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SSO Brief

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The State of Knowledge Concerning Haplosporidium costale,

or "Seaside Organism" (SSO), in the Eastern Oyster

SSO is a haplosporidian oyster parasite (*Haplosporidium coastale*) distantly related to the major pathogen MSX (*H. nelsoni*). SSO has been known since the early 1960s, when it was discovered in the seaside lagoons of Virginia's Eastern Shore (Wood and Andrews 1962). Unlike MSX, which thrives in euryhaline estuarine conditions, SSO is primarily active where salinities are above 25 (Ford and Tripp 1996). Disease in infected oysters is associated with proliferation of plasmodia and sporulation of the parasite within oyster connective tissues.

How pathogenic is SSO? Prevalence of SSO can exceed 50% among older oysters, and associated oyster mortality historically has been reported to reach similarly high levels (Andrews 1988). For this reason, it has been justifiably regarded as a pathogen of concern. In recent years, however, there have been few indications that SSO is a significant cause of oyster disease and mortality, particularly relative to MSX and *Perkinsus marinus* (dermo). The presence of SSO in southern New England (Connecticut waters of Long Island Sound) was confirmed by surveys in 1997-1999, but the parasite was observed at well under 1% prevalence (Sunila et al. 2002). Carnegie and coworkers (unpublished) evaluated a seaside Virginia oyster population in 2008 and observed SSO prevalence to reach 56%, but with few serious infections; the peak prevalence of infections of moderate or greater intensity was 12%. While most spring oyster mortality in higher salinity lagoons of the mid-Atlantic may be caused by SSO, it seems unlikely that this amounts to more than 10% annual mortality, and the actual figure may be far lower. More recently, SSO has been reported at higher prevalence at several sites in New England using more sensitive PCR methods (see below).

How is SSO transmitted? The life cycle of SSO is unresolved, but transmission may be indirect via one or more intermediate hosts. Ford and Tripp (1996) note that "experimental transmission of *H. costale* by feeding and injection of spores has not been successful (Andrews 1979)", which, as in the case of MSX, points to an indirect life cycle. These authors suggest that a direct life cycle for SSO is more plausible than for MSX given the temporal coincidence of sporulation and infection acquisition in the mid-Atlantic spring. If assuming an indirect life cycle for SSO, both oysters and any intermediate hosts would need to be present making transmission more difficult. If oysters are an aberrant host, SSO infections of oysters may be a dead end for the pathogen.

What is the distribution of SSO? There are reliable records of the presence of SSO in higher salinity waters from Virginia north to Canada, although its distribution is unlikely to be continuous through this range. Sunila et al. (2002) identified SSO in Connecticut waters of Long Island Sound, as noted above, and Roxanna Smolowitz (Roger Williams University) has communicated that she observed SSO on both shores of Cape Cod as well as at Martha's Vineyard while working in Massachusetts earlier in her career. A sample of 30 aquacultured *C. virginica* sent to Carnegie (VIMS) by a grower in Casco Bay in July 2009 revealed one heavy SSO infection, confirmed by PCR. PCR and DNA sequencing have identified SSO from oysters as far north as Nova Scotia, Canada (WGPDMO 2003). SSO has been detected as far afield as British Columbia, Canada, infecting *Crassostrea gigas* (WGPDMO 2009) and, apparently, New South Wales, Australia, infecting *Saccostrea glomerata* (Carnegie et al. 2014).

Recent history of SSO in New England. Cem Giray (Kennebec River Biosciences), Diane Murphy (Cape Cod Cooperative Extension and Woods Hole Sea Grant), Marta Gomez-Chiarri (University of Rhode Island) and Smolowitz have provided perspective based on their analyses of oysters from the region (personal communications). Gomez-Chiarri reported a relatively widespread increase in SSO prevalence in RI waters in recent years. Giray and Murphy shared detailed records of analyses conducted in 2013 and 2014 showing regular detection of SSO by PCR in pools of 3 or 5 oysters at sites on both the north and south shores of Cape Cod. In some cases, all pooled samples were positive, indicating a prevalence of 20% or 33% to 100%. They reported histological prevalence up to 60%, although all infections were very light in intensity as visualized microscopically and consisted exclusively of spore forms. It is conceivable that many of these apparent SSO cells were ceroid produced by the oyster host, and that the actual prevalence of infection was much lower; PCR prevalence can reflect detection of environmental SSO cells; see Ford et al. (2009) regarding environmental detection of MSX by PCR. Smolowitz indicated that her histological analyses more than a decade ago revealed SSO only "rarely" on either shore of Cape Cod. Overall, the data suggest a wide parasite distribution but with limited infection and disease development in the oyster host.

Resistance and susceptibility of genetic lines to SSO. There are not explicit studies of resistance and susceptibility to SSO. Given the production and evaluation of multiple lines of oysters in SSO-enzootic waters and the close scrutiny such lines receive, it seems acute susceptibility to SSO would likely have been noted at some point before the present.

What do we know about the seasonality of SSO infection and associated mortality? As summarized by Ford and Tripp (1996), infection acquisition in Virginia consistently occurs in May or June of each year, which is coincident with the timing of parasite sporulation in, and mortality of, oysters infected the previous year, or perhaps earlier. In the Northeast, infections

have been noted in fall (Sunila et al. 2002, and Giray), suggesting a seasonal cycle that is different than in southern waters, however poorly resolved.

References

Andrews, J.D. 1979. Oyster diseases in Chesapeake Bay. Marine Fisheries Review 41: 45---53

Andrews, J.D. 1988. *Haplosporidium costale* disease of oysters. Pp. 296---299 in Disease Diagnosis and Control in North American Marine Aquaculture (C.J. Sindermann and D.V. Lightner, eds.). Elsevier, Amersterdam

Carnegie, R.B., K.M. Hill, N.A. Stokes, and E.M. Burreson. 2014. The haplosporidian *Bonamia exitiosa* is present in Australia, but the identity of the parasite described as *Bonamia roughleyi* is uncertain. Journal of Invertebrate Pathology 115: 33---40

Connecticut Department of Agriculture. 2021 Statewide Shellfish Disease Update. https://portal.ct.gov/-/media/DOAG/Aquaculture/Pathology/2021-Statewide-Shellfish-Disease-Update.pdf. Accessed August 29, 2022.

Ford, S.E., and M.R. Tripp. 1996. Diseases and defense mechanisms. Pp. 581---660 in: The Eastern Oyster *Crassostrea virginica* (V.S. Kennedy, R.I.E. Newell, and A.F. Eble, eds.). Maryland Sea Grant, College Park

Ford, S.E., B. Allam, and Z. Xu. 2009. Using bivalves as particle collectors with PCR to investigate the environmental distribution of *Haplosporidium nelsoni*. Diseases of Aquatic Organisms 83: 159---168

Sunila, I., N.A. Stokes, R. Smolowitz, R.C. Karney, and E.M. Burreson. 2002. *Haplosporidium costale* (Seaside Organism), a parasite of the eastern oyster, is present in Long Island Sound. Journal of Shellfish Research 21: 113---118

WGPDMO. 2003. Report of the Working Group on Pathology and Diseases of Marine Organisms. ICES CM 2003/F:03

WGPDMO. 2009. Report of the Working Group on Pathology and Diseases of Marine Organisms. ICES CM 2009/MCC:01

Wood, J.L., and J.D. Andrews. 1962. *Haplosporidium costale* (Sporozoa) associated with a disease of Virginia oysters. Science 136: 710---711