

5.2.4 Velar Virus Disease of Pacific Oysters

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A. Name of Disease and Etiological Agent

Oyster velar virus disease (OVVD) is also referred to as "blisters" by oyster hatchery biologists. Morphologically, the etiological agent is an iridovirus.

B. Known Geographical Range and Host Species of the Disease

1. Geographical Range

The disease has been reported only from oyster hatcheries in Washington state. Given the historical commerce of this oyster, it is likely that the range is much broader along the Pacific Rim.

2. Host Species

The etiological agent is known to infect only veliger larvae of the Pacific oyster *Crassostrea gigas*. Morphologically similar viruses have been reported in adult Portuguese and Pacific oysters, but have not been definitively linked to OVVD.

C. Epizootiology

Oyster velar virus disease can cause nearly 100% mortality in affected hatchery tanks. Environmental factors that affect the disease or the oysters' susceptibility are not known. The disease typically appears in March to May, but it has also been reported throughout the summer. Mortalities occur in larvae greater than 150 μm in shell length and typically begin at about 10 days of age when cultured at 25 to 30°C.

D. Disease Signs

Larvae with advanced lesions accumulate on tank bottoms and show deciliation of the velum. Loss of ciliated velar epithelial cells, which may appear as blebs on the periphery of the velum as they are sloughed, form the characteristic "blisters" associated with the disease (see Figure 1 and Figure 2).

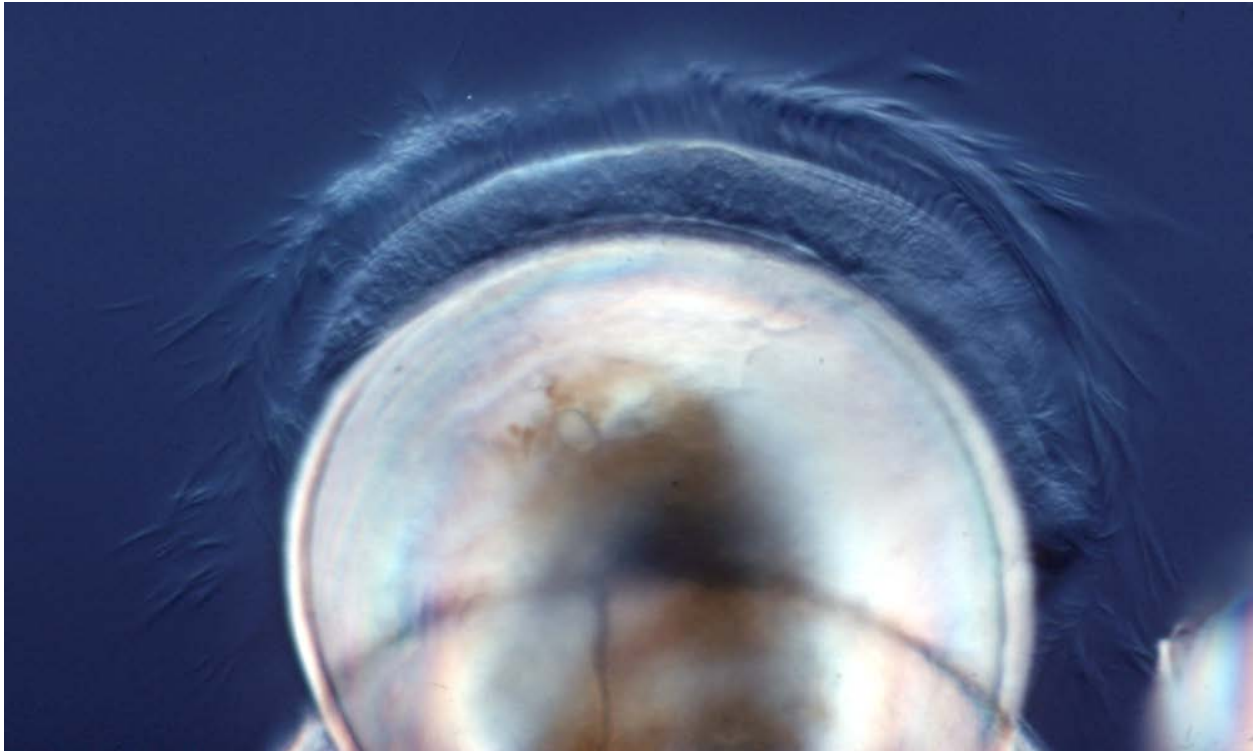


Figure 1. Wet mount of normal Pacific oyster larvae showing the extended intact ciliated velum, 383X. From Elston and Wilkinson 1985, with permission.

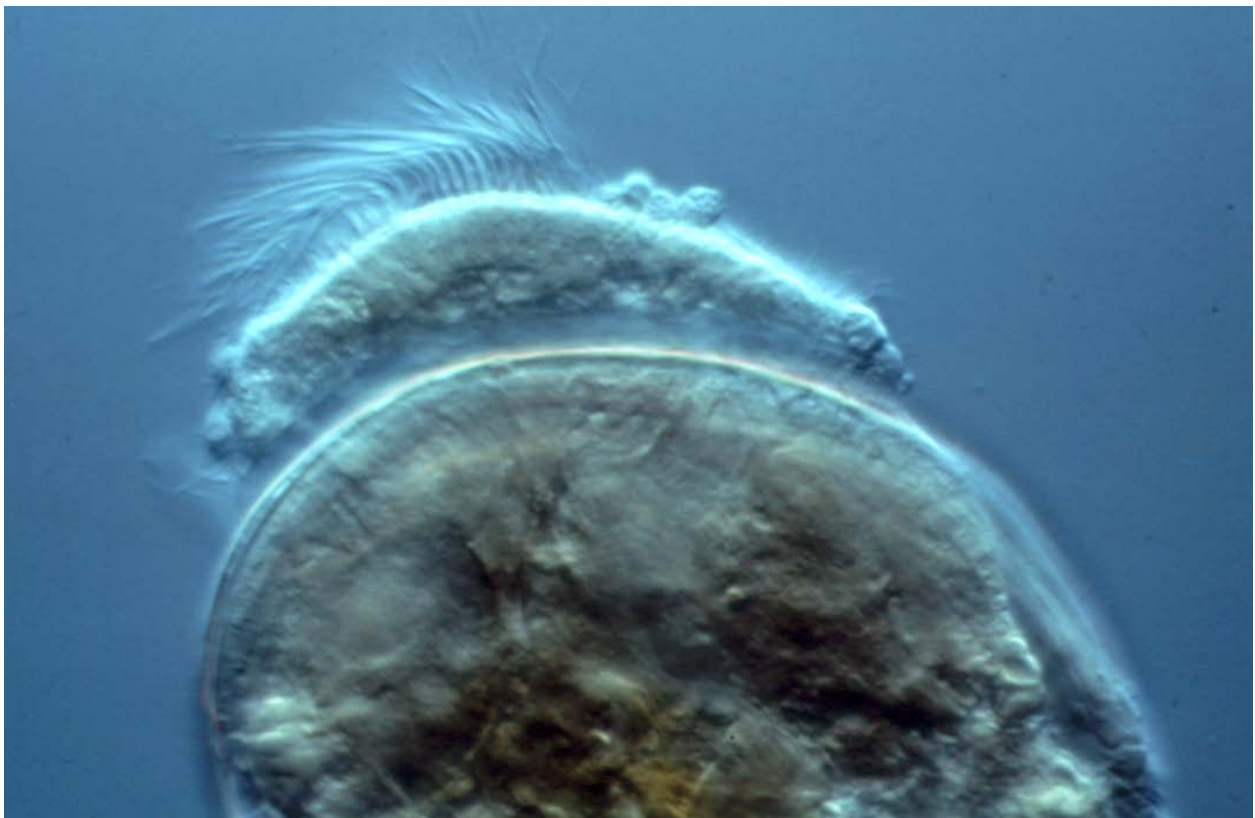


Figure 2. Wet mount of OVVD infected Pacific oyster larva showing velar degeneration including loss of cilia and loss of epithelial cells. As the cells round up and are in the process of detaching from the velum, they form the characteristic blisters.

E. Disease Diagnostic Procedures

1. Presumptive Diagnosis

A strong presumptive diagnosis can be made if characteristic intracytoplasmic inclusion bodies and associated lesions (Figure 3) are observed in conjunction with the clinical signs noted above. Inclusion bodies are located most commonly in ciliated velar epithelium, but may occasionally be observed in velar supporting epithelium, esophageal, and oral epithelium. In velar ciliated epithelium, inclusion bodies are often found in sloughing cells, which may be rounded and detached within the velar cavity as the velar architecture degenerates. Inclusion bodies are spherical, dense, and basophilic in early stage infections, but become irregular and less basophilic as virions form.

2. Confirmatory Diagnosis

Electron microscopic observation of virus-like particle profiles in velar or other infected epithelium. These particles have icosahedral symmetry are about 228 nm in diameter.

F. Procedures for Detecting Asymptomatic Infections

No methods are reported.

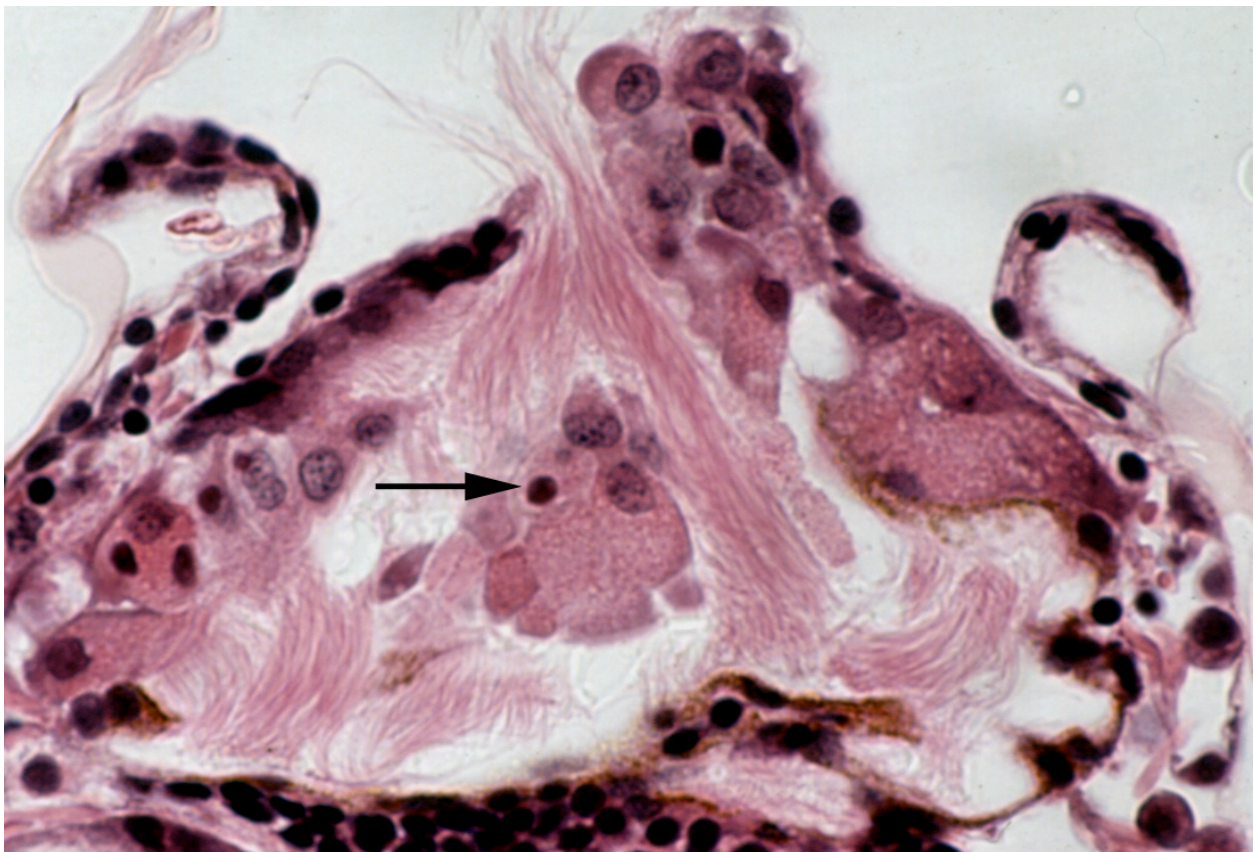


Figure 3. The intracytoplasmic inclusion body in detached velar epithelial cell (arrow) of *Crassostrea gigas* is characteristic of oyster velar viral disease, 2225X.

References

- Elston, R. 1979. Virus-like particles associated with lesions in larval Pacific oysters (*Crassostrea gigas*). *Journal of Invertebrate Pathology* 33:71-74.
- Elston, R. A., and M. T. Wilkinson. 1985. Pathology, management, and diagnosis of oyster velar virus disease (OVVD). *Aquaculture* 48:189-210.