

Northeastern Regional

Aquaculture Center

University of
Massachusetts Dartmouth
North Dartmouth
Massachusetts 02747

History and Impact of MSX and Dermo Diseases on Oyster Stocks In the Northeast Region

John W. Ewart, University of Delaware Sea Grant Marine Advisory Service

Susan E. Ford, Haskin Shellfish Research Laboratory, Rutgers University

Introduction

Diseases have been a recognized problem in the culture of the eastern oyster, *Crassostrea virginica*, since 1915 when the first reported epidemic or “epizootic” mortality of oysters occurred in Prince Edward Island, Canada. The outbreak, now referred to as “Malpeque Bay disease” remains a mystery to scientists who have been unable to identify a cause for the epizootic.

Two other infectious oyster diseases, however, commonly known to oyster growers as “MSX” and “Dermo”, have had a much more significant and chronic effect on oyster populations in the northeastern region of the United States. The combined impact of both diseases has caused hundreds of millions of dollars in losses over the last 35–45 years in mid-Atlantic states (especially Chesapeake and Delaware Bays) and seriously threatens both natural and cultivated oyster populations in the New England states. Although both parasites are lethal to eastern oysters, they are not harmful to humans. This publication provides a current overview of both diseases by summarizing information on their natural history and distribution, methods of transmission, diagnostic techniques, environmental influences, control measures, and recommended management practices.

MSX Disease

Natural History and Distribution

MSX disease was first recognized as the cause of massive oyster mortalities (90–95%) in lower Delaware Bay in 1957. Two years later, the disease was discovered in the lower Chesapeake Bay. The causative agent, a single-celled parasite, *Haplosporidium nelsoni*, was originally given the acronym “MSX” because it was observed as a Multinucleated Sphere with unknown affinity (“X”).

In the early and mid- 1960s, *H. nelsoni* was found in coastal waters from North Carolina to Connecticut, Oyster mortality clearly linked to *H. nelsoni* activity occurred predominantly in Delaware Bay and Chesapeake Bay. In the New England states, *H. nelsoni* was found in 1967 in Wellfleet Harbor on Cape Cod Bay. Periodic sampling along the entire Atlantic and Gulf Coasts between the mid 1960s and the early 1980s failed to detect the parasite south of North Carolina. By 1984, however, *H. nelsoni* was reported from the Damariscotta River, Maine to Biscayne Bay, Florida, although there were no associated mortalities at the extremes of this range (Figure 1, page 2).

The earliest infections of *H. nelsoni* are found in the oyster’s gill, leading to the conclusion that the infective stage is water-borne. The infective stage itself has never

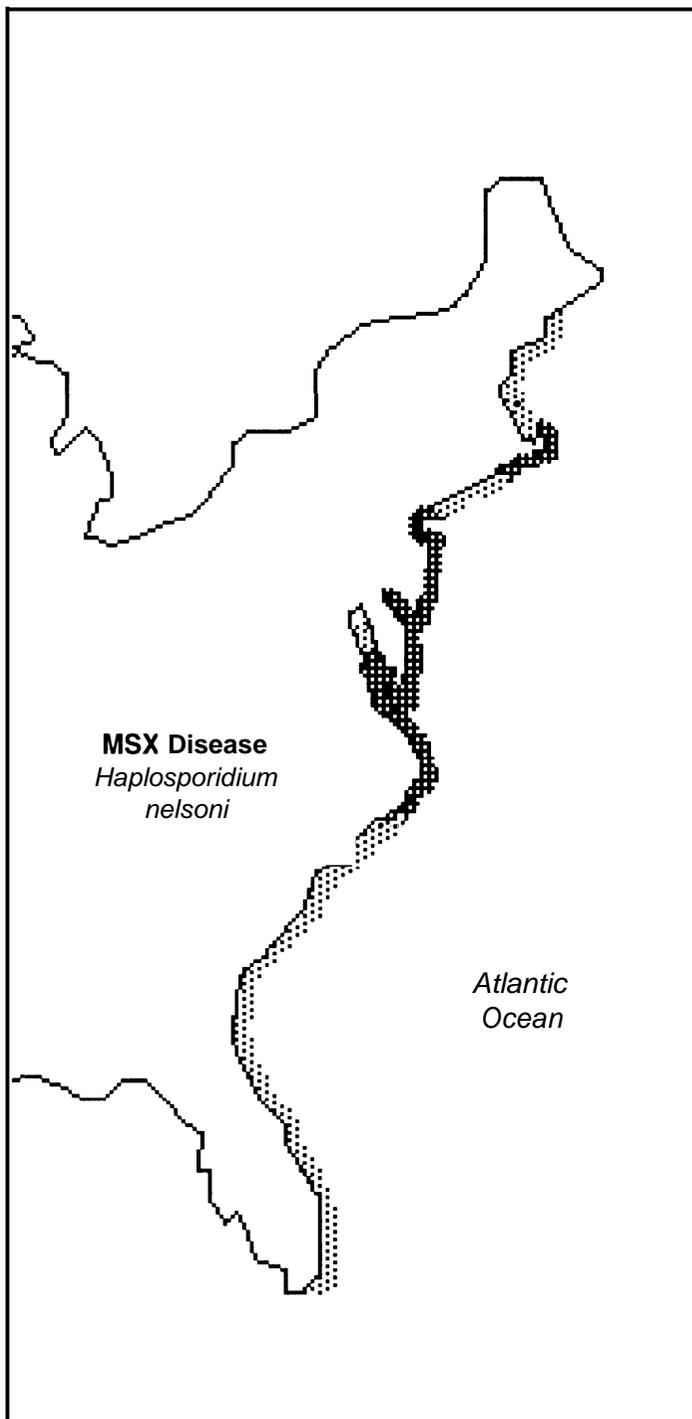


Figure 1. Distribution of MSX disease, caused by *Haplosporidium nelsoni* in eastern oysters. The heavy line represents regions where MSX disease has caused epizootic mortalities. The lighter shading represents areas where *H. nelsoni* has been reported, but is not known to have caused mortalities.

been recognized and the parasite is commonly present as a multinucleated cell (“plasmodium”), which enters the blood stream after lodging in the gill. Oysters die after plasmodia have become abundant in all tissues. The manner in which *H. nelsoni* causes death is not completely

understood, but overwhelming numbers of parasites damage tissues and interfere with normal functions such as respiration and feeding.

Transmission

The means by which MSX disease is transmitted is not known. All attempts to pass *H.nelsoni* from infected to uninfected oysters in the laboratory have been unsuccessful. Further, the prevalence of infections in a newly exposed population of oysters is not related to the presence, density, or infection level of surrounding oysters. Many investigators believe that an alternate or intermediate host may be involved in the life cycle. Distribution of MSX disease is fairly uniform over a large area within the higher-salinity (greater than 15 ppt – parts of salt per thousand parts of water) portion of affected estuaries, even where natural oyster populations are absent. With favorable salinities, the disease is capable of spreading rapidly over large distances.

Diagnostic Methods

Diagnosis of *H. nelsoni* infections is typically made through microscopic examination of specially prepared sections of oyster tissue. Another approach involves microscopic examination of blood smears, which is more rapid and economical, but generally unreliable for detecting early stages of the disease. Other assays using molecular probes adapted from recent advances in medical research are being developed to expedite early disease diagnosis. Accurate diagnosis of infections is an essential management tool enabling the grower to determine the intensity of MSX disease in a particular locale and to select the most effective strategy to minimize disease related mortality.

Environmental Influences

Infections of *H. nelsoni* are rare at salinities below 10 ppt and intensify above 15 ppt. Significant increases in parasite activity and distribution often occur during extended periods of reduced rainfall when higher salinity waters advance up the estuary. Conversely, increased freshwater runoff into estuarine areas substantially limits or lessens disease activity and distribution.

Field studies have shown that infection and mortality periods for MSX disease are similar from the lower Chesapeake Bay, Virginia to Cape Cod, Massachusetts, but are slightly delayed towards the north. Oysters usually become infected beginning in mid-May in Chesapeake Bay, early June in Delaware Bay, and late June on Cape Cod, Sus-

ceptible oysters usually show early gill infections about 3 weeks after initial exposure and begin dying 2–3 weeks later. In Delaware Bay, native oysters with some resistance to MSX disease typically display new infections in late July and heavy mortalities occur during late August and September. Additional mortalities may occur in late winter.

Temperature is also a factor influencing activity and distribution of the MSX parasite. If oysters are not exposed until late in the season when temperatures are falling, infections often remain at a low level until the following spring and mortalities usually do not occur until the following June and July, 8–9 months after exposure. As a result, delayed planting to avoid the major early-summer infection period may be a useful method for reducing exposure time and disease-related mortality.

Historically, periods of low *H. nelsoni* activity in Delaware Bay lasting between 1–2 years have been pre-

ceded by an extremely cold winter. Researchers have speculated that extreme cold might diminish the supply of infective particles by killing a possible alternate or intermediate host. Within the last few years, however, reductions of *H. nelsoni* activity throughout most of its range along the eastern seaboard have not been clearly associated either with reduced salinity or cold winter temperatures, indicating that additional factors may influence the distribution and abundance of the parasite (Figure 2).

Control Measures and Management Recommendations

Management strategies for successfully dealing with MSX disease vary according to region but rely to a great extent on inhibiting the disease by low salinity. If possible, oysters should be grown where salinity is low enough (less

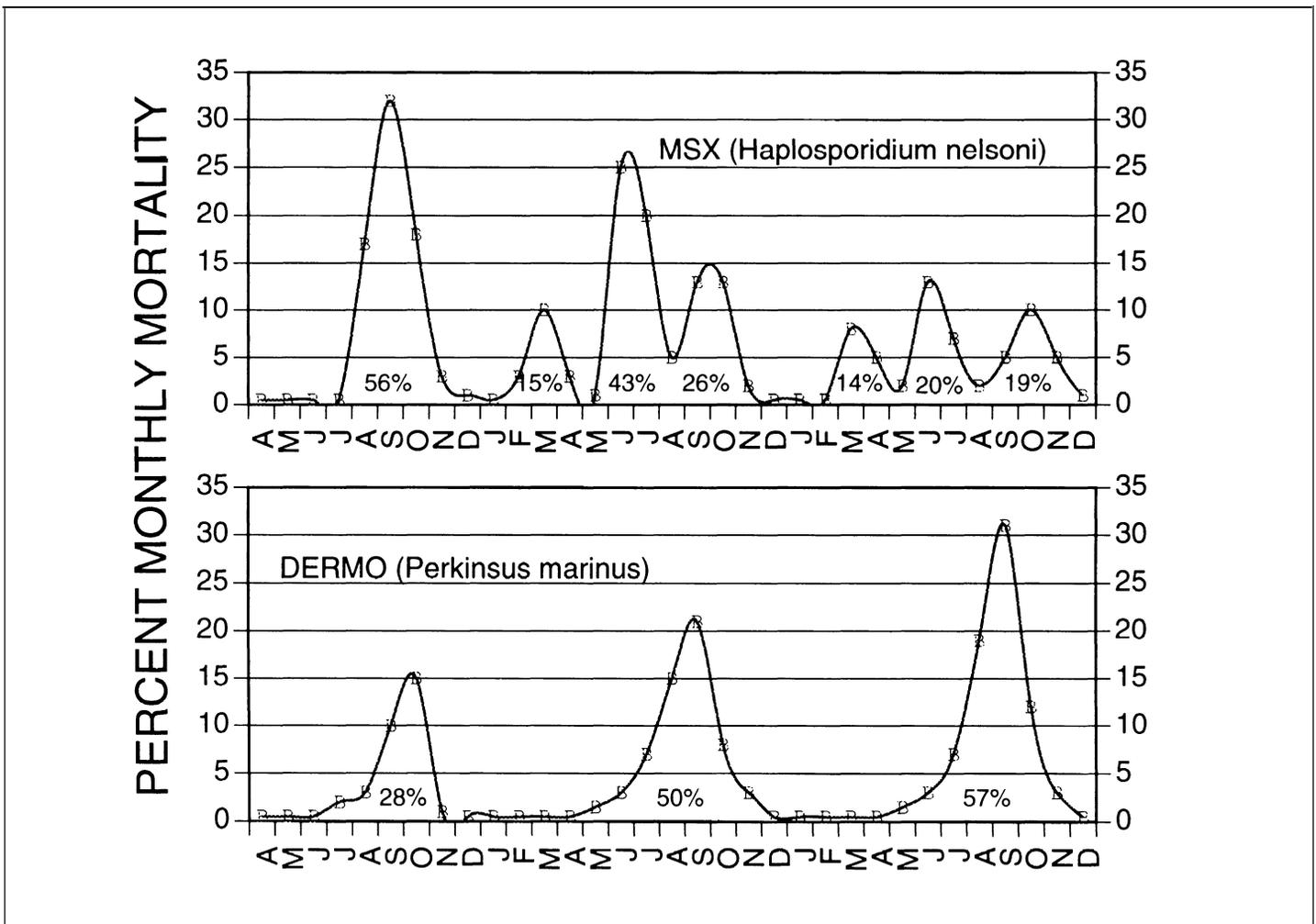


Figure 2. Typical mortality patterns caused by *Haplosporidium nelsoni* and *Perkinsus marinus* infections of eastern oysters in mid-Atlantic estuaries. Each scenario assumes that susceptible oysters are exposed to infections of one of the parasites for a 33-month period beginning in April or May of year one. Each point represents the monthly mortality, and the percentages under the peaks represent the cumulative mortality during that period.

than 15 ppt) to prevent serious MSX disease activity. If susceptible seed oysters must be moved from low to high salinity for final growth and conditioning prior to market, their exposure to high salinity should be limited to a single growing season. Planters can further minimize mortality by moving oysters after the early summer infection period (June and July) and harvesting by the end of the year or the following spring. Loss of time in the best growing areas must be balanced against better survival, and results may vary according to location and time.

Culture methods often used to protect oysters from predators, such as off bottom racks, floating rafts, intertidal bags or cages do not provide any protection from MSX disease because infective stages are present throughout the water column. Continuous monitoring and early diagnosis are essential management tools to define infection and mortality patterns and help growers determine when or whether to plant and harvest.

One of the most effective means to combat the disease is the use of resistant strains produced in hatcheries and reared using aquacultural techniques. Both natural epizootics and controlled selection and breeding experiments at Rutgers University and the Virginia Institute of Marine Science have produced strains of eastern oysters with increased resistance to MSX disease-related mortality. Resistance does not necessarily prevent infection but enables the exposed oysters to reach market size before infections become lethal. Oysters achieve resistance by restricting parasite development and increasing tolerance to infection. Oysters that survive MSX disease epizootics are likely to have some natural resistance and should be preserved as brood stock for future spat production. Because disease-resistant oysters must be spawned in a hatchery, their use by growers who already rely on hatchery-produced seed is particularly recommended.

Even though there is no evidence that transmission of *H.nelsoni* occurs directly from infected to uninfected oysters, transfer of wild oysters from infested areas into waters where the parasite is absent should be avoided. If an intermediate or alternate host does exist, introduction of this species could occur with movement of wild oysters. Limited transfer of oysters such as brood stock between sites should use quarantine facilities and protocols as a safety precaution.

Dermo Disease

Natural History and Distribution

Dermo disease was first identified as the cause of extensive oyster mortalities in the Gulf of Mexico nearly fifty

years ago. It was originally thought to be a fungus and was named *Dermocystidium marinum*. Even though subsequent research led to its reclassification as a protozoan parasite *Perkinsus marinus* in 1978, the disease is still commonly referred to as “Dermo.” From its discovery until 1990, Dermo disease was primarily restricted to southern waters, from the western Gulf of Mexico to Chesapeake Bay, where it caused chronic, and occasionally epizootic mortalities. In the mid 1950s, *P. marinus* was introduced into Delaware Bay when infected seed oysters were transplanted from lower Chesapeake Bay. Dermo disease effectively disappeared when further imports were prohibited and it was concluded that water temperatures were too cold in Delaware Bay to support a self-sustaining parasite population. Beginning in 1990, and coincident with a series of unusually warm winters, Dermo disease appeared in many northern locations from Delaware Bay (New Jersey waters), where epizootic mortalities resulted, to Massachusetts (Figure 3). At present, it is not clear whether the presence of *P.marinus* in northern areas is due to recent introductions, existing small concentrations of the parasite proliferating under higher temperatures, or a new, low-temperature tolerant strain.

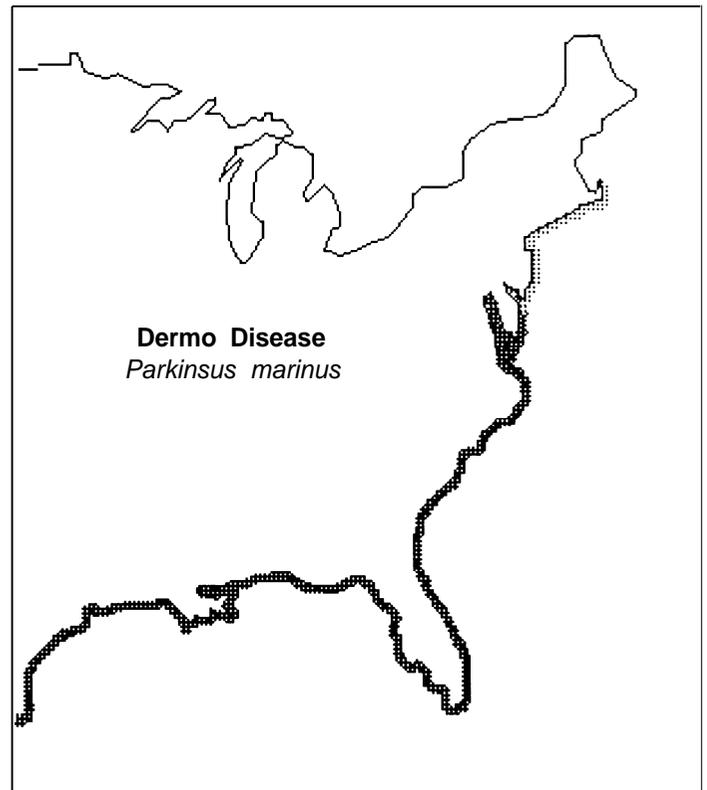


Figure 3. Distribution of Dermo disease, caused by *Perkinsus marinus* in eastern oysters. The heavy shading represents the known range of the disease before 1990. The lighter shading represents its range extension between 1990 and 1992.

Glossary

Disease: damage to cells sufficient to cause dysfunction of the organism.

Enzootic: a disease that is present, although not necessarily at high levels, in an animal population (equivalent to endemic in humans).

Epizootic: a disease that is rapidly spreading throughout an animal population (equivalent to an epidemic in humans).

Estuary: the lower end of a river where freshwater meets and mixes with saltwater.

FTM: fluid thioglycollate medium, a special medium used to detect *P. marinus* which causes parasites to enlarge, and accept a stain (Lugol's iodine), which turns them blue-lack for easy observation.

Hemolymph: circulatory fluid in invertebrates, comparable to blood in mammals.

Infection: the presence of an infectious or foreign organism in tissues of a host.

Molecular probe: a biochemical means of detecting molecules that are specific to a particular organism, such as a parasite.

Parasite: an organism living on or in another host organism to its advantage and the disadvantage of the host.

Pathogen: any disease causing organism.

Prevalence: the percentage of a population with a particular characteristics (e.g., disease) at a particular time.

Protozoan: a single celled organism often free living, but sometimes parasitic like the agents that cause MSX and Dermo diseases.

Quarantine: a state of isolation designed to prevent the spread of disease or pests.

Resistance: the relative ability of an organism to avoid infection or to withstand the effects of disease.

Systemic: throughout the body, involving the whole body.

Vector: any agent (living or inanimate) that acts as an intermediate carrier or alternative host for a pathogenic organism and transmits it to a susceptible host.

The life cycle of *P. marinus* consists of several distinct developmental stages that infect and proliferate rapidly within the oyster. Dead and disintegrating oysters release infective stages back into the water column where they infect other oysters and repeat the cycle. Most researchers have concluded that oysters infected with Dermo disease die primarily from extensive tissue lysis and blockage of major blood vessels.

Mortalities of susceptible oysters begin within a month or two of exposure. In a newly exposed population, a few oysters initially become infected and die. Death rates increase in each succeeding year as more infective stages are released by increasing numbers of dying oysters (Figure 2). Estimates have been made suggesting that a full-fledged epizootic of newly exposed oysters in Chesapeake Bay can develop within a period of 1 to 3 years.

Transmission

Unlike *H. nelsoni*, transmission of *P. marinus* between oysters is easily accomplished both in the field and the laboratory. The main method of transmission occurs when infective stages of the parasite, free in the water column, are ingested by oysters and then invade the lining of the digestive system. Earliest infections are most often found in the lining of the stomach and intestine, but apparently new infections have also been detected in gill and mantle tissue.

Natural infections are most often caused by parasites released from the disintegration of dead oysters. *P. marinus* may also be distributed by scavengers feeding on dead oysters or by *Boonea impressa*, a parasitic snail that feeds on oyster hemolymph and acts as a vector in the transmission of the disease between live oysters (Figure 4, page 6).

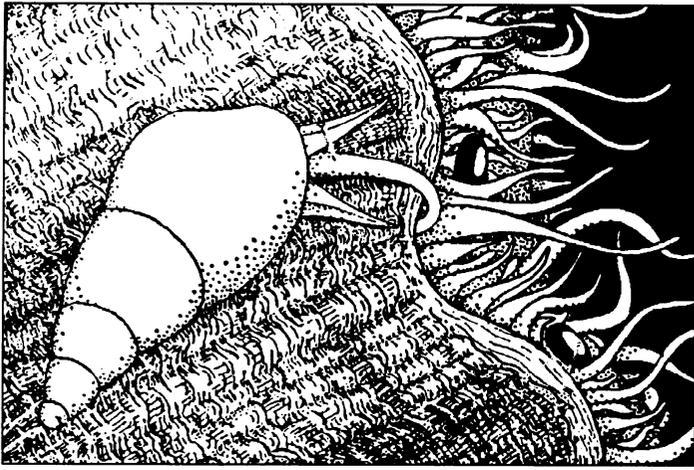


Figure 4. An example of *Boonea* sp. feeding on the hemolymph of a bivalve mollusc (in this case a scallop) via the mantle tissue. Parasitism by *Boonea* aids in the transmission of Dermo disease between living eastern oysters. After Fretter and Graham, 1962.

Diagnostic Techniques

Diagnosis of *P. marinus* infections is most often made by microscopic examination of oyster tissues that have been first conditioned in a special medium (fluid thioglycollate medium or FTM). As with *H. nelsoni*, blood samples cultured in FTM are also used to detect systemic infections. This technique permits repeated, nondestructive sampling of individual oysters, but is unreliable for detecting the earliest stages of the disease. Molecular probes capable of detecting both the early and advanced stages of *P. marinus* infections are also being developed.

Environmental Influences

P. marinus is considered a warm water pathogen because it proliferates and spreads most rapidly at temperatures above 25°C (77°F). The main infective period occurs during summer months and is more extended in southern than in northern areas. Both prevalence and intensity of *P. marinus* infections decrease over winter, especially at the northern end of its range. The parasite, however, is capable of surviving over winter and has been cultured in FTM even after being frozen.

Activity of *P. marinus* is suppressed, but not eliminated, at salinities below 8 to 9 ppt and salinities about 12 ppt or higher are usually required for a full epizootic. Drought conditions and associated higher salinities promote spread and increased activity of the parasite. Oysters from low salinity areas of estuaries where *P. marinus* is enzootic may have very light, non lethal infections (not even detectable by FTM culture), which can proliferate rapidly when those oysters are moved into higher salinity waters.

In mid-Atlantic estuaries, where both temperature and salinity vary substantially, temperature appears to be the more important factor controlling the distribution and intensity of Dermo disease. In the Gulf of Mexico, where temperature is never limiting for *P. marinus*, salinity is probably the more important factor.

Infective stages of *P. marinus* are generally present from May through October in Chesapeake Bay. Initial infections are usually detected in June, with a major mortality period between July and October. Prevalence of the disease in surviving oysters declines over the winter (Figure 2). However, some parasites remain dormant and proliferate as temperatures rise in the spring, releasing stages that initiate another round of infections. Warm winters allow more parasites to survive, and high spring or autumn temperatures extend the period during which epizootics occur.

Management Recommendations

Researchers have not yet been able to select strains for resistance to Dermo disease, and MSX disease-resistant oysters exposed to *P. marinus* in Chesapeake and Delaware Bays have experienced high mortalities. Efforts are continuing, at both Rutgers University and the Virginia Institute of Marine Science, to develop oysters with improved survival when exposed to both MSX and Dermo disease agents.

Because *P. marinus* is known to be easily transmitted, seed oysters from areas where Dermo disease is enzootic should not be transplanted under any circumstances. If disease-free seed is planted in areas where Dermo disease is present, they should be kept isolated from potential sources of infection such as native populations on natural beds, pilings, bridges, or piers. Placing uninfected seed in enzootic waters in early August will avoid two months of heavy infection (June and July) while still providing significant time for growth before winter. During periods of normal rainfall, a minimum distance of 1300 feet separating new plantings from potential sources of infection is recommended. During prolonged warm, dry periods, few management options are available.

Once *P. marinus* becomes established in even low salinity growing areas, years may be required to eradicate the pathogen. Growers should remove all residual oysters from a ground on which they intend to place seed stock

Important Facts About...

MSX Disease

- The complete life cycle and mechanism of transmission of its causative agent, *Haplosporidium nelsoni*, are not known.
- Activity and distribution of the disease is primarily regulated by salinity; *H. nelsoni* is absent or inactive at less than 10 ppt salinity, and becomes very active at 15 ppt or greater salinity.
- The major infective period occurs between May and July with a time delay from south to north within the region.
- The MSX parasite is most prevalent in higher salinity portions of estuaries and mortality increases with level of exposure. Stocks selected for improved survival may suffer high mortalities if infection pressure is extraordinarily high.
- Exposure of susceptible oysters to high salinity for final growth and conditioning before market should be limited to a single growing season.
- Mortality can be reduced by moving oysters after the early summer infection period (June and July) and harvesting by the end of the year or the following spring.
- Continuous monitoring and early diagnosis of infections are essential to define infection and mortality patterns that will suggest management options for specific localities.
- Resistance to MSX disease does not necessarily prevent infection but enables exposed oysters to restrict and tolerate parasites, and thereby reach marketable size.
- Use of oyster strains highly resistant to MSX disease is recommended for intensive culture operations that deploy hatchery produced seed.
- Transfer of wild seed oysters from infested areas into waters where the parasite is absent should be avoided. Movement of brood stock should be undertaken using quarantine facilities and protocols.

Dermo Disease

- The life cycle of *P. marinus* consists of several distinct developmental stages. Dead and disintegrating oysters release highly infective stages of the parasite into the water column where they come in contact with, and are ingested by other, uninfected oysters.
- Dermo disease is most prevalent in warm, high salinity estuaries. Most infections are transferred by close proximity of uninfected oysters to dead and disintegrating oysters with high intensity infections.
- Activity and distribution of the disease is primarily regulated by temperature in the northeast; low level infections persist but are not lethal at salinities of less than 5 ppt, and intensify at salinities greater than about 12 ppt.
- Mortalities of susceptible oysters begin within a month or two of exposure. Death rates increase in each succeeding year as more infective stages are released by increasing numbers of dying oysters.
- The major infective period occurs between May and October. Mortalities generally occur between July and October.
- Oysters with natural or genetically bred resistance to MSX disease are vulnerable to Dermo disease.
- Seed oysters from regions with Dermo disease should not be transplanted to non-disease areas under any circumstances.
- Disease-free seed should be kept isolated from other sources of infection such as native populations on natural beds, pilings, bridges, or piers.
- Placing uninfected seed in enzootic waters in early August will avoid two months of heavy infection (June and July) while still providing significant time for growth before winter.
- Growers should remove all residual oysters from a ground on which they intend to place seed stock and should let grounds lie "fallow" for one or two years after harvest so that remaining infected oysters can die.
- Planted oysters should be assayed for evidence of *P. marinus* infections in late summer or early fall.

and should let grounds lie “fallow” for one or two years after harvest so that remaining infected oysters can die. Planted oysters should be assayed for evidence of infections in late summer or early fall. Growers should also inspect beds for dead or dying oysters to determine the best schedule for continued growth or harvest. Because Dermo disease is so persistent and difficult to eradicate, elimination of parasitized oysters as a source of infective stages and maintaining normal freshwater input into affected estuaries appear, at present, to be the most reasonable ways of controlling the disease.

References/Suggested Additional Reading

- Andrews, J. D. and S. M. Ray, 1988. Management Strategies to Control the Disease Caused by *Perkinsus marinus*. American Fisheries Society Special Publication 18:257-264.
- Ford, S. E. and H. H. Haskin, 1988. Management Strategies for MSX (*Haplosporidium nelsoni*) Disease in Eastern Oysters. American Fisheries Society Special Publication 18:249-256.

- Ford, S. E. 1992. Avoiding the spread of disease in commercial culture of molluscs, with special reference to *Perkinsus marinus* (Dermo) and *Haplosporidium nelsoni* (MSX). J. Shellfish Res. 11:539-546.
- Ford, S. E. and M. R. Tripp. 1993. Diseases and defense mechanisms. To be published in The Eastern Oyster *Crassostrea virginica*. (A. F. Eble, V. S. Kennedy and R. I. E. Newell, editors). Maryland Sea Grant, College Park, Maryland.
- Fretter, V. and A. Graham. 1962. *British Prosobranch Molluscs*, The Ray Society, London, 748 p.

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