Oyster Diseases of the Chesapeake Bay

— Dermo and MSX Fact Sheet —

Virginia Institute of Marine Science

Scientific Name: *Perkinsus marinus*

Common Name: Dermo, Perkinsus

Taxonomic Affiliation:
Kingdom = Protista, Phylum = Undetermined

Species Affected:
Crassostrea virginica (eastern oyster)

Geographic Distribution:
East coast of the US from Maine to Florida and along the Gulf coast to Venezuela. Also documented in Mexico, Puerto Rico, Cuba, and Brazil.

Dermo

History:
Dermo disease was first documented in the 1940s in the Gulf of Mexico where it was associated with extensive oyster mortalities. The causative agent was initially thought to be a fungus and was called *Dermocystidium marinum*. Based on structural characteristics the organism was reclassified *Labyrinthomyxa marina* in 1966 and as *Perkinsus marinus* in 1978. The disease was found in Chesapeake Bay in 1949 and it has consistently been present in the Bay since that time. The parasite was observed in Delaware Bay in the mid 1950s following the importation of seed from the Chesapeake Bay. An embargo of seed resulted in a disappearance of the disease from Delaware Bay for more than 3 decades. However, an epizootic recurred in Delaware Bay in 1990 and since 1991 the parasite has been found in Connecticut, New York, Massachusetts, and Maine. This apparent range extension is believed to be associated with abnormally high winter temperatures, drought conditions, and the unintentional introduction of infected oysters or shucking wastes.

In the Chesapeake Bay, Dermo disease has increased in importance since the mid 1980s. Several consecutive drought years coupled with above average winter temperatures resulted in expansion of the parasite's range into upper tributary areas and the parasite became established at all public oyster grounds in Virginia. The parasite has persisted in these areas despite a return to normal salinity conditions. In addition to its baywide distribution, Dermo is also present in the embayments along the Atlantic coast of Virginia.

Biology and Epizootiology:
The seasonal cycle of *P. marinus* has been well documented in Chesapeake Bay. Transmission of the parasite is direct from oyster to oyster. Waterborne infective stages are present throughout the warm months, May through October. Initial infections are typically observed in July and peak prevalences and maximum mortalities are observed in September and October. Prevalence in surviving oysters declines dramatically during the late winter and spring and infections may become undetectable by the standard diagnostic assay. However, low numbers of parasites remain and these parasites proliferate once temperatures increase in late spring. Infective stages of the parasite are released from infected and dying oysters, thereby initiating another infection cycle. The infective stages become waterborne and are acquired as oysters feed. Within the oyster, early infections are observed in digestive gland tissues. The most prominent stage is a single cell stage called the trophont. These cells divide forming a multicellular stage called a meront. Meronts enlarge and rupture releasing many small single cells. Under certain conditions, in artificial media and occasionally in moribund oysters, the parasite produces a third stage known as a biflagellate zoospore.

Infections are usually not acquired in oysters less than a year old but prevalences may be high during the oysters second year and mortality may result. Moderately to heavily infected oysters usually exhibit a reduction in growth rate, poor condition, and reduced reproductive capacity. Oyster death results as a consequence of hundreds of thousands of parasites "taking over" the oyster, lysing tissues, and occluding hemolymph vessels.

Environmental Influences:
Temperature and salinity are the two most important environmental factors influencing Dermo disease. The parasite proliferates and infections intensify above a threshold of 20°C. At temperatures above 25°C, the parasite rapidly multiplies, spreads, and kills oysters. Infections decline at temperatures below 15-20°C. In nature the most dramatic decline is observed in late winter and early spring. Abnormally warm winters may result in a higher proportion of over-wintering parasite cells.

Prevalence and infection intensities of *P. marinus* increase with increasing salinity. During drought years, elevated salinities result in an intensification of the disease. High intensity infections and high mortalities often occur in areas with salinities above 12-15 ppt. Infection intensities remain low in areas with salinity consistently below 9 ppt. Once established in a low salinity area the parasite can persist for years.

Control Measures:
Dermo disease is easily transmitted from oyster to oyster so it is imperative to avoid moving infected oysters into an area containing uninfected oysters. Holding oysters at salinities less than 9 ppt will retard disease development and restrict disease associated mortalities. If possible, let grow out areas remain fallow for one to two years before planting seed stocks.

Diagnostic Method:
Fluid thioglycollate media (FTM) culture assay is the standard diagnostic technique. This method involves culturing small pieces of oyster tissue in FTM for 4-7 days. Following culture the tissue is stained with Lugol's iodine and examined using a light microscope. *Perkinsus marinus* cells will appear as blue to black stained spheres. Modifications of this assay exist for the examination of oyster hemolymph and total parasite burdens in whole oyster tissues. Polymerase reaction chain (PCR) assays have also been developed; however, PCR is not employed for routine diagnosis.

Scientific Name: *Haplosporidium nelsoni*

Common Name: MSX (multinucleated sphere unknown)

Taxonomic Affiliation:
Kingdom = Protista, Phylum = Haplosporidia

Species Affected:
Crassostrea virginica (eastern oyster), possibly *Crassostrea gigas* (pacific oyster)

Geographic Distribution:
In the US, MSX disease ranges from Damariscotta River, Maine to Biscayne, Florida. It is not present in the Gulf of Mexico. Epizootic mortalities have been limited to Chesapeake and Delaware Bays and recently Long Island Sound. The parasite has also been found in *C. gigas* from Korea and Japan.

History:
The disease was first documented in 1957 in Delaware Bay where it caused massive oyster mortalities and two years later it was found in the lower Chesapeake Bay. At that time the disease agent was given the acronym MSX-multinucleated sphere X (unknown). In the 1960s the parasite was found in coastal bays of North Carolina, Virginia, Maryland, Delaware, New Jersey, Connecticut and New York, but associated oyster mortalities did not occur south of Virginia or north of New Jersey. In the 1980s, an apparent range extension occurred as the parasite was reported as far north as Maine and as far south as Florida. Since 1995, MSX associated mortalities have occurred in Maine and New York. Description of the spore stage of the parasite led to it being named *Minchinia nelsoni* in 1966. The parasite was later renamed *Haplosporidium nelsoni* in 1980.

MSX

Biology & Epizootiology:
Haplosporidium nelsoni is a spore-forming protozoan. The predominant stage of the organism in the oyster is a multinucleated plasmodium, which ranges in size from 5-100 m. The production of spores (sporulation) is rare in adult oysters but has been observed at prevalences as high as 40% in spat. Sporulation occurs in late June through early July and in the autumn. The infective stage has never been recognized but it is believed to be a uninucleate form that is released by a spore. The complete life cycle remains unknown. Controlled transmission of the parasite has not been achieved despite many laboratory attempts. The inability to transmit the parasite combined with the rarity of spore stages, the lack of correlation of the disease with oyster density and its ability to spread rapidly over long distances has led to the hypothesis that an alternate or intermediate host exists.

In the Chesapeake Bay, oysters become infected from mid-May through October; however, infection pressure during late summer and autumn is quite variable from year to year. Infections develop rapidly in susceptible oysters resulting in mortalities from July through October. Surviving oysters may maintain a high prevalence of the disease through the winter and a second period of mortality may occur in spring. Oysters acquiring infections in late autumn may harbor low level infections, which intensify the following summer. These infections often proliferate as temperatures warm in June causing early summer mortalities.

The disease can affect all ages of oysters, spat to adult. Infections are acquired through gill and mantle tissue, and can rapidly spread throughout the oyster.

Environmental Influences:
Temperature and salinity play an important role in regulating MSX. Infections are acquired at temperatures above about 20°C. Three critical temperatures have been proposed for oyster-*H. nelsoni* interactions. Both parasite and oyster are inactive at temperatures <5°C. At 5-20°C, the parasite proliferates more rapidly than the oyster can control it. Above 20°C, resistant oysters can overcome the parasite while susceptible oysters are killed.

Salinity is important in determining the distribution of the disease within an estuary. A salinity of 15 ppt is required for infection, 20 ppt is required for rapid and high mortality, and 10 ppt or below results expulsion of the parasite at temperatures above 20°C.

Control Measures:
Use MSX disease resistant oyster strains. Maintain oysters in disease-free areas (low salinity). If oysters must be moved to high salinity areas for growth and conditioning, the move should be timed to avoid the early summer infection period. Low-salinity immersion to expulse MSX infections may be valuable, but critical time-temperature-salinity combinations have not been thoroughly determined. Avoid importation of infected oysters into grow-out area.

Diagnostic Method:
Histological examination using light microscopy of paraffin embedded tissue sections is the standard diagnostic technique for MSX. Alternative methods include screening of hemolymph and employment of molecular tools including polymerase chain reaction (PCR) and DNA probe assays.

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