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Status Of Researches On Oyster Diseases In North America

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NEEDLER (1931) FIRST CALLED ATTENTION to disease of oysters and the related mortalities in Malpeque Bay, Prince Edward Island. This disease has come to be known as Malpeque Bay disease (Logie 1958). Although studied over a long period, less is known about Malpeque Bay disease and its effects than is known about any of several diseases studied much later. Prytherch (1938, 1940) believed that *Nematopsis ostrearum*, a gregarine described by him, was highly pathogenic to oysters and that mortalities in Louisiana and Mobjack Bay, Virginia, were caused by this protozoan. Prytherch's belief in the pathogenicity of *Nematopsis* has not been corroborated by later studies, but his work served to focus attention on the highly important field of disease researches. Mackin, Owen, and Collier (1950) described *Dermocystidium marinum* and initiated a long series of studies of this pathogen. Andrews and Hewatt (1957), working at the Virginia Fisheries Laboratory, made significant contributions to the study of this parasite in the period from about 1952 to the present. Ray (1954) working at Rice institute and at the Grand Isle Laboratory, contributed his diagnostic technique and other outstanding researches.

Mackin, Korringa and Hopkins (1952) suggested that Hexamita might be a factor in epidemics in America, and Stein, Denison and Mackin (1960) experimentally infected *Ostrea lurida* with Hexamita (and perhaps other disease-producing organisms) but were unable to do the same with *Crassostrea gigas*. Davis and Loosanoff (1955) described a fungus disease of oyster larvae and Mackin and Loesch (1955) reported on a Haplosporidian parasite of Bucephalus in oysters. In 1957, Haskins and associates began studies of mortalities due to disease in Delaware Bay and were soon joined by a number of other workers on the middle Atlantic coast. At this time, diseases associated with these mortalities are being studied closely. Mackin (1960) reported on "Mycelial disease" of oysters on the Gulf Coast, and added data on several other new parasites and diseases of problematical nature.

It is the purpose of this paper to discuss some of the parasites and diseases mentioned above with the intention to point out progress of researches and problems solved and to be solved. The latter are in the majority. No attempt is made to exhaust the field, but only to discuss those features of most importance at this stage of the studies.

Malpeque Bay Disease

Needler (1931, 1941), Needler and Logie (1947) described a disease which struck oysters in bays of Prince Edward Island, Canada, in about 1915. These were pioneering studies of disease of oysters in America. Although these authors and other Canadian workers have never solved the problem of etiology their work is nevertheless distinguished because of the demonstration that disease-restistant strains of oysters may be developed through exposure to disease. According to Needler and Logie the advent of disease coincided first with the advent of oyster farming and second with imports of oysters from the United States. Planted oysters died over two or three years time and cumulative mortalities reached ninety per cent. Oysters affected were thin and emaciated and sometimes had yellowish pustules and abscesses. Epidemics appeared successively in various bays of Prince Edward Island, but required a period of many years to affect all populations of the Island. Recovery in Malpeque Bay, the original focus of disease, required about thirteen years. Although local stocks apparently became largely immune to attack after that time, the introduction of foreign stock from time to time showed that the disease was still endemic and is still so today (Logie 1958).

Logie (1958) described extensive experiments testing for development of resistance of oysters to Malpeque Bay disease. These indicated that the development of resistant strains occurred under stress of natural selection. This was a noteworthy achievement and points the way to the most promising of the methods in controlling other diseases of oysters. But if the Prince Edward Island experiments are any indication, this will be a very slow and painful method of rehabilitation.

Certain epidemiological data presented by Logie (1958) indicate that Malpeque Bay disease may in fact be two diseases or possibly even more. Separation of mortality peaks into winter-spring and late summer peaks may mean that there are two corresponding diseases. Resolution of the problem of etiology in the case of the Canadian diseases becomes most important.

Needler (1941) showed that prior to the onset of disease, said to have begun in about 1915 in Malpeque Bay, the oyster industry had already undergone severe reduction. The year 1882 showed a peak production of more than 50,000 barrels of oysters; the production was cut in half the following year, and never again attained the peak production. By 1894 production was down to less than 20,000 barrels. By 1915, when Malpeque Bay disease was said to appear, the oyster industry was down to about 4000 barrels, or about 8 per cent of peak production. By 1918 the industry in Malpeque Bay disappeared, presumably because of disease. Needler thinks that the decline prior to 1915 was due to overfishing of natural reefs. After decimation of these natural reefs the oystermen turned to oyster farming and introduced the disease with seed. However, the data do not indicate that the disease was a new introduction. Here are the reasons why this is so:

(1) The natural reefs, said to have been decimated by over-fishing, were abandoned one by one over the years. They did not rebound when abandoned, which they would have if they had been healthy reefs.

(2) The sparking of the epidemic in Malpeque Bay could just as logically have been due to infection of highly susceptible imports with a local disease as to infection of local oysters by an imported disease.

(3) In fact the description of the beginning of the epidemics by Needler and Logie (1947) suggests that it was the imports and not the locals which were first decimated by disease. They stated that "In 1915 large numbers of oysters were found dead or dying in the central part of Malpeque Bay, in the area where the imported oysters had been planted. In 1916 the mortality spread throughout the bay and its immediate tributaries." The imports were, therefore, highly susceptible to the disease and were wiped out first. It appears odd that imports bringing in a disease from an endemic area should themselves be victims of a disease to which they should have been largely immune. This violates all concepts of epidemiology. The imports should have survived to become the back bone of a new industry. But they did not. They may have survived just long enough to cross breed with the natives, thus producing new crops of susceptibles. The thirteen years which elapsed before the industry revived could have been the time required to eliminate the introduced susceptible strain of oysters. This does not invalidate the concept of the development of resistant strains as described by Logie (1958). Indeed it makes the case stronger.

It should be observed also that severe epidemic disease followed the introduction of the practice of making concentrated plantings in small areas. It is the opinion of the author that this one factor has made those areas where crowded planting is the rule the areas of most intense development of oyster disease (Louisiana and Virginia). This is a concept which should be closely studied: Has modern planting method itself precipitated the most acute crisis in oyster production ever observed?

***Mortalities associated with* Haplosporidium spp.**

Excessive loss of oysters in Delaware Bay became apparent in the spring of 1957 (Haskins, 1959). In a six week period in April and May, 35 per cent to 85 per cent of planted oysters died, the number depending on location and origin of the oysters in Delaware Bay on the New Jersey side. Losses continued in 1958, 1959, and 1960. In each year the mortalities were in two periods, the first in the late winter and early spring, the second in late summer and fall. Mortalities extended to the seed bed area in the fall of 1958 (Haskins 1960). Generally speaking, Haskins' reports indicate that, so far as disease is concerned, the spring mortality period is about as severe as is the summer-fall period.

Seiling studied mortalities in Chincoteague Bay (see report by Fred Seiling in Haskins, 1960, 1960a). This bay has a history of recurrent mortalities going back to the mid-forties, and perhaps earlier. Seiling reported mortalities at nine stations in Chincoteague Bay and on Virginia seaside in the period from March, 1959, to January, 1960. The average for native oysters was 27.6 per cent (tray oysters) and for "control" imports was 7.5 per cent. Peak mortality at all nine stations was in the May-June period, but there was also a September-October peak at several stations. Andrews and Wood (in Haskins, 1960) also reported that appreciable mortality occurred on the Virginia seaside in the May-June period, but not in the summer-fall period. Andrews (personal communication) reported that the seaside May-June mortality was repeated in 1960, with identical timing with the 1959 losses.

Haskins (1959) reported the finding in diseased oysters of a new organism, termed in manuscript reports the "MSX" organism. This was determined by Mackin to be one of the Haplosporidia, probably genus *Haplosporidium*, (see Haskins 1960). This organism has been consistently reported to be associated with the summer-fall mortalities in Delaware Bay, and appears to be present

in the spring period of mortality but in reduced incidence. Haskins (1959b, fig. 2) showed that weighted incidence of this organism in tray oysters (Cape May area) rose steadily from July to November, 1959, thus correlating with the summer-fall mortalities, but the same diagram showed that it had almost disappeared by April of 1959. These data were also presented by Canzonier (in Haskins 1959a) in table form. Haskins (1959b, 1960) showed that the mortalities continued into 1959 and 1960, with about the same pattern of peaks. If the data on weighted incidence have continued in the same pattern in 1959 and 1960 as was established in 1958 the spring mortalities have a different etiology from that of the summer-fall peaks. A careful study of all distributed data shows that exact calculations of the incidences of the "MSX" organism have not been presented by Haskins and associates in the critical period of the spring mortalities (about late April to late June), or for the earlier winter-spring mortalities. Haskins (1959b) believes that the winter-spring mortalities result from infections picked up in the preceding fall which so weaken the oysters that they die under stress of winter hibernation. There appears to be a better alternative explanation, namely that "MSX" is not responsible for the winter-spring mortalities but that some other pathogen is responsible. This could be any of several known to be in the area, (excepting *Dermocystidium marinum*).

"MSX" organism is in the Chesapeake Bay area where it is now spreading slowly (Wood in Haskins, 1960; Andrews, Wood and Hoese 1960). The organism appears to be in fairly heavy concentration in the Mobjack Bay-lower Chesapeake Bay area, where heavy losses have occurred in the past, and where steady increase in mortality has occurred in 1960. "MSX" appeared in heavy concentration after June, 1960. Hampton Roads also had heavy losses in the summer of 1960.

The consistent mortalities of oysters in seaside bays concentrated in the May-June period led to studies of epidemiology and the parasites concerned. The Maryland-Virginia seaside mortalities differed from those of the Delaware Bay Cove in that spring peaks stood alone and were not followed by the summer-fall peaks (see Andrews and Seiling, in Haskins, 1960). A species of Haplosporidium, similar to the "MSX" is associated with these seaside mortalities. Haskins and Stauber believe that this Halosporidium is "MSX" but it differs in that spore stages are prominent in sections, whereas in "MSX" spore stages may not have been observed at all. Andrews and Wood (1960) believe that this Halosporidium is a different species from "MSX." The author concurs in this opinion, which is based on observed differences in the parasite and on the epidemiological differences which have been worked out by Drs. Andrews and Wood. Thus there may be two species of Haplosporidium, overlapping ecologically, in the Mid-Atlantic area, one concentrated in the lower reaches of the large estuaries, the other in the smaller sea-side bays behind the barrier islands.

Additionally, Mackin (1960) reported an organism causing "amber disease" in Louisiana oysters which may be related to the Haplosporidia, and there is another species in Louisiana associated with short term spring mortalities which perhaps should be included in the genus Haplosporidium. The Haplosporidian parasites appear to form a group of considerable importance in oyster mortality and within the next ten years it is believed that the group will be studied intensively.

Haskins (1959b) assumed that "MSX" was new to Delaware Bay, basing his belief on the fact that his studies showed that the parasite apparently

appeared first in a central area of the Delaware Bay Cove, and spread from there to outlying areas. This is what would have happened if the parasite developed a cyclic epidemic wave after a period of subsidence, and may not constitute evidence of a new introduction. A study of oyster production in Delaware Bay shows that there were earlier cyclic declines in oyster populations. Indeed, the present decline appears to have begun in about 1954 rather than in 1957. It forced attention in 1957 but, unless one mortality-producing agent suddenly dropped out of sight and another took its place in 1957, a most unlikely event, "MSX" and/or other pathogens, began the present epidemic wave in 1954. If this parasite was new in 1957 the pathogen suddenly displayed a tremendously accelerated capacity for dispersal, since it is now known to exist in the lower Chesapeake and in various sea-side bays up at least as far as the Great Bay area. One would have to assume that (1) the "new" organism spread from the Delaware Bay Cove to all these outlying areas in a few years time, or (2) it was present in these other areas in the past, but never penetrated to Delaware Bay until the mid 1950s, a concept difficult to believe when one considers (a) all the multiple opportunities for distribution of a pathogen in Atlantic coastal waters, and (b) the history of variable production, and overall declining production in Delaware Bay.

The studies of Pixell-Goodrich (1915) on *Haplosporidium chitonis* are pertinent in this matter. Lankester reported the presence of *H. chitonis* in English waters in the mid 1890s; Pixell-Goodrich, beginning about 10 years later, searched for the parasite for four or five years before finding it. When found it was abundant in an area near the Plymouth Laboratory. This suggests that there are periods of cyclic abundance in these parasites, alternating with periods of scarcity.

Infection experiments with "MSX" (Scheltema, in Haskins, 1960) have not been successful because *Dermocystidium marinum* invaded the control and experimental tanks and produced such severe mortality that interpretation of results was complicated. Andrews and Wood (personal communication) have tried to infect oysters with "MSX" but results of these studies have not been reported to date.

Hexamitiasis

Certes (1882) recorded *Hexamita inflata* from the digestive tract of oysters, *O. edulis,* and noted that the flagellates were true parasites since they reproduced in the host. Orton (1924) recorded flagellates which probably were Hexamita from diseased oysters in England. Richardson (1939) recorded Hexamita from Canadian oysters, *Crassostrea virginica.* The latter author associated Hexamita and a ciliate with mortality in a few oysters. He used the generic name Urophagus interchangeably with Hexamita. Mackin, Korringa and Hopkins (1952) named Hexamita as the etiological agent in "pit disease" of Dutch oysters and called attention to intracellular stages. Stein, Denison, and Mackin (1960) showed experimentally that *Ostrea lurida,* dying with heavy Hexamita infections, could be used to transmit lethal disease to uninfected oysters; but that *Crassostrea gigas* was not affected. It was uncertain in these experiments whether or not the actual etiologic agent of disease was Hexamita, or whether there was an association of Hexamita with some other disease agent which latter was the actual lethal agent. At the present time the most pressing problem related to Hexamita is to resolve this question. There are three schools of thought so far as Hexamitiasis is concerned. Tenets of these

are as follows. (1) Hexamita is a saprophyte or mild parasite, and the association of the flagellate with mortalities of oysters is due to the universal distribution of the flagellates. They are always ready to attack dying oysters in the late winter or spring. The intracellular stages, according to this theory, are phagocytized leucocytes. (2) Hexamita is a parasite but is not of itself sufficiently pathogenic to destroy oysters. It may finish off weakened oysters; weakness is assumed to derive from cold or other adverse chemical or physical conditions, or from some other disease. (3) Hexamita is a highly pathogenic parasite. It can of itself kill oysters, and extensive mortality is attributed to it. The intracellular cells are developmental stages, and the disease attacks oysters when they are alive and in excellent condition.

Each of these positions has supporting data. In support of the theory that the Hexamita of oysters is a saprophyte is the fact that trophozoites do not ordinarily appear in oysters until they are dead or moribund. Smears of stomach contents or gill of apparently healthy oysters may show light infections with trophozoites but rarely are they found in numbers. The supposition that "intracellular stages" are phagocytized leucocytes rests solely on a resemblance of some intracellular stages to leucocytes.

The concept that Hexamita is a weak parasite, and is effective in destruction of oysters when host restistance is reduced by adverse environmental agencies, rests on somewhat firmer ground. Mortalities laid to Hexamita predominantly follow cold winters, occurring when oysters are subjected to low temperature sufficient to affect ciliary activity. In the case of *Hexamita salmonis* attacking trout, young trout in hatcheries are particularly subject to mortalities, especially after handling, and after transportation in confining containers. Oysters in holding basins in Holland, when artificial low temperatures were maintained, and where the water was recirculated, had heavy mortality not equalled, so far as known, on bedding grounds in the same general area. Experimentation on the effect of Hexamitiasis on *Ostrea lurida* showed that excessive mortality occurred only when the temperature in aquaria was held below 10°C. The dikes in which *O. lurida* are grown in the South Sound area in the state of Washington, and where "Hexamita" epidemics occur, depend for their usefulness on concentration of oysters in very large numbers in very small areas. This overcrowding appears to be a prime factor in development of epidemics in that area.

Those data supporting the concept that Hexamita is a highly pathogenic organism may be summarized as follows:

a) The fact that Hexamita is associated with extensive mortalities of oysters is itself the strongest evidence of pathogenicity. The association is not local: the syndrome accompanying the mortalities, which includes the production of trophozoites as an end phase, occurs in Australia, Holland, the Pacific Northwest, the Atlantic East Coast, and rarely, the Gulf Coast of the United States.

b) Several other species of Hexamita are considered by principal researchers to be pathogenic (McNeil et al, 1941; Moore, 1923, 1923a; Davis, 1926). McNeil et al produced disease by transmitting the parasites. In fact, the group is basically parasitic, and the "freeliving" forms are actually the questionable ones. There is evidence that the trophozoite may be the motile distibuting stage of a life cycle in which tissue-infection cells make up the effective parasitic stage.

c) The intracellular stages seen in oyster tissues, epithelium of the gut, Leidig cell tissue, gonad, and other organs of oysters are developmental stages

of Hexamita. The resemblance of some of these stages to oyster leucocytes is superficial and most developmental stages bear little resemblance to leucocytes. It is admitted that these stages may be another organism, but if it is then this other organism is quite definitely tied to Hexamita, not only in the Hexamita of oysters, but in the Hexamita of trout *(H. salmonis,* Moore) as well. It also appears wherever in the world that the trophozoites appear. The close resemblance of the aflagellate stages in the oyster to the developmental cycle as worked out by Moore (1923a); and Davis (1923, 1924, 1926) for *H. salmonis* is remarkable and extends to basic cytological features in mitosis. It is known (Swezy, 1915) that *Hexamita nodulosa* of amphibians also develops multinucleate stages closely resembling the multinucleate stages of certain so-called Haplosporidians and to those in oysters associated with Hexamita.

d) Experiments by Stein, Denison, and Mackin (in press) transmitted intracellular and tissue infecting stages along with the Hexamita trophozoites. It is obvious that a disease was transmitted which was accompanied by a complete syndrome of the Hexamita cycle as worked out by Moore and Davis. It remains to be shown that some obscure disease, rather than Hexamita, was the lethal factor.

e) Extensive field studies in Puget Sound have shown that live, apparently healthy oysters show all stages of development of disease, including the end stage, the trophozoite.

f) Cold of itself cannot be the cause of mortality in those cases where mortalities are associated with both cold and Hexamita. Roughley (1926) showed that the disease was not actually tied up with extreme cold, when studying the Australian "Winter Disease." Those oysters subjected to most intense cold failed to die, while those subjected to slightly higher temperatures died. The author has studied oysters from Australia dying of Winter Disease and they have the same Hexamita syndrome as do American and Dutch oysters, Stein et al (1960) showed that controls subjected to the same low temperatures as were experimental oysters failed to die in comparable numbers. *Crassostrea gigas,* subjected to both cold and Hexamita infection, failed to die. Conversely, it is apparent that Hexamitiasis is a winter-spring disease just as *Dermocystidium marinum* is adapted to high temperature. Most disease-producing organisms are environmentally controlled to some extent as are free living organisms.

These differing view points all have merit. But it is obvious that those most closely associated with experimental studies favor the thesis that Hexamitiasis is a winter-spring disease of *Ostrea spp,* but it has not been demonstrated that *Crassostrea spp.* are also affected.

Sheltema (see Haskins 1960b) inoculated Hexamita into oysters held at low and high temperatures and failed to get a mortality difference between the controls and the experimental oysters. It was not stated how the controls were prevented from becoming infected or indeed whether they were or not. It was stated in the report by Haskins that Scheltema failed to confirm the works of Stein, Denison, and Macklin (1960). Since he was not repeating experiments of these authors (he used a different host and completely different methods) it is not clear in what respects he failed to confirm work of these authors.

Dermocystidium marinum

D. marinum was described by Mackin, Owen, and Collier (1950). Since that time extensive and intensive studies of this organism have been carried out.

Mackin (1951) studied histopathologic effects and suggested methodologies of study in that field. Since the appearance of the original description of the disease and its causative organism, many studies of distribution, epidemiology and pathogenicity have been carried out. The disease caused by *D. marinum* affects oysters from Delaware Bay (Anonymous, 1958) to Mexico (Mackin, 1960a). The range is not continuous, since certain areas appear to be free of the parasite (Virginia seaside). Areas such as Aransas Bay, Texas, appear to have an increasing incidence (Hofstetter and Hefferman, 1959). In the more northerly part of the range, *D. marinum* disappears from the oyster populations in winter, but reappears in the summer to produce epidemics. This indicates that an overwintering stage (the hypnospore) exists. In the Gulf States, temperatures may not be low enough to eliminate the parasite, and in mild winters considerable mortality may result from epidemics. The principal effect of mild winters is to insure severe epidemics the following summer and fall. Low salinities and effective flushing of estuaries tend to decrease incidence of the fungus.

Diagnosis of the disease has been simplified through the studies of Ray (1954), making possible extensive studies of distribution, development of epidemics, and pathogenicity. Experimental studies by Mackin, Ray, and Andrews and Hewatt (See Mackin, 1960a) have established that *D. marinum* is highly pathogenic, and the disease caused by this fungus is a major threat to oyster production within its range. In the Virginia-Chesapeake Bay area (Andrews and Hewatt, 1957) a summer epidemic may destroy fifty percent of oyster populations. In Louisiana (Mackin, 1960a) oyster plantings may be virtually destroyed in a single summer.

Other Diseases

Mackin (1960) reported on several parasites and diseases not heretofore known. At the present time none of these seem to be major causes of mortality, but this may be because they have not been tested in the ecological areas best suited to them. The "mycelial" disease for example is now known to be widely distributed in the Gulf, Atlantic, and Pacific Northwest. There are indications that it may at times be destructive in Louisiana and Texas. The causative organism of "amber disease" appears to be rare, but may be rare in the Louisiana area where it was first observed, but common in some as yet unknown area where ecological conditions favor the parasite. The author has observed a species of *Dermocystidium* in Australian oysters, and a plasmodial parasite in *Crassostrea gigas* from Japan. These parasites must sooner or later be studied in more detail.

Problems of Disease

The pressing problems confronting students of oyster diseases are many and difficult. There are no short term solutions for these problems. The major ones are as follows:

(1) Before serious work can be undertaken, the kinds and relationships of the organisms parasitizing oysters must be resolved. It is unfortunate that most parasites causing disease in oysters seem to be aberrant types and belong to little-known groups of fungi, *(D. marinum),* protozoa, ("MSX" and related species), or bacteria, ("Mycelial disease"). Failure to properly solve the problems of etiology is a solid hindrance to further studies. We are much too prone to believe that basic taxonomic studies can be relegated to the background while

studies of "practical" nature are pursued. This belief has delayed the solution of some problems of Malpeque Bay disease for more than thirty years.

(2) Solution of the problems of epidemiology is the essence of studies of disease. This means the study of those ecological conditions which augment disease or control its incidence, hosts and reservoir hosts, transmission agencies, parasite physiology, distribution, form of epidemics and many others. Management of oyster crops depends upon accumulation of data of this kind in sufficient amount.

(3) Studies of life cycles of parasites probably should be included as a part of epidemiology. But the obvious paramount importance of these studies justifies singling them out. If counter measures against disease become possible, it will be because there are "weak points" in the parasite life cycle which make it vulnerable to artificial control.

(4) The search for races of oysters resistant to disease, and the management of oyster plantings to further development of resistant populations is at least one area in which progress may be made in spite of lack of specific basic knowledge in taxonomy and life cycles (example: Malpeque Bay disease). It is evident that development of resistant races for specific disease has more chance of successful accomplishment than any other control method. Here also is a type of study in which collaboration of planters and scientists in the studies is feasible and probably necessary.

(5) It is predicted that in the near future highly artificial methods of oyster production must replace the bulk methods now in use in the United States. Control of disease may be possible under artificial conditions, such as pond and tray culture, while it may not be possible under the planting methods now used. Part of the increasing toll of disease may be due to heavily crowded plantings, constant reintroduction of large numbers of susceptible oysters to areas of endemic disease, and repeated reintroduction of disease by exotic oyster populations. In anticipation of the day when the industry will be dependent on the development of artificial culture methods, researches in this field should be pressed even stronger than is now the case. Tray culture is not new, and neither is pond culture, but all too little is known of the problems involved.

(6) Studies of natural biological controls for disease may possibly produce results of value. It is believed that this is a rather slender thread considering that its application would depend on finding natural controls existing in some unexplored part of the world. Since most parts of the world are totally unexplored for the disease-producing entities found in American waters, it would involve much spade work.

(7) Chemical control is considered much too risky to be attempted. Even if chemical control, selective of the parasite could be found in laboratory studies, large scale treatment of estuarial waters is not advised because all factors of the environment may not be explored in the laboratory. It is believed that chemical control is on its way to reduced use in agriculture because of hazards of modern highly toxic compounds.

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Delaware Bay Oyster Mortalities

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Abstract

In approximately six weeks in the spring of 1957 over 50 per cent of the oysters planted on leased grounds in Delaware Bay died in an epizootic. The kill was most intense in the center of the leased grounds and decreased in intensity with distance from this center. No unusual mortalities were detected in the eastern planted areas nor on the seed beds in the Upper Bay. By fall of 1958 oysters were dying in the Upper Bay seed beds and in all areas on the planted grounds. Seed oysters planted in the spring of 1958 had an average mortality of 52 per cent by that same fall. A year later the minimal kill of this population was 80 per cent.

A new sporozoan has been identified as the causative agent in this epizootic kill. Experimental studies of several imported stocks have shown differences in susceptibility to the pathogen and have demonstrated the seasonal patterns of kill.

Rehabilitation of Disease-Depleted Oyster Populations in Eastern Canada

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INTRODUCTION

IN EASTERN CANADA, oysters, *Crassostrea virginica*, Gmelin, occur in those portions of the provinces of Prince Edward Island, New Brunswick and Nova Scotia bordering the southwestern Gulf of St. Lawrence.

In 1915 oysters started dying in large numbers in Malpeque Bay, Prince Edward Island. Mortalities fanned out from an apparent epidemic centre and soon involved Cascumpeque and Bedeque Bays. By 1920 the fishery of these waters was prostrate. Subsequent investigation, ten or more years after the subsidence of spectacular mortality, suggested:

(1) that in infectious, epidemic disease was involved;

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