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Leaflet No. 38

MSX disease of oysters caused by *Haplosporidium nelsoni*

Original by Jay D. Andrews

Revised and updated by Susan E. Ford



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MSX disease of oysters caused by *Haplosporidium nelsoni*

Original by Jay D. Andrews, Revised and updated by Susan E. Ford

Susceptible species

Eastern oyster, *Crassostrea virginica*; rarely in the Pacific oyster, *C. gigas*

Disease name

MSX disease

Aetiological agent

Haplosporidium (= *Minchinia*) *nelsoni*, Phylum Haplosporidia. Complete life cycle and mode of transmission is unknown. Attempts at experimental transmission by proximity, feeding, injection, and tissue transplantation have all failed to cause infections. Proximity to infected oysters in the field is not needed to transmit the parasite. Spores are presumed to be a transmission stage. They are observed only rarely in adult oysters, but commonly in juveniles (≤ 20 mm) with advanced infections. A second host is suspected, but none has been identified. Comparison of genetic sequences between *H. nelsoni* and a previously observed, morphologically identical parasite in *C. gigas* indicates that they are the same organism and that *H. nelsoni* was introduced from the Pacific to the east coast of North America, where it found a highly susceptible host.

Geographical distribution

Infected oysters are found along the east coast of North America from Nova Scotia, Canada to Florida, USA. Persistent epizootics with high mortality have been restricted to the Mid-Atlantic States and New England, USA, and Canada. The parasite is present in Korea, Japan, the west coast of the US, and France in the Pacific oyster, *C. gigas*, where it is rare and has not caused noticeable mortality.

Associated environmental conditions

Experimental and observational data indicate that *H. nelsoni* proliferates *in vivo* at temperatures $\geq 10^{\circ}\text{C}$. The parasite is highly sensitive to low salinity and is rarely found in oysters living at 10 ppt or less. It is expelled from oysters in the upper reaches of estuaries during high run-off periods. A salinity of 15 ppt or higher is associated with epizootics and drought conditions allow the parasite to move up-estuary. In regions where epizootics occur, a distinct seasonal cycle is observed. Infections are acquired from mid-May through October, mostly during the early portion of that period. Parasites multiply rapidly during summer and early autumn, causing heavy mortalities in late summer and autumn. Prevalence remains high overwinter and mortalities occur again in late winter and spring when the temperature rises. A second prevalence peak may occur in late May or early June, with consequent mortalities. A multiyear cycle of infection prevalence, with low-prevalence years following cold winters, has been documented in lower Delaware Bay, USA.

Significance

The parasite can cause annual mortalities of up to 90% during an initial outbreak and chronic losses of 50% per year thereafter, with reductions in shell growth, meat quality, and reproductive capabilities among still-infected survivors. Oyster production in Chesapeake and Delaware bays has been severely depressed since the appearance of *H. nelsoni* in the late 1950s. Outbreaks in Long Island Sound, USA in 1997–1998 and Nova Scotia, Canada in 2002 also caused heavy losses.

Gross clinical signs

MSX disease cannot be diagnosed by gross clinical signs.

Control measures and legislation

Selective breeding has been successful in producing highly resistant strains and the development of resistance through natural selection has been reported in some wild populations subjected to heavy infection pressure. Management strategies include maintaining oysters at reduced salinities (<15 ppt) as long as possible. If final conditioning at higher salinity is needed for market, the conditioning should be done late in the season to avoid the major early-summer infection period. Immersion of oysters at ≤ 10 ppt for 2–3 weeks at $\geq 20^\circ\text{C}$ or more should eliminate the parasite from infected oysters. Particle filtration (1- μm cartridge filter) and UV irradiation will eliminate infective stages from water coming into hatcheries or nurseries. Although direct transmission has not been demonstrated, the introduction of oysters, especially juveniles that may contain spores, from an enzootic area to an area where the pathogen is not present should be avoided. MSX disease is an OIE notifiable disease.

Diagnostic methods

Plasmodia (the most common stage in oysters) are from 5 to $>50 \mu\text{m}$ in diameter, depending on the number and size of nuclei they contain. Nuclei are spherical, 1.5 to 3.0 μm in diameter with a peripheral endosome, or elongated and up to 7.5 μm . Infections are extracellular, although phagocytes ingest dead plasmodia. Parasites occur initially in the gill epithelium and subsequently become dispersed through all tissues. Sporulation, when observed, generally occurs in the epithelium of the digestive diverticula in oysters with advanced infections. During sporulation, plasmodia develop into sporocysts, with spore walls forming around each nucleus. Spores are approximately $5.5 \times 8 \mu\text{m}$ and have a cap with an overhanging lid. It is estimated that the histological method does not become reliably accurate until the parasite density is ca. 10^3 to 10^4 parasites gramme^{-1} wet weight. Diagnosis can also be made of hemolymph or tissue smears, although this method is less sensitive than tissue section histology. Molecular diagnosis using specific DNA primers and PCR is considerably more sensitive, although it is not currently in routine use.

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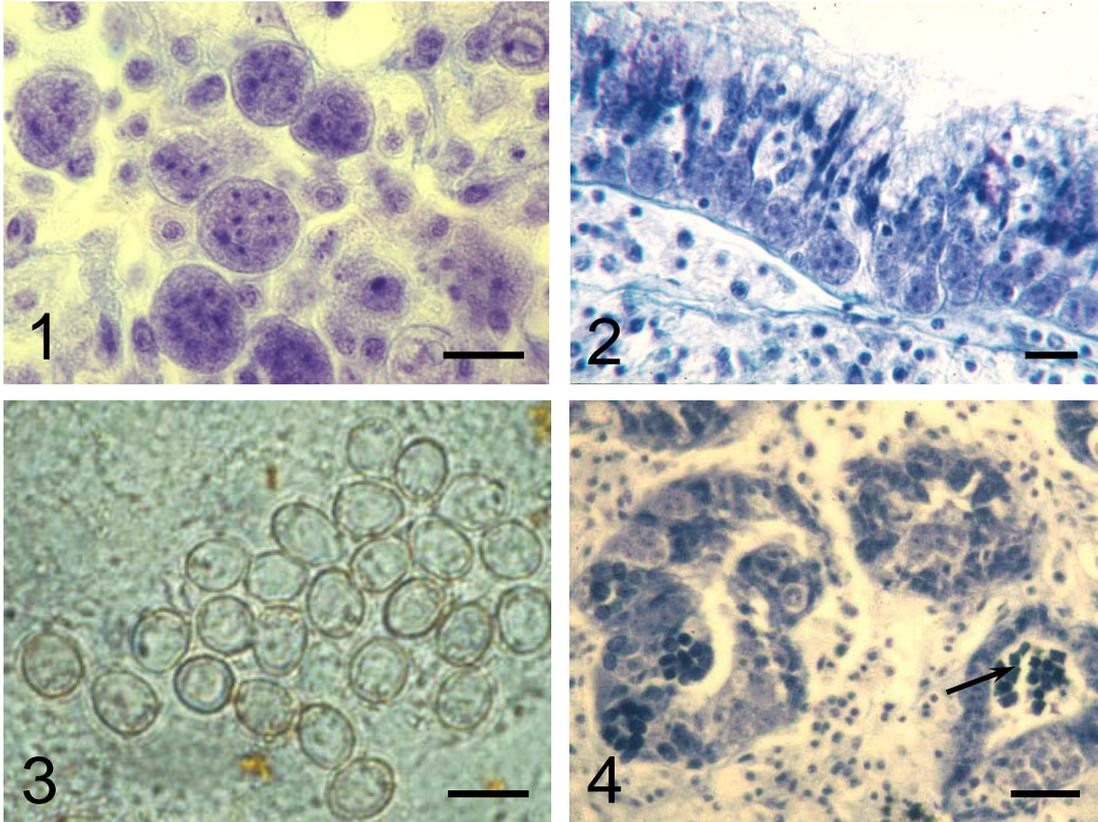


Figure 1. Multinucleated plasmodia of *Haplosporidium nelsoni* in a stained tissue section of the eastern oyster. Scale bar = 10 μm .

Figure 2. Early-stage infection of *Haplosporidium nelsoni*. Plasmodia are lined up along the basal lamina of the gill epithelium. Scale bar = 10 μm .

Figure 3. Spores of *Haplosporidium nelsoni* in a fresh mount of infected eastern oyster tissue. Scale bar = 10 μm .

Figure 4. Developmental stages of *Haplosporidium nelsoni* spores in digestive tubule epithelia and mature spores shed into tubule lumen (arrow). Scale bar = 20 μm .

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