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## Roseovarius Oyster Disease (ROD) caused by *Roseovarius crassostreae*

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# Roseovarius Oyster Disease (ROD) caused by *Roseovarius crassostreae*

by Susan E. Ford

## Susceptible species

Eastern oyster, *Crassostrea virginica*

## Disease name

Roseovarius Oyster Disease (ROD), formerly Juvenile Oyster Disease (JOD)

## Aetiological agent

*Roseovarius crassostreae*, an alpha-proteobacterium, which is the numerically dominant bacterium isolated from ROD-affected oysters and has been found in all ROD outbreaks so far examined. Challenge with *R. crassostreae* induces ROD-like conchiolin deposits and the bacteria can be re-isolated from affected individuals. *R. crassostreae* has also been detected in oysters in concert with the earliest microscopic tissue lesions and the development of gross disease symptoms.

## Geographical distribution

Major outbreaks and associated mortality have been reported in cultured oysters from New York to Maine (USA), with few or no reports from more southern sites. A similar syndrome has been reported in eastern oysters experimentally kept in a French hatchery.

## Associated environmental conditions

ROD outbreaks typically occur in rapidly growing juvenile eastern oysters during their first summer of culture. Onset of disease symptoms occurs when water temperature has exceeded about 21–22°C. Outbreaks have been limited to high salinity (25–32) sites. Experimental transmission was ineffective at salinity and temperature below 18 and 18°C, respectively. The disease seems to be related to a variety of high-density culture conditions (upwellers, trays, bags).

## Significance

Mortalities of up to 90% occur in animals <25 mm; larger juveniles show characteristic inner-shell organic deposits, but have much lower mortalities. For nearly ten years, ROD was a major impediment to hatchery-based oyster culture in the northeastern United States, but the incidence of the disease has diminished significantly since the late 1990s.

## Gross clinical signs

Sharply decreased growth rates often precede the appearance of gross ROD signs. The principal signs include (i) extreme cupping of the lower valve, (ii) breaking off of the growing edge of the upper valve, leaving a band of exposed, fouled, inner shell on the lower valve, and (iii) secretion of a conchiolin layer, on both valves, that surrounds the soft tissues, which retract well into the shell cavity. The conchiolin layer is raised into a ridge at the periphery of the mantle, resulting in the appearance of a distinct “brown ring” on the inner shells of affected oysters. The conchiolin layer

frequently disrupts the adductor muscle attachment, causing the soft tissues to fall out of the shell. Oysters that survive an ROD outbreak often have a pronounced growth check visible on both the inner and outer shell.

### Control measures and legislation

Selective breeding has produced oyster strains with significantly better survival than unselected controls. Some of the improvement appears to be due to more rapid growth so that oysters reach the critical 25 mm size before the onset of disease in midsummer. A similar outcome can be achieved by early season production and deployment of juveniles to give them a head start on growth. Decreasing density in trays and bags, increasing mesh size, and increasing flow rate through upwellers have all proven effective in reducing losses. ROD is not an OIE listed disease.

### Diagnostic methods

The disease is typically diagnosed by its shell signs, principally the anomalous conchiolin deposit. The prevalence of the conchiolin deposit is highly correlated with mortality in oysters between about 10 and 25 mm shell height, but smaller oysters in which *R. crassostreae* is detected may die without the appearance of shell anomalies. Mantle and gill lesions are the principal histopathological correlates with ROD, but cannot be considered specific for the condition. A PCR assay for the detection of *R. crassostreae* is available based on the 16S-23SrDNA internal transcribed spacer region. The PCR sample should be collected by swabbing the inner shell surface. This method, which can detect as few as ten bacteria, is preferable to tissue sampling, which can reduce the assay's sensitivity.

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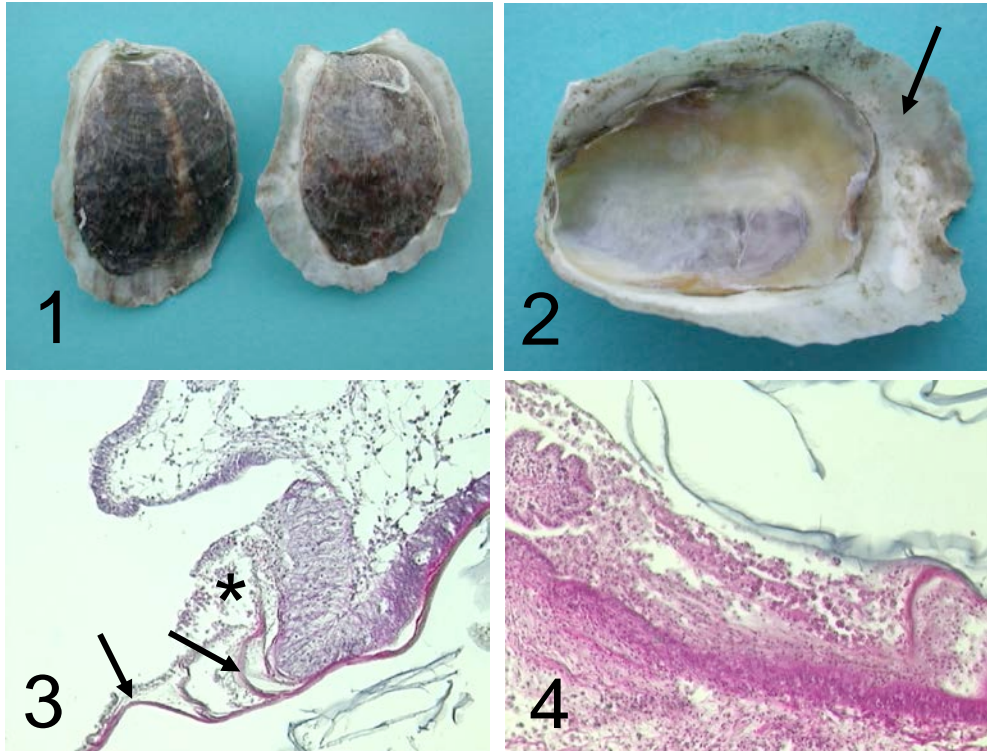


Figure 1. Example of uneven shell margins caused by the loss of the fragile growing edge of the upper valve.

Figure 2. Example of conchiolin deposits on inner valve, showing fouling outside the raised ridge (arrow).

Figure 3. Histological section through mantle edge, showing anomalous conchiolin deposit (arrows) and exudate of hemocytes (\*).

Figure 4. Severe lesion on mantle surface. Epithelium has been almost completely eroded.

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