Southern hemisphere mollusc diseases and an overview of associated risk assessment problems

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Summary: In Australia and New Zealand, Bonamia sp. causes epizootics in flat oysters (Tiostrea chilensis, Ostrea angasi); Marteilia sydneyi and Mikrocytos roughleyi cause mortality in farmed rock oysters (Saccostrea commercialis); and Perkinsus olseni is pathogenic in abalone (Haliotis spp.). Marteilia lengehi, Marteiloides branchialis, other Marteiloides spp. and two species of Haplosporidium are regarded as potential pathogens. A review of the pathogens causing diseases listed in the Office International des Epizooties 'notifiable diseases' of molluscs shows major gaps in the information available. The life cycles and transmission of Haplosporidium nelsoni and Marteilia refringens are unknown, Bonamia spp. and Mikrocytos spp. cannot be diagnosed with certainty, monoclonal antibodies and molecular probes are not generally available, and little is known of survival parameters or treatment of the pathogens. The author concludes that stringent guidelines and protocols are needed to minimise the high risks involved in translocation of molluscs.


INTRODUCTION

Nearly all reports of serious disease in bivalve molluscs in the southern hemisphere are from Australia and New Zealand, with little information available from South America (53) or Southern Africa. In Australia, the main industries are based on pearl oysters (Pinctada maxima), and pearl exports are worth approximately AUS$120 million a year. In addition, exports of Sydney rock oysters (Saccostrea commercialis) (64) are worth approximately AUS$35 million a year, and exports of Pacific oysters (Crassostrea gigas) are worth about AUS$13 million a year. Other molluscs farmed on a smaller scale include abalone (Haliotis spp.) and giant clams (Tridacna spp.). In New Zealand, molluscs form the core of the aquaculture industry, earning NZ$155.7 million in exports in 1994. The bulk of this income was derived from greenlip mussels (Perna canaliculus) and Pacific oysters, which brought in NZ$70.3 million and NZ$10.3 million, respectively. The serious diseases prevalent in these industries and in wild stocks are reviewed below.

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The risks inherent in the translocation of molluscs are then considered in relation to current knowledge on serious molluscan diseases, particularly the Office International des Epizooties (OIE) notifiable diseases. Risk may be minimised by the procedures adopted to effect translocation, such as choice of stock and health certification in the exporting country, quarantine and requirement to breed an F1 generation followed by destruction of imported animals, and post-introduction health monitoring, as outlined in guidelines published by the International Council for the Exploration of the Sea (ICES), the European Inland Fisheries Advisory Committee (EIFAC)/Food and Agriculture Organisation of the United Nations (FAO) and others. Initially, assessment of the risks inherent in any translocation involves knowledge of likely pathogens; this should include knowledge of life cycles, modes of transmission, host specificity, reliability of detection, and ways of neutralising, treating or controlling the pathogens. The current state of knowledge of the pathogens causing the OIE notifiable diseases of molluscs are considered below, to put into perspective the current ability of mollusc pathologists to manage translocations of molluscs.

DISEASES IN BIVALVE MOLLUSCS IN THE SOUTHERN HEMISPHERE

Cultured and wild bivalves in Australia and New Zealand are known to harbour several pathogenic and potentially pathogenic organisms (Table I). *Bonamia* sp., a pathogenic protistan causing bonamiosis – an OIE notifiable disease – has caused epizootics in flat oysters (*Tigriopus chilensis*) in New Zealand (48, 49) and mortalities in *Ostrea angasi* in Australia (Victoria, Tasmania, Western Australia). A species of *Bonamia* has also been reported in *T. chilensis* in Chile and has been compared to *Bonamia ostreae* in the northern hemisphere (56); there is no evidence for introduction of *B. ostreae*, however, and occurrence in *T. chilensis* makes it more likely that the pathogen detected in Australia and New Zealand is the southern hemisphere *Bonamia*. If so, the distribution of this *Bonamia* sp. may result from the previous existence of the southern continent, ‘Gondwanaland’.

Another pathogen causing an OIE notifiable disease is *Mikrocytos roughleyi*, the cause of winter mortality in Sydney rock oysters (*Saccostrea commercialis*) in the Georges River and surrounding estuaries in New South Wales, Australia. Up to 70% of cultured oysters up to three years old (26) are killed in their third winter (July-September) before marketing (10). *S. commercialis* also experiences epizootics caused by *Marteilia sydneyi*, which occurs on the eastern (77) and north-western (unpublished findings) coasts of Australia, and may kill up to 80% of infected oysters, with up to 100% prevalence. Death occurs within 60 days of infection (77). Restrictions on movement to control diseases of *S. commercialis* exist in New South Wales (64), but not in Queensland (T. Anderson, personal communication).

*Perkinsus olseni*, which occurs around Australia, excluding Tasmania, causes epizootics in abalone (65) but also occurs in many bivalve species (38), and may cross-infect between them (39) with little if any pathogenicity.

Potentially serious pathogens include the following:

- *Marteilia lengehi* in oysters (*Saccostrea cucullata*)
- *Haplosporidium* spp. in *S. cucullata* and in pearl oyster (*Pinctada maxima*) spat in northern areas of Western Australia
### Table I

**Serious and potentially serious pathogens in molluscs in Australia and New Zealand**

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Host(s)</th>
<th>References</th>
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<tr>
<td><strong>Australia</strong></td>
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<tr>
<td><em>Bonamia</em> sp.</td>
<td><em>Ostrea angasi</em></td>
<td>(P.M. Hine, unpublished findings)</td>
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<tr>
<td><em>Mikrocytos roughleyi</em></td>
<td><em>Saccostrea commercialis</em></td>
<td>(26)</td>
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<tr>
<td><em>Marteilia sydneyi</em></td>
<td><em>S. commercialis</em></td>
<td>(70, 76)</td>
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<tr>
<td><em>Marteilia lengehi</em></td>
<td><em>S. cuccullata</em></td>
<td>(P.M. Hine, unpublished findings)</td>
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<tr>
<td><em>Marteilioides branchialis</em></td>
<td><em>S. commercialis</em></td>
<td>(1)</td>
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<tr>
<td><em>Marteilioides sp.</em></td>
<td><em>S. echinata</em></td>
<td>(P.M. Hine, unpublished findings)</td>
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<tr>
<td><em>Perkinsus olseni</em></td>
<td><em>Haliotis spp.</em></td>
<td>(64)</td>
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<td></td>
<td>Many bivalves</td>
<td>(33, 39)</td>
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<tr>
<td><em>Haplosporidium</em> sp.</td>
<td><em>S. cuccullata</em></td>
<td>(P.M. Hine, unpublished findings)</td>
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<tr>
<td></td>
<td><em>Pinctada maxima</em></td>
<td>(P.M. Hine, unpublished findings)</td>
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<tr>
<td><strong>New Zealand</strong></td>
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<tr>
<td><em>Bonamia</em> sp.</td>
<td><em>Tiostrea chilensis</em></td>
<td>(47, 48, 49, 50)</td>
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- *Marteilioides branchialis* in *S. commercialis* (1)
- *Marteilioides* sp. resembling *M. chungmuensis* (22) in the ova of *Saccostrea echinata* in Darwin harbour, Northern Territory (76).

There are no examples of disease spread as a result of translocation of molluscs in the southern hemisphere, reflecting the low level of trade in molluscs between southern hemisphere countries. There are very few examples of possible translocation from the southern hemisphere to the northern hemisphere. An unidentified parasite reported in the eggs of Pacific oysters from Humboldt Bay, California (6) was very similar to *Marteilioides* sp. in *S. echinata* in Darwin harbour, but patterns of stock movement and host species mean that this is more likely to be *M. chungmuensis*, reported from Pacific oysters in Korea (22). *Perkinsus atlanticus*, infecting clams (*Ruditapes decussatus*) in Portugal and Spain, appears to be conspecific with Australian *P. olseni* on the basis of ribosomal ribonucleic acid (rRNA) base sequences (37), but there is no evidence for *P. olseni* in North America, the route by which clams were introduced into Europe. As there is currently some confusion in *Perkinsus* taxonomy, it would be premature to conclude that Portuguese *P. atlanticus* originated from *P. olseni* in Australia.
PATHOGENS CAUSING OFFICE INTERNATIONAL DES EPIZOOTIES NOTIFIABLE DISEASES

LIFE CYCLES, TRANSMISSION AND HOST SPECIFICITY

Iridoviral disease in oysters (*Crassostrea gigas*)

Gill necrosis virus disease and haemocytic infiltration virus disease of Portuguese oysters (*Crassostrea angulata*) and, to a lesser extent, Pacific oysters, spread very quickly through oyster stocks in France and neighbouring countries in the 1970s (21), suggesting that transmission is direct and horizontal. Both diseases are attributed to one or two iridoviruses (21) but identification as an iridovirus, and relationship to the oyster velar virus disease iridovirus of *C. gigas*, have yet to be determined (10). These iridoviruses appear to be host-specific in *C. gigas* (syn. *C. angulata*) (12).

Haplosporidiosis caused by *Haplosporidium nelsoni* in oysters (*Crassostrea virginica*)

Both the life cycle and the modes of transmission of *H. nelsoni* (also referred to as MSX: multinucleate sphere X) are unknown. An intermediate host or vector may be needed, as *H. nelsoni* cannot be transmitted directly between oysters using plasmodia, either by injection or by cohabitation, although there have been few attempts to effect transmission using spores (32, 44). Other *Haplosporidium* spp. and *Minchinia* spp. cannot be transmitted directly under experimental conditions. It is unlikely that a period of maturation is required outside the host, as sporoplasms of a similar organism (*Minchinia dentali*) were viable and active after release from fresh spores (24). Spread of *H. nelsoni* between and within oyster beds is not related to oyster density (S.E. Ford, personal communication), whereas direct horizontal transmission is likely to be dependent on host density, also supporting the theory that an intermediate host or vector is required. Such a host or vector would probably be small and possibly pelagic, as initially uninfected *C. virginica* held in a hatchery seawater system over summer became infected (27). *H. nelsoni* may be closely related to *Haplosporidium* sp. from *C. gigas* (36, 55), as a gene probe used by E.M. Burreson and N.A. Stokes at the Virginia Institute of Marine Science found strong binding between these organisms (S.E. Ford, personal communication). It seems unlikely, however, that *H. nelsoni* derived from any of the *Haplosporidium* spp. of *C. gigas* from around the world (23, 36, 54, 55), as *C. gigas* exposed to conditions in which *C. virginica* became infected did not themselves become infected (27). *H. nelsoni* is probably host-specific to *C. virginica*.

Bonamiosis caused by *Bonamia* spp. in flat oysters (*Ostrea* spp., *Tiostrea chilensis*)

The transmission of *Bonamia ostreae* is horizontal and direct between flat oysters (*Ostrea edulis*) (43), infecting granulocytic haemocytes and branchial epithelial cells (62). Infection can occur throughout the year (43), with the parasite appearing three to five months after infection (2, 61, 74); prevalence is low in young oysters (73), but infection is related more to size than age, and there is little involvement of the gonad in the infection cycle (16). *B. ostreae* infects *Ostrea* spp. (7, 66) and *Tiostrea chilensis* (42).
In New Zealand, *Bonamia* sp. is also transmitted directly between flat oysters (*T. chilensis*) (P.M. Hine, unpublished findings), infecting agranular haemocytes (51). The parasite is not readily detectable for approximately three months after infection; the gonad (particularly the ovary) is often infected (48, 49), probably as a source of lipids for energy metabolism (50). *Bonamia* sp. causes a very different pathology in flat oysters (*O. angasi*) in Australia. Apparently, small numbers of parasites in granulocytic haemocytes and the branchial epithelium cause a massive inflammatory response, without proliferation in the ovary (P.M. Hine, unpublished findings), and the agent therefore more closely resembles *B. ostreae* in *O. edulis* than *Bonamia* sp. in *T. chilensis*. Like *B. ostreae*, the host range of *Bonamia* sp. is probably restricted to flat oysters (*Ostrea* spp., *Tiostrea* spp).

Although the above may indicate that the life cycles of *Bonamia* spp. are well known, the existence of a symmetrical stage of *Bonamia* sp. which superficially resembles a spore but lacks a sporoplasm (49) suggests that an alternative life cycle may occur under different conditions. Furthermore, in New Zealand, *Bonamia* sp. appears to cause epizootics at intervals of approximately thirty years but remains in the population at sub-clinical levels between epizootics. The factors triggering epizootics are unknown. Contaminated fishing gear and boats may be responsible for spreading *B. ostreae* (52).

**Mikrocytosis caused by Mikrocytos mackini in Pacific oysters (Crassostrea gigas) and by M. roughleyi in Sydney rock oysters (Saccostrea commercialis)**

Ultrastructural evidence suggests that *Mikrocytos mackini* and *M. roughleyi* may not be congeneric (10). Unpublished transmission studies suggest that transmission is horizontal and direct by the known vegetative stages. Studies in which *M. mackini* was injected into *O. edulis*, *O. conchaphila* and *C. virginica* suggest that these three hosts may be more susceptible to *M. mackini* infection than *C. gigas* (10, 46). *S. commercialis* is the only known host of *M. roughleyi*.

**Marteiliosis caused by Marteilia refringens in flat oysters (Ostrea spp.), Pacific oysters (Crassostrea gigas), mussels (Mytilus edulis), cockles (Cerastoderma edule) and possibly other bivalves**

Both life cycle and transmission are unknown (3, 35), but *M. refringens* cannot be transmitted directly between hosts, and an alternative host is suspected (29). Another pathogenic species, *Marteilia sydneyi*, is thought to use a filter-feeding or detritivorous invertebrate rather than a scavenger as intermediate host (71). Natural transmission of *M. refringens* occurs in July-August (10). *M. refringens* appears to have a wide host range, but this situation is confused due to uncertainties in the taxonomic discrimination of *Marteilia* spp. (10). *M. sydneyi* is probably host-specific to *S. commercialis*. A highly pathogenic *Marteilia* sp. has recently been associated with devastating epizootics in calico scallops (*Argopecten gibbus*) (63).

**Perkinsosis caused by Perkinsus marinus in oysters (Crassostrea spp.)**

Transmission is direct and horizontal by any developmental stage (32) or by snail vectors (75). *P. marinus* is highly infectious under optimum conditions, but even a distance as short as 15 m substantially delays transmission (32). *P. marinus* experimentally infects clams (*Mercenaria mercenaria*) and Pacific oysters.
C. gigas) (5). C. virginica can become infected via contact with snails (Boonea impressa), flat oysters (O. lurida) and small fish (e.g. Gobiosoma bosc). The parasite occurs in a wide variety of scavengers, and Perkinsus-like organisms occur in many bivalve groups (32). Infection may also be spread via moist contaminated shells, fouling on boats and vectors (including birds) (S.E. Ford, personal communication).

RELIABILITY OF DETECTION

Iridoviral disease in oysters

In epizootics, iridoviral infections of oysters may cause gross signs of diagnostic importance, such as extensive gill erosion, lesions turning from yellow to brown (which result in V-shaped indentations of the gill margin) and yellow/green pustules on the mantle and on the adductor muscle (10). Electron microscopy is required for confirmation of infection (21). Neither the epidemiology nor the factors causing virulence are understood, and light infections with indistinct viral inclusion bodies may go undetected. The lack of cell-lines for the culture of bivalve viral pathogens greatly exacerbates the problems of diagnosis.

Haplosporidiosis

Spores up to 4 µm in length are more readily detectable by histopathology than intra- and extracellular plasmodia of 2-5 µm diameter, but spores are infrequently present (except in spat) (4, 13). Examination of haemolymph is more rapid than histology, but may miss light or localised infections (34), especially in the gills (14). An enzyme-linked immunosorbent assay (ELISA) has been developed (28), and deoxyribonucleic acid (DNA) probes are being developed (58, 72). As the life cycle is unknown, however, and as it is unknown whether there is a pre-patent ‘latent’ period following infection, testing for infection must be carried out over a prolonged period.

Bonamiosis

The only signs of B. ostreae may be yellow discolouration and/or perforated ulcers on the gill and mantle, while there are no clinical signs in Bonamia sp. infections. The three- to five-month pre-patent period (2, 61, 74) and subsequent light infections make routine histological diagnosis unreliable. Monoclonal antibody techniques have been developed for B. ostreae (8, 20, 70), with some cross-reaction with Bonamia sp. (60). An ELISA is reported to have been used in diagnosis (11).

Mikrocytosis

Pacific oysters infected with M. mackini may have a discoloured digestive gland and green surface pustules which may be confused with nocardiosis. M. mackini is intracellular in haemocytes, and hard to detect by histopathology as it is only approximately 2 µm in diameter. Tissue imprints and purification by centrifugation in sucrose are the most reliable methods of detection (47). M. roughleyi does not cause gross signs but, conversely, the disease tends to develop in oysters in apparently good condition. M. roughleyi is also intracellular and approximately 2 µm in diameter, and
is not detectable by histopathology for 2.5 months after initial infection (26); even then, it is very difficult to detect. Monoclonal antibodies and DNA probes are being developed, but are not likely to be available in the near future.

**Marteiliosis**

Oysters infected with *M. refringens* become emaciated, the digestive gland is brown to pale yellow, tissue necrosis occurs, and the mantle is sometimes translucent (29). The parasite is readily detectable histologically. Sydney rock oysters infected with *M. sydneyi* are shrunken by one- to two-thirds, and may have a translucent appearance (77). Again, infection is easy to detect histologically.

**Perkinsosis**

Macroscopically, *P. marinus* causes emaciation, gaping, pale digestive gland, mantle shrinkage, gonadal inhibition, and sometimes abscesses. *P. marinus* is readily grown in thioglycollate medium (15), but light infections can be missed using histology only. Polyclonal and monoclonal antibodies have been developed for detection of the parasite (25), and flow cytometry has been used to detect the pathogen in water samples (68) and host tissues (69). The gene sequence of *P. marinus* small subunit rRNA has been determined (30). Monoclonal antibodies and DNA probes have also been developed for *P. atlanticus* (41).

**KILLING, TREATMENT OR CONTAINMENT OF PATHOGENS**

**Iridoviral disease in oysters**

The survival parameters of oyster iridoviruses have not been reported, as further studies have been limited by the scarcity of infected oysters after the 1970s epizootics, by the lack of bivalve cell-lines and by low virion production in natural infections (21). Destruction of infected stocks and movement controls are the only possible methods of containment.

**Haplosporidiosis**

*H. nelsoni* appears to be able to move over several kilometres, but whether these are 'new' or 'new patent' infections is uncertain, because of difficulty in diagnosing light infections (S.E. Ford, personal communication). Holding *in vivo* for up to 2 weeks in 10 ppt salinity seawater at 20°C kills the parasite but not the host (31), and *H. nelsoni* does not cause disease at salinities below 15 ppt (10). Therefore, prolonged holding of oysters at low salinities (45), along with controls on movement (32) and use of selectively-bred resistant oysters in culture (33, 59), may lead to eradication or control.

**Bonamiosis**

Survival parameters have not been reported for either of the *Bonamia* spp. occurring in oysters, but transmission of *B. ostreae* occurs at temperatures as low as 4-5°C (43). No chemotherapeutants are known, and control measures are thus limited to movement restrictions.
Mikrocytosis

*M. mackini* is thought to be limited by temperature, with a maximum water temperature requirement of 12°C, although subclinical infections may persist for three months at 15°C (S.M. Bower, personal communication). The pathogen has been contained within a small geographical area by restrictions on movement. Husbandry practices may reduce disease (9). The survival parameters of *M. roughleyi* have not been reported, but the parasite thrives in saline waters (30-35 ppt) at low temperatures (10), and this disease problem can therefore be alleviated by culture at low salinity.

Marteliosis

Survival parameters of *M. refringens* have not been reported, but high salinities (35-37 ppt) limit development. As *M. refringens* is primarily spread by human agency, controls on movement may be effective in controlling the spread of infection.

Perkinsosis

Presporangia can survive at 4-28°C (18), the rate of development increasing with temperature. Low temperatures may prevent *P. marinus* from persisting in Delaware Bay, and from becoming epizootic in northern Chesapeake Bay (32). Although the parasite can survive in 3 ppt salinity (19), it is less virulent at < 9 ppt salinity (67). Controls on stock movement can be implemented (32), but this may only slow the spread of the pathogen (S.E. Ford, personal communication). *In vitro*, *P. marinus* can be controlled by a wide range of chemicals (57). Although lasalocid, malachite green, cycloheximide, monensin and sulphadimethoxine kill the parasite *in vitro*, only cycloheximide effectively kills the parasite *in vivo*; infection persists, however, in oysters treated with cycloheximide at 10 mg/l after 30 days (17). *P. olseni* survives at least one day at 0°C and 4°C, and at least 197 days at –60°C. Free prezoosporangia are killed within 30 minutes in 6 ppm chlorine, while those enclosed in tissue survive for > 2 hours. Free prezoosporangia do not survive for < 6 hours in distilled water, > 6 hours in 7 ppt sea water and < 10 minutes in 120 ppt saline solution at 50°C (40).

CONCLUSIONS

The life cycles and transmission of the pathogens causing two (*H. nelsoni*, *M. refringens*) of the six OIE notifiable diseases are unknown, and the life cycle of *Bonamia* sp. may prove to be more complex than it appears at present. The possible role of vectors in the spread of *H. nelsoni*, *Bonamia* spp., *M. refringens* and *P. marinus* is suspected but not understood. The lack of cell-lines makes identification of sub-clinical viral infections unreliable, and light infections with *Mikrocytos* spp. and *Bonamia* spp. cannot be diagnosed with certainty. Neither nucleic acid probes, nor monoclonal antibodies and the immunoassays based on them, are generally available for any of the pathogens causing OIE notifiable diseases. The unknown life cycles of *H. nelsoni* and *M. refringens* and the known pre-patent periods of *Bonamia* spp. complicate the establishment of diagnostic protocols. *Perkinsus* spp. are the only organisms for which considerable data on survival parameters (17, 18, 19, 40, 67) and information on chemical inhibition exist (57).
Therefore, although these are among the most intensively-studied bivalve pathogens, information on which to base decisions concerning the translocation of the host species is insufficient. The data would only permit an incomplete qualitative risk analysis, based largely on expert opinion, rather than a quantitative risk analysis based on published information. Although the high-risk hosts appear to be oysters (Ostrea spp., Crassostrea spp., T. chilensis and S. commercialis), M. refringens and P. marinus may be translocated in other hosts. The long periods during which organisms such as Bonamia sp. may exist in a non-virulent sub-clinical state, the sudden appearance of devastating pathogens such as Marteilia sp. in scallops (63), the relatively few countries in which – and brief time over which – mollusc diseases have been studied, make it likely that new pathogens will emerge. It is therefore premature to consider that only oysters pose a high risk, or to assume that only known pathogens may be involved in any translocation.

Those concerned with supervising the translocation of molluscs must deal with a high-risk situation, due to the lack of information, inadequate diagnostic techniques, and the problem of pathogens in a medium which both supports and transports them. If the risk is considered acceptable, and international translocation is to proceed, a translocation protocol must be developed which minimises the risks involved. Bearing in mind our incomplete knowledge of even the basic biology of serious bivalve pathogens, any such protocol must include stringent pre-export health certification and post-import quarantine, and the ICES and EIFAC/FAO guidelines should be regarded as minimum standards for the translocation of molluscs.

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LES MALADIES DES MOLLUSQUES SÉVISSANT DANS L'HÉMISPHERÉ SUD ET LES PROBLÈMES DE L'ÉVALUATION DES RISQUES ASSOCIÉS À CES MALADIES. – P.M. Hine.

Résumé : En Australie et en Nouvelle-Zélande, Bonamia sp. provoque des épidémies chez les huîtres plates (Tiostrea chilensis, Ostrea angasi) ; Marteilia sydneyi et Mikrocytos roughleyi sont à l'origine de maladies provoquant une forte mortalité dans les élevages d'huîtres creuses d'Australie (Saccostrea commercialis), et Perkinsus olseni affecte les ormeaux (Haliotis spp.). Marteilia lengehi, Marteillioide branchialis, d'autres espèces de Marteillioide ssp. et deux espèces de Haplosporidium sont considérées comme des agents pathogènes potentiels. L'étude des agents pathogènes responsables des maladies de mollusques devant être déclarées à l'Office international des épidémies révèle l'existence de lacunes importantes dans l'information disponible. Les cycles de vie et la transmission de Haplosporidium nelsoni et de
Marteilia refringens sont inconnus. Il n’existe pas de méthode sûre pour détecter Bonamia spp. et Mikrocytos spp.; on dispose rarement de sondes moléculaires et d’anticorps monoclonaux spécifiques; enfin, les connaissances sur les critères de survie des agents pathogènes et sur les traitements nécessaires sont très incomplètes. Des directives et des protocoles rigoureux s’imposent, conclut l’auteur, pour réduire au minimum les risques importants liés au transfert de mollusques.


LAS ENFERMEDADES DE LOS MOLUSCOS DEL HEMISFERIO AUSTRAL Y LOS PROBLEMAS DE EVALUACIÓN DE LOS RIESGOS ASOCIADOS A DICHAS ENFERMEDADES. – P.M. Hine.

Resumen: En Australia y Nueva Zelanda, Bonamia sp. provoca epizootias en la ostra plana (Tiostraea chilensis, Ostrea angasi); Marteilia sydneyi y Mikrocytos roughleyi son responsables de altas tasas de mortalidad en los viveros de ostras australianas (Saccostrea commercialis); Perkinsus olseni ataca a la oreja marina (Haliotis spp.). Marteilia lengehi, Marteilioides branchialis, otras especies de Marteilioides y dos especies de Haplosporidium son considerados como potencialmente patogénicos. El examen de los patógenos responsables de enfermedades de los moluscos de declaración obligatoria a la Oficina Internacional de Epizootias pone de manifiesto la existencia de importantes lagunas en la información disponible. Se desconocen los ciclos vitales y modos de transmisión de Haplosporidium nelsoni y Marteilia refringens; no pueden diagnosticarse con certeza las infecciones causadas por Bonamia spp. y Mikrocytos spp.; no se dispone generalmente de pruebas de anticuerpos monoclonales ni de sondas moleculares; y se sabe muy poco sobre los parámetros de supervivencia de estos patógenos y los métodos de tratamiento adecuados. El autor concluye que la minimización de los elevados riesgos asociados a los movimientos de moluscos requiere tanto protocolos estrictos como rígidas directrices.


REFERENCES


