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Diseases of Sydney rock oysters

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The Sydney rock oyster (*Saccostrea glomerata*) is considered a gourmet's delight, and is the mainstay of the oyster industry in New South Wales.

Since 1990, production has averaged 8 million dozen oysters per annum and has been worth around $34 million. Oyster farming is the oldest aquaculture industry in NSW, and has been a valuable contributor to regional economies of the state for over 100 years.

Unfortunately, like other farm animals (such as cattle, sheep, pigs, poultry and fish), oysters also suffer from diseases. Just like in other farm animals, there is an interaction between environment and disease, and oyster farmers can modify their farming practices to minimise both the incidence and impact of some diseases. The practice of 'highway oyster farming', which involves transfer of oysters between estuaries for on-growing, was established during the 1960s to increase production, which it has done – but it also increases the risk of spreading diseases.

Winter mortality

Winter mortality is caused by a protozoan parasite, *Bonamia roughleyi*, which occurs over the southern or cooler half of the range in which Sydney rock oysters are farmed. The area between Port Stephens and the Victorian border is particularly susceptible to winter mortality. Outbreaks of the disease are patchy within this range and, while mortality may occur in winter, most of the oysters do not die until the warmer spring weather of September or October. Winter mortality may kill up to 80% of oysters in a local area and, in general, oysters in their third winter (just before they reach market size) are most susceptible. In severe outbreaks, small spat (baby oysters) may also be affected. The severity of the kill can vary markedly between years, but also between estuaries, between adjacent leases and even within leases. Oysters grown on trays are no more susceptible to winter mortality than oysters grown on sticks. Dry autumns (high salinities), early winters and low temperatures increase the likelihood of a severe kill.

Reducing the impacts of winter mortality

Farmers can, to a large extent, reduce the impact of this disease by moving oysters further upstream to areas of lower salinity before the end of autumn (May), and by increasing the growing height of the oysters to 150 mm above normal growing height. Alternatively, farmers may sell their oysters for consumption before the onset of the disease.

Although triploid Sydney rock oysters are not currently available commercially, they suffer less than half the mortality of diploids exposed to winter mortality. Triploid oysters (which have three sets of chromosomes instead of the usual two sets), reach plate size (50 g) six months earlier than wild caught oysters (diploids) of the same age, and hold their meat condition better in winter and spring. Although triploids may suffer discolouration of meat in summer and autumn, for farmers who want to grow a winter crop that is partially resistant to winter mortality, triploid oysters may be a future option.

NSW DPI has developed breeding lines that are resistant to two major diseases of Sydney rock oysters. Winter mortality disease-resistant breeding lines are available for commercial production. So far (April 2007), mortality from this disease has been cut by half through selective breeding. Further progress is being made to reduce kill from winter mortality much further. Tests need to be carried out to determine if the reduction in losses from winter mortality by triploidy and selective breeding can be combined in the same oyster to enhance survival.

QX disease

'Queensland unknown' or 'QX' disease is caused by another protozoan parasite, *Marteilia sydneyi*, and is often responsible for the deaths of large numbers of oysters. QX disease is seasonal; infections usually occur from January to April, with

diseased oysters losing condition and dying through the winter and early spring. The life cycle of the parasite is thought to include an intermediate host, the identity of which remains unknown. Current understanding is that healthy oysters do not become infected with QX disease by association with infected oysters – there is currently no evidence of direct oyster-to-oyster transmission of this disease. In addition, the presence of the disease-causing agent, *Marteilia sydneyi*, does not necessarily result in expression of QX disease. Recent research by Queensland Museum in association with NSW DPI found that the parasite was present without causing disease in the majority of oyster-growing estuaries in NSW. What turns infection from benign to virulent is not known, but interactions between environmental and nutritional factors, oyster health and susceptibility, and elements of the parasite's life cycle are being investigated.

Effects of QX disease in NSW

QX disease was first detected in the northern, warmer estuaries, and was responsible for a decline in the oyster industry in southern Queensland and in the Tweed, Richmond and Clarence rivers of northern NSW during the 1970s.

Prior to the commercial availability of a hatchery and breeding program, farmers in northern NSW and southern Queensland resorted to importing half-grown oysters from non-QX infested southern estuaries. These oysters would then rapidly grow to harvest size and be sold for consumption during summer, before the main QX disease risk period (January–April). Some farmers in QX-affected estuaries still employ this 'QX avoidance' strategy.

In 1994, QX disease was first diagnosed in Georges River. This is the most southerly outbreak of the disease to date (April 2007), and it devastated oyster production in Georges River, which has not recovered.

The Hawkesbury River was hit by QX disease in 2004, and by 2006 production had been reduced to virtually nil. Fortunately, oyster farmers in the Hawkesbury River had two alternate crop options which were not available to Georges River farmers in 1994 – triploid Pacific oysters and hatchery-produced, QX disease-resistant Sydney rock oysters. By 2005, NSW DPI's oyster breeding program had developed QX disease-resistant Sydney rock oysters which were ready for a commercial trial by oyster farmers. These QX disease-resistant Sydney rock oysters reached market size in 24 months and suffered approximately 13% mortality, whereas non-resistant control oysters suffered 84% mortality.

Industry uptake of disease-resistant, hatchery-produced stock has been rapid, and commercial-scale farming of disease-resistant stock now occurs in most estuaries. In addition, research continues, and further improvements in QX disease resistance have been made.

Although resistance to QX disease has not conferred resistance to winter mortality, a dual disease-resistant breeding line was developed by exposing broodstock to QX disease over summer (December–March) and winter mortality over winter (April–September). These oysters also grow much faster: by 2007, after six generations of selection

Comparison of QX disease resistant (background tray) and control oysters (foreground tray) in Hawkesbury River. Equal numbers of similarly sized spat were placed in each tray in July 2005. The photo shows the number of survivors and their size in November 2006.

for fast growth, the time to market size (50 g whole weight) had been reduced by 10 months from the usual 38 months.

Managing QX disease in NSW

Although farmers on the Tweed, Richmond and Clarence rivers have for many years employed strategies to avoid problems with QX disease, oyster production in these rivers has not recovered since QX disease was first identified there in the 1970s. Many oyster farmers in these rivers have now left the industry. It should be noted that the practice of farming oysters imported from QX-free estuaries may have inhibited the development of resistance in the local oysters in these estuaries.

Current NSW DPI quarantine regulations for QX disease prohibit the movement of oysters from QX-recurrent estuaries into disease-free estuaries. Movement restrictions were first put into place in 1986. At present, NSW DPI, in collaboration with other researchers, NSW Farmers Association and the oyster industry, is developing a QX-risk based approach to managing inter-estuary oyster movements.

Mudworm

The mudworm, *Polydora websteri*, is thought to be the most damaging of four species of polychaete worms which infest and kill large numbers of oysters. The siltation of shell beds and dredge beds and the appearance of mudworm early in the 20th century forced the industry into intertidal culture, which protected oysters from mudworm. Dredge bed culture, which is an effective way of oyster farming, has virtually disappeared from NSW because of siltation and mudworm.

Apart from the Sydney rock oyster, mudworm also infests most commercial molluscs, including Pacific oysters (*Crassostrea gigas*), blue mussels (*Mytilus galloprovincialis*), flat oysters (*Ostrea angasi*), scallops (*Pecten fumatus*) and abalone (*Haliotis rubra*). The adult mudworm is up to 25 mm long, 1 mm wide and red in colour. It lives on the inside of the oyster shell, where it gives rise to the formation of a mud blister, but maintains a tube across the lip of the shell to the outside.

Healthy, rapidly growing oysters are able to cover the worm and its mud patch with shell and recover from the attack, whereas weak oysters usually succumb and die. Losses of oysters due to mudworm are often very high, and the remaining infested oysters become unsaleable because of their poor condition and unsightly, foul-smelling mud blisters, which rupture easily when the oysters are opened.

Infested oysters can be left out of water in the shade for up to ten days to kill mudworm and/or flatworm (see below); some oyster farmers wash mud from oysters on their leases using boom-sprays on punts, to lower the risk of infestation.

Successful control methods

Some farmers have successfully used the following method to kill mudworm: Oysters are left out to dry for 4–5 days, followed by a 2–3 hour bath in an iodine-based disinfectant solution at 0.1 g of active iodine per litre of seawater, prior to returning oysters to the lease. It is claimed that this treatment is more successful in treating bad outbreaks of mudworm than drying alone.

Flatworm

Also known as wafers or 'leeches', flatworms are common predators of oysters and other commercial bivalves around the world.

In NSW, the flatworm (*Imogine mcgrathi*) was identified as a threat to oyster production as early as the 1890's, and more recently has been observed to feed directly on oyster tissue.

In laboratory studies, flatworms have been found to eat oysters and mussels by entering the shells and everting their stomachs to engulf their prey. Generally, flatworms have been found to consume oysters at a rate of approximately 1 oyster per flatworm per month. When flatworms occur in high numbers they can have pronounced effects.

Recently, *I. mcgrathi* were found at an average of 376 per metre of mussel culture rope in Twofold Bay, and were thought to be responsible for considerable mussel losses. Since the recent increased use of fine mesh trays, cylinders and baskets for oyster nursery culture of hatchery-produced stock, mortality from flatworm has become a serious problem for Sydney rock, Pacific and flat oyster production in NSW. It seems that the fine mesh protects the flatworm from desiccation, allowing them to feed on oyster spat. Regular inspection of spat in nursery culture and increasing the mesh size assist in protecting against this predator.

Anecdotally, it would appear that flatworm numbers are greatest in periods of prolonged drought when estuary salinities are relatively high for extended periods. The reason for this is unclear, as adult *I. mcgrathi* can tolerate reduced salinities for some time, although reduced salinity may affect other aspects of flatworm survival (e.g. reproductive behaviour). Regardless, exposure to freshwater or saturated brine solutions is an effective means of controlling flatworms, with baths of 15 minutes sufficient to kill adult flatworms.

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