AUSTRALIAN AQUATIC VETERINARY EMERGENCY PLAN

AQUAVETPLAN

Edition 1.0

Disease Strategy

Infection with *Candidatus* Xenohaliotis californiensis

(Withering syndrome of abalone)

Version 1.0, 2006

AQUAVETPLAN is a series of technical response plans that describe the proposed Australian approach to aquatic animal disease incursions. The documents provide guidance based on sound analysis, linking policy, strategies, implementation, coordination and emergency-management plans.

Primary Industries Ministerial Council

This disease strategy forms part of:

AQUAVETPLAN Edition 1.0

This strategy will be reviewed regularly. Suggestions and recommendations for amendments should be forwarded to:

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AQUAVETPLAN is available on the internet at:
http://www.affa.gov.au/aquavetplan

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IMPORTANT NOTE: Important regulatory information for infection with *Candidatus* Xenohaliotis californiensis is contained in the OIE *Aquatic Animal Health Code* (OIE 2006a), which is updated annually and is available on the internet at the OIE website:
http://www.oie.int/eng/normes/fcode/en_sommaire.htm
Further details are given in Appendix 1 of this manual.

DISEASE WATCH HOTLINES

These telephone numbers connect callers to the relevant state or territory officer to report concerns about any potential emergency disease situation. Anyone suspecting an emergency disease outbreak should use this number for immediate advice and assistance.

New South Wales	**1800 043 536**	**Northern Territory**	**1800 720 002**
Queensland	**07 3830 8550 (after hours)** **13 25 23 (DPI&F Call Centre Mon–Fri, business hours)**	**Victoria**	**136 186**
South Australia	**1800 065 522**	**Western Australia**	**1800 815 507**
Tasmania	**1800 005 555**		

Preface

This disease strategy for the control and eradication of infection with *Candidatus* Xenohaliotis californiensis (withering syndrome of abalone) is an integral part of the **Australian Aquatic Veterinary Emergency Plan**, or **AQUAVETPLAN (Edition 1)**.

AQUAVETPLAN disease strategy manuals are response manuals and do not include information about preventing the introduction of disease.

The Australian Quarantine and Inspection Service (AQIS) provides quarantine inspection for international passengers, cargo, mail, animals, plants and animal or plant products arriving in Australia, and inspection and certification for a range of agricultural products exported from Australia. Quarantine controls at Australia's borders minimise the risk of entry of exotic pests and diseases, thereby protecting Australia's favourable human, animal and plant health status. Information on current import conditions can be found at the AQIS ICON website.[1]

This strategy sets out the disease control principles for use in an aquatic veterinary emergency incident caused by the suspicion or confirmation of infection with *Ca* Xenohaliotis californiensis in Australia. The strategy was scientifically reviewed by the National Aquatic Animal Health Technical Working Group of the Aquatic Animal Health Committee, before being endorsed by:

- the Aquatic Animal Health Committee of the Primary Industries Standing Committee in June 2006; and
- the Primary Industries Standing Committee, out of session (PISC 11), in July 2006.

Infection with *Ca* Xenohaliotis californiensis is listed by the World Organisation for Animal Health (OIE, formerly Office International des Epizooties) in the *Aquatic Animal Health Code*.[2]

Detailed instructions for the field implementation of AQUAVETPLAN are contained in the disease strategies, operational procedures manuals and management manuals. Industry-specific information is given in the enterprise manual. The full list of AQUAVETPLAN manuals that may need to be accessed in an emergency is shown below:

Disease strategies
Individual strategies for each disease

Operational procedures manuals
Disposal
Destruction
Decontamination

Management manual
Control centres management

Enterprise Manual
Includes sections on:
– open systems
– semi-open systems
– semi-closed systems
– closed systems

[1] http://www.aqis.gov.au/icon32/asp/homecontent.asp
[2] http://www.oie.int/eng/normes/fcode/fcode2006_back/en_sommaire.htm

Aquatic Animal Diseases Significant to Australia: Identification Field Guide (Herfort 2004) is a source of information about the aetiology, diagnosis and epidemiology of infection with *Ca* Xenohaliotis californiensis and should be read in conjunction with this strategy.

This manual was prepared by Karina Scott, Australian Government Department of Agriculture, Fisheries and Forestry. The author was responsible for drafting the strategy, in consultation with a wide range of stakeholders from aquaculture, recreational fishing and government sectors throughout Australia. However, the text was amended at various stages of the consultation and endorsement process, and the policies expressed in this version do not necessarily reflect the views of the author. The author would like to thank Dr Carolyn S Friedman of the School of Aquatic and Fishery Sciences at the University of Washington, and Dr Judith Handlinger of the Fish Health Unit of the Tasmanian Department of Primary Industries, Water and Environment, for their help and support for this project. Contributions made by others not mentioned here are also gratefully acknowledged.

The format of this manual was adapted from similar manuals in AUSVETPLAN (the Australian veterinary emergency plan for terrestrial animal diseases) and from the AQUAVETPLAN **Enterprise Manual**. The format and content have been kept as similar as possible to these documents, in order to enable animal health professionals trained in AUSVETPLAN procedures to work efficiently with this document in the event of an aquatic veterinary emergency. The work of the AUSVETPLAN writing teams and the permission to use the original AUSVETPLAN documents are gratefully acknowledged.

The revised manual has been reviewed and approved by the following representatives of government and industry:

Government	**Industry**
Commonwealth of Australia	Australian Abalone Growers Association
State of New South Wales	
State of Victoria	
State of Queensland	
State of Tasmania	
State of South Australia	
State of Western Australia	
Northern Territory	
Australian Capital Territory	

The complete series of AQUAVETPLAN documents is available on the internet.[3]

[3] http://www.daff.gov.au/aquavetplan

Contents

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1 Nature of the disease

Withering syndrome is a disease of abalone (of the genus *Haliotis*) that has the potential to cause high levels of mortality in both farmed and wild abalone populations.

Infection of abalones with the causative organism causes body shrinkage and pathologic changes to epithelia. Moore et al (2001) suggest that the body shrinkage is not due solely to the malabsorption that results from the epithelial changes, but that decreases in the host's ability to consume food also play a role.

Withering syndrome has not been reported in Australia.

1.1 Aetiology

The aetiological agent of withering syndrome is the intracellular bacterium *Candidatus* Xenohaliotis californiensis (abbreviated *Ca* Xenohaliotis californiensis). *Ca* Xenohaliotis californiensis is a new genus and new species of intracellular prokaryote in the family Rickettsiaceae (Berthe 2003, Bower 2004). The term '*Candidatus*' in the literature indicates that it has been given provisional status as a new species.

Ca Xenohaliotis californiensis has a dimorphic rod-to-spherical shape. The average size of the bacillus form is 332 nm × 1550 nm, and the spherical morphotype has a mean diameter of 1405 nm. Reproduction is within intracytoplasmic vacuoles 14–56 µm in diameter (Friedman et al 2000a).

1.2 Susceptible species

Ca Xenohaliotis californiensis infects abalone of the genus *Haliotis*. Susceptibility varies between *Haliotis* species (Berthe 2002). Infection is most commonly observed in Californian black abalone (*H. cracherodii*), but also occurs in Californian red abalone (*H. rufescens*), pink abalone (*H. corrugata*), green abalone (*H. fulgens*), white abalone (*H. sorenseni*) and Japanese abalone (*H. discus hannai*) (Godoy and Muňoz 2003). Bower (2004) suggests that *H. midae* may also be susceptible. *Ca* Xenohaliotis californiensis can infect both wild and cultured abalone (Friedman et al 2003).

The susceptibility of other species of *Haliotis*, including Australian species, to infection has not been tested; however, until the full range of host susceptibility has been determined conclusively, it is reasonable to assume that all *Haliotis* species are susceptible.

Ca Xenohaliotis californiensis has not been reported as a human pathogen. Although infected abalone are suitable for human consumption, those with advanced withering syndrome will have a very atrophied foot muscle and may be rejected by processors for quality and aesthetic reasons (C Friedman, School of Aquatic and Fishery Sciences, University of Washington, pers comm, September 2005).

1.3 World distribution and occurrence in Australia

Withering syndrome is endemic on the southwest coast of North America — from Baja California, Mexico, north to San Francisco County, California (Friedman et al 2003), and has also been reported from Chile (Godoy and Muňoz 2003). The presence of *Ca* Xenohaliotis californiensis (without presence of withering syndrome) was reported in Iceland in June 2004 on farms that imported broodstock from California in 1988 (OIE 2004).

A disease with similar presentation but unknown aetiology has been reported from cultured *Haliotis discus hannai* on the northern coast of China (Bower 2004).

The finding of *Ca* Xenohaliotis californiensis in Iceland supports earlier suggestions that the geographic range of *Ca* Xenohaliotis californiensis could include any areas where California red abalone (*H. rufescens*) are cultured, including locations that have imported live abalone from other countries (Berthe 2003, OIE 2006b).

Ca Xenohaliotis californiensis has not been reported in Australia.

Rickettsial-like organisms were found in Australian abalone during a national survey; however, the appearance of the organisms and the characteristic histological changes in the digestive tubules were quite dissimilar from those described for withering syndrome (Handlinger et al 2005).

1.4 Diagnosis of infection with *Ca* Xenohaliotis californiensis

A number of methods are available to help diagnose infection with *Ca* Xenohaliotis californiensis. However, a positive result from any single method is not considered confirmatory.

The following sections outline available diagnostic methods.

1.4.1 Field methods: clinical signs and gross pathology

Abalone can be infected with *Ca* Xenohaliotis californiensis and show no clinical signs of disease. When infection results in disease, the incubation period is prolonged (245 days in black abalone, 130 days in red abalone) but comparable to the range of incubation periods reported for other known rickettsial diseases (Friedman et al 2003). Death occurs within one month of development of clinical signs of withering syndrome at temperatures of 18–20°C (Friedman 1996).

One of the earliest clinical signs of adverse health linked to withering syndrome is a diminished feeding rate (Moore et al 2001); however, this is also a common response to other diseases. The primary clinical sign of withering syndrome is shrinkage of the body mass and retraction of the mantle (Moore et al 2000a). The clinical signs of withering syndrome may include:

- weakness (Moore et al 2000a);
- lethargy (Moore et al 2000a);

- mantle retraction, and atrophied (or greatly diminished) and flaccid foot muscle, so that it appears that the animal does not fit its shell (Haaker et al 1992, VanBlaricom et al 1993, Friedman et al 1997);
- decreased response to tactile stimuli (Haaker et al 1992, VanBlaricom et al 1993, Friedman et al 1997);
- easy detachment from the substrate by hand (Bower 2004);
- inability of individuals to right themselves when turned upside down (Bower 2004);
- in the wild, loose attachment of individuals to rocks; they may hang from the rocks and have greater vulnerability to dislodgement by waves (Haaker et al 1992, VanBlaricom et al 1993);
- poor, or lack of, gonadal development (Friedman et al 1997, Moore et al 2000a); and
- death (Friedman et al 1997, 2000a; Moore et al 2000a).

Collectively, these signs have been termed 'withering syndrome'. Clinical signs alone are not considered sufficient to definitively diagnose infection with *Ca* Xenohaliotis californiensis; however, the foot withering is an important clinical sign of this disease.

1.4.2 Laboratory methods

Sample submission

In the first instance, samples should be submitted to the relevant state or territory laboratory. The laboratory should be contacted directly to ensure that samples are collected using techniques that will satisfy its requirements. In the event that the laboratory cannot be contacted (eg out of hours), formalin-fixed and paraffin-embedded material should be submitted for histopathology and in situ hybridisation. For analysis by polymerase chain reaction (PCR), frozen tissues (or, if this is not possible, tissue preserved in 95% ethanol) should be submitted (M Crane, Australian Animal Health Laboratory, pers comm, February 2005).

Sampling equipment may be available on farm or may be obtained from state or territory fisheries or agricultural officers (see the AQUAVETPLAN **Enterprise Manual**[4] for contact details). Advice on packaging of samples for shipment is also available from state and territory laboratories and the Fish Diseases Laboratory at the Australian Animal Health Laboratory.

Microscopy

Tissue imprints

Tissue imprints (from sections of the posterior oesophagus) may be used to detect moderate to high intensities of infection with *Ca* Xenohaliotis californiensis. However, histology is more sensitive than tissue imprints.

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[4] http://www.daff.gov.au/aquaticanimalhealth

Histopathology

Ca Xenohaliotis californiensis infects the epithelial cells of the posterior oesophagus (post-oesophagus), digestive gland and, to a lesser extent, intestine (Berthe 2003). The main pathological lesions characterising the disease are the presence of intracellular bacteria in the digestive epithelia and morphological changes in these areas. The morphological changes in the digestive gland vary between species, but may include metaplasia and/or degeneration.

Degeneration is characterised by an increase in connective tissues, inflammation, and atrophying of the digestive tubules (Berthe 2003). Metaplasia refers to the substitution of one mature cell type with another. The metaplastic changes in abalone with withering syndrome involve the replacement of terminal secretory or absorptive acini with absorptive or transport ducts similar in appearance to the post-oesophagus (Gardner et al 1995; Friedman et al 2000a, 2002; Braid 2004). Some hyperplasia of the absorptive or transport ducts may also be involved.

The foot of affected individuals contains fewer and less organised muscle bundles than in unaffected individuals, and abundant connective tissue. It may also contain more brown pigmented serous cells than in unaffected individuals (VanBlaricom et al 1993).

Infection with *Ca* Xenohaliotis californiensis may be detected through histological examination of sections of the post-oesophagus, digestive gland, and foot muscle. Presumptive diagnosis by histology must include the observation of morphological changes to the digestive gland and the presence of the bacterium.

Transmission electron microscopy

Transmission electron microscopy can be used to confirm the presence of intracellular bacteria with rickettsia-like morphology. However, this method cannot differentiate *Ca* Xenohaliotis californiensis from other nonpathogenic members of the group, which have also been detected in abalone (Diggles et al 2002; J Handlinger, Senior Veterinary Pathologist (Aquatic Animals), DPIWE, Tasmania, pers comm, August 2004).

Detailed information on the conduct of these tests is available in the OIE *Manual of Diagnostic Tests for Aquatic Animals* (OIE 2006b).

Molecular techniques

Polymerase chain reaction

A PCR test has been developed for detecting the rickettsia-like prokaryotes (RLP) associated with withering syndrome (Andree et al 2000). PCR detects the presence of the agent's deoxyribonucleic acid (DNA) but cannot determine whether viable agent or infection is present. A positive result may therefore provide a presumptive diagnosis but is not confirmatory.

In situ hybridisation

In situ hybridisation (ISH) techniques have been developed for detecting the RLP associated with withering syndrome (Antonio et al 2000). ISH is a useful tool that allows a specific probe hybridised to the target organism to be visualised in infected tissues.

1.4.3 Confirmation of diagnosis

For the purposes of this manual:

- confirmation of withering syndrome caused by infection with *Ca* Xenohaliotis californiensis requires the observation of clinical signs, presence of digestive gland metaplasia and a positive result from either PCR or ISH; and
- confirmation of subclinical infection with *Ca* Xenohaliotis californiensis requires a positive result from ISH.

1.4.4 Differential diagnosis

The clinical signs of withering syndrome are not specific to the disease. They are also seen in starvation (Moore et al 2000a), poor food supply, poor environmental conditions and diseases other than withering syndrome (Moore et al 2001).

The presence of digestive gland metaplasia (where functional tissue, including secretory cells, is replaced with cells similar in appearance to those of transport ducts) appears to be typical of withering syndrome (Moore et al 2001) and is quite pronounced in this condition (C Friedman, School of Aquatic and Fishery Sciences, University of Washington, pers comm, March 2006).

Low numbers of rickettsia-like organisms have also been reported, without associated gut pathology, in the digestive tract of abalone (*Haliotis midae*) from culture facilities in South Africa (Mouton 2000), from New Zealand (Diggles et al 2002), and recently from Australia (J Handlinger, Senior Veterinary Pathologist (Aquatic Animals), DPIWE, Tasmania, pers comm, August 2004). Those from New Zealand have been shown to be negative for *Ca* Xenohaliotis californiensis using the ISH test (C Friedman, School of Aquatic and Fishery Sciences, University of Washington; and B Diggles, Principal Consultant, DigsFish Services, pers comm, August 2005), as have those from South Africa (J Handlinger, Senior Veterinary Pathologist (Aquatic Animals), DPIWE, Tasmania, pers comm, August 2004). The histological appearance of the rickettsia-like organisms found in Australia was quite dissimilar to *Ca* Xenohaliotis californiensis, and cellular evidence of withering syndrome was not present (Handlinger et al 2005

1.5 Resistance and immunity

1.5.1 Innate and passive immunity

Abalone haemocytes have a nonspecific immunity function that involves chemotactic, phagocytic and chemiluminescent abilities. Friedman et al (2000b) showed experimentally that this immune capacity is affected in abalone with withering syndrome: haemocytes displayed increased chemotactic activity but also displayed a compromised ability to engulf and destroy foreign particles. This compromised ability may play a role in the mortality associated with withering syndrome. It has been suggested that the increase in chemotactic activity may be due to the degeneration of the digestive gland and the use of the foot muscle as an energy source (Friedman et al 2000b).

One study has suggested that Californian red abalone (*H. rufescens*) may be more refractory to withering syndrome, although severe losses of red abalone with clinical signs of withering syndrome have been recorded (Moore et al 2000b). It has also been implied that heat stress reduces the immune function of abalone (Moore et al 2000a).

1.5.2 Active immunity

Molluscs do not have B/T-type lymphocytes and do not produce antibodies (Berthe 2002). There is no evidence that molluscs can generate long-term acquired immunological memory comparable to that found in vertebrates (Benkendorff 2003; D Raftos, Department of Biological Sciences, Macquarie University, pers comm, February 2005).

1.6 Epidemiology

1.6.1 History of *Ca* Xenohaliotis californiensis

The first reports of withering syndrome came from commercial abalone fisherman, who observed gross signs typical of the syndrome on the south side of Santa Cruz Island, California, around 1985 (Haaker et al 1992, Lafferty and Kuris 1993). The aetiology of the syndrome was undescribed until 2000 (Friedman et al 2000a).

Withering syndrome was initially recorded in Californian black abalone. It was reported in wild red abalone from San Miguel Island, California, in 1994, and was first observed in cultured red abalone during the 1997–1998 El Niño event (Friedman et al 2003).

1.6.2 Reservoirs

Water

Transmission experiments suggest that *Ca* Xenohaliotis californiensis can survive in sea water (Moore et al 2001) for an undetermined time (C Friedman, School of Aquatic and Fishery Sciences, University of Washington, pers comm, February 2005).

Sediment

No data are available on the survival of *Ca* Xenohaliotis californiensis in sediment or other environmental reservoirs.

Animal reservoirs

The presence of *Ca* Xenohaliotis californiensis has not been reported in species other than the genus *Haliotis*. However, PCR analysis suggests that some colonial ascidians may concentrate the bacterium via feeding (C Friedman, School of Aquatic and Fishery Sciences, University of Washington; and J Moore, California Department of Fish and Game, pers comm, July 2004).

1.6.3 Predisposing factors

Intensity of *Ca* Xenohaliotis californiensis infection

A clear association has been observed between the severity of withering syndrome and level of *Ca* Xenohaliotis californiensis infection in both cultured and wild

Californian red abalone; however, this relationship is not well defined in the more heavily impacted Californian black abalone (Friedman et al 2002).

Temperature

Elevated water temperatures (4.5°C above the average culture temperature of 14°C) have been shown to increase mortality, expression of clinical signs and RLP burdens in cultured asymptomatic Californian red abalone (Moore et al 2000a, Friedman et al 2003). In Californian black abalone, elevated water temperature accelerates mortality associated with withering syndrome (Friedman et al 1997).

The actual increase in water temperature that may produce these effects in Australian abalone may differ from that seen overseas and would depend on the normal local seawater temperatures and the temperature threshold of the species under culture. It should be noted, however, that cool-water *Haliotis* species are stressed by heat, and heat is a predisposing factor to almost all diseases of abalone (Dr Stephen Madigan, Primary Industries and Resources South Australia, pers comm, September 2005).

1.6.4 Modes of transmission

Horizontal spread

Several studies have demonstrated horizontal transmission of *Ca* Xenohaliotis californiensis.

Ca Xenohaliotis californiensis is transmissible between abalone by cohabitation, although close or direct physical contact is not required (Moore et al 2001, Friedman et al 2002). The movement of infected animals can introduce the causative agent to new areas. For example, the out-planting of hatchery-reared abalone in California, as part of restoration efforts, has been linked to the present distribution of *Ca* Xenohaliotis californiensis (Friedman and Finley 2003).

Friedman et al (2002) suggest that *Ca* Xenohaliotis californiensis is transmissible via a waterborne, faecal–oral route, stating that *Ca* Xenohaliotis californiensis may be shed in the faeces of infected abalone (Friedman et al 2002). This is supported by the finding of bacterial foci in the digestive epithelium and the observation of both intact and lysed RLP foci in lumina of the digestive tract (Friedman et al 2002). Moore et al (2001) suggest that ingestion of food contaminated with *Ca* Xenohaliotis californiensis in the marine environment is the most likely mode of transmission.

Vertical spread

No studies have examined the vertical transmission of *Ca* Xenohaliotis californiensis.

1.6.5 Environmental impact

Since 1985, widespread mass mortalities associated with withering syndrome have caused significant reductions in the wild populations of Californian black abalone on the mainland coast of central California. The largest decline was seen at the southernmost site, with a 97% reduction in abalone numbers between 1992 and 1995 (Altstatt et al 1996). The wild abalone fisheries in southern California have been severely depleted by the disease (Davis 1993, Lafferty and Kuris 1993, Friedman et al 1997).

1.6.6 Inactivation of the organism

No information is currently available on the efficacy of specific disinfection agents against *Ca* Xenohaliotis californiensis.

2 Principles of control and eradication

2.1 Introduction

The discovery of *Ca* Xenohaliotis californiensis in Australia (with or without the clinical signs of withering syndrome) may present a serious threat to both the wild and farmed abalone industries. A number of different control measures may be effective in minimising the impact of such an event. This section provides background information to enable the choice of the most appropriate control measure following detection of *Ca* Xenohaliotis californiensis in Australia.

There are essentially two main control options should *Ca* Xenohaliotis californiensis be detected in Australia:

- *Eradication* — eradication of *Ca* Xenohaliotis californiensis from Australia (highest level of control measure and cost); and
- *Containment, control and zoning* — containment of *Ca* Xenohaliotis californiensis to areas with endemic infection, prevention of further spread and protection of uninfected areas.

A third option, control and mitigation of the disease, is not favoured because it could lead to the establishment of *Ca* Xenohaliotis californiensis in both the farmed and wild abalone industries.

The basic principles of eradication and other control responses are described in the AQUAVETPLAN **Enterprise Manual**[5] and the AQUAVETPLAN **Control Centres Management** manual.[6] The AQUAVETPLAN **Enterprise Manual** lists the state and territory legislation relating to disease control and eradication.

Within these overall options, the general principles for the control and eradication of *Ca* Xenohaliotis californiensis include:

- rapid detection and identification of infection;
- rapid definition of the nature and extent of the problem;
- rapid definition and implementation of control measures;
- prevention of spread of *Ca* Xenohaliotis californiensis by controlling stock movement, within and between farms and water bodies; and
- maintenance of good management practices and high standards of hygiene.

The most appropriate option will depend on:

- presence or absence, and location, of a reservoir of infection;
- chances of success of eradication;

[5] http://www.daff.gov.au/aquaticanimalhealth
[6] http://www.daff.gov.au/aquaticanimalhealth

- level of risk accepted for the potential future spread of infection (eg associated with grow-out of infected populations);
- short-term costs of control and disruption to production;
- long-term costs of production with or without the presence of *Ca* Xenohaliotis californiensis; and
- long-term costs of control should *Ca* Xenohaliotis californiensis become endemic.

In Australia, abalone are collected from the wild (open), grown in tanks on shore (semi-closed) or grown in floating barrels moored off ropes, in concrete hides situated on the sea floor or in plates in ring cages (all classified as semi-open systems). The type of system in which the infected abalone exist will greatly influence the response options, as outlined below.

Open systems

If an outbreak occurs in an open system (*Ca* Xenohaliotis californiensis found in wild abalone), eradication will be extremely difficult. Methods such as removal of all abalone from the water body by hand would be time consuming, and it would be difficult to ensure 100% removal. The use of any chemicals to aid removal of individuals may also be impractical and environmentally controversial. Establishment of 'fire breaks' (that is, artificial barriers created by dredging) in an open system for the control of *Bonamia ostrae* in *Ostrea edulis*[7] had limited success in Holland (van Banning 1988). This technique was also trialled in New Zealand's Foveaux Strait oyster fishery in 1990, to minimise the spread of *B. exitiosa* in *O. chilensis*[8]; however, there was no evaluation of its effectiveness (K Michael, National Institute of Water and Atmospheric Research, New Zealand, pers comm, February 2005). It was not included as a management tool in a fisheries management plan for the Foveaux Strait drafted in 1995 (Anon 1995).

Semi-open systems

In semi-open systems, the movement of abalone can be controlled, but there is little or no control over the flow of water. The chemical treatment of abalone in semi-open systems may impact on the surrounding environment and this must be taken into consideration before the use of any antibiotic or disinfectant treatments. In addition, disease may have spread beyond the farm into local wild abalone, which would reduce the suitability of eradication as a first-response option.

Semi-closed systems

In semi-closed systems, the movement of abalone can be controlled and there is partial control of the distribution and flow of water; however, the extent to which input and output water can be contained may differ between farms. Control of seawater temperatures may reduce parasite transmission and disease expression (Friedman et al 2003), but farms typically lack the ability to do this. The use of any antibiotic or disinfectant treatment would be easier and more effective in a semi-

[7] *Bonamia ostrae* is an intracellular parasitic pathogen reported from the oyster *Ostrea edulis.*
[8] *Bonamia exitiosa* is an intracellular parasitic pathogen reported from the oyster *Ostrea chilensis.*

closed system than in an open or semi-open system. The use of pathogen-free saltwater from a bore to establish disease-free farms could also be investigated. Disinfection of water entering and/or exiting the facility may also be an option. Any decision to use chemicals to control infection (or create buffer zones) would need to take into account the environmental legislation and responsibilities in each state or territory.

In both semi-open systems and semi-closed systems, the critical step in deciding how to proceed will be an assessment of the extent of disease spread that has taken place.

2.2 Methods to prevent spread and eliminate pathogens

2.2.1 Quarantine and movement controls

The following quarantine and movement controls could be implemented immediately upon suspicion of withering syndrome or infection with *Ca* Xenohaliotis californiensis.

Establishment of quarantine areas

Specified areas for quarantine control (shown in Figure 1) include the following (see the AQUAVETPLAN **Enterprise Manual** for more details):

- *declared area* — includes restricted area and control area
- *restricted area* — an area around infected premises or areas
- *control area* — a buffer between the restricted area and free areas
- *free area* — noninfected area (this area is not considered a 'declared area' and may include large areas of Australia in which the presence or absence of *Ca* Xenohaliotis californiensis remains unassessed).

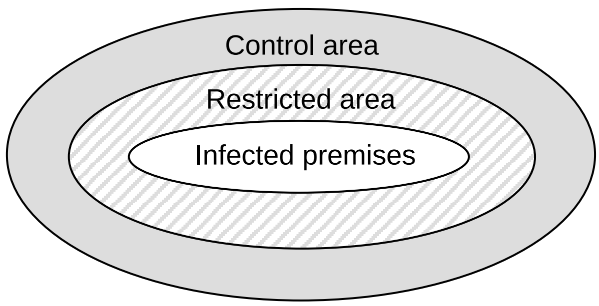

Figure 1 Establishment of specified areas to control withering syndrome

In the declaration of quarantine areas, the following factors need to be taken into account:

- industries involved;
- environmental factors;
- processing options (animals and products);
- natural vs artificial barriers or boundaries;

- nature of the outbreak; and
- presence of native abalone populations.

The following practices must be considered when implementing control strategies:

- possible poaching activities within the area;
- recreational abalone fishing within the area;
- abalone harvesting and transportation to processing plants;
- discharge of processing plant effluent;
- movement of farm vehicles and personnel; and
- disposal of dead abalone.

Movement controls

Movement controls could include:

- bans on the movement of live abalone into, within, or out of restricted and control areas;
- bans on the movement of live abalone into noninfected areas;
- restrictions or bans on movement of people, vehicles or equipment within and between farms or water bodies containing abalone within the declared area; and
- suspension of recreational abalone fishing within the declared area.

The implementation of bans and restrictions will be a dynamic process, determined by the location and extent of the disease outbreak and whether the aim is to eradicate the agent or to control its spread. Some restrictions may be impractical or unnecessary, but others will be of critical importance to eradication or control.

The feasibility of the restrictions and bans and the extent to which they are enforced will depend on the location of infection, the location and type of enterprises affected and the control response option chosen.

Zoning

If *Ca* Xenohaliotis californiensis were to become endemic in specific regions of Australia, a zoning policy specific for withering syndrome may be necessary to protect noninfected areas and to prevent further spread of infection. Zones would be based on the distribution of abalone species and of any vector species present, the geographical and hydrological characteristics of water bodies and landform, and predictions of the most likely method of spread of disease. Zoning may rely on the identification of biogeographic barriers. A corresponding surveillance and monitoring program for detection of *Ca* Xenohaliotis californiensis would be required to support the zoning policy. Principles of zoning for infected and noninfected zones in Australia are outlined in the AQUAPLAN Zoning Policy Guidelines[9] and in the OIE *Aquatic Animal Health Code* (OIE 2006a).

2.2.2 Tracing

Tracing the spread of an agent is the process of retrospectively determining its mode and pattern of spread. Tracing investigations are crucial in determining all confirmed and potential locations of the agent, as well as in defining restricted and control areas.

The information gathered from tracing will assist in determining the most appropriate response action. The immediate steps required are to trace-back all contacts with infected abalone, premises and sites (to establish the origin of the outbreak) and to trace-forward all contacts with infected abalone, premises and sites (to establish the current location and potential spread of infection).

The following must be traced:

- abalone — broodstock and seed;
- abalone products — abalone for consumption or for use as bait, effluent and waste products from processing;
- water — input and output;
- vehicles — transport vehicles, feed trucks, visitors' cars, boats;
- materials — feed, tanks, floating installations (where appropriate), tools and instruments;
- personnel — farm workers, sales and feed representatives, tradespeople, veterinarians, scientists, technicians and visitors; and
- abalone diving industry — movement of divers (recent dive sites, deliveries of broodstock).

Tracing an outbreak in an open system may be very difficult.

Neighbouring abalone populations

Neighbouring abalone farms and processing plants may become, or may already be, infected. Maps showing the location of neighbouring abalone farms, processing plants and waterways, and hydrographic data, are necessary to monitor the potential spread of *Ca* Xenohaliotis californiensis. The location of susceptible abalone species and animal reservoirs in the vicinity of the infected site should also be noted. Other sites of infection may be identified if a number of facilities share common water.

For information on the location of farming establishments and wild abalone populations at risk of infection, the relevant state or territory fisheries or

[9] http://www.daff.gov.au/aquaticanimalhealth

agriculture agency should be contacted (see the AQUAVETPLAN **Enterprise Manual**,[10] for contact details).

2.2.3 Surveillance

Surveillance, using screening for clinical signs and laboratory testing, is necessary to:

- define the extent of infection;
- support tracing activities;
- detect new outbreaks;
- establish restricted and control areas to which quarantine and movement restrictions are applied;
- establish disease-free and infected areas or zones for a withering syndrome zoning program; and
- monitor the progress and success of an eradication strategy.

2.2.4 Treatment of infected abalone

Reducing water temperatures may reduce parasite transmission and disease expression, but farms do not typically have the ability to regulate water temperatures (Friedman et al 2003).

Tetracycline can reduce or eliminate the development of symptoms of withering syndrome upon injection into the foot muscle of infected asymptomatic abalone (Friedman et al 2003). Oral administration of oxytetracycline-medicated feed (for 14 consecutive days) to asymptomatic abalone has also been shown to effectively control withering syndrome. Oxytetracycline residues declined to less than two parts per million between 15 and 22 days post-treatment. The treatment caused significant and persistent long-term reductions in both the degree of morphological changes in the digestive gland and the intensity of bacterial infection (Friedman et al 2003).

However, once clinical signs are observed, tetracycline is not effective in treating withering syndrome (Friedman et al 2003). Due to the influence of temperature on the development of clinical signs, and the chronic nature of withering syndrome, treatments must therefore be administered in infected individuals before the onset of warm water events and the onset of clinical signs. Treatment by injection would not be practical for large numbers of animals; however, farm populations may feasibly be treated with medicated feed.

The decision to treat abalone depends on the control measures implemented and the availability of effective antibiotics. Considerations for the use of antibiotics against withering syndrome include:

- availability of the treatment — no antibiotics, including oxytetracycline and tetracycline, are currently registered in Australia for use in food-producing

[10] http://www.daff.gov.au/aquavetplan

molluscs; for information about the process for gaining approval of antibiotics for use in Australia, see Appendix 2;

- cost of treatment, including labour, administration and equipment; and
- drug withholding periods — where antibiotic treatment is available, drug withholding periods must be adhered to; treatment of abalone populations close to harvesting may preclude their sale for human consumption.

Before the administration of any treatment, it is preferable to obtain confirmatory advice from the diagnostic laboratory that *Ca* Xenohaliotis californiensis is present. However, it should be noted that the need to wait for results will delay treatment.

2.2.5 Destruction of abalone

Harvest and destruction must be hygienic and there must be no spillage of the waste produced by these activities. The most appropriate method of destruction (see the AQUAVETPLAN **Operational Procedures Manual — Destruction**[11]) will depend on the following factors:

- size and number of abalone;
- deadline for harvest or destruction, which depends on the pressure of infection and the risk of further spread;
- slaughter facilities — site, equipment and methods available; and
- experience and availability of personnel.

If necessary, anaesthetic may be administered to aid in harvesting abalone. Information on anaesthetics that are registered for use in abalone can be found in the Public Chemical Registration Information System of the Australian Pesticides and Veterinary Medicines Authority.[12] This database contains details of agricultural and veterinary chemical products that are registered for use in Australia, including the product name, active constituents and details of the registering company. Most products will only be available for use on veterinary prescription.

There is little risk of spread of *Ca* Xenohaliotis californiensis through the movement of shell, provided that the shell is empty and free from all digestive gland residues (C Friedman, School of Aquatic and Fishery Sciences, University of Washington, pers comm, February 2005), although it is likely to be more practical to dispose of the whole animal.

2.2.6 Disposal

Disposal must be immediate to decrease infection pressure on the site (see the AQUAVETPLAN **Operational Procedures Manual — Disposal**[13] for details). Diseased and dead abalone are the main source of *Ca* Xenohaliotis californiensis in

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[11] http://www.daff.gov.au/aquavetplan

[12] http://services.apvma.gov.au/PubcrisWebClient/welcome.do;jsessionid=GzFC5KrfzbtkJB6KrV8ZGKL6d2vZDhNFvnXhG6kJZbkYg3yT7HXL!-804521097

[13] http://www.daff.gov.au/aquavetplan

the environment. They should be removed as soon as possible and disposed of, together with other waste, to prevent further spread of infection. If burial is the preferred option, burial sites must be chosen carefully to prevent contact (both current and future) with waterways, groundwater or vectors.

2.2.7 Decontamination

Effective decontamination requires that equipment, materials, tanks and buildings are thoroughly cleaned before disinfection. Due to differences in farming enterprises, disinfection protocols may need to be determined on an individual basis, involving discussions between the farm manager and the state or territory chief veterinary officer (CVO) and/or director of fisheries. The protocol should take into consideration the factors outlined in Section 1.6, in particular:

- the source and location of infection;
- the type of enterprise (eg farm or processing plant);
- the construction materials of the buildings or structures on the site;
- the design of the site and its proximity to other buildings or waterways;
- current disinfection protocols;
- environmental impact of the disinfectant protocol; and
- availability of approved, appropriate and effective disinfectants.

See the AQUAVETPLAN **Operational Procedures Manual — Decontamination**[14] for details of disinfectant chemicals and their indications.

2.3 Environmental considerations

Environmental considerations in the control of withering syndrome include the following:

- Discharge of infected, or potentially infected, effluent into catchment areas or natural waterways may lead to further spread of infection and the establishment of reservoirs of infection in wild abalone populations and waterways.
- The use of disinfectants or antibiotics may impact on the environment, especially if used in larger than normal quantities or concentrations, as is possible in a disease control situation. The local environmental protection agency may need to be consulted (see the AQUAVETPLAN **Enterprise Manual**[15] for appropriate contact information).
- The destruction and disposal of infected material may have an impact on the environment, and this must be minimised while preventing the dissemination of infection.

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[14] http://www.daff.gov.au/aquavetplan
[15] http://www.daff.gov.au/aquaticanimalhealth

2.4 Sentinel and restocking measures

Restocking should only occur once it has been ascertained that *Ca* Xenohaliotis californiensis is no longer likely to be present in the water body or aquaculture system. This may involve the holding of RLP-free, susceptible sentinel abalone in cages at different locations in the water body. It may be difficult to ascertain that *Ca* Xenohaliotis californiensis is no longer present, as sentinel abalone may not be exposed to the organism if there are low population densities of infected abalone in the water body. The length of exposure time for sentinel stock would also be critical.

Abalone used for restocking must be disease free.

Surveillance (using methods such as video or diver transects) to verify that no abalone remain following an outbreak may also be required. The survival time of *Ca* Xenohaliotis californiensis outside an abalone host is not known.

2.5 Public awareness

Any public awareness campaign must emphasise education, surveillance and cooperation from industry, governments and the community in order to control potential outbreaks of withering syndrome in Australia. Such campaigns should also emphasise that *Ca* Xenohaliotis californiensis does not pose a human health risk.

2.6 Feasibility of control in Australia

The feasibility of controlling an outbreak of withering syndrome in Australia depends upon both the nature of the outbreak (including whether it occurs in an open, semi-open or semi-closed system) and the control management strategy adopted. As outlined in Section 2.1, there are two broad control options for withering syndrome in Australia:

- eradication — eradication of *Ca* Xenohaliotis californiensis from Australia (highest level of control measure and highest initial cost); and
- containment, control and zoning — containment of *Ca* Xenohaliotis californiensis to areas with endemic infection, prevention of further spread and protection of uninfected areas.

2.6.1 Eradication

Eradication is likely to be successful in a closed system. In open or semi-open systems, the success of an eradication strategy would depend on the availability of resources for surveying and destocking wild abalone in the immediate area. Due to the long incubation period associated with infection with *Ca* Xenohaliotis californiensis, the first observations of clinical signs in an open system would be made some time after any introduction of the agent, increasing the likelihood of spread of the agent before its detection and reducing the chance of eradication.

Eradication is unlikely to be successful or feasible if epidemiological investigations determine that infection is widespread, has no point source, is unable to be contained, or is present or potentially present in wild abalone species or the sea.

This also reflects the lack of a full understanding of how the pathogen is transmitted and how it survives in the aquatic environment.

An eradication strategy would need to be accompanied by intensive surveillance of the wild abalone population. It should aim to prevent any further exposure of unexposed abalone populations to *Ca* Xenohaliotis californiensis and prevent the spread of infection.

Unexposed abalone

Young (pre-market sized) unexposed abalone may be allowed to grow out, provided that there has been no risk of infection and that there is no risk of future infection. Older abalone that have had no possible exposure to infection may be harvested for human consumption.

Strict hygiene protocols for the farm, transportation and processing would be necessary. On-farm processing may be preferable, if the site is infected, to prevent the spread of infection during transport to off-site processing plants.

Immediate destruction of unexposed abalone populations located within a declared area will decrease the chance of spread of infection to abalone stocks.

Exposed or potentially exposed, clinically normal abalone

Rapid removal of these populations from the water is essential. Normal, or controlled, grow-out is not an option in an eradication campaign for exposed or potentially exposed, clinically normal farmed abalone populations because they may harbour *Ca* Xenohaliotis californiensis, and so pose a high risk of a future outbreak. There are two options for the destination of these abalone:

- The abalone may be harvested for human consumption. This activity will carry a risk of further transfer of infection, which may jeopardise the success of an eradication strategy unless it is carried out under strict control measures.
- Abalone that cannot be harvested for human consumption (ie not market size) should be immediately destroyed (under strict control measures). This is very effective at decreasing the infectious load on a site and minimising the spread of infection.

Control measures necessary to prevent further spread of infection include:

- disinfection of all equipment and personnel involved in harvesting, destruction and processing;
- application of quarantine restrictions and procedures to the infected site, including for personnel, equipment and vehicles;
- on-site processing — possibly the only option if quarantine restrictions are in place;
- strict movement and disinfection procedures for the transport of abalone to off-site processing plants, which will then become infected sites that are subject to quarantine procedures;
- holding, treatment and safe disposal of harvest or processing effluent (including holding water and any waste material); and
- ensuring that the final product will not result in the spread of infection.

Clinically diseased abalone

Immediate removal, destruction and disposal of all diseased and dead abalone are essential to the success of an eradication strategy. These abalone, along with infectious waste, are the main source of *Ca* Xenohaliotis californiensis in the environment.

2.6.2 Containment, control and zoning

Unexposed abalone

Control options for unexposed abalone from within the declared zone are the same as those outlined in Section 2.6.1. The implementation of a zoning policy, and associated control measures, to maintain uninfected zones would be necessary. There would be no restrictions on production in the free zones.

Exposed or potentially exposed, clinically normal abalone

A successful zoning program will rely on the implementation of movement restrictions for exposed or potentially exposed abalone that prevent spread of *Ca* Xenohaliotis californiensis to uninfected zones. The feasibility of a zoning program will depend on farm management practices, the extent to which infection has already spread, and the location of reservoirs of infection. This can only be assessed at the time of the outbreak, taking into account movement restrictions required on abalone, people, vehicles and boats, and market access for the abalone products and byproducts. If young abalone are allowed to grow out, they must be treated as infected.

In a declared area, normal or controlled grow-out and slaughter may be feasible without further spread of infection or onset of clinical signs in covertly infected abalone. This may be achieved through either reducing water temperatures or treatment with an approved antibiotic (Friedman et al 2003). These measures are likely to be feasible only in a farmed semi-closed system. To prevent spread of infection, care must also be taken when processing and transporting final products to the designated market.

Treatment of exposed or potentially exposed but clinically normal abalone may be possible, as outlined in Section 2.2.4.

Immediate destruction of the abalone is an option for containment, control and zoning, as it is very effective at decreasing the infectious load on a site and minimising the spread of infection.

Clinically diseased abalone

These abalone, along with infectious wastes, are considered to be the main source of *Ca* Xenohaliotis californiensis in the environment and constitute the greatest risk for spreading the infection to uninfected zones. The only real option for clinically diseased abalone is immediate destruction. Antibiotic treatment for these individual abalone is not effective (Friedman et al 2003). Burial sites should be chosen carefully to prevent contact with waterways or vectors (refer to the AQUAVETPLAN **Destruction**[16] and **Disposal**[17] manuals for further details).

2.6.3 Trade and industry considerations

Where withering syndrome is endemic, the only industries that have been affected by the disease are the abalone farming industries. However, it is impossible to predict whether other aquatic farming industries in Australia would be affected. There may be some 'collateral damage' should the public believe that all molluscs are diseased.

Withering syndrome is not restricted to aquaculture situations and, as previously mentioned, the disease has had severe impacts on wild abalone fisheries in southern California. The impact on wild fisheries should be taken into account as it could be considerable.

Trade regulations, market requirements and food safety standards must be considered as part of a control strategy.

Export markets

Withering syndrome is endemic along the southwest coast of North America from Baja California, Mexico, north to San Francisco County, California, and infection with *Ca* Xenohaliotis californiensis has been reported in Iceland. Because the disease is listed by the OIE, some countries may require imports to be certified free from *Ca* Xenohaliotis californiensis infection. The Australian Quarantine and Inspection Service is responsible for the health certification of all exports and should be contacted for further information about export requirements.

Domestic markets

A cautious approach is required for the salvage of exposed or potentially exposed but clinically normal product for the domestic market. Decisions regarding the release of abalone to the domestic market will depend on the control strategy implemented. Abalone in the late stages of disease (ie severe clinical signs) would not be suitable for human consumption because it is the saleable product (the foot muscle) that is withered by the syndrome; in affected abalone, meat quality and appearance are inferior.

Abalone without clinical signs that have been treated for withering syndrome (should treatments become available) may be harvested for human consumption, provided that appropriate withholding periods are observed to avoid antibiotic residues in the abalone tissue. Provided that normal seafood safety practices are maintained, there are no public health risks associated with harvesting potentially infected abalone for human consumption. If healthy, potentially infected or infected abalone are destined for human consumption, the chief medical officer and health authority of the relevant state or territory must be notified that there are

[16] http://www.daff.gov.au/aquavetplan
[17] http://www.daff.gov.au/aquavetplan

no human health concerns associated with *Ca* Xenohaliotis californiensis and that withering syndrome is not a zoonotic disease.

Eradication

For successful eradication, decisions relating to the release of product for the domestic market must prevent the spread of *Ca* Xenohaliotis californiensis.

Containment, control and zoning

The release of exposed or potentially exposed but clinically normal product to the domestic market must be carefully controlled to prevent the spread of viable *Ca* Xenohaliotis californiensis to areas or zones declared free from withering syndrome.

3 Preferred Australian response options

3.1 Overall policy for infection with *Ca* Xenohaliotis californiensis

_Ca_ Xenohaliotis californiensis has the potential to cause severe, long-term production and economic losses in the abalone farming industry and associated production, sales and export industries. It will therefore be necessary to act immediately to control or eradicate the disease if it is detected in Australia.

The control of an outbreak of _Ca_ Xenohaliotis californiensis in Australia will depend on the nature of the outbreak. By drawing on the previous sections of this manual, Section 3 provides guidance to directors of fisheries and chief veterinary officers (CVOs) to choose and implement the most appropriate control option for the circumstances.

There are two preferred response options for _Ca_ Xenohaliotis californiensis in Australia:

- **Option 1 — eradication of _Ca_ Xenohaliotis californiensis from Australia; and**
- **Option 2 — containment, control and zoning, with the aim of containing _Ca_ Xenohaliotis californiensis within known endemic areas, thus preventing further spread and protecting uninfected areas.**

Both of these options involve the use of a combination of strategies as outlined in Section 2, including:

- **quarantine and movement controls on fish, fish products and things in declared areas to prevent spread of infection;**
- **destruction and disposal of clinically diseased and dead abalone to prevent release of _Ca_ Xenohaliotis californiensis into the environment;**
- **decontamination of facilities;**
- **surveillance to determine the extent of possible infection, and to provide proof of freedom from _Ca_ Xenohaliotis californiensis;**
- **zoning to define infection-free zones;**
- **restocking with sentinel abalone; and**
- **a public awareness campaign to encourage cooperation from aquaculturalists and the community.**

The director of fisheries and/or the CVO in the state or territory in which the outbreak occurs will be responsible for developing an emergency animal disease response plan (EAD Response Plan). This plan will be submitted to the aquatic

Consultative Committee on Emergency Animal Diseases (AqCCEAD), which will provide advice on its technical soundness and consistency with AQUAVETPLAN.

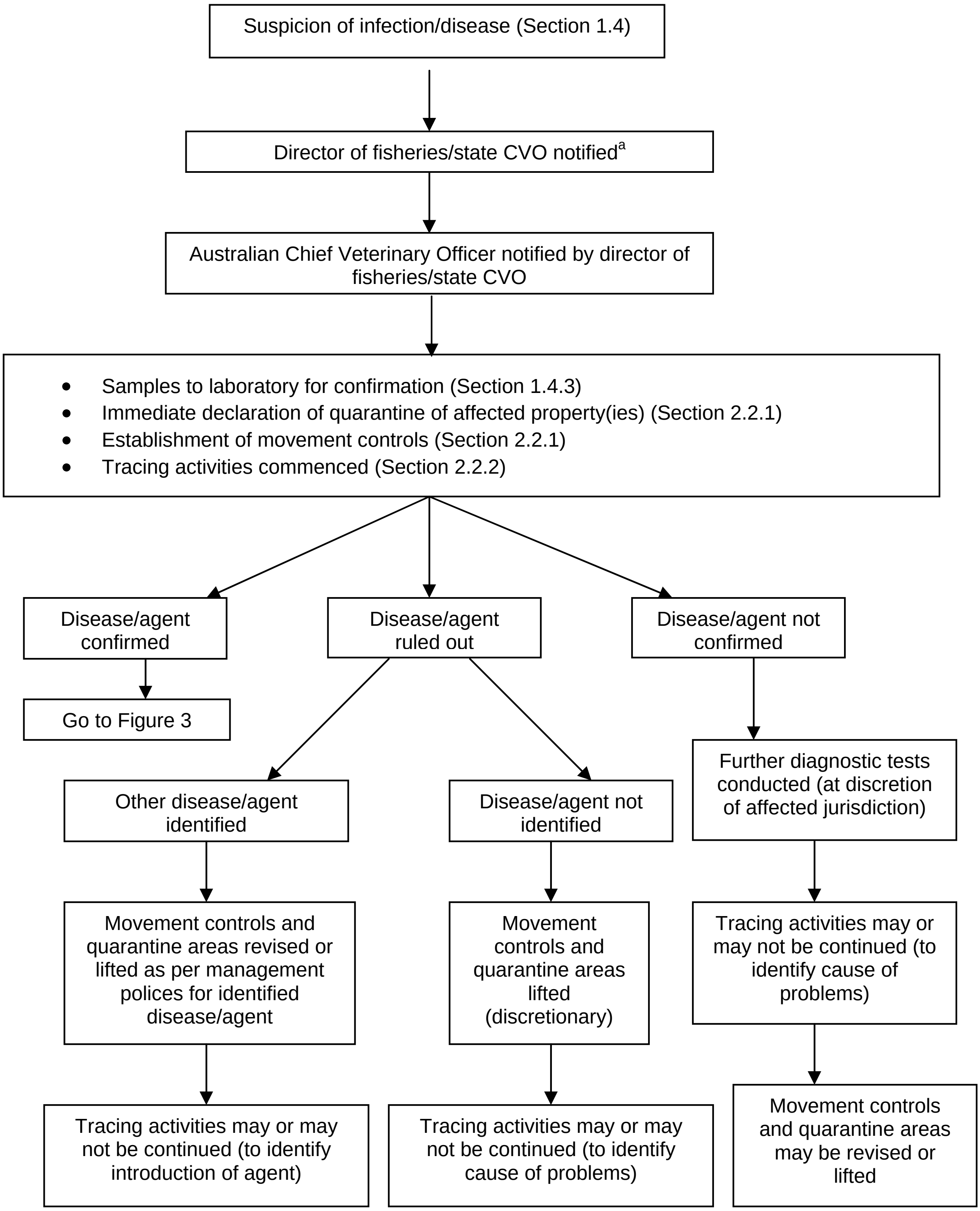
Directors of fisheries and/or CVOs will implement the disease control measures as agreed in the EAD Response Plan and in accordance with relevant legislation. They will make ongoing decisions on follow-up disease response measures in consultation with AqCCEAD. The detailed response measures adopted will be determined using the principles of control and eradication (see Section 2), epidemiological information about the outbreak, and the financial feasibility of the option.

For information on the responsibilities of the state or territory disease control headquarters and local disease control centres, see the AQUAVETPLAN **Control Centres Management** manual.[18]

3.2 Control options

The circumstances surrounding an outbreak of withering syndrome associated with *Ca* Xenohaliotis californiensis will greatly influence selection of the most suitable control option. Figure 2 details the actions that should occur on initial suspicion of infection with *Ca* Xenohaliotis californiensis. Figure 3 has been developed to help identify the most appropriate control option. These decision trees are flexible, depending on the specific situations experienced. However, in Figure 3, in any instance when a response is 'unknown' this should be treated in a cautionary manner and the 'no' option should be followed.

[18] http://www.daff.gov.au/aquavetplan



a As appropriate in the affected jurisdiction

Figure 2 Decision matrix/flow chart

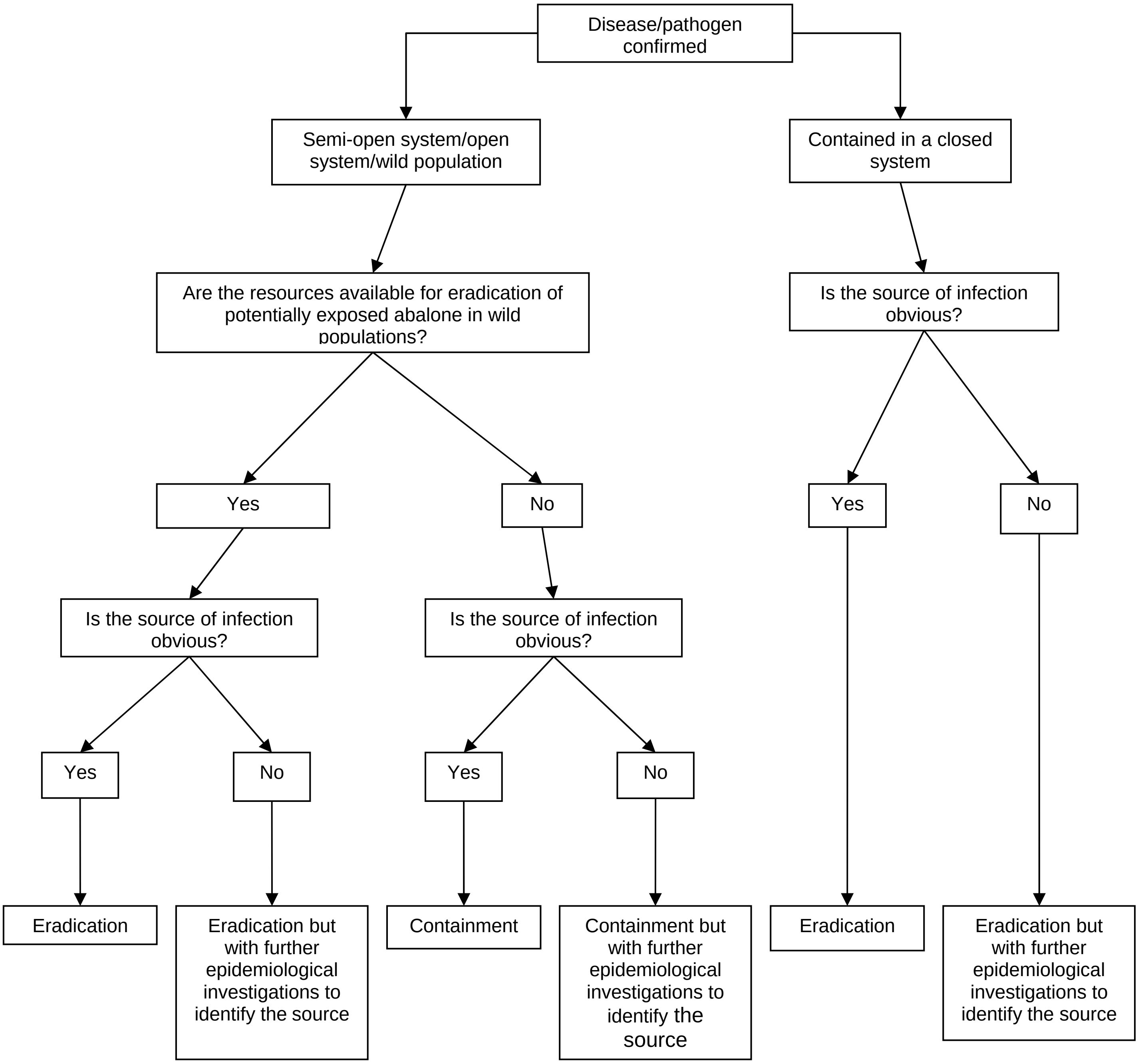


Figure 3 Determination of most appropriate response option

If the source of infection cannot be identified, consideration should be given to surveillance of neighbouring populations of abalone (if possible) to determine whether *Ca* Xenohaliotis californiensis was already present in the abalone population before detection of the disease. If it was present, the outbreak could have been brought on by external factors (eg increased water temperatures).

3.2.1 Option 1 — Eradication

Eradication has the highest short-term economic costs; however, if *Ca* Xenohaliotis californiensis were successfully eradicated, long-term economic benefits could outweigh the short-term costs.

Eradication is likely to be successful in a closed system. In open or semi-open systems, the success of an eradication strategy would depend on the availability of resources for surveying and destocking wild abalone in the immediate area.

An eradication plan must include the following activities:

- Quarantine and movement controls must be declared immediately and stringently enforced on abalone, abalone products, water, and any vectors located in declared areas. Restrictions must apply to movement out of the infected area of anything capable of transmitting *Ca* Xenohaliotis californiensis from infected to uninfected abalone, and to aquaculture facilities or processing plants. Movement controls should be maintained until the agent is either eradicated or declared endemic.
- All diseased and dead abalone must immediately be removed, destroyed and disposed of.
- Any exposed or potentially exposed abalone must immediately be removed, destroyed and disposed of.
- Any product from exposed or potentially exposed but clinically normal abalone must immediately be destroyed and disposed of.
- All buildings, tanks, materials and equipment that may be contaminated — including nets, boats and vehicles — must be decontaminated.
- All infected abalone, wastes, effluent and equipment that cannot be decontaminated effectively must immediately be disposed of safely.
- Effluent must be treated.
- Restocking with sentinel abalone can occur only after the site has been thoroughly decontaminated.

3.2.2 Option 2 — Containment, control and zoning

Eradication is unlikely to be successful if infection is widespread, no point source of infection is immediately identified, or infection is potentially present in wild abalone. Under these conditions, containment, control and zoning are feasible options.

A containment, control and zoning plan must include the following activities:

- Quarantine and movement controls must be declared immediately and stringently enforced on abalone, abalone products, water, and any vectors located in declared areas. Restrictions must apply to movement out of the infected area of anything capable of transmitting *Ca* Xenohaliotis californiensis from infected to uninfected abalone, and to aquaculture facilities or processing plants. Movement controls should be maintained until the agent is either eradicated or declared endemic.
- All diseased and dead abalone must immediately be removed, destroyed and disposed of.

- Any exposed or potentially exposed abalone must immediately be removed, destroyed and disposed of OR treated.
- Any product from exposed abalone must immediately be destroyed and disposed of.
- All buildings, tanks, materials and equipment that may be contaminated — including nets, boats and vehicles, must be decontaminated.
- All infected abalone, wastes, effluent and equipment that cannot be decontaminated effectively must immediately be disposed of safely.
- Effluent must be treated.
- If zoning is implemented, a targeted surveillance program for *Ca* Xenohaliotis californiensis would be necessary within the uninfected zone; state- or territory-based legislation would also be required to support the declaration of zones. Surveillance programs should take into consideration the need to detect covert infections in clinically normal abalone.
- Thorough cleaning and disinfection of water and equipment — including nets, boats and vehicles that may move from an infected to an uninfected zone — are important. (See Section 2.2.6 and the AQUAVETPLAN **Operational Procedures manuals — Decontamination** and **Disinfection**[19] for further details).
- Restocking with sentinel, marked abalone is one method of ascertaining freedom from infection in open systems where sparse populations of infected abalone could still remain. Large-scale restocking with susceptible species should only occur once the water body is considered, or likely, to be uninfected.

3.3 Criteria for proof of freedom

Proof of freedom from *Ca* Xenohalitos californiensis, which may be important for trade, can be demonstrated at the aquaculture establishment, zone and country level. Criteria for proof of freedom at each level are given in the OIE *Aquatic Animal Health Code* (OIE 2006a).

3.4 Funding and compensation

There is currently no cost-sharing arrangement in place for aquatic animal diseases.

[19] http://www.daff.gov.au/aquavetplan

Appendix 1 OIE *Aquatic Animal Health Code* and *Manual of Diagnostic Tests for Aquatic Animals*

OIE Aquatic Code

The objective of the OIE *Aquatic Animal Health Code* (OIE 2006a) is to prevent the spread of aquatic animal diseases, while facilitating international trade in aquatic animals and aquatic animal products. This annually updated volume is a reference document for use by veterinary departments, import and export services, epidemiologists and all those involved in international trade.

The current edition of the OIE Aquatic Code (8^{th} edition) was published in 2006 and is available on the OIE website at:

http://www.oie.int/eng/normes/fcode/fcode2004/en_acode.htm
(Accessed on 7 September 2006)

The following chapter is relevant to this manual:

Chapter 2.2.8 Infection with *Xenohaliotis californiensis*

OIE Aquatic Manual

The purpose of the OIE *Manual of Diagnostic Tests for Aquatic Animals* (OIE 2006b) is to contribute to the international harmonisation of methods for the surveillance and control of the most important aquatic animal diseases. Standards are described for laboratory diagnostic tests and the production and control of biological products (principally vaccines) for veterinary use across the globe.

The current edition of the OIE Aquatic Manual was published in 2006 and is available on the OIE website at:

http://www.oie.int/eng/normes/fmanual/A_summry.htm
(Accessed on 7 September 2006)

The following chapter is relevant to this manual:

Chapter 2.2.8 Infection with *Xenohaliotis californiensis*

Further information

Further information about the OIE Aquatic Code and Aquatic Manual is available on the OIE website at:

http://www.oie.int/eng/normes/en_acode.htm
(Accessed on 7 September 2006)

Appendix 2 Approval of chemicals for use in Australia

The Australian Pesticides and Veterinary Medicines Authority (APVMA) evaluates, registers and regulates agricultural and veterinary chemicals. Before an antibiotic or vaccine can enter the Australian market, it must go through the APVMA's rigorous assessment process to ensure that it meets high standards of safety and effectiveness. (In addition, an import permit is required from the Australian Quarantine and Inspection Service if a product containing biological material is to be sourced from overseas.)

Detailed data about the product and its proposed use pattern must be submitted to the APVMA with the application for registration or permits. Since the assessment process is so detailed, the evaluation may take some time to complete.

Minor use permit system

The minor use permit (MUP) system is a temporary approval system for the use of drugs and chemicals. The system was devised by the APVMA for Australia, and allows the restricted use of a limited amount of a drug or chemical in a specified species when inadequate data are available to satisfy APVMA requirements for registration. Conditions are applied to the permit, which often include the collection of data related to the use of the product. The MUP system aims to enable restricted use of a drug or chemical until sufficient data are available to enable full registration.

For example, the APVMA may set a temporary withholding period with a wide margin of safety for a MUP. This withholding period may have been extrapolated from data relating to the use of the product in other species. In such cases, a condition of the MUP will be the collection of residue testing data. Results from the data are assessed by the APVMA (usually after 12 months — the duration of most permits) and used to more accurately set a withholding period for the product.

Emergency use permits

The APVMA has a permit system for the emergency use of a product that is either unregistered in Australia or registered for use in a different species or for a different use pattern. The APVMA will verify with the appropriate state and territory coordinators that the emergency is genuine.

For further details or permit application forms, visit the APVMA website.[20]

[20] http://www.apvma.gov.au

Glossary

Aquatic Animal Health Committee	A committee comprising representatives of the Australian Government; state and territory governments; the major aquaculture, wild capture, aquarium and recreational fishing industries; and CSIRO. The committee provides advice to Primary Industries Ministerial Council on aquatic animal health matters, focusing on technical issues and regulatory policy. *See also* Primary Industries Ministerial Council
Australian Chief Veterinary Officer	The nominated senior veterinarian in the Australian Government Department of Agriculture, Fisheries and Forestry who manages international animal health commitments and the Australian Government's response to an animal disease outbreak. *See also* Chief veterinary officer
AQUAVETPLAN	*A*ustralian A*qua*tic *Vet*erinary Emergency *Plan*. A series of technical response plans that describe the proposed Australian approach to an emergency aquatic animal disease incident. *See also* AUSVETPLAN
AUSVETPLAN	*Aus*tralian *Vet*erinary Emergency *Plan*. A series of technical response plans that describe the proposed Australian approach to an emergency animal disease incident. The documents provide guidance based on sound analysis, linking policy, strategies, implementation, coordination and emergency-management plans.
Chief veterinary officer (CVO)	The senior veterinarian of the animal health authority in each jurisdiction (national, state or territory) who has responsibility for animal disease control in that jurisdiction. *See also* Australian Chief Veterinary Officer
Control area	A buffer between the restricted area and areas free from disease. Restrictions on this area will reduce the likelihood of the disease spreading further afield. As the extent of the outbreak is confirmed, the control area may reduce in size. The shape of the area may be modified according to circumstances, eg water flows, catchment limits etc. In most cases, permits will be required to move animals and specified product out of the control area into the free area.
Covert infection	Clinically inapparent infection that is transmissible and that may eventually lead to clinical disease.

Dangerous contact premises or area	That which has had a direct, and possibly infectious, contact with an infected premises or area. The type of contact will depend on the agent involved in the outbreak but, for example, may involve animal movements or net/equipment movements.
Declared area	A defined tract of land or water that is subjected to disease control restrictions under emergency animal disease legislation. Types of declared areas include *restricted area, control area, infected premises, dangerous contact premises* and *suspect premises*.
Decontamination	Includes all stages of cleaning and disinfection.
Disease agent	A general term for a transmissible organism or other factor that causes an infectious disease.
Disinfectant	A chemical used to destroy disease agents outside a living animal.
Disinfection	The application, after thorough cleansing, of procedures intended to destroy the infectious or parasitic agents of animal diseases, including zoonoses; applies to premises, vehicles and other objects that may have been directly or indirectly contaminated.
Disposal	Sanitary removal of fish carcases and things by burial, burning or some other process so as to prevent the spread of disease.
Emergency animal disease	A disease that is (a) exotic to Australia or (b) a variant of an endemic disease or (c) a serious infectious disease of unknown or uncertain cause or (d) a severe outbreak of a known endemic disease, and that is considered to be of national significance with serious social or trade implications. *See also* Endemic animal disease, Exotic animal disease
Endemic animal disease	A disease affecting animals (which may include humans) that is known to occur in Australia. *See also* Emergency animal disease, Exotic animal disease
Enterprise	*See* Risk enterprise
Epidemiological investigation	An investigation to identify and qualify the risk factors associated with the disease.
Exotic animal disease	A disease affecting animals (which may include humans) that does not normally occur in Australia. *See also* Emergency animal disease, Endemic animal disease
Free area	An area known to be free from the disease agent.

Infected premises or area	The area in which the disease has been confirmed. Definition of an 'infected area' is more likely to apply to an open system, such as an oceanic lease.
Local disease control centre	An emergency operations centre responsible for the command and control of field operations in a defined area.
Monitoring	Routine collection of data for assessing the health status of a population. *See also* Surveillance
Movement control	Restrictions placed on the movement of fish, people and other things to prevent the spread of disease.
OIE Aquatic Code	OIE *Aquatic Animal Health Code* (OIE 2006a). Published on the internet at: http://www.oie.int/eng/normes/fcode/en_sommaire.htm (Accessed 7 September 2006) *See* Appendix 1 for further details
OIE Aquatic Manual	OIE *Manual of Diagnostic Tests for Aquatic Animals* (OIE 2006b). Describes standards for laboratory diagnostic tests and the production and control of biological products (principally vaccines). The current edition is published on the internet at: http://www.oie.int/eng/normes/fmanual/A_summry.htm (Accessed 7 September 2006). *See* Appendix 1 for further details
Operational procedures	Detailed instructions for carrying out specific disease control activities, such as disposal, destruction, decontamination and valuation.
Polymerase chain reaction (PCR)	A method of amplifying and analysing DNA sequences that can be used to detect the presence of DNA from a disease agent.
Premises or area	A production site, which may range from an aquarium to an aquaculture lease in the open ocean.
Primary Industries Ministerial Council	The council of Australian national, state and territory and New Zealand ministers of agriculture that sets Australian and New Zealand agricultural policy (formerly the Agriculture and Resource Management Council of Australia and New Zealand).
Quarantine	Legal restrictions imposed on a place, fish, vehicles, or other things, limiting movement.

Restricted area	The area around an infected premises (or area), likely to be subject to intense surveillance and movement controls. It is likely to be relatively small. It may include some dangerous contact premises (or area) and some suspect premises (or area), as well as enterprises that are not infected or under suspicion. Movement of potential vectors of disease out of the area will, in general, be prohibited. Movement into the restricted area would only be by permit. Multiple restricted areas may exist within one control area.
Risk enterprise	A defined livestock or related enterprise, which is potentially a major source of infection for many other premises. Includes hatcheries, aquaculture farms, processing plants, packing sheds, fish markets, tourist angling premises, veterinary laboratories, road and rail freight depots and garbage depots.
State or territory disease control headquarters	The emergency operations centre that directs the disease control operations to be undertaken in that state or territory.
Surveillance	A systematic series of investigations of a given population of fish to detect the occurrence of disease for control purposes, and which may involve testing samples of a population.
Susceptible animal	Animal that can be infected with a particular disease.
Tracing	The process of locating animals, persons or other items that may be implicated in the spread of disease, so that appropriate action can be taken.
Vector	A living organism that transmits an infectious agent from one host to another. A *biological* vector is one in which the infectious agent must develop or multiply before becoming infective to a recipient host. A *mechanical* vector is one that transmits an infectious agent from one host to another but is not essential to the lifecycle of the agent.
Zoning	The process of defining disease-free and infected areas.

Abbreviations

APVMA	Australian Pesticides and Veterinary Medicines Authority
AQUAVETPLAN	Australian Aquatic Veterinary Emergency Plan
AqCCEAD	Aquatic Consultative Committee on Emergency Animal Diseases
AUSVETPLAN	Australian Veterinary Emergency Plan
Ca Xenohaliotis californiensis	*Candidatus* Xenohaliotis californiensis
CVO	chief veterinary officer
DAFF	Department of Agriculture, Fisheries and Forestry (Australian Government)
DNA	deoxyribonucleic acid
DPIWE	Department of Primary Industries, Water and Environment, Tasmania
EAD	emergency animal disease
ISH	in situ hybridisation
OIE	World Organisation for Animal Health (formerly Office International des Epizooties)
PCR	polymerase chain reaction
RLP	rickettsia-like prokaryote

References

Altstatt JM, Ambrose RF, Engle JE, Haaker PL, Lafferty KD and Raimondi PT (1996). Recent declines of black abalone *Haliotis cracherodii* on the mainland coast of central California. *Marine Ecology Progress Serie*s 142:185–192.

Andree KB, Friedman CS, Moore JD and Hedrick RP (2000). A polymerase chain reaction assay for the detection of genomic DNA of a Rickettsiales-like prokaryote associated with withering syndrome in California abalone. *Journal of Shellfish Research* 19:213–218.

Anon (1995). A draft plan for the Foveaux Strait oyster fishery. Unpublished report. (Unpublished report held in the Ministry of Fisheries library, Dunedin, New Zealand).

Antonio DB, Andree KB, Moore JD, Friedman CS and Hedrick RP (2000). Detection of *Rickettsiales*-like prokaryotes by *in situ* hybridization in black abalone, *Haliotis cracherodii*, with withering syndrome. *Journal of Invertebrate Pathology* 75:180–182.

Benkendorrf K (2003). Immune indicators for monitoring abalone health. In: *Proceedings of the 10th Annual Abalone Aquaculture Workshop*, Fleming AE (ed), Port Lincoln, Australia, 19–21 November 2003, Fisheries Research and Development Corporation, Canberra.

Berthe FCJ (2002). Pacem in terries pathogenibus bonae voluntatis: molluscs–pathogen relationships prospects. *Bulletin of the European Association of Fish Pathologists* 22(2):52.

Berthe FCJ (2003). *Disease card – Withering Syndrome of Abalone*. http://library.enaca.org/Health/DiseaseLibrary/Disease_card_for_WSA.pdf (Accessed 12 September 2006)

Bower SM (2004). *Synopsis of Infectious Diseases and Parasites of Commercially Exploited Shellfish: Withering Syndrome of Abalone*, Fisheries and Oceans Canada. http://www-sci.pac.dfo-mpo.gc.ca/shelldis/pages/fwsab_e.htm (Accessed 29 August 2006)

Braid BA (2004). Biochemical and histological changes in red abalone subjected to eight different treatment combinations of exposure to the agent of withering syndrome, water temperature and food availability. MS thesis, University of California, Davis, California.

Davis GE (1993). Mysterious demise of southern California Black Abalone, *Haliotis cracherodii* Leach, 1814. *Journal of Shellfish Research* 12(2):183–184.

Diggles BK, Nichol J, Hine PM, Wakefield S, Cochennec-Laureau N, Roberts RD and Friedman CS (2002). Pathology of cultured paua *Haliotis iris* infected with a novel haplosporidian parasite, with some observations on the course of disease. *Diseases of Aquatic Organisms* 50:219–231.

Friedman CS (1996). Update on abalone withering syndrome, *Alolkoy* Vol 9, Fall 1996.

Friedman CS and Finley CA (2003). Anthropogenic introduction of the etiological agent of withering syndrome into northern California abalone populations via conservation efforts. *Canadian Journal of Fisheries and Aquatic Sciences* 60(11):1424–1431.

Friedman CS, Thomson M, Chun C, Haaker P and Hedrick RP (1997). Withering syndrome of the black abalone, *Haliotis cracherodii* (Leach): water temperature, food availability, and parasites as possible causes. *Journal of Shellfish Research* 16:403–411.

Friedman CS, Andree KB, Robbins TT, Shields JD, Moore JD, Beauchamp K and Hedrick RP (2000a). 'Candidatus *Xenohaliotis californiensis*,' a newly described bacterial pathogen and etiological agent of withering syndrome found in abalone, *Haliotis* spp., along the west coast of North America. *International Journal of Systematic and Evolutionary Microbiology* 50:847–855.

Friedman CS, Robbins T, Jacobsen JL and Shields JD (2000b). The cellular immune response of black abalone, *Haliotis cracherodii* Leach, with and without Withering Syndrome (abstract). *Journal of Shellfish Research* 19:514.

Friedman CS, Biggs W, Shields JD and Hedrick RP (2002). Transmission of Withering Syndrome in black abalone, *Haliotis cracherodii* Leach. *Journal of Shellfish Research* 21:817–824.

Friedman CS, Trevelyan G, Robbins TT, Mulder EP and Fields R (2003). Development of an oral administration of oxytetracycline to control losses due to withering syndrome in cultured red abalone *Haliotis rufescens*. *Aquaculture* 224:1–23.

Gardner GR, Harshbarger JC, Lake JL, Sawyer TK, Price KL, Stephenson MD, Haaker PL and Togstad HA (1995). Association of prokaryotes with symptomatic appearance of withering syndrome in black abalone *Haliotis cracherodii*. *Journal of Invertebrate Pathology* 66:111–120.

Godoy MG and Muñoz GP (2003). Major diseases encountered in Japanese abalone (*Haliotis discus hannai*) and red abalone (*Haliotis rufescens*) reared in Chile. Abstract, 5th International Abalone Symposium, Qingdao, China, 12–17 October 2003.

Haaker PL, Parker DO, Togstad H, Richards D, Davis GE and Friedman CS (1992). Mass mortality and withering foot syndrome in black abalone, *Haliotis cracherodii*, in California, In: *Abalone of the World: Biology, Fisheries and Culture*, Shepard SA, Tegner MJ and Guzman del Proo SA (eds), Proceedings of the 1st International Symposium on Abalone, 21–25 November 1989, La Paz, Baja California Sur, Mexico, Fishing News Books, Cambridge, 214–224.

Handlinger J, Bastianello S, Carson J, Callinan R, Creeper J, Deveney M, Freeman K, Forsyth M, Hooper C, Jones B, Lancaster M, Landos M, Loh R, Oyay BS, Phillips P, Pyecroft S, and Stephens F (2005). Abalone Aquaculture Subprogram: A national survey of diseases of commercially exploited abalone species to support trade and translocation issues and the development of health surveillance programs, Draft Final Report, June 2005, FRDC Project No. 2002/201.

Herfort A (2004). *Aquatic Animal Diseases Significant to Australia: Identification Field Guide*, Australian Government Department of Agriculture, Fisheries and Forestry, Canberra.

Lafferty KD and Kuris AM (1993). Mass mortality of abalone *Haliotis cracherodii* on the California Channel Islands: tests of epidemiological hypotheses. *Marine Ecology Progress Series* 96:239–248.

Moore JD, Robbins T and Friedman CS (2000a). Withering syndrome in farmed red abalone, *Haliotis rufescens*: thermal induction and association with a gastrointestinal Rickettsiales-like procaryote. *Journal of Aquatic Animal Health* 12:26–34.

Moore JD, Robbins T and Friedman CS (2000b). The role of a *Rickettsia*-like prokaryote in withering syndrome in California red abalone, *Haliotis rufescens* (abstract). *Journal of Shellfish Research* 19:525–526.

Moore JD, Robbins TT, Hedrick RP and Friedman CS (2001). Transmission of the Rickettsiales-like procaryote, *Candidatus* Xenohaliotis californiensis, and its role in withering syndrome of California abalone *Haliotis* spp. *Journal of Shellfish Research* 20(2):867–874.

Mouton A (2000). Health management and disease surveillance in abalone, *Haliotis midae*, in South Africa (abstract). *Journal of Shellfish Research* 19:526.

OIE (World Organisation for Animal Health) (2004). *Candidatus Xenohaliotis Californiensis* in Iceland. *Disease Information* 17(25), 18 June 2004.

OIE (World Organisation for Animal Health) (2006a). *Aquatic Animal Health Code*, 8th edition, OIE, Paris.
http://www.oie.int/eng/normes/fcode/fcode2006_back/en_index.htm
(Accessed 29 August 2006)

OIE (World Organisation for Animal Health) (2006b). *Manual of Diagnostic Tests for Aquatic Animals*, OIE, Paris.
http://www.oie.int/eng/normes/fmanual/A_summry.htm (Accessed 29 August 2006)

VanBlaricom GR, Ruediger JL, Friedman CS, Woodard DD and Hedrick RP (1993). Discovery of withering syndrome among black abalone *Haliotis cracherodii* Leach, 1814, populations at San Nicolas Island, California. *Journal of Shellfish Research* 12:185–188.

van Banning P (1988). Management strategies to control diseases in the Dutch culture of edible oysters. *American Fisheries Society Special Publication* 18:43–245.

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