6-1967

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Proceedings of the National Shellfisheries Association
Volume 57 — June 1967

INTERACTION OF TWO DISEASES OF OYSTERS IN NATURAL WATERS[1]

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ABSTRACT

A localized epizootic caused by Dermocystidium marinum *was induced in oysters in the York River, Virginia, to simulate natural epizootics in timing of infections and mortalities. Relative isolation was achieved by use of trays located on barren bottom. Decimation by* Minchinia nelsoni *(MSX) since 1960 has insured low populations of oysters and a relative scarcity of* Dermocystidium. *Trays spaced 15 feet apart exhibited epizootics which were progressively earlier and more vigorous in proportion to the number of infected oysters added (foci of infection).* Minchinia *infections were severe and at least 50 per cent of the oysters died from this parasite.* Dermocystidium *spreads more rapidly and kills more quickly but requires dense populations and high temperatures. MSX is not localized and is much less affected by these limiting factors. In lower York River, new imports of disease-free oysters require one to three years to acquire* Dermocystidium *cases which initiate epizootics. During this period MSX decimates oyster populations, thereby preventing* Dermocystidium *from becoming epizootic.*

INTRODUCTION

Fifteen years experience in field and laboratory indicates that *Dermocystidium marinum* causes a typical infectious disease transmitted from oyster to oyster by close proximity. Isolation has proven to be an effective method of controlling the disease caused by this fungus pathogen. Nature has performed a large-scale isolation experiment on *Dermocystidium* in Virginia waters through the activities of *Minchinia nelsoni* (MSX), a virulent sporozoan pathogen which decimated oysters and forced oystermen to stop planting oyster beds in high-salinity areas. Before 1960 the fungus was common in oyster-planting areas throughout the lower Chesapeake Bay. Now it is rare except in areas where susceptible oysters are imported regularly and and where appreciable numbers of oysters survived the MSX epizootic.

Infection experiments carried out in natural waters before and during the *Minchinia nelsoni* epizootic have provided valuable information on transmission of *Dermocystidium* (Andrews, 1965). Failure to obtain timely infections in laboratory oysters, which were to have served as foci of infection, limited the value of the 1963 experiment conducted in a period of relative scarcity of *Dermocystidium*. A new proximity experiment conducted in 1965 is reported here. Continuation of the MSX epizootic insured low populations of oysters in the area of York River around the Virginia Institute of Marine Science (VIMS), hence low natural prevalence of *Dermocystidium* during the experiment.

METHODS

Methods and area of experimentation were the same as reported in the 1965 paper. Disease-free oysters (experimentals) were imported 27 May 1965 from Horsehead Rock, a low-salinity site in the James River. After 11 days at VIMS pier, four trays of oysters were placed about 15 feet

[1] Contribution No. 207 from the Virginia Institute of Marine Science, Gloucester Point, Virginia.

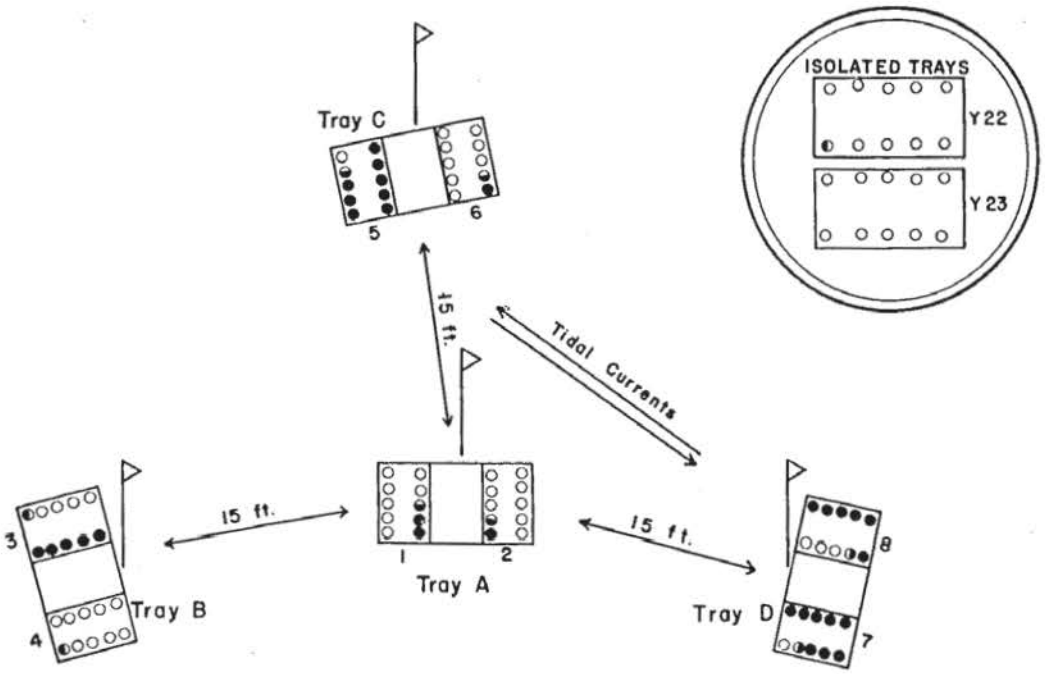

FIG. 1. *Percentages of live oysters infected with* Dermocystidium *on 8 October 1965. Each circle represents 10% of a compartment population with closed circles indicating fungous infections in experimental oysters.*

apart off from the pier on sandy bottom free of wild oysters. Each tray contained 400 oysters, segregated into lots of 200 in end compartments by two wooden partitions and monitored separately. Numbering of compartments in letter-designated trays is shown in Table 1 and Figure 1. The control tray (A) was placed by a center stake and three experimental trays by stakes arranged in a triangle around it at distances of about 15 feet.

On 16 June, 75 oysters marked with orange paint were fed macerated *Dermocystidium*-infected gapers in an aquarium. On 18 June these fungus-exposed oysters were added to compartments of the trays as follows: tray A—none; tray B, compartment 3—2 oysters; tray C, compartment 5—10 oysters; and tray D, compartment 7—61 oysters. Hence comparisons of prevalences and mortalities could be made between trays and between compartments in the same trays.

Gapers and boxes (dead oysters with and without meats) were usually collected from the trays by divers using SCUBA but occasionally, when the trays were brought up for cleaning, oysters were examined in the boat. All gapers and live oysters in samples were tested for *Dermocystidium* by the thioglycollate method of Ray (1952). All were examined also for MSX in stained tissue sections.

RESULTS

Monitoring of Artificially-Infected Oysters

Three paint-marked boxes were found in compartment 7 on 16 July. By 30 July, 12 more marked oysters were dead. Two gapers had heavy infections of *Dermocystidium* and nine live oysters revealed seven fungous infections, of which five were advanced cases. By mid-August marked gapers with heavy cases had been recovered from each of compartments 3, 5 and 7. These early samples indicated that laboratory infections had been successfully induced and that timing of infections was about right to simulate typical summer epizootics of the fungous disease. Epizootics of varying intensities were expected from the number of infected oysters introduced in some compartments and the absence of foci of infection in others. No attempt has been made to present complete data on marked oysters. Both MSX and *Dermocystidium* were found in gapers, with the fungus predominating. Nearly all of these oysters died before the experiment was ended.

Mortalities of Experimental Oysters

Deaths in the experimental oysters began in mid-July from MSX but less than 5 per cent had died by 30 July. By this date compartment 7 had

TABLE 1. *Accumulative mortalities in proximity experiment trays, 1965 (Initial count — 200 per compartment).*

Tray no.	Compartment	30 July	17 August	8 September	8 October	31 December
A	1	4	12	23	44	54
	2	2	11	26	47	63
B	3[a]	5	14	32	60	70
	4	5	16	43	60	72
C	5[b]	2	11	28	55	67
	6	4	12	16	36	54
D	7[c]	6	13	49	75	—
	8	4	11	24	40	65

[a]Foci of infection consisted of 2 fungus-infected oysters added.
[b]Foci of infection consisted of 10 fungus-infected oysters added.
[c]Foci of infection consisted of 61 fungus-infected oysters added.

one well-established infection of *Dermocystidium* in 25 live oysters. None were found in 25 live oysters from compartment 8. Eleven gapers from six different compartments of experimental oysters had no fungus on 30 July. By 13 August, 12 more gapers from seven different compartments revealed only one case of fungus (in compartment 7). Thus MSX had been killing experimental oysters for a month before *Dermocystidium* caused deaths.

Accumulative mortalities by dates of examination are given in Table 1. Mortalities began by 30 July and increased rapidly in August and September. By 8 September, compartment 7 had lost half its oysters and was ahead of other trays in cumulative mortality. A month later, compartmen 7 had reached 75 per cent mortality and other compartments had about 50 per cent dead. Losses in all trays for the six-month period ending 31 December were higher than those usually experienced for either MSX or *Dermocystidium* alone.

Prevalences of Diseases in Live Oysters

Prevalences of the two diseases in live oysters are shown in Table 2. Exposure to MSX infection can be assumed from 27 May, the day of import (Andrews, 1966). Gapers from fungus-inoculated oysters began to appear in late July at which time experimental oysters were first exposed to

TABLE 2. *Prevalence of diseases in live oysters, 1965 (Percentages in samples of 25 oysters).*

Tray	Compartment	17 August		8 October		31 December	
		MSX*	Dermo	MSX	Dermo	MSX	Dermo
A	1	64	0	48	24	56	0
	2	80	0	40	16	52	0
B	3[a]	40	0	28	52	64	8
	4	40	4	20	4	56	4
C	5[b]	32	8	56	88	40	8
	6	40	0	28	16	56	8
D	7[c]	36	75	52	88	—	—
	8	60	12	40	64	40	16

*An exceptional number of localized rare cases in gills in these samples.
[a]Foci of infection consisted of 2 fungus-infected oysters added.
[b]Foci of infection consisted of 10 fungus-infected oysters added.
[c]Foci of infection consisted of 61 fungus-infected oysters added.

Dermocystidium. This is typical timing of exposure for new imports in Virginia. By 17 August, two months after mixing with newly-infected oysters and no more than one month after first exposure to gapers killed by *Dermocystidium*, compartment 7 had 75 per cent infection of the fungus in live oysters. It had also appeared in three of the remaining seven compartments. At this time deaths from MSX were occurring rather uniformly in all trays. However, prevalences were more variable than usual for oysters of uniform history and exposure. This is due partly to an unusually high proportion of localized (very recently patent) cases. Finding of these by sections is somewhat fortuitous and samples of 25 oysters permit considerable variation by chance.

By 8 October, the compartments with fungus-inoculated oysters (3, 5, and 7) were showing high levels of infection compared to control compartments in the opposite ends of the same trays (Fig. 1). Apparently the trays were grouped too closely to prevent transmission to the control oysters in the center tray. However, fungous prevalence was no greater in compartments 4 and 6 adjacent to foci of infection in 3 and 5 than in the control oysters in tray A. Figure 1 shows the relative postions of trays and direction of currents but no attempt was made to keep compartments aligned as illustrated. Despite the prevalence of *D. marinum* in all compartments by 8 October, it is difficult to deduce that fungous infections had increased mortality rates differentially except in compartment 7 (Table 1). By this date all compartments had fungous infections. Despite the rising level of fungous infections, MSX was still causing most deaths in all compartments except 7. Curiously, tray A, with higher levels of MSX infection, had lower death rates than tray B. It is unfortunate that live-oyster samples were not taken about mid-September.

By 31 December, most *Dermocystidium* infections had disappeared from the populations, but about half of the surviving oysters had cases of MSX. Since deaths from the fungus usually stop about 1 November each year due to inhibition of multiplication by falling temperatures, samples should have been taken in November for post-mortality season prevalences of *Dermocystidium*. Oysters usually discharge those stages which respond to thioglycollate tests as soon as winter temperatures are reached in December. Furthermore, some oysters with mixed infections of MSX and the fungus were probably killed by the sporozoan parasite after 1 November.

A high level of initial infections by MSX from early-summer exposure can be deduced. Two-thirds of the oysters had died by the end of the year and probably 50 per cent were killed by MSX. Half the survivors were infected at the end of the year, hence a minimum of 75 per cent of the initial populations were infected. Unlike the fungus, MSX continues to kill throughout the winter at reduced rates, and infection levels remain high. Prevalences above 50 per cent in early winter of the first year of exposure (Table 2) are above usual levels of infection for this period (Andrews, 1966).

Prevalences of Diseases in Gapers

All gapers were examined for occurrence of *Dermocystidium* and MSX. Cases of the fungus are shown by date of occurrence in Table 3 to demonstrate timing and magnitude of activity in trays of similar history but variable exposure. The only known modification of the ecosystem of tray populations was the addition of varying numbers of fungus-inoculated oysters in compartments 3, 5, and 7. Usually oysters do not die from *Dermocystidium* until "heavy" cases are developed. These appeared first in experimentals on 27 August and by 8 September heavy cases had been found in nearly all trays.

Diagnoses of gapers for *M. nelsoni* are given in Table 4. No attempt has been made to list cases by intensities because all levels of infection were found in gapers. MSX infections were first observed in gapers on 16 July and continued in abundance through October. Prior to September nearly all gapers had MSX infections but thereafter through October a significant number of gapers with negative diagnoses were observed. These negative gapers were mostly killed by *Dermocystidium* as the data in Table 3 indicate. There was no pattern of variation of MSX infections among trays or compartments. All lots became infected and exhibited deaths with similar timing and intensities. MSX was diagnosed in 95 of 126 gapers (76 per cent), hence it was obviously the dominant mortality agent in these groups of oysters.

It is difficult to assign precise figures for the number of oysters killed by each disease. An attempt to summarize the prevalence of *Dermocystidium* and MSX in gapers is given in Table 5. For *Dermocystidium*, prevalence of serious cases (heavy and moderate infections) was related to the number of inoculated oysters added. In each comparison, oysters in compartments and trays with infected oysters added showed a greater number of serious infections than controls. Tray A, with no infected oysters added, had 6 serious cases of fungus in 27 gapers. Trays B through D, with increasing numbers of infected oysters added in the odd-numbered compartments, had: 6 in 29, 12

TABLE 3. *Chronological record of thioglycollate tests for* Dermocystidium marinum *in gapers* [1], *proximity experiment, 1965.*

Tray	Compartment	16 July	30 July	13 August	17 August	27 August	8 September	20 September	23 September	8 October	20 October	16 December
A	1			N		N		3N		N	N	L
	2		N	2N	M*	4N	L* H	H	2N	3N	H M* L N	N
B	3		2N	N		N	H* M* N		H*	H* 2L**	M*	
	4	N	N	3N	2L**	2N	H* 3N			N L*	3N	
C	5	N	N			4N	H* N 2H*	H	3H	2H	4N	
	6		4N	N			N	3N	L*	2H L* N	H	
D	7[2]			M* 2N	L*	3H**	4H**	3H M*	3H*	2H M		
	8		2N	N	L* N			3H** 2L*	M* N	H* M*	5H*** L*	
Totals		2N	11N	1M 11N	1M 4L	12N	4H L 3N	9H M 4N	8H M 2L 8N	7H M L 5N	8H 2M 4L 2N	7H 2M 2L 9N L N

[1] Intensity of infections was rated heavy (H), moderate (M), light (L), or negative (N).

[2] A negative gaper on 18 June.

* Each asterisk designates a gaper infected with both MSX and *Dermocystidium*.

TABLE 4. *Diagnoses of* Minchinia[1] nelsoni *(MSX) in gapers*[1], *proximity experiment, 1965.*

Tray	Compartment	18 June	16 July	30 July	13 August	17 August	27 August	8 September	20 September	23 September	8 October	20 October	16 December	31 December	Totals	Per cent MSX by trays
A																
	1				P		P		3P		P	P	N		7P N	82
	2				4P	5P*	P*	N	P 2N	2P		2P* N	P		16P 4N	
B																
	3			P N	P			3P**		P*	3P***	P*			10P N	93
	4		N	P	3P	4P**		4P*			P*	3P			16P N	
C																
	5		P	P		2P	2P*	P* N	N	3N	N				7P 6N	72
	6			2P	P				3P	P*	2P* 2N	4P			13P 2N	
D																
	7	N			3P*	P*	2P** N	2P** 2N	P* 3N	P* N	3N				10P 11N	62
	8			2P	P	2P*		N	3P*** 2N	2P*	2P**	4P**** 2N			16P 5N	
Totals		N	P N	7P N	14P	14P	6P N	10P	5N 11P 8N	7P 4N	9P 6N	15P 3N	P N		95P 31N	

[1] Positive or negative

* Each asterisk designates a gaper infected with both *M. nelsoni* and *Dermocystidium*

TABLE 5. *Summary of diagnoses of diseases in gapers from proximity trays.*

Tray	Compt.	Number of gapers	Cases of *Dermocystidium**				Cases of MSX
			H	M	L	N	
A	1	8			1	7	7
	2	19	4	2	2	11	16
B	3	12	3	2	3	4	10
	4	17	1		3	13	16
C	5	16	9			7	7
	6	21	3		2	16	13
D	7	20	14	3	1	2	10
	8	19	10	2	3	4	16
Totals		132	44	9	15	64	95

* Intensity ratings: heavy (H), moderate (M), light (L), negative (N).

in 37, and 29 in 39 cases, respectively (Table 5). In all trays the most cases of fungous disease occurred in the compartment with infected oysters added.

Figure 2 is a graphic presentation of the data in Table 5 by trays only. MSX exhibited high prevalences in all trays but particularly those with less fungus. *Dermocystidium,* especially serious cases, increased in proportion to the number of artificially-infected oysters added. Concurrent infections varied from about 10 to 50 per cent, more or less in proportion to the amount of fungus present.

Figure 3 shows the percentages of gapers for each date that had *Dermocystidium* and MSX infections. Number of gapers varied from 8 to 20 except the first and last dates which had only 2 (see Tables 3 and 4). The graph shows that MSX preceded the fungus in occurrence in gapers. In mid-August 28 consecutive gapers were positive for MSX although five of these also had *Dermocystidium* infections. After the fungus appeared, MSX prevalence in gapers dropped from 100 per cent to about two-thirds. Fungous infections peaked in August through October and most cases were advanced ones. All trays were grouped for this graph regardless of degree of exposure to infection. During its active period, *D. marinum* probably caused half of the oyster deaths in the combined populations.

Figure 4 depicts chronological occurrence of cases of MSX, *Dermocystidium,* and concurrent infections. The vertical time scale is approximate except for the first and last dates which are "closed in." Each symbol represents a gaper, hence a gross scan of the graph conveys the timing and the proportion of deaths by each pathogen. MSX appeared in mid-July and occurred in 6 to 15 gapers per examination in a rather steady pattern. *Dermocystidium* appeared in mid-August and was found in about 5 to 14 oysters in each examination. Most fungous infections were advanced and all except three light cases were associated with MSX infections. Nearly one-third (29 per cent) of all gapers had mixed infections and two-thirds of these involved serious fungous infections (closed triangles). Thirty-seven of the gapers tested had infections of both MSX and *Dermocystidium.* Over half of these (19) were in tray D which received the greatest number of artificially-infected oysters. Serious infections of the fungus (heavy and moderate) occurred in 25 of the 37 gapers with concurrent infections.

From the thioglycollate tests, 53 of 132 gapers (39 per cent) had serious infections of *D. marinum.* With high prevalences of both diseases, a fairly large number of mixed infections could be expected. All combinations of intensities of infections occurred but usually it can be deduced which pathogen was the probable killer. Many light infections of the fungus were barely established and can be discounted as contributing to morbidity. Consequently, after subtracting serious *Dermocystidium* infections, at least 56 per cent of all gapers were probably killed by MSX. The percentage would be considerably higher if tray D, which had a large proportion of the fungous infections, were not included.

DISCUSSION

An attempt to induce and monitor a localized epizootic of *Dermocystidium* in the open waters of the York River was surprisingly successful in

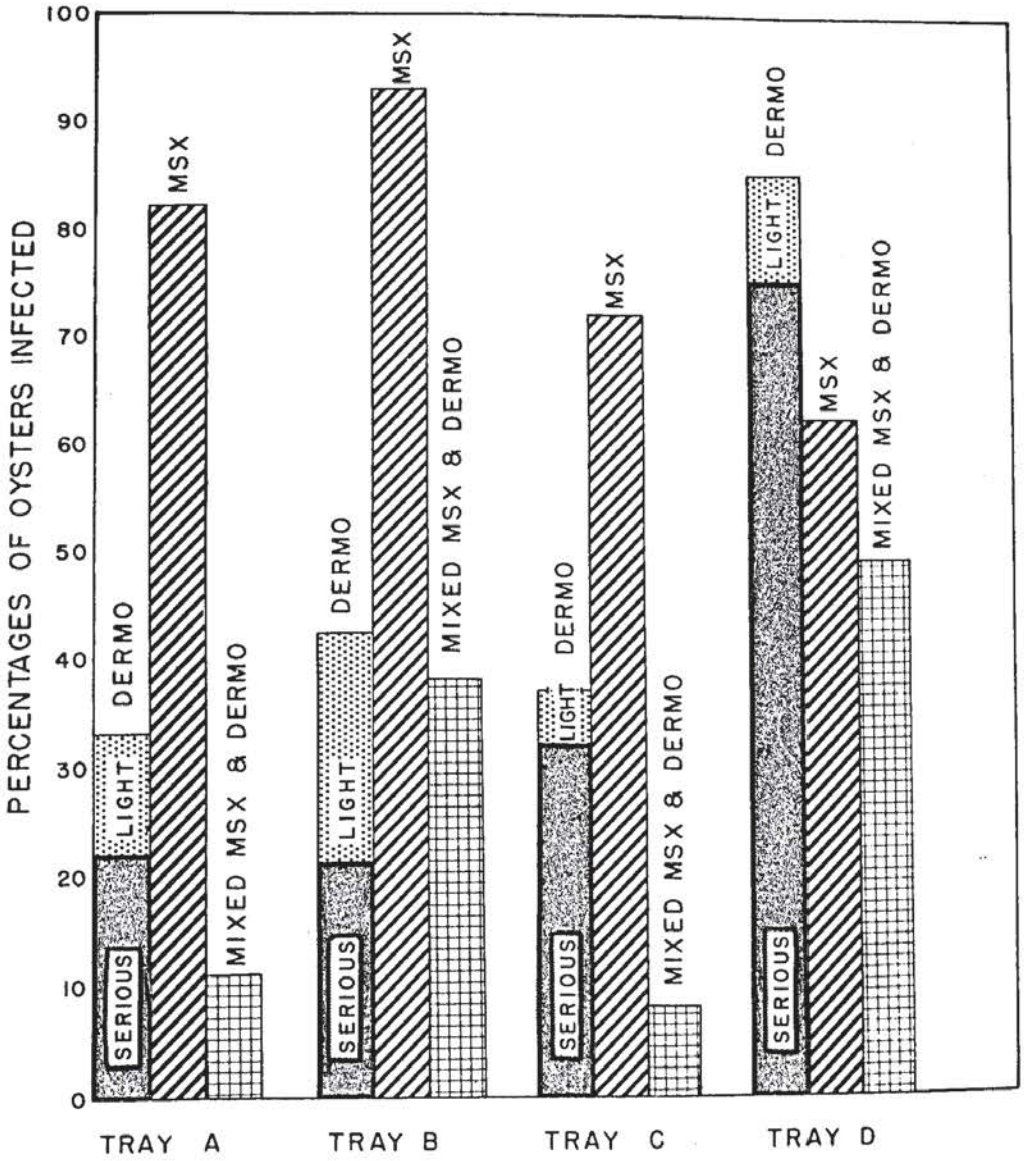

FIG. 2. *Comparison of* Dermocystidium *and MSX in gapers by trays. Concurrent infections are included in the percentages for each pathogen and also shown for each tray by separate bars.*

1965. Artificial infections have been induced many times in aquaria and in trays in open waters but limited circulation in the former and exposure to wild infections in the latter have complicated results. Since 1960, MSX has kept oyster populations severely reduced in the lower bay, hence *Dermocystidium* has been suppressed except in places such as VIMS pier where new populations have been imported regularly. Experience with trays of disease-free oysters on abandoned oyster grounds off VIMS has shown that from one to three years are required for an epizootic of the fungus to get started from natural sources.

The objectives were to initiate a localized epizootic which could be controlled in respect to timing and distances between various lots of oysters. One important but uncontrollable factor was continued epizootic activity of *M. nelsoni*. MSX appears unaffected by proximity of infected oysters hence it is assumed that all lots of oysters were affected about equally by this parasite. Although the data from individual samples or dates may not appear to justify this assumption, it seems to be verified by results of six-months' observations. Unfortunately, preoccupation with other work left three periods unsampled which

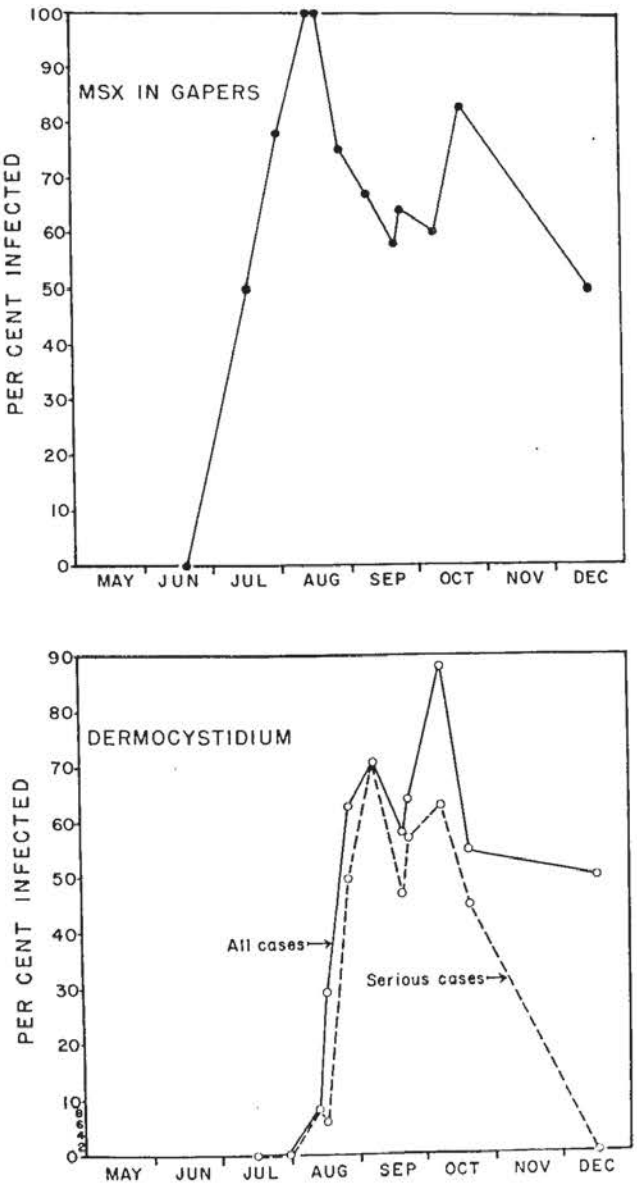

FIG. 3. *Percentage occurrence of MSX and* Dermocystidium *in gapers from all four trays. Number of gapers tested per date varied from 8 to 20 oysters.*

would have clarified the progress of the epizootics. Perhaps hindsight is involved, too, in the conclusion that samples should have been taken in mid-July, mid-September, and mid-November.

Experimental oysters were imported in late May, which is believed to be the beginning of the infection period for MSX. Six weeks later in mid-July, mortalities had begun and MSX-infected gapers were recovered. Both timing and prevalence of infections continued typically throughout the rest of the year. By mid-August prevalence reached levels of about 50 per cent which were maintained throughout the experiment, despite 60 per cent mortality. This straggling in the clinical appearance of MSX is characteristic, although all late-summer and fall cases can be traced to infections initiated in about a six-week period in June and early July.

The laboratory infections of *Dermocystidium*, induced in mid-June, began to kill marked oysters about one month later in mid-July. This is about typical of natural infections (over-wintering cases) which become apparent in late June and kill in July, thereby initiating a second round of infections. By mid-August, disintegrating marked gapers had initiated infections in 75 per cent of the experimental oysters in compartment 7. This rapid rise of *Dermocystidium* prevalence is noted also in compartments 3 and 5 between 17 August and 8 October. By 8 October it had spread to other compartments and trays including the control tray A in the middle of the cluster. The rapid climb in prevalence of the fungus at warm temperatures (about 25°C) is countered by an equally precipitous decline, usually in December. *Dermocystidium* spreads rapidly in crowded populations, once infected gapers begin dying. Even one gaper in a tray (only one of the two laboratory-infected oysters added to compartment 3 died by 17 August and the other much later) can initiate an epizootic.

It is presumed that the four clustered trays exhibited a self-contained epizootic of the fungous disease. This cannot be verified except by comparison with other trays introduced in the same year. Examples are trays Y22 and Y23, comparable to the proximity series except they were placed farther offshore of VIMS and at least 100 feet from other trays. Y22 had one light case in 25 live oysters on 20 October 1965 and Y23 had none (Fig. 1). One of 65 gapers collected from these two trays in 1965 had a light case of *Dermocystidium*. Furthermore, in 1963 natural infections were rare in a similar experiment at the same location (Andrews, 1965).

It is apparent from Table 2 that 15 feet was not an adequate distance to prevent infections being transmitted from one tray to another. Yet significant differences in prevalences were obtained in opposite ends of the same tray with only a pair of partition boards to prevent direct flow of water. The control tray in the center obtained as many infections as compartments 4 and 6 in trays with infected oysters added.

Interaction of Two Pathogens

It is impossible to integrate the many factors which contribute to deaths of oysters. Mackin and Sparks (1962) list over nine agents which cause

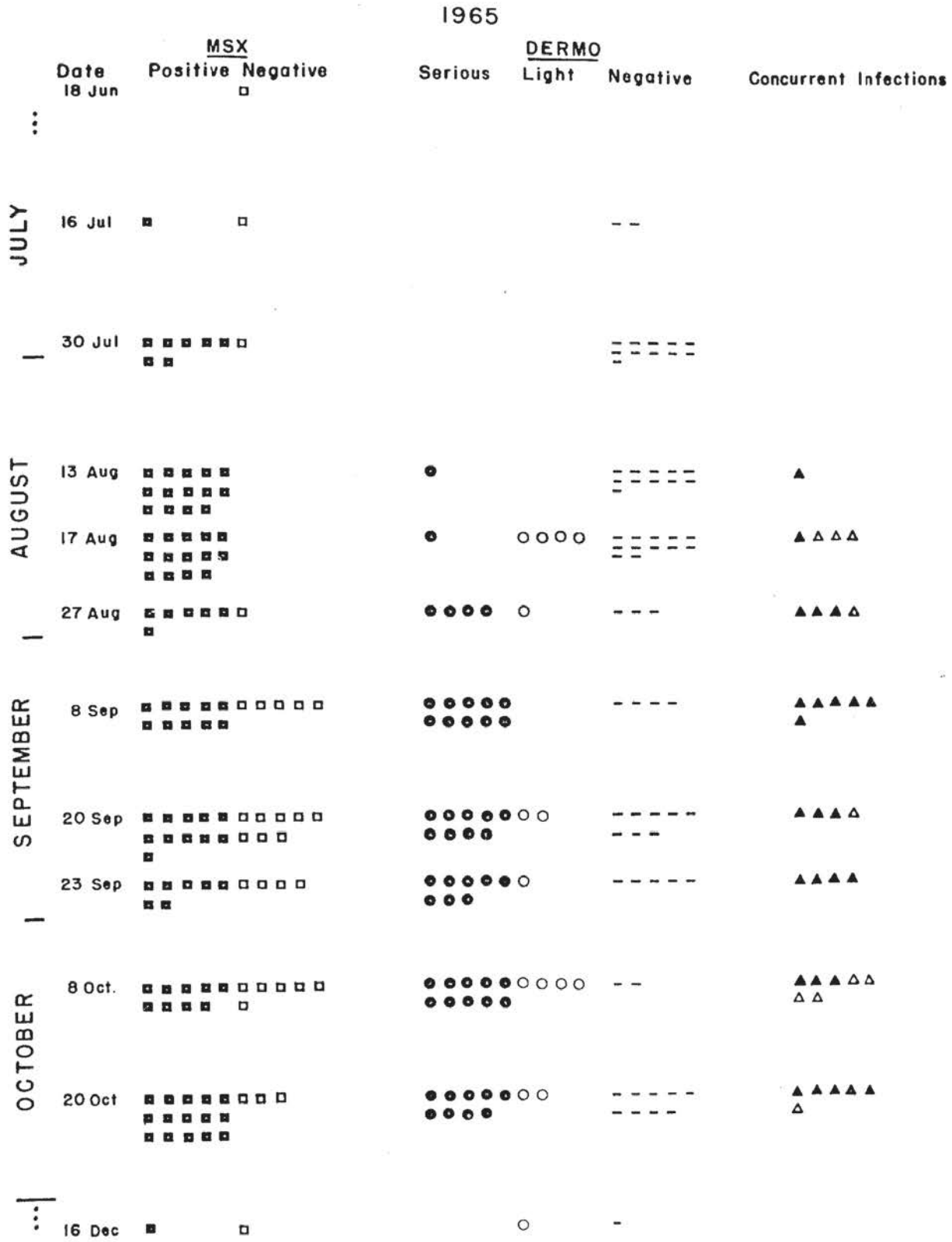

FIG. 4. *Occurrence of MSX and* Dermocystidium *infections in gapers. Each symbol represents one gaper. In concurrent infections, closed triangles represent serious fungous infections and open triangles light ones.*

oyster mortalities in Louisiana. Of those present in Virginia, only *Dermocystidium* is considered important in my studies. *Bucephalus* and *Polydora* species are minor agents compared to some undescribed pathogens, and *Odostomia* is not considered to be a cause of mortality. Blue crabs and mud crabs may kill spat occasionally but usually are scavengers. Boring sponges seem to play accessory roles to other agents in old oysters, including accidental breakage of shells. Predators were excluded and only diseases and parasites had free access to experimental oysters.

MSX and *Dermocystidium* were obviously the major causes of death, yet a few gapers exhibited neither parasite. A rough approximation of the roles of the two pathogens can be obtained in Table 3 by chronologically comparing the number of gapers diagnosed by intensity of infection as light or negative (probably killed by MSX) with those designated moderate and heavy (probably died from *Dermocystidium*). MSX predominated prior to about 1 September, whereas the two pathogens were about equally destructive from that time through October. Of 37 mixed infections, 25 had serious cases of the fungus, implying that *Dermocystidium* is quicker to kill when both pathogens have become established. In addition, MSX had nearly two months head start over the fungus in terms of exposure of experimentals to infection. Since both MSX and *Dermocystidium* may inhibit growth for two or more weeks prior to death (Andrews, 1963), some reduction of each disease can be expected from the presence of the other. If an oyster does not feed, it is unlikely to contract another disease, whereas if both diseases are already present, a synergistic lethal effect may occur. Mortality data imply that, except for compartment 7, exposure to *Dermocystidium* did not appreciably increase accumulative mortalities. Tray Y22 without the fungus had 60 per cent mortality from MSX (Andrews and Wood, 1967) which is about comparable to losses shown in Table 1.

Both pathogens are highly lethal to susceptible oysters given appropriate environmental conditions. It is extremely important therefore to know the epizootiology of both diseases as to seasonality of infections, duration of incubation periods, susceptibility of oyster populations, and inhibiting factors. With this knowledge, time and area of planting and harvesting, and choice of seed sources can be regulated to minimize losses. The interactions of two diseases in nature, one of which can be controlled by isolation whereas the other cannot, are important in management procedures. This experiment has provided some insight of these interactions.

One must conclude that MSX, within its range, is by far the most serious disease of oysters in Chesapeake Bay. Its ubiquity in most Virginia waters and tolerance of wide temperature range leaves little room for manipulation of oyster populations. Mackin and Sparks (1962) concluded that *Dermocystidium* overshadowed all other agents combined in Louisiana. Ten years ago, the fungus was the major agent of mortality in Virginia (Andrews and Hewatt, 1957).

Dermocystidium can be controlled by avoiding infested seed and by cleaning and fallowing oyster beds. It is subject to fairly rigid temperature controls. In short, being an infectious disease directly transmitted, it is amenable to manipulation of populations and planting grounds. The major purpose of this study was to demonstrate the importance of controlling or eliminating foci of infection for *Dermocystidium*. There is probably no "safe" distance for effective isolation but distances of 15 to 100 feet are useful in slowing the spread of epizootics. Probably the size of a bed of oysters is important in commercial operations in respect to distances needed for limiting infestations.

ACKNOWLEDGMENTS

The slides for diagnoses of MSX occurrence were prepared by the Microtechnique Section of the Microbiology Department of VIMS under the supervision of Dr. J. L. Wood. Grateful appreciation for this essential help is acknowledged.

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