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Diseases of cultured molluscs in Australia

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Abstract — *Three mollusc species are cultured commercially in Australian tropical and warm temperate waters : the Sydney rock oyster* ***Sacccostrea commercialis****, the pearl rock oyster is susceptible to two protozoans : Marteilia sydneyi, which is the cause QX Disease and infects oysters during the summer; and* ***Mikrocytos roughleyi*** *which is associated with winter mortality. It also harbours mudworm,* ***Polydora websteri*** *a parasite farmers try to avoid by growing oysters on racks. Transported pearl oysters* ***Pinctada maxima*** *die with heavy infections of* ***Vibrio harveyi****. Cultured giant clams* ***Tridacna gigas*** *are pestered by pyramidellid snails and some carry* ***Perkinsus*** *sp., a protozoan common in reef bivalves. Deaths in mollusc hatcheries have been associated with* ***Vibrio tubiashi****,* ***Vibrio*** *spp., and* ***Alteromonas*** *spp.*

INTRODUCTION

The value of cultured molluscs in Australia in 1987/88 was A$104 million p.a., equivalent to 91,300 million CPF or US$83 million. This included A$65 million CPF from the pearl oyster industry, A$30 million from the sale of Sydney rock oysters (*Saccostrea commercialis*) and A$8 million from Pacific oysters (*Crassostrea gigas*). Smaller but developing industries are based on the culture of mussels *Mytilis edulis* and mud oysters *Ostrea angasi* (Victoria), abalone *Haliotis ruber* (Tasmania), blacklip oysters *Crassostrea echinata*, milky oysters *C. amasa* and giant clams *Tridacna gigas* (Queensland).

Bivalve culture throughout the world has been plagued by epizootics, the most severe of which have been those caused by Protozoa. Australia is no exception. These and other diseases known from Australian molluscs are described below.

PROTOZOA QX DISEASE

Aetiology. The disease is caused by *Marteilia sydneyi* Perkins and Wolf, 1976, a protozan in the phylum Ascetospora.

Pathogenesis and epizootiology. The parasite multiplies within the digestive gland of *Saccostrea commercialis*, the Sydney rock oyster. First signs usually appear within a few days of a « fresh », i.e. a sudden drop in salinity (Lester, 1986a). Some oysters will die. Histological sections of survivors show foci of inflammation in the Leidig tissue around individual tubules. The parasite is presumably within these masses of haemocytes. Within 3 weeks parasites move to the cytoplasm of epithelial cells lining the tubule and begin to develop into sporangia.

In the summer oysters die about 6 weeks after infection. Some of those infected in early fall will survive through the winter. Oysters in the laboratory with light infections apparently shed all their parasites via the digestive tract and recovered (Roubal et *al.*, in press). Recovery from infection has not yet been observed in the field.

The parasite causes epizootics in estuarites in southern Queensland and northern New South Wales, down to the Mcleay River (Nell and Smith, 1988).

Clinical signs and lesions. When the digestive gland is cut open, its interior is pale yellow-brown rather than the deep green of healthy oysters. This is not a diagnostic test, however, as other conditions will also stop an oyster from feeding. In the winter, oysters with chronic infections are largely devoid of white gonad tissue; they appear watery and have an enlarged pale brown digestive gland. In the summer, the « fatness » or amount of gonad present is not a reliable guide as oysters close to spawning often become infected and may carry extremely high parasite loads beneath a full gonad.

Diagnosis. Confirmation of infection is based on the demonstration of sporonts. These are easily seen in wet smears of digestive gland examined under the high power of a microscope; the refractile granules show up particularly well. Sporangia and sporonts are also readily visible in the tuble epithelium of fixed tissues sectioned and stained with H. and E..

Treatment. No treatment is known.

Prevention. Oyster farmers in the endemic area try to avoid having oysters on leases in estuarine waters during the summer months, specifically from the beginning of January to the second week of April. Oysters in high salinity areas, though they grow less well, remain free of infection. The life cycle of *M. sydneyi* is not known. It probably requires an intermediate host (Roubal et *al.*, in press).

WINTER MORTALITY

Aetiology. This disease is believed to be caused by a minute protozoan *Mikrocytos roughleyi* Farley, Wolf and Elston, 1988, related to the oyster pathogens, *M. mackini* from North America, and *Bonamia* spp. from Europe and New Zealand. Their taxonomic position in the subkingdom Protozoa is unclear.

Pathogenesis and epizootiology. Parasites develop in the cytoplasm of haemocytes. The disease is reported only from S. commercialis from the George's River and adjacent estuaries of high salinity in New South Wales. It thrives in high salinity waters (30 to 35 °/°°), has an incubation period of 2.5 months and mortality does not occur in animals less than 3 years old.

Perkinsus olseni has been reported from four species of abalone (*Haliotis* spp.) from South Australia (Lester, Goggin and Sewell, 1988). The large trophozoites are typically extra-cellular and circulate in the haemolymph (Lester and Davis, 1981). In recovering abalone they are trapped and killed in tissue abscesses. The parasite is believed to be responsible for the die-back of greenlip abalone *Haliotis laevigata* off Stansbury, South Australia (Lester, 1986b).

Unknown *Perkinsus* species are wide-spread in bivalves on the Great Barrier Reef (Goggin and Lester, 1987) and have been linked to mortalities in giant clams. It seems more prevalent in the winter.

Treatment. In abalone, stress from high temperature seems to aid the spread of the parasite through the tissues (Lester and Davis, 1981). Removal of any source of stress on cultured animals may help to alleviate mortalities attributed to this parasite.

Prevention. Use filtered sea water. Avoid introducing infected molluscs.

COCCIDIA

An unidentified protistan resembling a coccidian was found in the ovary of blacklipped oysters *C. echinata* from Darwin harbour, Northern Territory (Wolf, 1977). The intracellular parasite was within the developing and mature ova of over half the female oysters examined. It was not detected in males.

SPHERICAL BODIES

Wolf (1978) reported spherical bodies 2 to 3 ñm in diameter in the digestive epithelium of moribund pearl oysters, *Pinctada maxima* from tropical Australia. Pass and Perkins (1985) concluded that they were probably normal constituents of the digestive cells.

METAZOA MUDWORM DISEASE

Aetiology. The disease is caused by four species of polychaete worms, of which *Polydora websteri* is thought to be the most damaging (Nell and Smith, 1988). It parasitizes *S. commercialis, C. gigas, Mytilus edulis* and *Ostrea angasi.*

Pathogenesis and epidemiology. Young worms creep under the mantle of the bivalve and develop a « U » shaped burrow from which they strectch

out to collect the detritus on which they feed. The mollusc tries to wall them off, together with the mud waste, by laying down shell over the burrow. In *S. commercialis* a large thin-walled « mud blister » eventually forms on the inside of the shell. Young oysters and heavily infected older oysters die. Mudworm has been blamed for the disappearance of the dredge oyster industry from Moreton Bay (Smith, 1982). Today, low levels of infection are a nuisance to those shucking oysters because blisters are easily broken and valuable time is lost washing mud off the meat.

Clinical signs. In oysters a dark blister 10 mm or more across is found on the inside of the shell. The mantle adjacent to the blister is often yellow and necrotic. Infected oysters are more likely to die from high temperature, low salinity, or storage out of water than uninfected oysters (Nell and Smith, 1988).

Diagnosis. The U shaped burrow is usually visible at least near the edge of the shell. The openings may be at the edge or, if the animal has grown since infection, may open to the outside some distance from the shell margin.

Treatment. Several crude methods have been used, e.g. oysters kept out of water in shade for 10 to 14 days; oysters dipped in dilute solution of phenol and detergent for 1 hr then left out of water for 1 day (Nell and Smith, 1988).

Prevention. To avoid mudworm disease, oysters are grown on racks so that they dry out each day. Under these conditions the larval worms are apparently not able to gain entrance.

TURBELLARIA

A large polyclad turbellarian, *Notoplana australis*, is found within the shells of weakened oysters, *S. commercialis*, on the New South Wales coast. Sometimes called the wafer or oyster leech, it feeds on the oyster but probably only enters the shell when the oyster is dying from some other cause (Prudhoe, 1982).

Small commensal turbellarians are occasionally found on the gills and in the gut of cultured bivalves but do no demonstrable harm (Lauckner, 1983; Goggin and Cannon, in press).

DIGENEA

Digenetic trematodes of the family Bucephalidae parasitize the gonads of oysters (Howell, 1966), scallops (Sanders and Lester, 1981) and tridacnid clams (Shelley et *al.*, 1988). The gonad is enlarged and eventually almost totally replaced by the branching sporocyste.

CESTODA

Larval tapeworms are common but do not cause problems. Encapsulated lecanicephalids (genera *Tylocephalum* and *Polypocephalus*) are abundant in the tissues of oysters from Queensland waters. Adults occur in rays.

PEA CRABS

The pinnotherid crab *Pinnotheres hickmani* is a frequent commensal in the mantle cavity of cultured mussels *Mytilis edulis* in Victoria. It reduces the meat yield of infected animals by up to 26 % (Pregenzer, 1981). A pinnotherid infection has been controlled with an insecticide in animals not for human consumption (Andrews et *al.*, 1968).

PYRAMIDELLIDAE

Pyramidellids are tiny gastropods that parasitize other molluscs. Large numbers of *Pyrgiscis* sp., 1 to 6 mm long, developed in the culture facility on Orpheus Island and killed juvenile giant clams (Cumming, 1988). During the day they hide outside the clam. At night they crawled to the lip of the shell and extended their probosces into the mantle to feed. Biological control using the crab *Thalamita sima* was suggested.

Pyramidellids reduce the growth rate of *C. virginica* and can transmit the protozoan *Perkinsus marinus* from oyster to oyster (White et *al.*, 1987).

BACTERIA VIBRIOSIS

Aetiology. *Vibrio tubiashi, V. anguillarum, V. siginolyticus* and *V. harveyi.*

Pathogenesis and epizootiology. Saprophytic vibrios and pseudomonads are an ongoing problem in mollusc hatcheries. Larval molluscs under adverse environmental conditions become covered in the bacteria. The gut and tissues are invaded and the larval molluscs die. The course of infection is rapid; mortality may be 100 % within 24hr.

Infections with *Vibrio tubiashi* and *Alteromonas spp.* were fatal to larval *C. gigas* in a Tasmanian hatchery. The pathogens were apparently taken in with food as fertilized eggs were bacteria-free. Fifteen to 100 % of larvae died (Garland, 1988).

Vibrio harveyi has been isolated from the haemolymph of dying adult pearl oysters. Up to 80 % of oysters frequently died after being transported from collection grounds to a lease site in Western Australia. The mortality was associated with cold water temperatures (19 %), crowding of oysters during transport, inadequate water circulation in carrier tanks, and infection with *Vibrio* sp. Experimentally, the *V. harveyi* isolated was shown to produce mortality in the oysters (Pass et *al.*, 1987).

Clinical signs and lesions. Larval molluscs show a decrease in motility and high sudden mortalities. They become pale and stop growing. Velum losses its cilia (Garland, 1988).

Adult pearl oysters had withdrawn mantles and brown-stained nacre between the withdrawn mantle and the edge of the shell. Foci of inflammation were evident in histological sections of the mantle and digestive gland. The epithelium of digestive tubules was atrophied.

Diagnosis. Direct microscopic examination of live affected larvae for swarming vibrios. Bacteriological culture.

Treatment. In hatchery, antibiotics (Chloramphenicol 10 ppm, erythromycin, neomycin); may be too late. For adult oysters, reduce stock density and improve water quality.

Prevention. Improve water quality.

VIRUS

Virus-like particles were found in the digestive gland of the pearl oyster (*Pinctada maxima*). They were in inclusions centrally located within hypertrophied nuclei in the epithelial cells of digestive tubules. Inclusions were basophilic or amphophilic with H. and E., and surrounded by a clear zone. They were not associated with any disease (Pass et *al.*, 1988).

PAPILLARY EPITHELIOMA

Papillary epitheliomas have been found in the mantle of *S. commercialis* from several locations in central New South Wales (Wolf, 1976). The tumours are ovoid or spherical, 1 to 16 mm in diameter, and have deep indentations like a cauliflower. They are composed of proliferating epithelium. They occur in older stunted oysters and are apparently unrelated to pollution.

MESENCHYMAL TUMOURS

Mesenchymal tumours have been reported from a pearl oyster *P. margaritifera* on the Great Barrier Reef. The tumours were firm, polyp-like growths attached by flexible stalks to the visceral mass near the gut loop and adductor muscle. Normal tissue elements with increased fibrosis and blood spaces composed the stroma and epithelium covered the surface (Dix, 1972).

Similar tumours were found in a Sydney rock oyster by Dinamani and Wolf (1973).

TRIBUTYL TIN

The tributyl tin (TBT) contained in many antifouling paints inhibits the growth of Sydney rock oysters at extremely low concentrations (5 ng TBTO/l; J.A. Nell, pers. com.). The sale of paint containing TBT is restricted in NSW and Tasmania.

HEAT KILL

Sydney rock oysters die from heat stress if a midday low tide coincides with a hot summer day. Trays are often covered in shadecloth or sprayed with water to reduce losses.

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