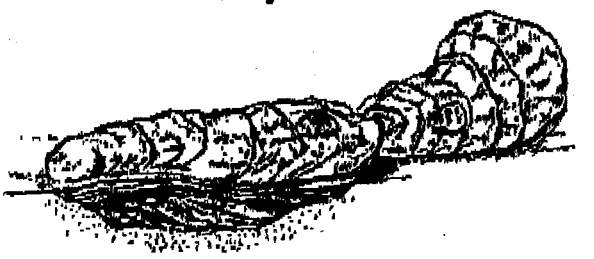
NOAA Technical Memorandum NMFS-SEFSC-404
(For use with NOAA Technical Memorandum NMFS-SEFSC-405, video)

JUVENILE OYSTER DISEASE FACT SHEET

C. Austin Farley and Earl J. Lewis



HOST SPECIES:

Common - scientific name:

AMERICAN OYSTER,
EASTERN OYSTER
Crassostrea virginica

DISEASE NAME:

Common or generic disease name:

JUVENILE OYSTER DISEASE (JOD)
(JUVENILE OYSTER MORTALITY)

ETIOLOGY:

Definitive etiology of the disease is unknown, although the disease agent is infectious, filterable and easily transmitted to susceptible oysters. Early hypotheses included involvement of a:

(1) toxic pollutant;
(2) toxic dinoflagellate;
(3) *Vibrio* sp.; and
(4) genetic disease.

These have been eliminated by experimental and epizootiological studies. Some evidence exists implicating an intracellular protist (possibly a parasitic ciliate such as a suctorian). The disease seems specific for *Crassostrea virginica* and affects juveniles, or older oysters, when exposed for the first time to the disease agent in aquaculture situations. The disease is rare in natural seed, even at heavily affected sites during periods of dense spat set. Other shellfish seed such as *Ostrea edulis*, *Mya arenaria*, and *Mercenaria mercenaria*, grown in JOD-infected culture facilities are not affected. Experimental transmission has been accomplished using infected oysters and filtrates of water from infected sites.

SIGNIFICANCE:

This disease has caused mortalities of 60 to nearly 100% in hatchery produced seed. This caused the demise, or near demise, of many oyster farming companies in New England and Long Island, NY. It has so far not affected naturally produced seed on the bottom of estuaries.

GEOGRAPHICAL DISTRIBUTION:

Thus far, the first known mortalities of oysters exhibiting the characteristic JOD syndrome were observed in September 1984. This occurred in second-year juvenile oysters from Maine, 20-30+ mm in size, cultured in Great Bay, NH (R. Langan, University of New Hampshire, Durham, NH, pers. commun.). By 1990, the disease had been documented in Maine, Massachusetts, Rhode Island, New York, New Hampshire, and Connecticut. Introductions of infected oysters into Maryland are not known to have established the disease. However, oysters introduced into Maryland and maintained in low salinity waters demonstrated the disease when held at 20° C in high salinity aquaria even after overwintering for 6 months at 5‰.

GROSS CLINICAL SIGNS:

(1) Cessation of growth in otherwise fast growing juveniles in good condition occurs 1 to 2 weeks prior to the onset of mortality. Affected oysters are generally in the size range of 5-30 mm;

(2) Presence of a characteristic ring of conchiolin on the internal surface of the shell(s) in affected animals that has not become fully incorporated into the shell matrix. However, conchiolin may not be seen by the unaided eye in the first oysters to die of JOD;

(3) Conspicuous shell check(s) which mark the size of the animal where active disease occurred;

(4) Severe mantle recession occurs which produces shell checks during regrowth of the shell; and

(5) Sometimes a deep cupping or directional growth change of the shell.

Mortality onset occurs rapidly. Initial losses of 2-3% the first week may be missed, but are followed by a rapid rise in heavy mortalities for 3-7 weeks before leveling off. Mortalities may continue at lower levels throughout the cold winter months.

HISTOPATHOLOGY:

The earliest evidence of infection is the presence of 2-5 µm cells with 1-3 µm Feulgen picromethyl blue (FPM) positive nuclei in vacuoles in the cytoplasm of healthy mantle epithelial cells. The second stage shows multiple cells in the vacuoles, sometimes with what appear to be macro- and micronuclei. The third stage is characterized by the rupture of the vacuoles to produce ulcers that in turn initiate an inflammatory response in the adjacent connective tissue composed of hyaline hemocytes. The ulcers cause hemorrhage and are believed to induce the deposition of conchiolin in concomitant locations on the shell. Necrosis is commonly observed in affected mantle tissue.

DISEASE ONSET:

The disease strikes susceptible oysters in infected areas 3-5 weeks after the water temperature exceeds and maintains 20°C. Areas of Long Island, NY, typically see the onset of JOD mortalities in late June or early July. In Maine, disease onset is delayed until mid-August to early September.

ASSOCIATED ENVIRONMENTAL CONDITIONS:

Salinities above 18‰ and temperatures above 18°C are conducive for initiating the disease process. Incubation period is 3-7 weeks, depending on water temperature. The disease is inhibited by salinities below 16‰ and erythromycin (a commercial product for marine aquaria). The disease may be exacerbated by stresses, such as poor nutrition, but is not caused by nutritional deficiencies since the disease is clearly transmissible and inhibited by low water temperature, low salinity and erythromycin.

CONTROL:

The first effective management approach was to produce seed earlier in heated water by spawning oysters in January through March. This allowed juveniles to grow above 30 mm in length by the time water temperatures attained 20°C. The strategy of spawning oysters late in the summer to take advantage of declining water temperatures

appears to work in Maine, but not in more southern, warmer water areas. The second and most effective approach was to produce resistant oysters from brood stocks that had survived the disease and had strong evidence of characteristic shell checks. Survival of these seed (0-13% mortality) was 4 - 20 times better than comparable-aged susceptible juveniles (55-85%) in field tests. No significant difference in survival was observed in F_1, F_2, or F_3 generations of resistant oysters. Increased water flow (≥ 21 L/min in a 30 cm silo) for upweller grown juveniles reduces, but does not eliminate, infection and resultant mortalities (G. Rivara, Cornell Cooperative Extension, Southold, NY, pers. commun.). In the same study, density of oysters was not found to reduce the effects of JOD significantly. These procedures have allowed one company to go from nearly total production failure, to the best production in 35 years of operation. There is some evidence that resistant seed produced in local waters may have better survival than those developed and brought in from other locations. Results of experimental transmission studies showed the disease agent was able to pass through 5 µm mesh bag filters typically used by aquaculture facilities, but not a 1 µm filter bag.

COMMENTS:

This is one of the few examples of a success story based on the application of techniques from observational and experimental research on a molluscan disease problem, and yet the etiological agent is still unidentified.

KEY REFERENCES:

BRICELJ, V.M., S.E. FORD, F.J. BORRERO, F.O. PERKINS, G. RIVARA, R.E. HILLMAN, R.A. ELSTON, and J.CHANG, 1992. Unexplained mortalities in hatchery-reared juvenile oysters, *Crassostrea virginica* (Gmelin). J. Shellfish Res. 13, 137-142.

DAVIS, C.V. and B.J. BARBER, 1994. Size-dependent mortality in hatchery-reared populations of oysters, *Crassostrea virginica* Gmelin, 1791, affected by juvenile oyster disease. J. Shellfish Res. 13, 137-142.

FORD, S.E. and M.R. TRIPP, 1996. Diseases and defense mechanisms. In: V.S. Kennedy, R.I.E. Newell and A.F. Eble, (eds.), The eastern oyster, *Crassostrea virginica*. Univ. Maryland Sea Grant College, College Park, MD. pp. 587-591.

LEWIS, E.J., C.A. FARLEY, E.B. SMALL, and A.M. BAYA, 1996. A synopsis of juvenile oyster disease (JOD) experimental studies in *Crassostrea virginica*. Aquat. Living Resour. 9, 169-178.

PAILLARD, C., K.A. ASHTON-ALCOX, AND S.E. FORD, 1996. Changes in bacterial densities and hemocyte parameters in eastern oysters, *Crassostrea virginica*, affected by juvenile oyster disease. Aquat. Living Resour. 9, 145-158.

ACKNOWLEDGMENTS:

The authors wish to acknowledge the NOAA Oyster Disease Research Program and the Northeastern Regional Aquaculture Center for

funding of scientific projects and the numerous oyster growers in the northeastern United States for their cooperation and assistance in achieving our research goals. Of special acknowledgment for their outstanding support are the employees of Frank M. Flower & Sons, Inc.; Pemaquid Oyster Co.; Moonstone Oysters; Blue Points; and Gregg Rivara, Cornell Cooperative Extension Agent.

Desktop Publishing by Derek Orner, NOAA Chesapeake Bay Office; editing by Jane Keller, NMFS Cooperative Oxford Laboratory.

Copies may be obtained from:
National Marine Fisheries Service, NOAA
Southeast Fisheries Science Center
Cooperative Oxford Laboratory
904 South Morris Street
Oxford, MD 21654-9724
(410) 226-5193
Fax (410) 226-5925

or

National Technical Information Service
5258 Port Royal Road
Springfield, VA 22161
(703) 487-4650, Fax (703) 321-8547
Rush orders: (800) 336-4700

Correct citation of this report is: Farley, C.A. and E.J. Lewis. 1997. Juvenile oyster disease fact sheet. NOAA Tech. Memo. NMFS-SEFSC-404, 8 pp.