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## Parasites and diseases of commercially important molluscs in New Zealand

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**Abstract** — Mass mortalities among flat oysters (*Tiostrea lutaria*) in Foveaux Strait, southern New Zealand, between early 1986 and the present, are primarily associated with a haplosporidan, *Bonamia* sp.. Highest infection rates occur in the densest beds, with more than 80 % mortality in these areas, and movement of high density/high infection foci over c.10 km in 3 years. In some oysters large numbers of coccidian merozoites may also be a cause of mortality. Rickettsial-like organisms, microsporidans, bucephalid sporocysts and neoplasia also contribute to the poor health of these stocks.

*Bonamia* has also caused 40 % - 60 % mortalities in two or three stocks of *O. lutaria* held on a mussel farm 800 km north east of Foveaux Strait. A third, slow-growing, local stock held among heavily infected oysters experienced no mortalities. Future studies on this stock, and options for management of the Foveaux Strait fishery are discussed.

Mass winter mortalities have been reported among native rock oysters, *Saccostrea glomerata*, until recently the basis of oyster farming. Investigations by Jones (pers. com.) found gaping in these oysters was associated with an idiopathic adductor muscle necrosis with pustular areas adjacent to brown rubber-like blisters on the shell. Similar blisters occur in the shell of the currently farmed *Crassostrea gigas* and may be associated with ectosymbionts. Shell disease, of probable fungal aetiology, occurs in *Haliotis iris*, but other than intranuclear inclusions in haemocytes, disease is not known from this species.

Mortalities and disease have not been reported in the intensively-farmed green-lipped mussel (*Perna canaliculus*) or blue mussel (*Mytilus edulis aoteanus*), but digenean sporocysts are common in these species.

All these bivalve species may contain the copepod, *Pseudomyicola spinosus*, in the gut, but pathogenicity has not been demonstrated. Pea-crabs are also common as ectosymbionts, but in *Perna* the gregarine *Nematopsis* cycles through the mussel and crab. The general health of the stocks is discussed.

### INTRODUCTION

There have been relatively few studies on the parasites and diseases of molluscs in New Zealand. Several mass mortalities, particularly of

bivalves, have been reported, but sparseness of population and the terrain have made it difficult to obtain adequately fixed material for diagnostic studies. Commercially dredged or farmed species are more readily obtainable and their diseases and parasites are considered here.

## OYSTERS

### *Tiostrea lutaria*

This species forms the basis of a dredge fishery at the south end of the South Island in Foveaux Strait. Normally 23 boats dredge < c. 138 x 106 oysters/year from autumn (1 March) to late winter (31 August). The fishery has experienced mass mortalities about every 20 years, but the cause of most of these events is unknown. However, mortalities in 1962-64 were attributed to *Bucephalus* sporocysts and cercariae heavily infecting the oysters (Howell, 1963). Two small fishes (*Acanthoclinus quadridactylus* and *Tripterygion* sp.) have experimentally been shown to act as second intermediate hosts, and *Scorpaena cardinalis* and *Kathetostoma giganteum* as definitive hosts (Howell, 1966). Infection of oysters occurs in summer and development occurs at the expense of the gonad, with subsequent parasitic castration and death (Howell, 1967).

Tab. 1. — Incidence (% infection) of *Bonamia* at 8 stations in Foveaux Strait. N = northern, S = southern, W = western, E = eastern

Station	Beds	Month/year						
		9/86	1/87	6/87	8/87	11/87	4/88	12/88
13	E	2	3	12	20	6	4	2
14	E	4	4	18	38	8	30	8
17	S	0	0	4	4	4	8	4
19	E	4	6	10	22	12	61	4
21	N	0	0	5	0	4	4	2
27	S	0	0	12	28	2	8	0
36	W	—	35	32	28	18	18	6
38	W	—	35	18	32	14	16	6

Mass mortalities in autumn 1986 were attributed to a haplosporidan, ultrastructurally (Dinamani et al., 1987) and serotypically (Mialhe et al., 1988) very similar to, but distinct from, *Bonamia ostreae*, a very pathogenic species in *Ostrea edulis* in Europe (Pichot et al., 1979) and North America (Elston et al., 1986). *T. lutaria* sampled in winter contain a « lighter » form under surface epithelia and in blood spaces, but by late winter this becomes larger and irregular (Fig. 1), with many mitoses, leading to dense forms in gonads and deeper Leydig tissue in summer (Dinamani et al., 1987). Change in distribution may reflect movement of infected haemocytes and egress may occur in shed gonad products, through digestive diverticulae or through the gills (Dinamani et al., 1987). Heavy infections and mortalities occur in autumn (April/May). Infection « hot spots » have moved c. 10 km down-current in 3 years, but both incidence and intensity of infection appear to be declining (Table 1).

Autumnal mortalities may also be attributable to merozoites of an apicomplexan (Fig. 2). The parasite initially radiates out of the ventral « blood sinus », between Leydig cells, in a stellate configuration. After phagocytosis by haemocytes the merozoites divide (Fig. 3) until the cell ruptures causing a responding haemocytosis, destruction of Leydig tissue and tissue loss (Fig. 4) and its replacement by amorphous eosinophilic and fibrous tissue. The parasite infects 80-100 % of oysters, is associated with tissue damage in 10-25 % of these, and numbers build up over summer, almost certainly causing mortalities in autumn. Ultrastructurally the merozoite has a typical apical complex, c. 84 sub-pellicular microtubules, and division is by *Semiopen pleuromitosis* (R. Entzeroth : pers. com.). With decline in *Bonamia* and increase in the apicomplexan, the latter currently appears to be the major pathogen in the oysters. *Bucephalus* occurred in < 4 % of oysters in 1986-88 samples, compared with 18-47 % in 1963-64 (Howell, 1967), and cannot be considered a major contributor to recent mortalities.

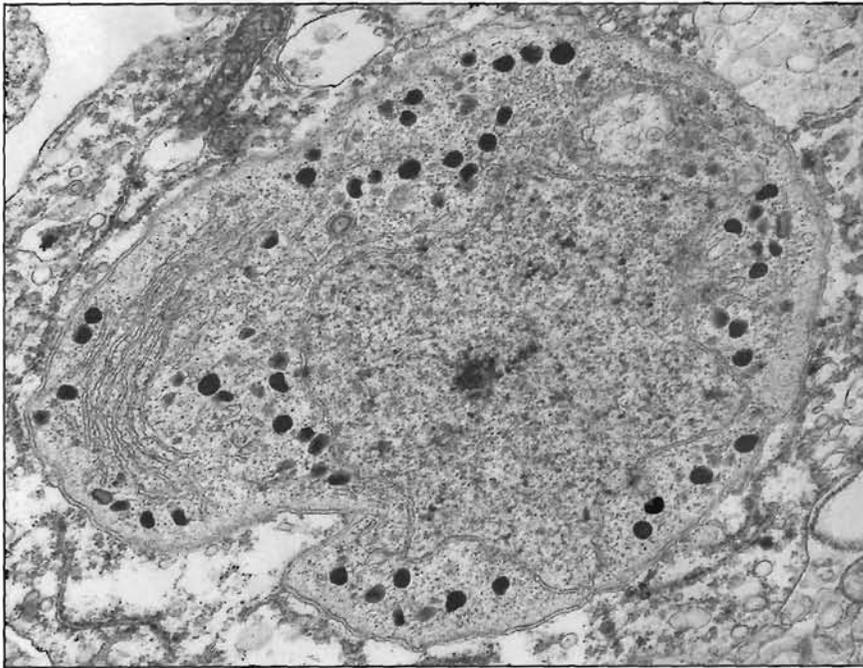
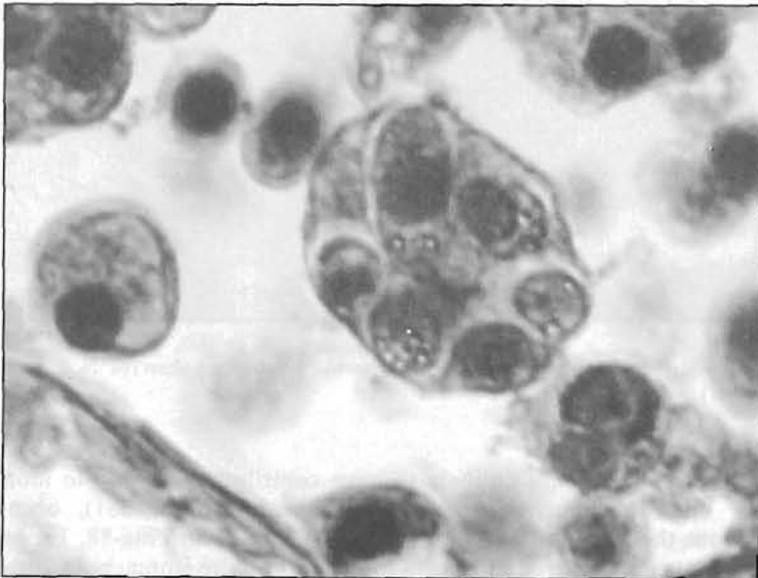


Figure 1. — Irregular plasmodial form of *Bonamia*, 5.5µm dia., observed in early spring (September).

Other parasites are unlikely to have contributed directly to mortalities. A microsporidan, *Microsporidium rapuae*, (Jones, 1981), occurred throughout the year in 10-40 % of stations sampled in 1986-88. Pre-spore and spore stages occurred in Leydig tissue around the stomach. Infiltrating haemocytes were occasionally observed phagocytosing spores from ruptured cysts, leaving a sheath of haemocytes and fibrous tissue around the



**Figure 2.** — Merozoite of the apicomplexan, 6.5 $\mu$ m long. Note intranuclear fibrils.



**Figure 3.** — Haemocyte replete with apicomplexans.

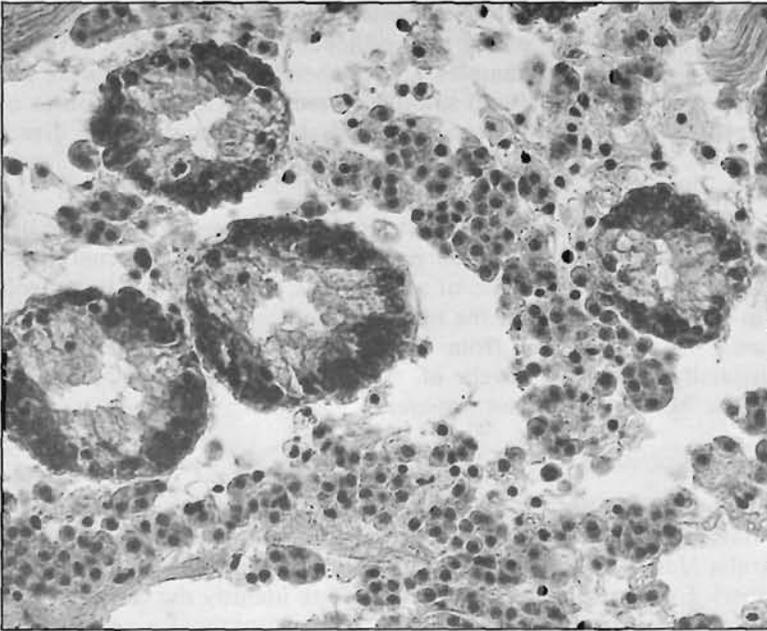


Figure 4. — Apicomplexans among digestive diverticulae. Note loss of ground substance.

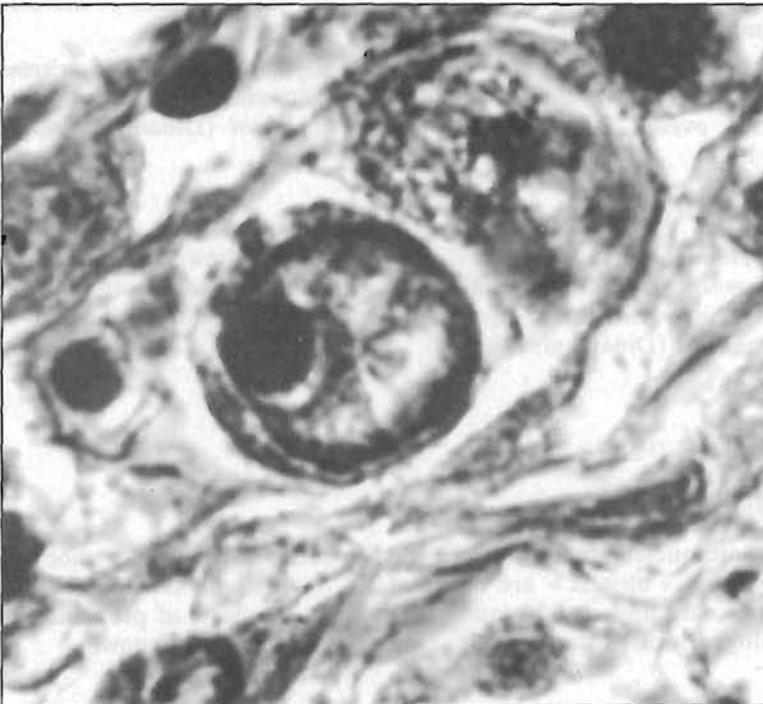


Figure 5. — Thick-walled spore (11.5 $\mu$ m dia.) with eccentric nucleus.

stomach. Oval or reniform bodies containing rickettsia-like organisms were observed intermittently among the epithelial cells of digestive diverticulae in spring and summer samples (November-February), but were absent from oysters in winter (June) samples. Rarely, thick-walled spores of an unidentified species have been observed among the digestive diverticulae (Fig. 5). The life-cycles of the apicomplexan, *M. rapuae* and the rickettsia-like organisms are unknown.

In general the Foveaux Strait stocks of oysters are in poor health, but bad weather over 1986-1988 probably contributed to general loss of condition. With the exception of an outbreak of bonamiasis in an isolated bay at Port Underwood, at the north of the South Island, in winter 1988, *Bonamia* is only known from Foveaux Strait. Oysters in other areas occasionally have low levels of the apicomplexan, *M. rapuae* or the rickettsia, but disease is not apparent.

#### *Saccostrea glomerata*

Mass mortalities (< 100 %) among rock oysters were noted in 1971 and named « winter mortalities » because of similarities to mortalities in Australia (Jones, 1975a), although mortalities occurred in spring (August-October). Examination for parasites failed to identify the cause of disease. However, it was noted the adductor muscle contained « yellow necrotic pustules » that extended onto the mantle, and the inner surface of the shell had « brown rubber-like warts and spots ». Histologically the adductor muscle showed a gradual reduction in muscle fibres and an extensive haemocytosis (Jones, 1975a). The cause and current status of the disease are unknown.

Rock oysters are parasitized by the copepod *Pseudomyicola spinosus* which causes gut epithelia to change from columnar to low-squamous type, but the overall effect on the host is unknown (Dinamani and Gordon, 1974).

#### *Crassostrea gigas*

Disease surveys of Pacific oysters have shown the presence of *P. spinosus*, turbellarians, chironomids, nematodes, mudworms and peacocks which, with the exception of *P. spinosus*, are non-pathogenic ecto-commensals (Dinamani, 1986).

### ABALONE (*Paua*)

#### *Haliotis* sp.

Intranuclear inclusions are rarely observed in the haemocytes of *H. iris*, and « blisters » are occasionally reported on the foot of *H. iris*. Shell disease under and around the adductor muscle, of probable fungal aetiology, occurs intermittently. Otherwise disease is unknown.

## MUSSELS

*Perna canaliculus*

The green-lipped mussel forms the basis of a growing industry which produced 10500 tonnes of mussels in 1985. There have been no reports of disease-associated mortalities in aquaculture systems. Examination of wild and cultured *Perna* has revealed cercariae and sporocysts of *Tergestia agnostomi*, which uses mullet (*Aldrichetta forsteri*) as definitive host, a weakly parasitic copepod (*Lichomolgus*) on the gills, and *P. spinosus* infections (Jones, 1975a, 1978). A gregarine, *Nematopsis*, infects labial palps (Jones, 1975b) with transmission by its other host, the pea-crab (*Pinnotheres*) (Jones : pers. com.).

*Mytilus edulis*

Blue mussels are not favoured for aquaculture and are only known to harbour *P. spinosus*, *Tergestia* and pea-crabs (Jones, 1975a).

## SCALLOPS

*Pecten novaezelandiae*

Scallops are reared for enhancement of the wild fishery. Although wild stock undergo large fluctuations in population, no diseases have yet been identified in this species.

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