



Figure 8. European flat oyster blood cells, highly magnified. The cell in the center of the photograph (arrow) is infected with *Bonamia*, the smaller round spheres within the blood cell. (From Elston et al. 1986)

area on a commercial scale, test batches should be introduced and examined over a two-year period. Thus, the total eradication period will take at least five years.

If economic or other practical considerations prevent the eradication approach, some steps can be taken to reduce the effects of the disease. It is known that mortalities due to bonamiasis are reduced in off-bottom culture methods.

Reducing the density of oysters is also believed to reduce the transmission of the disease, as is the use of subtidal rather than intertidal growing areas. In addition, it appears that some stocks of flat oysters may acquire resistance to the disease. These populations of oysters still carry the infectious parasite and some individual oysters succumb to the disease, but many appear to tolerate and grow well in spite of the infection.

Infected oyster populations should not be used as brood stock for seed to be planted into disease-free areas. There is no reason, however, to avoid the introduction of infected oyster seed into areas known to be infected and in which eradication is not possible.

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Marteiliasis of the European Flat Oyster (*Ostrea edulis*)

Marteiliasis (sometimes called Aber disease) is caused by a parasite, *Marteilia refringens*, that infects the connective and digestive tissues of the oyster. Spores (the mature stage of the parasite) are formed in the epithelium of the digestive tubules. The disease is responsible for flat oyster mortalities that began in 1967 along certain regions of Atlantic France and Spain.

A related parasite of the Australian rock oyster, *Saccostrea commercialis*, is *Marteilia sydneyi*. This parasite has caused heavy mortalities of the rock oyster in Moreton Bay, Queensland, Australia.

Geographic Range and Species Infected

Marteiliasis occurs only on the Atlantic coast of Europe. Serious disease resulting from the parasite infection, first reported from Aber Wrach in Brittany in 1967, occurs in other areas of France and in Spain as well. *Marteilia* parasites have been observed in Dutch flat oysters but without significant disease or mortality.

The disease occurs only in *Ostrea edulis*, but the parasite has been found, according to a single report from France, in a few specimens of the Pacific oyster, *Crassostrea gigas*. No significant detriment to health was reported in the Pacific oyster as a result of the infection, but the identity of the parasites observed needs to be confirmed as *Marteilia refringens* before accepting this as a definitive observation.

Mortality Rate, Environmental Factors, and Seasonality

Mortality rates of 90% annually were reported in the first epizootics of disease in France. When disease-free spat or two- and three-year-old oysters were planted in infected areas in March, they became infected between the first of May and the end of August. Severe mortalities occurred before the end of the first winter, but the parasite could not be found in the surviving oysters. The fact that the parasite occurs in oysters in some areas without causing disease suggests that environmental factors or oyster stock differences are important in determining whether or not the disease becomes a significant problem.

In addition, mortality seems to be related to the formation of the spore stages (known as sporulation) of the parasite within the oyster tissues. The sporulation process may result in the release of toxic substances that affect the oyster.

Diagnosis

Heavily infected oysters may have normally dark colored digestive glands and abundant glycogen stored in the connective tissues. In some cases, however, in diseased

oysters with advanced infections, the mantle is colorless, the digestive gland is pale yellow rather than brown, and the visceral mass is slimy and shrunken. Definitive diagnosis requires the histological examination of the tissues by a professional pathologist.

Prevention and Management

Disease-free Areas

Infected oysters should not be moved into disease-free areas.

Areas Known to Have the Disease

No management methods are known for the disease.

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Gill Disease of the Portugese Oyster (*Crassostrea angulata*)

Gill disease is a term used to describe the condition of Portuguese oysters (*Crassostrea angulata*) subject to a massive mortality which eventually resulted in the elimination of the culture of this oyster from all parts of France and Portugal. The cause is not certain, but several infectious agents have been proposed, among them parasites and a virus. Severe losses were reported in 1967-68 and 1970-73 in the Ile de Oleron and Archachon regions of France and in regions of Portugal.

Geographic Range and Species Infected

Gill disease in the Portuguese oyster has been reported in France, Portugal, and Great Britain. A disease with similar signs has been reported in the European flat oyster,

Ostrea edulis, in several European countries, but it has not been identified definitely as the same disease as afflicts the Portuguese oyster.

Mortality Rate, Environmental Factors, and Seasonality

Because of the devastating effect of gill disease, the Portuguese oyster is no longer cultured in the Ile de Oleron, a major production area for oysters in France. That region now cultures the Pacific oyster, *Crassostrea gigas*.

When Portuguese oysters were imported to Great Britain, it was reported that within three weeks the percentage of oysters showing the "active" disease, in which the gills eroded and were found to contain dead tissue, increased from 2% to 60%. This active stage of the disease was found primarily in spring and summer.

Diagnosis

A preliminary diagnosis can be made on the basis of visible signs. The disease first appears as yellow spots on the gills. As these spots enlarge, the centers become brown and necrotic, resulting in a perforation of the gill, or a V-shaped indentation if the lesion occurs at the edge of the gill. Yellow or green pustules may appear on the adductor muscle or mantle; on the mantle they may develop into perforations. These perforations or indentations of the gill may be found in recovering oysters, but the lesions lack the decaying yellow and brown tissue typical of the active stage of the disease.

Diagnosis can be confirmed by a shellfish pathologist. However, as noted above, the exact cause of the disease has not been determined, although the gills of some oysters with lesions contain a virus.

Prevention and Management

Little is known about the prevention and management of the disease. However, since there is some evidence that a virus or other infectious agent causes the disease, it is not advisable to move oyster stocks known to have had the disease to areas where the disease has not been reported.

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Hexamitiasis of *Ostrea* and *Crassostrea* Oysters

Hexamitiasis is caused by a parasite known as *Hexamita nelsoni*. The disease is also known as "pit disease," a name derived from the belief that it has been responsible for flat oyster mortalities in recirculating water basins, or pits, in Holland. The parasite is considered to be cosmopolitan, that is, to occur commonly throughout the world under suitable conditions.

The parasite is often found in the blood stream and within blood cells in dying oysters, and there is some controversy as to whether it actually causes a disease or simply takes advantage of an oyster already sick from some other cause. The only oyster for which the true disease-causing nature of the parasite has been shown is the Olympia oyster, *Ostrea lurida*, although hexamitiasis has been reported in several other species.

Geographic Range and Species Infected

As noted above, a true disease-causing relationship to the oyster has been established only in *Ostrea lurida* in Puget Sound, Washington. Other species and locations of infection have been reported as follows: *Crassostrea commercialis* (Australian rock oyster), Australia; *Crassostrea gigas* (Pacific oyster), Pacific Northwestern United States; *Crassostrea virginica* (American oyster), Prince Edward Island, Canada; and *Ostrea edulis* (European flat oyster), Holland and the maritime provinces of Canada.

Mortality Rate, Environmental Factors, and Seasonality

Mortality rates have not been recorded precisely, but in certain years oyster farmers have estimated mortalities of about 75% over a two-month period in association with hexamitiasis in *Ostrea lurida*. This is definitely a cold-temperature disease in this species. Experiments show that infection and debilitating disease occur at 6°C and lower but not at 12°C or higher. Mortalities associated with this disease are usually reported in winter, but in Alaska and other northern zones the disease has been found at other times of the year as well.

Diagnosis

A preliminary diagnosis can be made microscopically on a drop of oyster blood. The causative organisms are highly motile by means of their flagella. Confirmation by examining tissues must be made by a shellfish pathologist.

Prevention and Management

Since the causative organism is considered to be cosmopolitan, any oyster-growing area is potentially subject to hexamitiasis. In one of the original publications on the disease in *Ostrea edulis* in Holland (see reference by Mackin et al. 1952), it was suggested that cold temperature, poor circulation over the oyster basins, and overcrowding are optimal conditions for an outbreak of the disease. This is the only published information that provides any hint toward disease management of hexamitiasis.

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Shell Disease of Oysters

Shell disease, first described in 1894, is caused by a fungus known as *Ostracoblabe implexa*. Technically, the disease is known as oyster ostracoblabiasis in reference to the fungus. Serious mortalities are thought to have resulted from the disease in Europe at various periods during the 19th and 20th centuries. The disease has been known as *maladie du pied* ("disease of the foot," even though the adult oyster does not have a foot) and *maladie de la charnière* ("disease of the hinge ligament").

The filamentous fungus grows through the shell, weakening it and causing dark raised warts on the interior shell surface. In advanced cases, warts occur in the hinge region and cause excessive and abnormal hinge development. The result may be a beaked appearance to the hinge area of the shell and malformed valves that do not close properly.

Geographic Range and Species Infected

The full disease syndrome, including the formation of warts, occurs only in the European flat oyster (*Ostrea edulis*) in the Netherlands, France, Great Britain, and Nova

Scotia in North America. Part of the disease syndrome, not usually including the wart stages, occurs in *Crassostrea* species. In the Portuguese oyster (*Crassostrea angulata*) a form of shell disease is reported in the Netherlands, France, and Great Britain. Recently the disease was reported in *Crassostrea cucullata* in India. There are reports that a similar disease occurs in the American oyster (*Crassostrea virginica*) on the Atlantic seaboard of North America and in *Crassostrea gryphoides* in India, but these are not confirmed to be shell disease. The fungus that causes the disease is probably common to all marine coastal environments.

Mortality Rate, Environmental Factors, and Seasonality

Shell disease may have caused massive mortalities of *Ostrea edulis* in the Netherlands at various times and has also been claimed to be associated with severe oyster kills in France. Definitive proof that the disease is responsible for the oyster kills is lacking. Oysters are infected above 20°C, either by a waterborne fungus or by direct growth of the fungus from one oyster to adjacent oysters. Young oysters are reported to be more susceptible than older oysters. In the Netherlands, cockle shells, used as spat collectors, were suspected of containing the disease-causing fungus, ensuring that new oyster spat would become infected at an early age.

Diagnosis

A strong presumptive or probable diagnosis can be made on the basis of lesions on the oyster shell. The initial stage of shell disease in one-year-old oysters is the occurrence of small, bright white spots in the growing margin of the shell. This early stage can be cured by chemical treatment, but not the later stages characterized by the "warts" described below.

As the disease progresses, white spots from 0.5 to 3.0 mm in diameter occur on the inner surface of the shell. These spots form a small, slightly raised rough area. A dark indentation in the center of the area indicates that the fungus has penetrated into the mantle cavity. These infected spots coalesce to form the typical "cloud," also with a characteristic rough surface, as the infected area of the shell matrix enlarges. The pallial surface of the shell may acquire a brownish tint in advanced infections.

Formation of "warts" is common. These consist of small green to black protrusions attached to the inner shell surface, often in the area of the adductor muscle attachment and the hinge region but also at other sites on the inner shell surface. Excessive and abnormal hinge deposition may occur and result in a beaked appearance of the dorsal region and inability to effect normal shell closure.

Diagnosis can be confirmed by microscopic examination of the warts and weakened infected shell for the typical forms of the fungus. Fresh shell material should be submitted to the pathologist or preserved for later examination.

Prevention and Management

Disease-free Areas

Since the geographic extent of the disease is not known for certain, it is advisable not to import shell or live oysters from areas known to have the disease into areas where the disease is not known to occur.

Areas Known to Have the Disease

The disease was controlled in the Netherlands by dipping the oysters in a solution of mercuric chloride. However, given our current knowledge of mercury toxicity, this method should not be attempted. It is likely that other methods of killing the fungus would also be effective, such as dipping the oysters in a solution of 15 parts per million sodium hypochlorite (bleach) for 10 minutes or longer. This concentration is made by diluting household bleach containing 5.25% sodium hypochlorite by a factor of 3,500. In the Netherlands, old shell was removed from the oyster beds in order to eliminate a source of the fungus, and areas where young oysters are placed were kept free of dead shells in order to limit the effects of the disease.

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