

ICES IDENTIFICATION LEAFLETS FOR DISEASES AND PARASITES OF FISH AND SHELLFISH

Leaflet No. 39

SSO disease of oysters caused by *Haplosporidium costale*

Original by Jay D. Andrews

Revised and updated by Susan E. Ford



ICES

International Council for
the Exploration of the Sea

CIEM

Conseil International pour
l'Exploration de la Mer

**International Council for the Exploration of the Sea
Conseil International pour l'Exploration de la Mer**

H.C. Andersens Boulevard 44–46
DK-1553 Copenhagen V
Denmark
Telephone (+45) 33 38 67 00
Telefax (+45) 33 93 42 15
www.ices.dk
info@ices.dk

Recommended format for purposes of citation:

ICES. 2011. SSO Disease of Oysters Caused by *Haplosporidium costale*. Revised and updated by Susan E. Ford. ICES Identification Leaflets for Diseases and Parasites of Fish and Shellfish. Leaflet No. 39. 4 pp.

Series Editor: Stephen Feist. Prepared under the auspices of the ICES Working Group on Pathology and Diseases of Marine Organisms.

For permission to reproduce material from this publication, please apply to the General Secretary.

ISBN 978-87-7482-092-5

ISSN 0109–2510

© 2011 International Council for the Exploration of the Sea

SSO Disease of Oysters Caused by *Haplosporidium costale*

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

Susceptible species

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

Disease name

SSO disease

Aetiological agent

Haplosporidium costale (= *Minchinia costalis*), Phylum Haplosporidia. Complete life cycle and mode of transmission are unknown.

Geographical distribution

Infected oysters are detected from Nova Scotia, Canada, south along the east coast of the USA to the mouth of Chesapeake Bay. Prevalence is low in most areas, but associated mortalities are reported. Also detected from eastern China (in *C. gigas*).

Associated environmental conditions

Infections are typically restricted to high (≥ 25 ppt) salinity regions. No specific temperatures are documented for infection acquisition and development, but in Virginia, USA, where the parasite has been studied most extensively, new infections occur during spring (April–June), concurrent with deaths of previously infected oysters. Infections remain histologically undetectable for 9–10 months then develop rapidly in spring of the following year with concurrent mortalities. Most histologically detectable infections disappear by late summer, although some infections can still be detected in autumn.

Significance

H. costale inhibits the growth of infected oysters and kills them one to two months after infections become histologically detectable. The parasite can kill 20 to 50% of oysters in an affected population annually.

Gross clinical signs

SSO disease cannot be diagnosed by gross clinical signs.

Control measures and legislation

Maintaining oysters at salinities < 20 ppt should minimize the acquisition of new infections. Transfer of infected oysters to salinities < 20 ppt is likely to eliminate, or at least control proliferation of, parasites in infected oysters. Because of the apparent long incubation period after infection (9–10 months), oysters placed in enzootic water

immediately after an infection period (i.e. August in Virginia) and harvested within 18 months, should suffer little or no mortality. Particle filtration (1- μm filters) and UV irradiation of water coming into hatcheries or nurseries should eliminate infective stages, as it does for the related pathogen, *Haplosporidium nelsoni*. Although direct transmission has not been documented, transportation of infected oysters into nonenzootic areas should be avoided. SSO disease is not an OIE-notifiable disease.

Diagnostic methods

The recommended assay is a microscopic examination of a stained tissue section that includes gill, mantle, digestive diverticula, stomach, and intestine. Initial plasmodia stages are small (<5 μm), with few nuclei, and found beneath gut epithelia. In advanced infections, plasmodia and sporocysts containing spores in various stages of development are found in all tissues except the epithelia. Plasmodia are typically <10 μm in diameter, and nuclei average about 1.6 μm in diameter. During sporulation, plasmodia develop into sporocysts, containing 50 or more spores, with spore walls forming around each plasmodial nucleus. Unfixed spores are approximately 3.3 \times 4.3 μm , each having a cap with an overhanging lid. *Haplosporidium costale* can be distinguished from the related oyster pathogen, *H. nelsoni* by several features. Plasmodia, nuclei, and spores of *H. costale* are smaller than those of *H. nelsoni* and appear less clear in histological section. Nuclei of *H. costale* plasmodia have central, rather than peripheral, endosomes. Plasmodia of *H. costale* are present in all tissues except epithelia, whereas *H. nelsoni* is also found in epithelia. Sporulation of *H. costale* is common and takes place in all the tissues except the epithelia, in contrast to sporulation of *H. nelsoni*, which is rare in adults, and takes place in the epithelium of the digestive diverticula. It is difficult to distinguish *H. costale* plasmodia from *H. nelsoni* plasmodia in oysters heavily infected with both parasites and dual infections do occur. Molecular diagnosis using specific DNA primers and PCR, as well as *in situ* hybridization, is considerably more sensitive than tissue-section histology, although it is not currently in routine use.

Key references

- Andrews, J. D. 1982. Epizootiology of late summer and fall infections of oysters by *Haplosporidium nelsoni*, and comparison to annual life cycle of *Haplosporidium costalis*, a typical haplosporidan. *The Journal of Shellfish Research*, 2: 15–23.
- Andrews, J. D., and Castagna, M. 1978. Epizootiology of *Minchinia costalis* in susceptible oysters in seaside bays of Virginia's Eastern Shore, 1959–1976. *Journal of Invertebrate Pathology*, 32: 124–138.
- Andrews, J. D., Wood, J. L., and Hoese, H. D. 1962. Oyster mortality studies in Virginia III. Epizootiology of a disease caused by *Haplosporidium costale*, Wood and Andrews. *Journal of Insect Pathology*, 4: 327–343.
- Couch, J. A. 1967. Concurrent haplosporidian infections of the oyster, *Crassostrea virginica* (Gmelin). *The Journal of Parasitology*, 53: 248–253.
- Couch, J. A., and Rosenfield, A. 1968. Epizootiology of *Minchinia costalis* and *Minchinia nelsoni* in oysters introduced into Chicoteague Bay, Virginia. *Proceedings of the National Shellfisheries Association*, 58: 52–59.
- Ford, S. E., and Tripp, M. R. 1996. Diseases and defense mechanisms. In *The Eastern Oyster Crassostrea virginica*. Ed. by R. I. E. Newell, V. S. Kennedy and A. F. Eble. Maryland Sea Grant College, College Park, Maryland, pp. 383–450.

- Ford, S. E., Xu, Z., and DeBrosse, G. 2001. Use of particle filtration and UV irradiation to prevent infection by *Haplosporidium nelsoni* (MSX) and *Perkinsus marinus* (Dermo) in hatchery-reared larval and juvenile oysters. *Aquaculture*, 194: 37–49.
- Perkins, F. O. 1969. Electron microscope studies of sporulation in the oyster pathogen, *Minchinia costalis* (Sporozoa; Haplosporida). *The Journal of Parasitology*, 55: 897–920.
- Russell, S., Frasca, S., Sunila, I., and French, R. A. 2004. Application of a multiplex PCR for the detection of protozoan pathogens of the eastern oyster *Crassostrea virginica* in field samples. *Diseases of Aquatic Organisms*, 59: 85–91.
- Stokes, N. A., and Burreson, E. M. 2001. Differential diagnosis of mixed *Haplosporidium costale* and *Haplosporidium nelsoni* infections in the eastern oyster, *Crassostrea virginica*, using DNA probes. *The Journal of Shellfish Research*, 20: 207–213.
- Sunila, I., Stokes, N. A., Smolowitz, R., Karney, R. C., and Burreson, E. M. 2002. *Haplosporidium costale* (seaside organism), a parasite of the eastern oyster, is present in Long Island Sound. *The Journal of Shellfish Research*, 21: 113–118.
- Wang, Z. W., Lu, X., Liang, Y. B., and Wang, C. D. 2010. *Haplosporidium nelsoni* and *H. costale* in the Pacific oyster *Crassostrea gigas* from China's coasts. *Diseases of Aquatic Organisms*, 89(3): 223–228.
- Wood, J. L., and Andrews, J. D. 1962. *Haplosporidium costale* (Sporozoa) associated with a disease of Virginia oysters. *Science*, 136: 710–711.

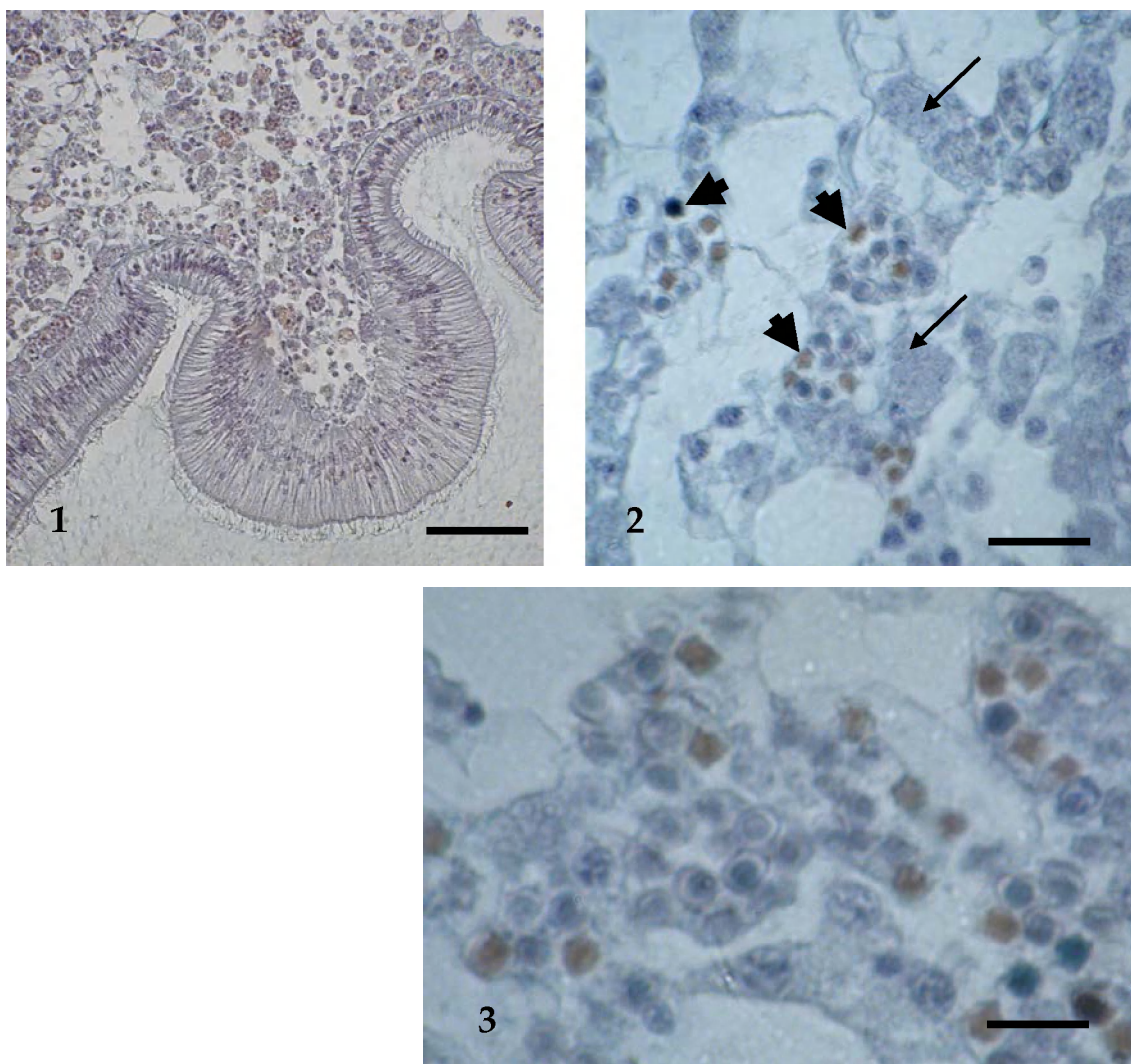


Figure 1. Tissue section of oyster heavily infected with *Haplosporidium costale*. Note the presence of all stages in the connective tissue and the absence of parasites in the stomach epithelium. Scale bar = 50 μm .

Figure 2. Plasmodial (long arrows) and spore (arrowheads) stages of *H. costale*. Scale bar = 20 μm .

Figure 3. Developing spore stages of *H. costale*. Scale bar = 10 μm .

Author Contact Information

Susan E. Ford
Rutgers University
Haskin Shellfish Research Laboratory
Port Norris, New Jersey, 08349 USA
susan@hsrl.rutgers.edu