Haplosporidium sp.

I. Causative Agent and Disease

Protozoa of the genus Haplosporidium sp. belong to the phylum Haplosporidia, class Haplosporea within the order Haplosporida in the family Haplosporidiidae. These are obligate parasites, some of which are extremely important pathogens of oysters. One such species, H. nelsoni, was responsible for MSX (multinucleate sphere unknown) later known as Delaware Bay Disease that caused the demise of the eastern oyster in higher salinity (>15 but < 25 ppt) areas of Delaware and Chesapeake Bays from 1957 through 1960. Systemic plasmodial life stages of the parasite can cause extensive necrosis of all tissues resulting in mass mortality. Occurring at about the same time another species, H. costalis or SSO (Seaside Organism), caused serious mortality in eastern oysters rearing in higher salinity waters (> 25 ppt) on the seaside coasts of Virginia and Maryland.

II. Host Species

Haplosporidium nelsoni has been reported in eastern oysters on the Atlantic coast of North America as far north as Bras d'Or Lakes. Nova Scotia. Canada south to Florida. A morphologically similar Haplosporidium sp. in Pacific (Japanese) oysters in Korea and Japan and on the Pacific coast of North America was determined by DNA sequence analysis to be H. nelsoni apparently introduced to the Pacific Northwest from Asia. The parasite was then introduced to the Atlantic coast with west coast Pacific oysters sometime before the epizootics occurred in Delaware Bay. *Haplosporidium armoricana* has been reported in the European flat oyster

in France, Britany and the Netherlands. Other Haplosporidium species have also been reported in California sea mussels (H. tumefacientis) from California, blue mussels (Haplosporidium sp.) from Maine, gaper clams and native oysters (Haplosporidium sp.) from Oregon, carpet-shell and Manila calms (*H.* tapetis) from France, Spain and Portugal. Unidentified Haplosporidium sp. were also found in cockles from France, black-footed abalone in New Zealand and in toredo ship worms from New Jersey, U.S.A. In Alaska, only one case of a bivalve haplosporidian is on record regarding an unidentified Haplosporidium sp. producing plasmodia in the gill connective tissues of a single razor clam collected from the southcentral area in 1991. No disease or mortality were associated with this finding.

III. Clinical Signs

Non-specific clinical signs of haplosporidiosis parasitism are more severe in oysters and can include mantle recession, gaping valves, watery emaciated tissues, pale digestive gland and rarely yellow-brown conchiolin deposits forming on the inner shell. Dissemination of plasmodial stages of the parasite throughout all tissues with associated host cell inflammatory infiltration and cell necrosis are evident by histological examination.

IV. Transmission

Haplosporidium sp. produce operculate spores which are not directly infectious but likely require, a yet unknown, intermediate host. The complete life cycle and infective stage are unknown but once in the bivalve host a modified type of shizogony gives rise to multinucleated

plasmodia. These plasmodia develop into sporonts that become sporocysts enclosing several spores that are released into the environment from moribund or dead bivalve hosts.

V. Diagnosis

Preliminary diagnosis for all Haplosporidium sp. is based on histological examination showing the typical plasmodia varying in size from 5 to 100 µm found in various host tissues. The larger spores of H. nelsoni (5.4 X 7.5 µm) are rarely found in adult oysters but are frequent in juveniles occurring only in the digestive gland epithelium. Smaller spores (3.3 X 4.3 µm) of H. costalis occur throughout the connective tissues but not in the digestive gland epithelium. Spores of both species (probably other haplosporidia as well) are red (acid-fast) using the Ziehl-Neelsen stain. Confirmatory tests using PCR and DNA probes are available for H. nelsoni and possibly other species of haplosporidia.

VI. Prognosis for Host

Various species of Haplosporidium sp. have caused significant bivalve mortality and are serious pathogens that should not be introduced into new areas via shellfish transports. Salinity and seawater temperature are important limiting factors for parasite development and disease that have been used to reduce oyster losses due to H. nelsoni and H. costalis. This strategy may also be useful to control other haplosporidia. Haplosporidiosis in Pacific oysters has not been associated with mortality of that species. On the east coast selectively bred eastern oysters that are genetically resistant to H. nelsoni have been deployed commercially on a limited basis.

VII. Human Health Significance

There are no zoonotic human health concerns associated with haplosporidian parasitism of bivalve mollusc tissues.



Haplosporidia-like sporonts (arrow) in the gill connective tissues of Alaskan razor clam

Haplosporidium sp.



Histological section showing various stages of haplosporidia-like sporonts (arrow) and spore development in the gill connective tissues of Alaskan razor clam



Histological section of eastern oyster with MSX plasmodia (arrow) in connective tissues



Histological section of eastern oyster with MSX acid-fast spores (red) within the digestive tubule epithelium



Histological section of eastern oyster with SSO plasmodia (arrow) and spores (arrowhead) in connective tissues



Histological section of eastern oyster exhibiting SSO acid-fast spores (red) in connective tissues