recent years and there was apprehension that the disease might spread to Chesapeake Bay along the high-salinity estuaries of the Delmarva (Delaware-Maryland-Virginia) Peninsula. It seemed imperative, there-

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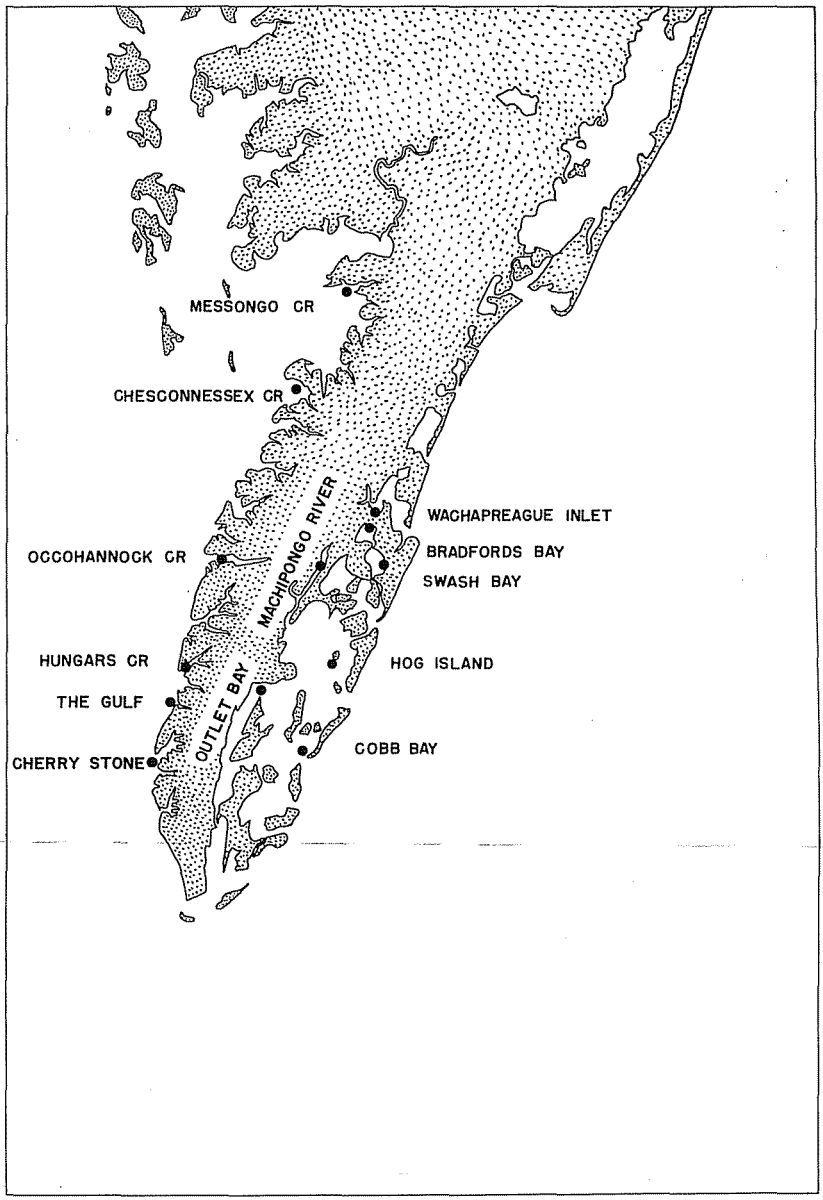

FIG. 1. A map of Eastern Shore of Virginia showing location of tray stations on Seaside and Bayside. Seaside is to the right; Bayside is to the left.

fore, to monitor Eastern Shore for occurrence or introduction of the disease.

In February 1959, with the help of oystermen, we placed trays of oysters at stations on both sides of the peninsula in Virginia (Fig. 1), and began the regular sampling of beds. In addition, the Biological Laboratory of the U. S. Bureau of Commercial Fisheries at Franklin City, Maryland has monitored the waters adjacent to Chincoteague, Virginia, and the Maryland Department of Research and Education has been observing oysters in Chincoteague Bay.

The Delaware Bay disease did appear in Chesapeake Bay in 1959 and it has been intensively studied (Andrews and Wood, 1960). But on Seaside of Eastern Shore our studies revealed a pattern of mortality which is distinctly different from that found in Chesapeake Bay (Hewatt and Andrews, 1954; Andrews and Hewatt, 1957) and in Delaware Bay (Haskin, 1960). This pattern, particularly the seasonal distribution of deaths, has drawn our attention to a new pathogen of oysters on Seaside. This pathogen, called Seaside organism (SSO) when first discovered, has now been described as a new species, *Haplosporidium costale* Wood and Andrews (Wood and Andrews, 1962). The pattern of oyster mortality and the epizootiology of "Seaside disease" are considered in the present paper.

Methods and Materials

Stations were chosen in major planting areas on both sides of the Eastern Shore peninsula (Fig. 1). Oysters from planted beds of known history were placed in fully enclosed trays on the same beds. Trays were designed with legs to raise oysters about one foot off the bottom to eliminate predation and smothering (Fig. 2). Known predation and smothering have been excluded from mortality data. Oysters were examined weekly or biweekly in warm weather, and monthly in cold. Gapers (dying oysters) and boxes (empty shells) were removed at each visit and accurate counts were made. Trays contained from 200 to 500 oysters initially. Disease-free control groups were obtained from James River.

Death rates have been expressed as number per thousand per month for each period between examinations. The length of these periods varied from a week to over a month. Annual mortality rates have been calculated using instantaneous rates to adjust for losses and removals. For annual mortality the year begins 1 April.

Incidence studies in live oysters were based on collections from certain planted beds. Gapers are difficult to recover from natural beds, hence most gapers were obtained from trays. The same oysters were held in trays through 1959 and 1960 whereas planted beds were harvested each year. Thus gapers collected in 1960 came from older oysters with heavier

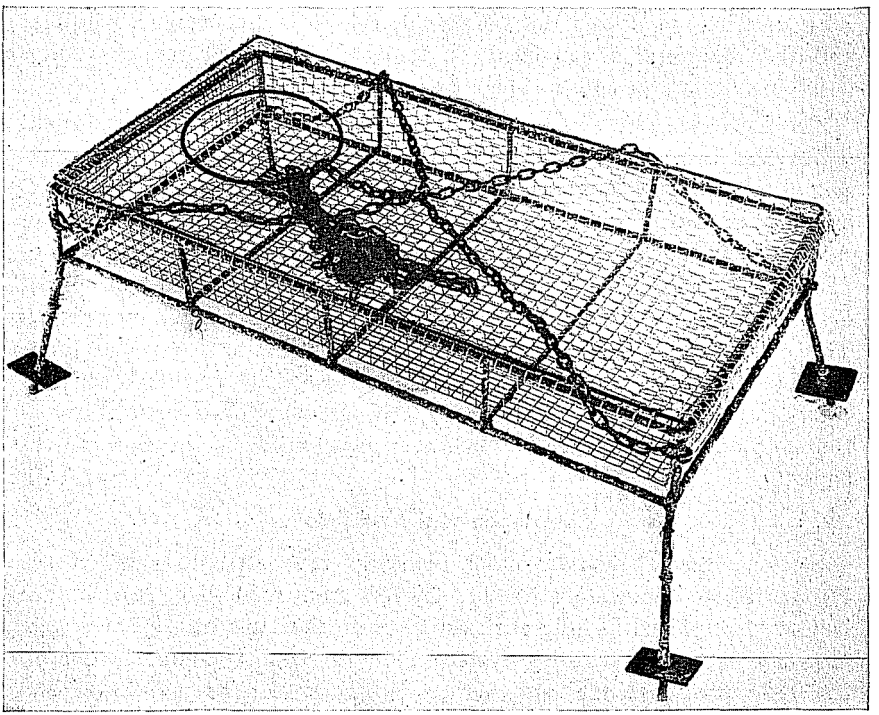

FIG. 2. An asphalt-covered wire tray of the type used to keep known numbers of oysters off the bottom to prevent predation and smothering.

death rates than the live oysters sampled. Incidence data reported in this paper are mostly from 1960 collections, therefore the differences in ages and history of live oysters and gapers must be noted.

Incidence or prevalence of *Haplosporidium costale* was determined from permanent slides of stained oyster-tissue sections. For purposes of identification and diagnosis, three stages have been designated: early plasmodium, late plasmodium, and mature spore with a thick glassy wall. Infections were categorized by the most advanced stage observed.

Results

Mortality Studies

The histories and annual mortalities of oysters at five stations on Seaside are given in Table 1. The oyster-growing areas represented are widely separated, and they usually have separate inlets from the ocean and rather intricate and often shallow tenuous connecting channels between them. Machipongo Creek is a rather enclosed seed area with lower salinities than the other areas. Oysters held here followed the time patterns of mortality but at reduced levels. Source and age of oysters are important items of history for disease studies. For oysters not native to an area the date of importation is often critical. Oysters on Seaside grow rapidly and are harvested at early ages. Most of the oysters placed in trays in 1959 were one- and two-year-olds from beds planted in the fall of 1958 and destined to be harvested in the winter of 1959-60.

Native oysters had annual death rates in 1959 ranging from 12 to 26 percent but typical figures for most trays were 12 to 14 percent (Table 1). The same trays of oysters had typical losses of 36 to 44 percent in 1960. Death rates in duplicate trays did not vary significantly during the period of May-June epizootics but often did after a full year of observation (Chi-square tests). In 1960 the tray oysters were overage as compared to oysters on most planted beds. Observations on planted beds indicated losses in both years at the level recorded in trays for 1959. However, a few beds held an extra year had heavy losses in 1960.

Seasonal distribution of deaths is illustrated in Fig. 3. Each curve represents death rates for a pair of duplicate trays. Each point is expressed as rate per month regardless of the length of the period of observation. Annual mortality cannot be accurately deduced from the death rate curves but is given in Table 1.

The pattern of mortality in trays was simple, and sharply defined. A short epizootic occurred between mid-May and mid-July with negligible death rates through the rest of the year. In 1960, death rates climbed as high as 40 percent per month for short periods. A remarkable similarity of timing was observed at all stations.

The Seaside epizootic was repeated in 1961 for the third consecutive year (Fig. 4). Two ages of oysters are represented in Fig. 4. The bottom curves show death rates in survivors of populations held in trays through 1959 and 1960. The top curves were obtained from oysters approximately one year younger. Both groups would be considered overage on Seaside

TABLE 1

SUMMARY OF HISTORIES AND ANNUAL MORTALITIES OF OYSTERS IN TRAYS, SEASIDE OF EASTERN SHORE, VIRGINIA, 1959 AND 1960

Location	Tray no.	Date started	Source of oysters	1959		1960	
				No. of oysters	Death rate (percent)	No. of oysters	Death rate (percent)
Cobb Bay	S1[a]	26 Feb 59	Local	300	14	250	37
	S2[a]	26 Feb 59	Local	300	13	245	39
	S12	16 Jun 59	J. R.[b]	500	4	464	63
Hog Island Bay	S3	25 Feb 59	J. R.	498	12	433	51
	S4[a]	25 Feb 59	Local[c]	300	14	287	44
	S5[a]	25 Feb 59	Local[c]	300	12	292	37
Outlet Bay	S7	15 May 59	J. R.	500	5	462	33[d]
	S8	15 May 59	Local	400	13	336	24
Swash Bay	S9[a]	5 Mar 59	Local	250	22	274	35
	S10[a]	5 Mar 59	Local	249	26	273	44
	S13	16 Jun 59	J. R.	500	8	450	47
	S15	24 Feb 60	J. R.	—	—	496	24
Machipongo Creek	S11	15 Jun 59	J. R.	500	5	456	21
	S14	15 Jun 59	Local	320	2	295	22

[a] Duplicate trays.

[b] J. R. = James River.

[c] Trays replenished with local oysters on 24 Jun 59 to original count.

[d] Record to Sept. 2 when tray damaged and oysters killed.

where oysters are usually harvested in 24 to 30 months. The older oysters, which had been through two previous epizootics, appear to have had lower losses in 1961. The younger oysters (not listed in Table 1) show different levels of mortality in stocks of similar age and history. Most other groups

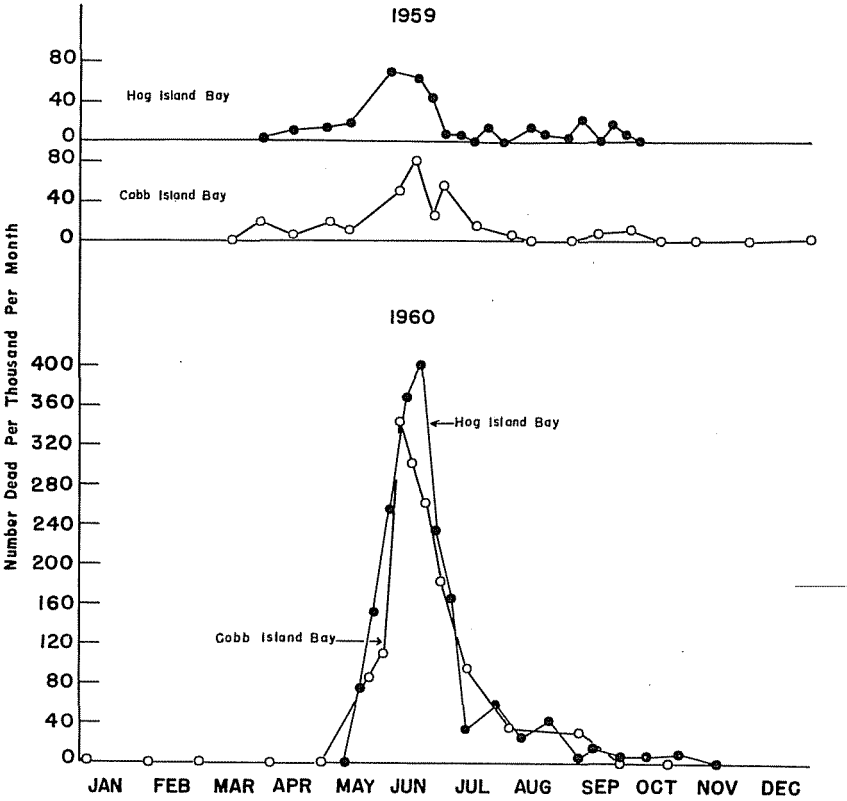

FIG. 3. Death rates of native Seaside oysters in trays. Each point represents the monthly death rate for the preceding interval.

have shown similar mortalities for a particular year at all stations (Table 1).

In comparing the timing and the level of deaths at various stations, it is important to know the history of each lot of oysters. The Hog Island Bay oysters (S4 and S5) were caught in Machipongo Creek in 1957 and were almost two, three, and four years of age at the respective May-June

epizootics of 1959, 1960, and 1961. The Cobb Bay oysters (S1 and S2) were obtained from a boat dredging for market and were three or more years of age in 1959. The Swash Bay and Hog Island Bay oysters depicted in Fig. 4 (upper curves) were placed in trays in the fall of 1960, and were from beds held over an extra year. Both beds experienced mild

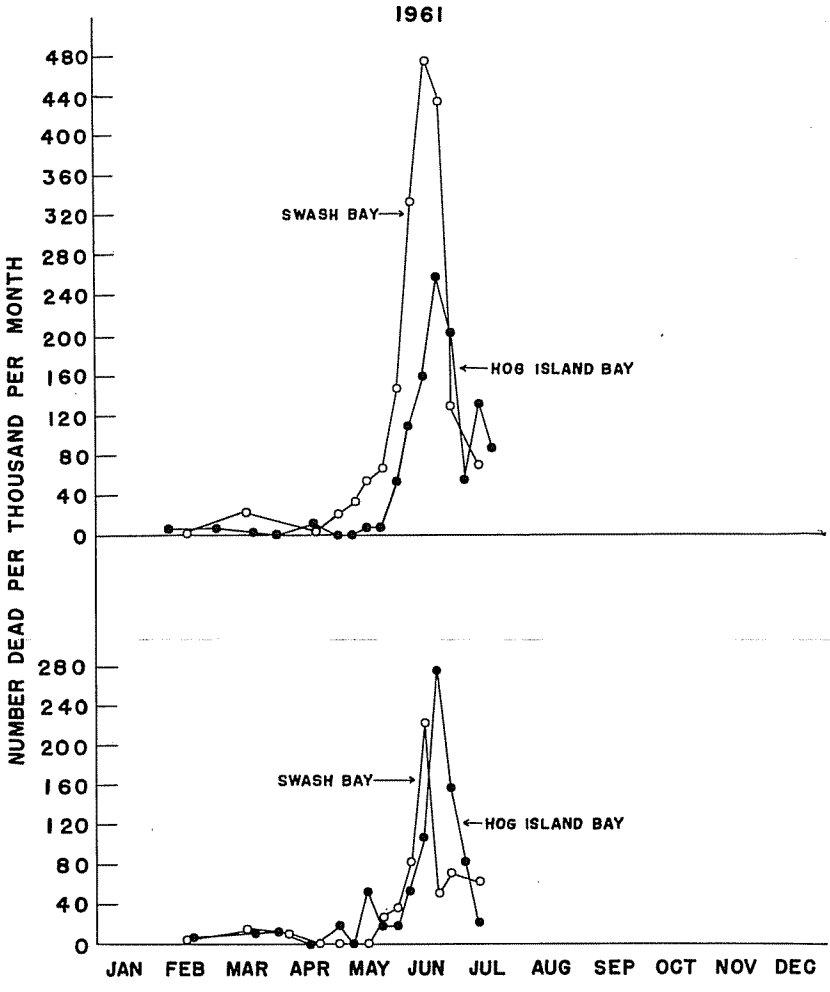

FIG. 4. Death rates of old oysters (lower curves) and acclimated oysters (upper curves) in trays on Seaside in 1961. All were native oysters.

losses in May and June 1960 and the oysters in trays were going through their second epizootic in 1961.

All discussions so far have been concerned with native oysters picked up and placed in trays at the locality in which they were planted commercially. For controls, oysters from Wreck Shoal of James River, known to have low death rates in their native low-salinity area, were imported. Some difficulties were encountered with midwinter transplants of James River seed from very low salinities (5 to 10 parts per thousand) to very high ones (25 to 35 parts per thousand) but in general these oysters were satisfactory as controls.

James River oysters had low mortalities the first year of exposure on Seaside but very high death rates the second year (Table 1). There was no sign of trouble during their first year of exposure in areas where SSO (*Haplosporidia*) epizootics were prevalent. The apparent exceptions to the generalization that survival was excellent in the first year were two winter transfers (S3 in 1959 and S15 in 1960). Deaths in these trays did not follow the typical SSO pattern of kill and we believe that these deaths have separate causes associated with change of environment. Gapers from these trays sometimes appeared sick in stained sections but did not contain recognized pathogens.

At all stations except Machipongo Creek, the death rate in the second year was significantly higher in James River oysters than in natives. This may be interpreted as indicating greater susceptibility of James River oysters to the disease or as a consequence of partial selection of native oysters in previous epizootics. The timing of deaths was identical in James River and native oysters, implying that the causes were the same.

Comparison of Bayside and Seaside Mortalities

Mortality data from one of six Bayside stations are presented to emphasize differences between Seaside and Bayside in timing and causes of deaths. The small embayment called The Gulf was chosen for report because oystering there is controlled by one man and imports of Seaside oysters have been minimal. Late summer and fall kills have predominated in the past with *Dermocystidium marinum* Mackin, Owens, and Collier as the chief agent of death. This pattern of losses was shown by The Gulf in 1959 and 1960 (Fig. 5). Late summer losses were observed in James River imports and native oysters. The absence of a May-June mortality is conspicuous.

In summary, Bayside creeks did not show May-June epizootics caused

by SSO, and Seaside bays did not exhibit late summer and fall epizootics which are associated with *Dermocystidium* and MSX. These respective mortality patterns persisted regardless of source and history of oysters used. In general, the Bayside creeks of Eastern Shore follow Chesapeake Bay rather than Seaside in their mortality patterns.

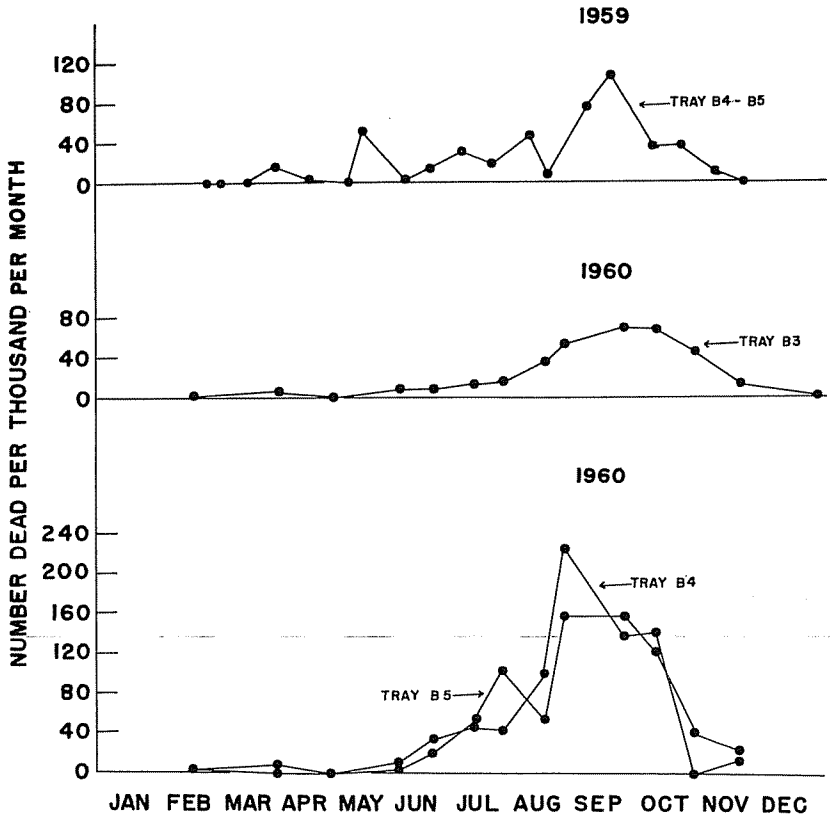

FIG. 5. Death rates of native Bayside oysters (B4 and B5) and James River controls (B3) in trays in The Gulf, Bayside of Eastern Shore. In Trays B4 and B5 the same population of oysters was observed in 1959 and 1960.

An intensive sampling program with tests for the presence of *D. marinum,* MSX, and SSO has been pursued on Bayside as well as Seaside. These results can only be touched upon here. SSO has been rare in Bayside oysters although the parasite was first seen in gapers from Tray B5

in The Gulf in 1959. In 1960 a few cases were observed in native oysters in Cherrystone Creek, and again in 1961 occasional SSO-infected gapers were found in The Gulf. This suggests that SSO can infect and develop in salinities appreciably lower (20-23 parts per thousand) than those found in Seaside waters (usually over 30 parts per thousand).

Relation of SSO to Seaside Mortalities

The problem of linking cause and effect in oyster mortality studies is difficult because so few diseases of oysters are known, and diagnostic methods are still limited. The pattern of kill on Seaside in 1959 suggested that agents other than MSX and *Dermocystidium* were involved. Gapers from the May-June epizootic were found to contain masses of cysts with haplosporidian-type spores. The thick-walled spores are particularly suggestive of a new organism since in Chesapeake Bay such spores have been absent from over a thousand live oysters and gapers with MSX infections. It was not until the May-June epizootic was repeated in 1960 that we were convinced that a new parasite was responsible. In the spring of 1960 an extensive collection of live oysters and gapers was obtained showing a logical sequence of diverse stages of SSO. This provided the basis for incidence and morphological studies which support the belief that SSO is the causative agent of the Seaside May-June mortalities.

Incidence in Live Oysters

Incidence of SSO in live oysters is given in Table 2. The samples for this series were all taken from the same planting of oysters in Hog Island Bay. The earliest infections of SSO were observed as early plasmodia in March. Two large samples taken in April and early May, prior to the beginning of deaths in mid-May, showed 33 and 19 percent incidence of SSO. Incidence in live oysters declined during the epizootic as infected individuals were removed by deaths and after mid-June no infections were found in live oysters. A comparison of incidence in live oysters before and during the epidemic can be made from the summary of samples from Hog Island Bay in Table 4.

In trays, samples of live oysters showed rather high levels of infection. For example, 96 oysters taken 24 May from Cobb Bay had 23 cases of Seaside disease. Only 5 to 7 percent of the oysters in these trays had died by this date; therefore, few infections had been removed by deaths. Incidence in live oysters appears to be adequate to account for observed death rates in trays. The abrupt disappearance of recognizable SSO

TABLE 2

INCIDENCE OF DISEASE IN NATIVE LIVE OYSTERS FROM PLANTED BEDS, HOG ISLAND BAY

Date	No. examined	SSO infections[a]			MSX infections	*Bucephalus*
		Early	Late	Spores		
1959						
25 Aug	5	—	—	—	—	—
1 Oct	15	—	—	—	—	—
1960						
23 Feb	6	—	—	—	1	—
9 Mar	10	3	—	—	1	—
6 Apr	5	2	—	—	1	—
13 Apr	5	3	—	—	—	—
27 Apr	21	4	3	—	1	—
11 May	21	2	1	1	1	—
7 Jun	16	—	—	2	—	—
11 Jun	21	1	2	—	3	—
18–26 Jun	25	—	—	—	—	—
6 Sep	25	—	—	—	—	1
27 Oct	25	—	—	—	—	—
16 Dec	25	2	—	—	5	2
1961						
12 Jan	25	—	—	—	1	—
7 Feb	25	1	—	—	3	—
1 Mar	25	1	—	—	—	—
20 Mar	25	2	—	—	—	—
5 Apr	25	9	—	—	—	—
18 Apr	25	4	1	—	1	—
1 May	25	3	—	—	—	—
8 May	23	4	4	—	—	—
15 May	50	9	3	—	2	—
22 May	25	2	2	—	—	—
29 May	25	3	1	1	—	—
5 Jun	25	—	1	—	3	—
12 Jun	25	—	—	1	2	—
19 Jun	25	—	—	—	3	—
10 Jul	25	—	—	—	2	—

[a] The terms early and late refer to plasmodial stages.

following the epizootic suggests that few if any oysters survived once infections had become patent.

Incidence in Gapers

Gapers began appearing about 10 May 1960 and were collected until slightly after 1 July. Very high death rates during a short period of time

facilitated collection of hundreds of gapers from trays. Data for the trays at the Hog Island Bay station are presented in detail.

Incidence of SSO was much higher in gapers than in live oysters (Table 3). In one collection of 32 gapers on 26 May, 31 were found to have SSO infections. About two-thirds of the infected gapers contained mature spores whereas in live oysters spores were relatively rare.

TABLE 3
INCIDENCE OF DISEASE IN HOG ISLAND BAY GAPERS

Date	Source	No. of gapers	SSO infections			MSX infections	*Bucephalus*
			Early	Late	Spores		
1959 Jun	S3 to S6	9	—	1	1	—	—
Aug-Sept	S3, S5, S6, S13	4	—	—	—	3	—
1960 23 Feb	N (Native)	6	—	—	—	1	—
11 Apr	N	1	—	—	—	—	—
11 May	N	2	—	1	1	—	—
18 May	S3-6	9	—	—	5	—	—
26 May	S3-5	32	1	8	22	—	—
3 Jun	S3-5	34	3	3	21	—	—
7 Jun	—	1	—	—	—	—	—
11 Jun	S3-5	28	2	7	15	—	—
18 Jun	S3-6	28	2	10	6	—	1
26 Jun	S4-5 & N	3	—	—	—	—	—
1961 12 Jan	S5	1	—	—	—	1	—

Gapers too decomposed to diagnose were listed as negatives. In three widely separated areas, incidence in gapers was 77, 78, and 90 percent for the 1960 epizootic (Table 4). No gapers were diagnosed as having SSO in any months of the year except May and June. Table 4 should be studied primarily for seasonal distribution rather than level of incidence in live oysters because samples from trays and planted beds of different ages are included. Levels of incidence in gapers can be compared since nearly all gapers came from trays.

MSX in Seaside Oysters

MSX was present on lower Seaside as early as June 1959 and it has been found at all times of the year in live oysters but never in abundance (Tables 3 and 4). Occasional gapers throughout the year have MSX infections. MSX infections in gapers may have been missed during SSO epizootics because diagnosis is difficult in damaged and deteriorating

tissues. However, incidence of MSX was low throughout 1960 as compared to levels experienced in the epizootic areas of Chesapeake Bay and it appears that this organism has been a negligible factor in May-June epizootics. In the winter of 1960-61 an increase in incidence of MSX in live oysters was followed by a low but persistent death rate during the summer and fall of 1961. Most gapers collected out of the SSO mortality season

TABLE 4
SUMMARY OF DISEASE INCIDENCE IN SEASIDE OYSTERS
1959 AND 1960

Location	Period	Kind of oysters	No. examined	No. of Infections			% SSO
				SSO	MSX	*Bucephalus*	
Cobb Bay	May-June 60	Live	142	24	1	0	17
		Gapers	60	54	0	1	90
	Other months	Live	81	0	1	2	0
		Gapers	0	—	—	—	—
Hog Is. Bay	March-April 60	Live	41	14	3	0	34
		Gapers	1	0	0	0	0
	May-June 60	Live	125	11	6	0	8
		Gapers	137	107	0	1	78
	Other months	Live	226	0	10	6	0
		Gapers	11	0	5	0	0
Swash Bay	April-June 60	Live	—	—	—	—	—
		Gapers	71	55	1	0	77
	Other months	Live	—	—	—	—	—
		Gapers	21	0	3	0	0

in 1961 were positive for MSX. *Bucephalus* was relatively rare in all areas of Eastern Shore.

Discussion and Conclusions

Seasonality of Epizootics in Chesapeake Area

Seaside disease caused by *Haplosporidium costale,* was discovered while monitoring for MSX on Eastern Shore. The term "MSX" refers to the organism discovered by Haskin, Stauber, and Mackin (Haskin, 1960), which has caused extensive losses of oysters in Delaware and Chesapeake bays in recent years. Haskin (1958) had reported finding MSX in Chincoteague Bay oysters collected in October 1958.

Questions have been raised whether SSO and MSX might be the same

organism in different environments or in different races of oysters. This viewpoint was encouraged by early losses of Seaside transplants in the Delaware Bay catastrophe immediately following some five or six years of intensive importation of seed from Seaside. Both organisms exhibit a multinucleate plasmodium, as do many Protista, but there the resemblance stops. Further comparisons must await publication of epizootiological and morphological detail of MSX.

After observing the periodicity of deaths for three years, it is apparent that Seaside epizootics do not follow the pattern known for MSX (unpublished data of authors) but are caused by a newly discovered disease that, as presently known, is confined mostly to Seaside of the Delmarva peninsula.

The only other known major disease in the Chesapeake area is caused by *Dermocystidium marinum,* which kills oysters in late summer and fall (Andrews and Hewatt, 1957). Mortality studies in lower Chesapeake Bay have revealed a pattern of high death rates from July to October and low death rates during the "cold" season of the year from November to June. This pattern of late summer losses only, in Chesapeake Bay, has been rapidly changed by the activity of MSX, which caused heavy losses in 1959 and 1960. *Dermocystidium* is inexplicably absent from Seaside waters, which greatly simplified the task of assigning causes for oyster losses. In contrast to Seaside waters, Bayside creeks follow Chesapeake Bay patterns of mortality.

Association of SSO with May-June Epidemics

Only a part of the life cycle of SSO is known. Experimental infections have not yet been attempted and nothing is known of stages and locations of the organism from July each year until it reappears in oysters the following March. Method and time of infection are unknown but field data suggest certain limitations. Oysters acclimated to Seaside, and thereby exposed to one SSO epizootic, die with high incidence of the disease the following May-June. Unexposed James River oysters brought in as early as February neither died nor showed SSO the following May-June. James River oysters moved in mid-June 1959 experienced the full epizootic of SSO in 1960. No infections were found during the 1961 epizootic in James River stock transplanted as late as November 1960.

The presumption that SSO is the cause of May-June epizootics on Seaside is based upon association in time and place and prevalence of the organism in live oysters and gapers. The regular occurrence of an organism

in the living tissues of another organism is rather strong evidence of parasitism. When the host shows strong leucocytosis and tissue disruption in a characteristic syndrome of morbidity, pathogenicity is indicated. The progression of stages in live oysters prior to the May-June losses is indicative of a disease-producing organism. The occurrence of spores in live oysters as well as gapers adds to the evidence. SSO shows higher incidence and more advanced stages in gapers than in live oysters—a sequence expected in pathogenic organisms. Repetition of this pattern of incidence and mortality in five separate localities of Seaside adds strongly to the evidence for pathogenicity. No other organisms were observed in abundance with any regularity. Bacteria were not evident except in gapers, and viruses have not been investigated.

The effects of age, source, and acclimation of oysters on SSO epizootics were only partly elucidated in these studies. All of these factors seem to be subordinate to the level of the epizootic in a particular year. The intensity of SSO in exposed populations of oysters seems to be similar at all stations in a given year, but rather large annual variations occur. In 1959, losses were low and uniform regardless of source, history, and locality. In 1960, losses were high in all localities but only in native oysters over two years of age and imported oysters acclimated for six months or more. In native oysters, exposure at an early age and selection by SSO during each May-June epizootic makes analyses of age and exposure factors difficult.

Importance of SSO on Seaside

Oystermen on Seaside have been plagued by serious drill predation for so long that all other causes of losses have been ignored or have gone unnoticed. They have learned by experience that successful oystering requires planting the largest native seed available, and seed planted one fall, winter, or spring is usually harvested the following year after 12 to 18 months of culture. Attempts to hold oysters longer result in heavy losses. It has been repeatedly said that James River seed will not survive on Seaside. Yet such oysters in trays lived 15 months with less than 10 percent losses; then an epizootic of Seaside disease killed over half in their second May-June period on Seaside. SSO appears to cause little trouble on Seaside if oysters are grown and harvested rapidly and if exposure to more than one May-June epizootic is avoided after one year of age is reached.

Minor losses in young oysters and much heavier losses in older oysters

are caused by SSO each year. Estimating losses on Seaside beds of heavily clumped oysters is difficult. Counts of dead oysters and talks with oystermen indicated only small losses on most planted beds—probably not in excess of 10 to 15 percent—but heavy losses on the very few beds of older oysters not harvested at the usual time.

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REFERENCES

ANDREWS, J. D., AND HEWATT, W. G. 1957. Oyster mortality studies in Virginia. II. The fungus disease caused by *Dermocystidium marinum* in oysters of Chesapeake Bay. *Ecol. Monogr.*, **27**, 1-25.

ANDREWS, J. D., AND WOOD, J. L. 1960. Unpublished data.

HASKIN, H. H. 1958. Personal communication, December.

HASKIN, H. H. 1960. Personal communications.

HEWATT, W. G., and ANDREWS, J. D. 1954. Oyster mortality studies in Virginia. I. Mortalities of oysters in trays at Gloucester Point, York River. *Texas J. Sci.*, **6**, 121-133.

WOOD, J. L., AND ANDREWS, J. D. 1962. *Haplosporidium costale* (Sporozoa) associated with a disease of Virginia oysters. *Science*, **136**, 710-711.