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IMPORT RISK ASSESSMENT: JUVENILE YELLOWTAIL KINGFISH (*SERIOLA LALANDI)* FROM SPENCER GULF AQUACULTURE, SOUTH AUSTRALIA

The attached import risk analysis was conducted and documented by a private consultant working on behalf of a would-be importer. It is not an official MAF Biosecurity Authority risk analysis.

Nevertheless, this risk analysis has been subjected to MAF's internal scientific review process and to external expert review. The risk analyst has addressed all the points raised by MAF and the external reviewers.

MAF considers this risk analysis to be technically sound and sufficiently robust for an Import Health Standard to be from.

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IMPORT RISK ASSESSMENT: Juvenile yellowtail kingfish (*Seriola lalandi*) from Spencer Gulf Aquaculture, South Australia

NIWA Client Report: WLG 2002/03
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1. Introduction

This Import Risk Assessment has been produced by request of Island Aquafarms Ltd. Nelson, for the consideration of MAF Biosecurity Authority, to allow importation of cultured juvenile (1 - 3 grams wet weight) kingfish (*Seriola lalandi*) from Spencer Gulf Aquaculture hatchery, Port Augusta, South Australia, for ongrowing at a proposed aquaculture facility in Nelson, New Zealand. It is proposed that the kingfish would be subjected to health assessments, certification procedures and import/export procedures which follow guidelines set out in the Office International des Epizooties (OIE) International Aquatic Animal Health Code (OIE 2001). It is proposed that after health certification in Australia (following procedures specified by the OIE (2000) and the recommendations herein), the kingfish would be placed in a sealed container (MAF/AQIS approved seal) and air freighted into Wellington from Sydney or Melbourne airports. After clearing customs in Wellington the kingfish would be freighted directly by road to an approved transitional facility at NIWA Mahanga Bay for 4 weeks quarantine. Upon reaching the quarantine facility the seal to the transport container would be broken only by authorised MAF Quarantine officers. Once cleared from quarantine the fish would then be shipped to the culture facility at Nelson for ongrowing in landbased tanks or seacages for human consumption. This IRA briefly reviews the information currently available on the stock structure of kingfish in Australia and New Zealand, but mainly examines the fish health risks associated with the importation of kingfish and proposes various safeguards and procedures to mitigate those risks.

1.1 Stock structure of kingfish populations in Australia and New Zealand

Seriola lalandi is a widely distributed species with a circum-global distribution in sub-tropical and temperate waters (Paxton *et al.* 1989). Three sub species of *S. lalandi* are generally recognised. Two occur in the northern hemisphere (*S. lalandi dorsalis* in California, and *S. lalandi aureovittata* in Asia) while the remaining sub species (*S. lalandi lalandi*) occurs in the southern hemisphere, including Australia and New Zealand (Smith -Vaniz *et al.* 1990, Paul 2000). *S. lalandi lalandi* (hereafter called *S. lalandi* or kingfish) occurs throughout southern Australian sub-tropical and temperate waters from Trigg Island in Western Australia through South Australia, Victoria, eastern Tasmania, New South Wales (NSW) and Queensland, including the southern areas of the Great Barrier Reef (Paxton *et al.* 1989, Kailola *et al.* 1993). In New Zealand *S. lalandi* occurs in the waters surrounding the North Island (including the Three Kings and Kermadec Islands) and the northern areas of the South Island (Paul 2000).

Little is known about the stock structure of kingfish in Australia (Kailola *et al.* 1993) and New Zealand (McGregor 1995). Evidence from some genetic loci (Smith *et al.* 1991) and parasites (Lester 1982) suggests that there may be some temporally discrete sub-populations of kingfish on the east coast of Australia, however no fixed allelic differences have been detected between samples of kingfish from NSW, New Zealand, and the United States (Smith 1987, Smith *et al.* 1991). A recent study examined microsatellite and mitochondrial DNA from kingfish from Australia, New Zealand and Japan (Nugroho *et al.* 2001). They found significant genetic divergence between kingfish from Japan and Australia/New Zealand, but no significant differentiation among the Australian and New Zealand population samples.

Tagging studies done in Australia indicate that most juvenile kingfish are relatively sedentary, but a small number of kingfish, (both juvenile and adult) move large distances, indicating that mixing of kingfish can and does occur throughout the range of the species off eastern Australia (Smith *et al.* 1991, Gillanders *et al.* 1997, 2001). A similar pattern of movements has also been recorded throughout the range of kingfish in New Zealand (Saul and Holdsworth 1992, Holdsworth and Saul 1998). To date kingfish tagged off NSW have been recaptured in Victorian waters, but not from South Australian waters (Gillanders *et al.* 1997, 2001). However, kingfish are regularly

captured along the coastline between the border of Victoria and Spencer Gulf (Smith 1987), suggesting that wild kingfish can range freely between these areas.

Of relevance to this risk assessment is tagging evidence which shows that wild kingfish do migrate naturally between Australia and New Zealand. A total of 5 trans-Tasman movements of kingfish have been recorded out of 2109 recaptures (0.23% of recaptures, see Holdsworth and Saul 1998, Gillanders *et al.* 2001). Three of these were for fish which moved from NSW to the North Island of New Zealand (Gillanders *et al.* 2001), while 2 fish moved from the North Island of New Zealand to NSW (McGregor 1995). All of these were relatively large fish, consistent with data that kingfish over 75 cm in length are far more likely to move long distances than are smaller fish (Gillanders *et al.* 2001). Because the majority of kingfish tagged in cooperative tagging programmes to date have been small, immature fish (40 - 70 cm, see Gillanders *et al.* 2001), and few sexually mature fish in the size range most likely to undertake large scale movements (>100 cm) were tagged, it is possible that trans-Tasman migration of adult kingfish occurs more frequently than the limited number of tag returns suggest. Since kingfish reach a maximum size of about 250 cm, Gillanders *et al.* (2001) suggested that the subset of small, immature fish tagged and recaptured to date may not be representative of the movements of the entire population. They suggested (Gillanders *et al.* 2001, page 189), and that future tagging studies need to target sexually mature, adult fish to examine whether large scale movements, such as trans-Tasman migrations, occur more frequently.

Juvenile kingfish less than 30 cm FL are rarely seen, as they are found far from land in the epipelagic zone associated with flotsam or floating weed which they use to provide camouflage (http://www.fishbase.org/Ecology/FishEcology/summary.cfm?stockcode=396&GenusName=Seriola &SpeciesName= lalandi). Juveniles eventually recruit to inshore waters at around 30 cm long, but the timing of this movement is unknown. Growth rates of juvenile kingfish in the wild have been difficult to estimate (Gillanders *et al.* 1999, Gillanders *et al.* 2001), but in culture, provided with ample food, kingfish grow rapidly to 30 cm in around 1 year or less (B. Diggles, personal observation). The length of time juvenile kingfish spend in the epipelagic stage during the wild is, therefore, likely to be up to around 1 year. During this time it would be possible that flotsam associated individuals could be carried long distances by ocean currents. Oceanographic data indicates that a significant proportion of the East Australian Current (EAC) which flows down the east coast of Australia separates eastwards into the Tasman Front once it reaches around 32-35°S, after which it meanders eastwards between 32 and 36°S on to northern New Zealand (Tilburg *et al.* 2001). Though trans-Tasman movements of juvenile kingfish are as yet undocumented, modeling suggests that passive transport by the Tasman Front can result in movements of 7% of phyllosoma larvae released by spiny lobsters (*Jasus verreauxi*) off New South Wales to northern New Zealand within 12 months (Chiswell *et al.* submitted). It is theoretically possible, therefore, that a small percentage of larval and juvenile kingfish spawned off New South Wales could be transported to New Zealand by the currents of the Tasman Front.

The natural trans-Tasman migrations of adult kingfish (and possibly also of juveniles transported by currents to New Zealand) probably account for the gene flow required between the two countries to maintain genetic homogeneity. These data indicate that translocation of kingfish from South Australia to New Zealand poses negligible risk to the genetic integrity of New Zealand kingfish populations if imported kingfish escaped from the proposed aquaculture facility.

1.2 Relevance of natural trans-Tasman movements of kingfish

The natural trans-Tasman movement of adult fish, and possibly juveniles during the epipelagic stage of their life cycle, must be viewed in the context of this risk assessment, which is concerned with the threat of introduction of disease into New Zealand from importation of many thousands

of juvenile kingfish 1 - 3 grams in weight. Trans-Tasman migration of mature adult kingfish or recruitment of 25 to 30 cm juveniles from epipelagic areas may be irrelevant when discussing diseases and parasites which are specific only to 1 - 3 gram juvenile kingfish, and which do not infect adult kingfish.

A hypothetical example of this could be a pathogenic bacterium or virus which only infects juvenile 1 - 3 cm cultured kingfish when they are reared at high densities in inshore waters of Australia. If such a disease agent existed, and all juvenile kingfish infected with it either died, or were cured spontaneously so that no surviving kingfish could act as asymptomatic carriers as adults, it is very likely that such a disease could pose a threat to kingfish, and possibly other fish species, in New Zealand.

On the other hand, trans-Tasman migration of juvenile and adult kingfish is relevant to this risk assessment when considering diseases which may be acquired when fish are juveniles, but which also persist in adult fish. An example of such a disease agent could be a virus which infects mainly juvenile fish, but which can persist in surviving asymptomatic adult fish (carrier fish). All vertically transmitted diseases, including viruses, would fall into this category. Also relevant are the various long lived protozoan and metazoan parasites which infect juvenile fish, without causing mortality, and then continue to persist in adult fish.

Furthermore, the theoretical possibility that juvenile kingfish spawned naturally off New South Wales could be transported by currents to New Zealand does not necessarily mean that those fish may present a similar risk of disease introduction as posed by cultured juveniles. This is because juveniles reared in a hatchery situation in Australia are exposed to coastal waters which may carry disease agents (including viruses, bacteria and protozoa) which would not normally be encountered by naturally spawned kingfish in the epipelagic oceanic environment.

1.3 Commodity Description

Species	Yellowtail kingfish (*Seriola lalandi*)
Commodity	Live hatchery reared juveniles, 1 to 3 grams liveweight
Origin	Spencer Gulf Aquaculture Ltd., Port Augusta, South Australia
Volume	1 to 3 batches of up to 15,000 fish per year
Use	Ongrowing in culture for human consumption
Processing	Kingfish would be reared from eggs obtained from broodstock caught and domesticated locally in South Australia. Larvae and juveniles would be reared in a hatchery in seawater that is filtered to 1 µm and then UV sterilised. Batches of juveniles destined for export would be separated from other fish after weaning and held in isolation (in separate tanks in a location physically and spatially separated from other batches) at that facility and reared until they reached 1 - 3 grams. Batches which experienced a cumulative mortality rate of above 5% from hatching would not be accepted for export. Subsamples of fish from each acceptable batch would be tested by the Australian Animal Health Laboratory (AAHL) and/or other agreed competent authorities. Documentation of the daily mortality rate of each batch from egg hatching would be submitted at the same time as the subsampled fish so the disease history of each batch could be better assessed. Batches declared clinically healthy and free of the diseases listed in the Import Health Standard for Kingfish would be approved for export and issued an International Aquatic Animal Health Certificate.
Processing Premises	Batches of kingfish destined for export would be reared in the hatchery facilities of Spencer Gulf Aquaculture Ltd, Port Augusta, South Australia. Testing of kingfish subsampled for disease from each batch would be carried out at the AAHL premises in Geelong, Victoria, or at the premises of an approved competent authority in South Australia as designated by Primary Industries and Resources, South Australia (PIRSA). After health certification in Australia, the remaining kingfish in each batch would be placed in a sealed container (MAF/AQIS approved seal) and air freighted into Wellington via Adelaide, then Sydney or Melbourne airports. There would be no water exchange during transport. After clearing customs at Wellington airport the kingfish would be transported 5 km by road directly to an approved transitional facility at NIWA Mahanga Bay for 4 weeks quarantine. Upon reaching the quarantine facility the seal to the transport container would be broken only by authorised MAF Quarantine officers. All wastewater discharged from the quarantine facility would enter directly into the municipal sewerage system. New Zealand authorities would be immediately informed if a disease outbreak was detected within 4 weeks of the fish being imported into New Zealand. Once cleared from quarantine the fish would then be shipped to the culture facility at Nelson for ongrowing in landbased tanks or seacages for human consumption.
Controlling Authorities	Australian Quarantine and Inspection Service (AQIS), CSIRO Australian Animal Health Laboratory, Primary Industries and Resources, South Australia (PIRSA), MAF Biosecurity.

1.4 The commodity as a vehicle for introducing fish diseases into New Zealand

For this commodity to serve as a vehicle for the introduction of fish diseases into New Zealand, the following criteria must be met:

a. The disease organism must be present in the waters of origin (Spencer Gulf) or hatchery of origin (Spencer Gulf Aquaculture Ltd).
b. The disease organism must be present in the batch of fish destined for export.
c. The disease organism must not be detected in fish sub sampled from each batch for disease screening by competent certifying authorities.
d. The disease organism must survive transport and quarantine upon arrival to New Zealand.

For this commodity to serve as a vehicle for the introduction of diseases into stocks of wild or cultured fish in New Zealand, the following additional criteria must be met:

e. The disease organism must escape from the culture facility in New Zealand into local waters.
f. The disease organism must be present at an infectious dose.
g. Infectious stages of the disease must come in contact with a susceptible host.
h. The disease organism must be able to establish infection by the oral route or by the host being bathed in infective stages.

2 The risk assessment process

This document will now focus on analysing the risk of introduction of exotic diseases into New Zealand during the proposed introductions. This risk assessment includes the following steps as described in the OIE International Animal Health Code (OIE, 2001) and Murray (2002).

2.1 Terminology

Hazard Identification

A hazard is defined as any pathogenic agent that could produce adverse consequences upon the importation of a commodity. Hazard identification is a process of identifying pathogenic agents that could potentially be introduced in the commodity in question.

Risk Assessment

A process by which the hazards (disease agents of concern) are evaluated in terms of the likelihood that they might be introduced in the particular commodity under consideration. A release assessment has been carried out for each identified hazard and also an exposure assessment where necessary. A release assessment consists of describing the biological pathways necessary for an imported commodity to 'release' or introduce a pathogenic agent into a particular environment and estimates the probability of that process occurring. An exposure assessment consists of a description of the biological pathways necessary which would result in exposure of fish in the importing country to a pathogenic agent released from a given risk source, and estimates the probability of exposure occurring. When exposure assessments determine that there is more than a negligible risk of introduction of a disease agent, consequence assessments have been carried out for each identified hazard to consider the possible effects on people, the New Zealand environment, and the New Zealand economy if, during the course of these activities, a disease agent is released into the New Zealand marine environment.

Risk Management

The process of identifying, selecting and implementing measures that can be applied to reduce or eliminate the level of risk. In this document risk management has taken the form of various recommendations which, if implemented, would significantly reduce or eliminate the risk of introduction of undesirable disease agents.

Terms used to describe the probability of an event occurring

The terms used here to describe the probability of an event occurring should be interpreted in a similar in context to those outlined by AQIS (1999). The terms used include:

High:	Event would be expected to occur
Moderate:	There is less than an even chance of the event occurring
Low:	Event would be unlikely to occur
Very low:	Event would rarely occur
Extremely low:	Event would occur very rarely
Negligible:	Chance of event occurring is so small that it can be ignored in practical terms

Terms used to describe the consequences of an event occurring

The terms used here to describe the consequences of an event occurring should be interpreted in a similar in context to those outlined by AQIS (1999). The terms used include:

Catastrophic: Establishment of diseases which would be expected to significantly harm economic importance at a national level, and/or cause serious and irreversible harm to the environment.

High: Establishment of diseases that would have serious biological consequences (e.g. high mortality or morbidity) and would not be amenable to control or eradication. Such diseases could significantly harm economic performance at an industry level and/or may cause serious harm to the environment.

Moderate: Establishment of diseases which would have less pronounced biological consequences and may be amenable to control or eradication. Such diseases could harm economic performance at an industry level and/or may cause some environmental effects, which would not be serious or irreversible.

Low: Establishment of diseases which would have mild biological consequences and would normally be amenable to control or eradication. Such diseases may harm economic performance at an industry level for a short period and/or may cause some minor environmental effects, which would not be serious or irreversible.

Negligible: Establishment of diseases which would have no significant biological consequences and would require no control or eradication. Such diseases would not affect economic performance at an industry level and would cause negligible environmental effects.

3 Hazard Identification

To help identify the disease agents potentially present in the imported commodity, this section will review the information currently available on the diseases of kingfish in Australia and New Zealand, and also the diseases recorded during the culture of *Seriola* spp. in other parts of the world.

3.1 Diseases recorded from kingfish in Australia

There is only limited information available on the diseases of kingfish in Australia. There appear to be relatively few studies which have examined kingfish in Australia primarily for the purposes of identifying parasites and other disease agents. Most of the information currently available appears to have resulted from studies undertaken in an opportunistic fashion. The known diseases currently recorded from kingfish from Australian waters (with the number of fish examined in each study when these data are available) include:

Viruses

Iridoviridae
Lymphocystis - body and fins, wild kingfish, Tuggerah, NSW (Reddacliff and Quartararo 1992, n = 2 fish).

Bacteria

Vibrio spp. - cultured kingfish, Spencer Gulf (A. Tindale, Hatchery Manager, Spencer Gulf Aquaculture personal communication. n = 1 fish).

Metazoa

Myxozoa

Unicapsula seriolae - muscle, wild kingfish, Moreton Bay, Queensland (Lester 1982, n = 26 fish).
Kudoa sp. - muscle, wild kingfish, Heron Island, Great Barrier Reef (Rohde 1976).

Copepoda

Brachiella sp. - gills, wild kingfish, Heron Island, Great Barrier Reef (Rohde 1977).
Caligus spinosus - gills, wild kingfish, Heron Island, Great Barrier Reef (Rohde 1977).

Monogenea

Monopisthocotylea
Benedenia seriolae - body surface, wild kingfish, Coffs Harbour, NSW, captive kingfish, Sydney, NSW (Whittington 1996), cultured kingfish, Spencer Gulf (Ernst *et al.* 2002).

Polyopisthocotylea
Paramicrocotyloides reticularis - gills, wild kingfish, Heron Island, Great Barrier Reef (Rohde 1978, n = 15 fish).
Zeuxapta seriolae - gills, wild kingfish, Heron Island, Great Barrier Reef (Rohde 1978, n = 15 fish), cultured kingfish, Spencer Gulf (Critchley 2000b, Ernst *et al.* 2002).

Unknown aetiology

Neurological disorder - 70 day old cultured kingfish, Spencer Gulf (Weaver 2001b).

3.2 Diseases recorded from kingfish in New Zealand

There is very limited information available on the diseases of kingfish in New Zealand. There has been only one study to date (Sharp 2001, Sharp *et al.* 2002) which examined kingfish in New Zealand primarily for the purposes of identifying disease agents. That study examined 50 wild caught kingfish from the northern region of the North Island for ectoparasites. Most of the other information currently available has resulted from studies undertaken in an opportunistic fashion. The known diseases currently recorded from kingfish from New Zealand waters (with the number of fish examined in each study when these data are available) include:

Bacteria

Vibrio spp. - cultured kingfish, Hauraki Gulf (B. Diggles, unpublished data). n = over 100 fish).

Metazoa

Copepoda

Caligus aesopus - skin (Jones 1988), gills (Sharp 2001). Sharp (2001) found prevalence = 74% (n = 39 fish).
Caligus lalandei - skin (Jones 1988, Sharp *et al.* submitted). Sharp (2001) found prevalence = 42% (n = 41 fish)
Lernanthropus sp. - gills (Sharp 2001). Prevalence = 26 % (n = 46 fish).
Neobrachiella sp. - gills (Sharp 2001). Prevalence = 24 % (n = 46 fish).

Monogenea

Monopisthocotylea
Benedenia seriolae - body surface (Hine *et al.* 2000, Sharp 2001). Sharp (2001) found prevalence = 88% (n = 42 fish).

Polyopisthocotylea
Zeuxapta seriolae - gills (Hine *et al.* 2000, Sharp 2001). Sharp (2001) found prevalence = 100% (n = 46 fish).

Nematoda

Anisakis spp. larvae - encysted on mesenteries , body cavity (Hewitt and Hine 1972).
Hysterothylacium aduncum - intestine (Hewitt and Hine 1972).
Hysterothylacium seriolae - stomach (Hewitt and Hine 1972).
Hysterothylacium spp. larvae - encysted on stomach, intestine, body cavity (Hewitt and Hine 1972).

3.3 **Significant diseases of cultured *Seriola* spp.**

Yellowtail (*Seriola quinqueradiata*) have been cultured on a commercial basis in Japan for over 50 years (Egusa 1983). During this time much information has been accumulated on the diseases of *S. quinqueradiata*, kingfish (*S. lalandi aureovittata*) and amberjack (*S. dumerili*) in that country. Viral, bacterial, fungal, protozoan and metazoan agents have all caused disease and have negatively affected production of *Seriola* spp. in Japan at some time or another (Egusa 1983, Kusuda and Salati 1993, Muroga 2001). The recent move towards aquaculture of *S. dumerili* in the Mediterranean has also resulted in increased knowledge of their disease agents in that region (Crespo *et al.* 1994, Grau *et al.* 1999). Below is a list of the most prominent parasites and diseases of cultured *Seriola* spp. The list is not exhaustive, but instead has been compiled to indicate the range of significant diseases affecting cultured *Seriola* spp. around the world.

Viruses

DNA Viruses
Iridoviridae
Lymphocystis - *S. quinqueradiata* Japan (Egusa 1983).
Red sea bream iridovirus - *S. quinqueradiata* Japan (Matsuoka *et al.* 1996, Nakajima *et al.* 1998b), *S. lalandi aureovittata* Japan (Matsuoka *et al.* 1996), *S. dumerili* Japan (Matsuoka *et al.* 1996).

RNA viruses
Birnaviridae
Yellowtail ascites virus (YAV) - *S. quinqueradiata* Japan (Sorimachi and Hara 1985), *S. lalandi aureovittata/S. dumerili* hybrid Japan (Isshiki and Kusuda 1987).
Viral deformity (VD) - *S. quinqueradiata* Japan (Nakajima *et al.* 1993).

Bacteria

Epitheliocystis - *S. dumerili* Mediterranean (Crespo *et al.* 1990).
Lactococcus garvieae (syn. *Enterococcus seriolicida*) - *S. quinqueradiata* Japan (Egusa 1983, Kusuda and Salati 1993).
Nocardia kampachi - *S. quinqueradiata* Japan (Egusa 1983, Kusuda and Salati 1993).
Photobacterium damsela subsp. *piscicida* - *S. quinqueradiata* Japan (Kusuda and Salati 1993, Kawakami *et al.* 2000).
Streptococcus iniae - *S. quinqueradiata* Japan (Sako 1998).
Vibriosis (*V. anguillarum, V. harveyi*) - *S. quinqueradiata* Japan (Egusa 1983, Kusuda and Salati 1993), *S. dumerili* China (Wu and Pan 1997), *S. lalandi lalandi* New Zealand (B. Diggles, unpublished data).

Fungi

Ichthyophonus hoferi - *S. quinqueradiata* Japan (Egusa 1983).

Protozoa

Ciliophora

Cryptocaryon irritans - *S. dumerili* Mediterranean (Rigos *et al.* 2001).

Microsporidia

Kabataia seriolae (Beko disease) - *S. quinqueradiata* Japan (Sano *et al.* 1998, Lom *et al.* 1999).

Metazoa

Copepoda

Caligus spinosus - *S. quinqueradiata* Japan (Egusa 1983).
Caligus curtus - *S. dumerili* Mediterranean (Grau *et al.* 1999).

Myxozoa

Kudoa amamiensis - *S. quinqueradiata* Okinawa, Japan (Yokoyama *et al.* 2000).
Kudoa pericardialis - *S. quinqueradiata* Japan (Egusa 1983).
Myxobolus buri - *S. quinqueradiata* Japan (brain, Egusa 1985).
Myxobolus spirosulcatus - *S. quinqueradiata* Japan (bile duct, Maeno *et al.* 1995a).
Myxobolus sp. - *S. dumerili* Mediterranean (Grau *et al.* 1999).

Monogenea

Monopisthocotylea
Benedenia seriolae - *S. quinqueradiata* Japan (Egusa 1983), *S. dumerili* Japan (Whittington *et al.* 2001c), *S. lalandi aureovittata* Japan, *S. lalandi lalandi* Australia (Ernst *et al.* 2002), New Zealand (Hine *et al.* 2000, Sharp 2001).
Neobenedenia girellae - *S. dumerili* Japan (Ogawa *et al.* 1995).
Neobenedenia melleni - *S. dumerili* China (Li and Yang 2002).

Polyopisthocotylea
Heteraxine heterocerca - *S. quinqueradiata* Japan (Egusa 1983), *S. dumerili* Mediterranean (Grau *et al.* 1999).
Zeuxapta seriolae - *S. lalandi lalandi* Australia (Critchley 2000b, Ernst *et al.* 2002), New Zealand (Diggles *et al.* 2002).

Digenea

Sanguinicolidae
Paradeontacylix grandispinus - *S. dumerili* Japan (Ogawa and Fukudome 1994).
Paradeontacylix kampachi - *S. dumerili* Japan (Ogawa and Fukudome 1994).
Paradeontacylix sp. - *S. dumerili* Mediterranean (Montero *et al.* 1999).

Nematoda

Philometra globiceps - *S. dumerili* Mediterranean (Grau *et al.* 1999).
Philometrioides seriolae - *S. quinqueradiata* Japan (Moravec *et al.* 1998).

Unknown aetiology

Neurological disorder - 70 day old cultured kingfish, Spencer Gulf (Weaver 2001b).

3.4 Assessment of current knowledge of the disease status of kingfish in Australia and New Zealand

Comparison of the list of disease agents of cultured *Seriola* spp. with the lists of known diseases of kingfish in Australia and New Zealand highlights at number of disease groups which appear under-represented in Australia and New Zealand at the present time. These groups include some viral diseases (particularly Birnaviridae), opportunistic bacterial pathogens such as *Nocardia*, *Photobacterium*, *Lactococcus* and *Streptococcus*, and obligate parasites such as microsporidia, some myxozoan groups (e.g. *Myxobolus*), and sanguinicolid digeneans. Given the limited study of diseases of kingfish in Australia and New Zealand, it is possible that representatives from these groups are already present in kingfish in the region, but are hitherto unreported.

As the kingfish culture industry matures in Australia and New Zealand it is possible that the "natural" emergence of at least some of these disease groups may occur. A very pertinent example of this is the discovery of a "neurological disorder" of unknown aetiology in 70 day old cultured kingfish from Spencer Gulf (Weaver 2001b). This emergence of such diseases and syndromes is due to the nature of fish farming, which by farming fish at high densities tends to concentrate disease agents which may occur at such low prevalences in wild populations that they are virtually undetectable, even if statistically sound numbers of fish are screened. Examples of groups which may fall into this category include the parasites such as microsporidia, myxosporeans and sanguinicolid digeneans. Other disease agents are opportunistic pathogens, ubiquitous in the marine environment, which become problematic when the fish are compromised by stress, injuries and the reduced water quality which are sometimes endured as part of normal farming practices. Examples of the latter may include birnaviruses, and opportunistic bacterial pathogens. Because there may still be a risk of disease translation even in the absence of disease identification in the exporting country (Gaughan 2002), these factors will be taken into consideration in this risk assessment and the possibility of introducing some of these apparently under-represented disease agents will be assessed.

3.4.1 Impact of the lack of knowledge of diseases of kingfish on the recommendations of this risk assessment

Due to the scarcity of information on diseases of kingfish in Australia and New Zealand, this document will be based on a purely qualitative assessment of the information currently available. Where necessary, this assessment will also rely on extrapolation from data obtained from other closely related species of *Seriola* cultured in the Northern Hemisphere. In all cases where the absence of available information has made assessment of the risks involved difficult, a precautionary approach has been followed towards the process of dealing with uncertainty. Nevertheless, the measures recommended here for reducing risk should be adopted only on a provisional basis (Murray 2002), and reviewed whenever significant additional information on the disease status of kingfish (or other relevant marine species) in Australian and New Zealand waters becomes available.

4 Diseases Under Consideration

The diseases listed in this table will be evaluated.

FISH DISEASES OF CONCERN	TYPE OF ORGANISM	STATUS IN NEW ZEALAND*	STATUS IN AUSTRALIA*	INCLUDED IN RISK ASSESSMENT
Fish diseases notifiable to the OIE				
Epizootic haematopoietic necrosis (EHN)	**Virus**	**X**	**E**	**YES**
Infectious haematopoietic necrosis (IHN)	Virus	X	X	
Oncorhynchus masou virus disease (OMVD)	Virus	X	X	
Spring viraemia of carp (SVC)	Virus	X	X	
Viral haemorrhagic septicaemia (VHS)	**Virus**	**X**	**X**	**YES**
Other significant fish diseases listed by the OIE				
Bacterial kidney disease (BKD)	Bacteria : *Renibacterium salmoninarum*	X	X	
Channel catfish virus disease (CCVD)	Virus	X	X	
Enteric septicaemia of catfish	Bacteria*: Edwardsiella ictaluri*	X	X	
Epizootic ulcerative syndrome (EUS)	**Fungus: *Aphanomyces invadans***	**X**	**E**	**YES**
Gyrodactylosis	Monogenea: *Gyrodactylus salaris*	X	X	
Infectious pancreatic necrosis (IPN) / Aquatic birnaviruses	**Virus**	**X/E**	**X/E**	**YES**
Infectious salmon anaemia (ISA)	Virus	X	X	
Piscirickettsiosis	**Rickettsia: *Piscirickettsia salmonis***	**X**	**E?**	**YES**
Red sea bream iridoviral disease (RSIV)	Virus	X	X	
Viral encephalopathy and retinopathy (VER)	**Virus**	**X**	**E**	**YES**
White sturgeon iridoviral disease	Virus	X	X	
Other fish diseases notifiable in New Zealand				
Enteric redmouth (ERM)	Bacteria*: Yersinia ruckeri* (exotic strains)	E	E	
***Aeromonas salmonicida* / furunculosis**	**Bacteria: *Aeromonas salmonicida***	**X/X**	**E/X**	**YES**
Whirling disease	Myxosporea: *Myxobolus cerebralis*	E	X	

FISH DISEASES OF CONCERN	TYPE OF ORGANISM	STATUS IN NEW ZEALAND*	STATUS IN AUSTRALIA*	INCLUDED IN RISK ASSESSMENT
Other fish diseases significant to New Zealand				
Ceratomyxosis	Myxosporea: *Ceratomyxa shasta*	X	X	
Cold water vibriosis or Hitra disease	Bacteria: *Vibrio salmonicida*	X	X	
Enterococcal infection	**Bacteria: *Lactococcus garvieae***	**X**	**E**	**YES**
Erythrocytic inclusion body syndrome (EIBS)	Salmon anaemia virus	X	X	
Henneguya salminicola	Myxosporea*: Henneguya salminicola*	X	X	
Kudoa thyrsites	**Myxosporea*: Kudoa thyrsites***	**X?**	**E**	**YES**
Loma salmonae	Microsporea*: Loma salmonae*	X	X	
Lymphocystis	Virus	E	E	
Nucleospora salmonis	Microsporea*: Nucleospora salmonis*	X	X	
Pancreas disease of salmon	Virus	X	X	
Parvicapsula species	Myxosporea*: Parvicapsula* species	X?	E	
Pasteurellosis (pseudotuberculosis)	Bacteria: *Photobacterium damsela* ssp. *piscicida*	X	X	
Plasmacytoid leukaemia	Salmon leukaemia virus (retrovirus?)	X	X	
Proliferative kidney disease (PKD)	Myxosporea: *Tetracapsula bryosalmonae*	X	X	
Rosette agent	Choanoflagellate-like organism	X	X	
Sanguinicolid digeneans	Digenea	E	E	
Streptococcosis	**Bacteria: *Streptococcus iniae***	**X**	**E**	**YES**
Unicapsula seriolae	**Myxosporea: *Unicapsula seriolae***	**X**	**E**	**YES**
Vibriosis	Bacteria*: Vibrio anguillarum* (exotic strains)	X?	E	
Viral deformity of yellowtail (VD)	Virus (aquatic birnavirus)	X	X	
Viral erythrocytic necrosis (VEN)	Virus	X	X	
Yellowtail ascites virus (YAV)	Virus (aquatic birnavirus)	X	X	
Kingfish diseases in Spencer Gulf				
Benedenia seriolae	Monogenea: *Benedenia seriolae*	E	E	
Zeuxapta seriolae	Monogenea: *Zeuxapta seriolae*	E	E	

- E = endemic, X = exotic, ? = status and/or identification of disease agent uncertain

A number of the diseases listed above can be removed from this assessment because there is no risk, or a negligible increase in risk, of them being introduced in live cultured juvenile kingfish. The following diseases will not be subject to further consideration in this risk assessment for the reasons described below.

Fish diseases notifiable to the OIE

Infectious haematopoietic necrosis (IHN)
Only salmonids have been shown to be naturally infected with the IHN virus (Wolf 1988, McAllister 1993, OIE 2000), however pike fry (*Esox lucius*), sea bream (*Sparus aurata*) and turbot (*Scophthalmus maximus*) have been infected experimentally, causing mortalities (Bootland and Leong 1999, OIE 2000). This disease has not been recorded from Australia (Herfort and Rawlin 1999). The disease agent must be present in the exporting country before there is a risk of introduction, hence IHN will not be subject to further consideration in this risk assessment.

Oncorhynchus masou virus disease (OMVD)
Only salmonids have been found to be infected with this virus (Wolf 1988, McAllister 1993, OIE 2000). Furthermore, this disease has not been recorded from Australia (Herfort and Rawlin 1999), hence OVMD will not be subject to further consideration in this risk assessment.

Spring viraemia of carp (SVC)

Only freshwater fishes (mainly cyprinids) are susceptible to this virus, which has only been found in Europe (Fijan 1999). This disease has not been recorded from Australia (Herfort and Rawlin 1999), hence SVC will not be subject to further consideration in this risk assessment.

Other significant fish diseases listed by the OIE

Bacterial kidney disease (BKD): *Renibacterium salmoninarum*
Natural outbreaks of BKD have only been reported in salmonids (Evelyn 1993, Shotts and Nemetz 1993, OIE 2000), however non salmonids (*Anoplopoma fimbria*) have been infected experimentally, causing mortalities (Wiens and Kaattari 1999). This disease has not been recorded from Australia (Herfort and Rawlin 1999), hence BKD will not be subject to further consideration in this risk assessment.

Channel catfish virus disease (CCVD)
CCVD is a herpesvirus infection of young channel catfish (*Ictalurus punctatus*). Channel catfish in the United States are the only species known to sustain natural outbreaks of the disease (Wolf 1988, OIE 2000), though the closely related blue catfish (*Ictalurus furcatus*) may also be infected (OIE 2000). These fish species and the disease do not occur in Australia, hence CCVD will not be subject to further consideration in this risk assessment.

Enteric septicaemia of catfish: *Edwardsiella ictaluri*
E. ictaluri is reportedly host-specific for ictalurids (catfish) (Plumb 1993), particularly the channel catfish (Thune 1993), but also catfish of the genus *Ameiurus* and *Clarias* and several ornamental catfish (OIE 2000). This disease has not been recorded from Australian catfish (OIE 2000), hence it will not be subject to further consideration in this risk assessment.

Gyrodactylosis: *Gyrodactylus salaris*
G. salaris is a parasite specifically adapted to salmonids (Cone 1995, OIE 2000). Though a number of other species of *Gyrodactylus* have been recorded from Australia (Dove and Ernst 1998, Ernst *et al.* 2000), *G. salaris* has not been recorded from Australian salmonids (Herfort and

Rawlin 1999), hence gyrodactylosis will not be subject to further consideration in this risk assessment.

Infectious salmon anaemia (ISA)
ISA is caused by a orthomyxovirus which causes disease in Atlantic salmon (Thorud 1991, OIE 2000). ISA virus has also been detected in apparently healthy sea trout (*Salmo trutta*), rainbow trout (*Oncorhynchus mykiss*) and Atlantic herring (*Clupea harengus*), which may act as carriers (OIE 2000). This disease has not been recorded from Australia (Herfort and Rawlin 1999), hence ISA will not be subject to further consideration in this risk assessment.

Red sea bream iridoviral disease (RSIV)
This iridovirus mainly infects red sea bream (or snapper, *Pagrus auratus*) in Japan, but also can also cause disease in yellowtail (*Seriola quinqueradiata*) and other marine species, including kingfish (*S. lalandi aureovittata*) and amberjack *S. dumerili* (see Matsuoka *et al.* 1996). Closely related viruses have been recorded from sheatfish (*Silurus glanis*) and turbot (*Scophthalmus maximus*) in Europe (Ahne *et al.* 1989, Bloch and Larsen 1993) and from grouper (*Epinephelus* spp.) in Thailand (Nakajima *et al.* 1998a, OIE 2000) and Singapore (Qin *et al.* 2001). These viruses are serologically distinct from Australian isolates of Epizootic Haematopoietic Necrosis Virus (EHNV) (Nakajima *et al.* 1998a). RSBIV has not been recorded from Australia (AQIS 1999, OIE 2000), though closely related ranaviruses do occur in Australian freshwater amphibians (Hengstberger *et al.* 1993, Zupanovic *et al.* 1998), and some of these can be pathogenic to barramundi (*Lates calcarifer*) in both freshwater and seawater (Moody and Owens 1994). As RSBIV is not known from Australia (AQIS 1999), this disease will not be subject to further consideration in this risk assessment, however the iridovirus/ranavirus group will be discussed in more detail in the risk assessment section on EHNV.

White sturgeon iridoviral disease
This virus causes disease in farm raised sturgeon in North America and Europe, occurs in wild populations of sturgeon in those areas only, and is known only from sturgeons (OIE 2000). These species of fish do not occur in Australia or New Zealand, hence this disease will not be subject to further consideration in this risk assessment.

Other fish diseases notifiable in New Zealand

Enteric redmouth (ERM) (Exotic strains)
The bacterium *Yersinia ruckeri* causes disease mainly in cultured freshwater fishes, mostly salmonids (Stevenson *et al.* 1993). Less virulent forms of *Y. ruckeri* occur in freshwater fish in both Australia (Herfort and Rawlin 1999) and New Zealand (Diggles *et al.* 2002), but there is no evidence that this bacterium infects kingfish or members of the genus *Seriola* (see Horne and Barnes 1999), hence ERM will not be subject to further consideration in this risk assessment.

Furunculosis
There are various strains of *A. salmonicida*, including *A. salmonicida* subsp. *salmonicida* (strains typically derived from salmonids), *A. s. achromogenes* and *A. s. masoucida* (Atypical strains derived from salmonids), and *A. s. nova* and *A. s. smithia* (Atypical strains associated with disease in non-salmonids). *Aeromonas s. masoucida* should be included in a restructured subspecies *achromogenes* according to Wiklund and Dalsgaard (1998)

A. s. salmonicida causes furunculosis disease in a wide spectrum of freshwater and marine species (Munro and Hastings 1993). While it is usually associated with salmonids, all species of freshwater and marine fish are considered to be susceptible (Shotts and Nemetz 1993). *A. s. salmonicida* has an essentially world-wide distribution including North America, South America,

Europe, Asia, and Africa (Shotts and Nemetz 1993, Austin and Austin 1993, Munro and Hastings 1993). *Aeromonas s. salmonicida* has not been reported from either Australia (AQIS 1999, Herfort and Rawlin 1999) or New Zealand (Anderson *et al.* 1994), and hence furunculosis will not be discussed further in this assessment. However, atypical strains of *A. salmonicida* do occur in Australia (Trust *et al.* 1980, Humphrey and Ashburner 1993, Whittington *et al.* 1995), and these organisms will be discussed later in the risk assessment section.

Whirling disease: *Myxobolus cerebralis*
Whirling disease is caused by *Myxobolus cerebralis*, a parasite of salmonids (Lom and Dykova 1995). Whirling disease has not been recorded from Australia (AQIS 1999, Herfort and Rawlin 1999), but has been detected in New Zealand salmonids (Diggles *et al.* 2002), hence it will not be subject to further consideration in this risk assessment.

Other fish diseases significant to New Zealand

Ceratomyxosis: *Ceratomyxa shasta*
C. shasta is only known to occur on the West Coast of North America, and only affects some salmonid species (Lom and Dykova 1995, Heckmann 1993). This parasite has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Cold water vibriosis or Hitra disease: *Vibrio salmonicida*
Vibrio salmonicida naturally affects Atlantic salmon (Austin and Austin 1993, Hjeltnes and Roberts 1993), and has also caused mortalities in cod (*Gadus morhua*). Disease normally occurs when water temperatures are low. The disease is restricted to Europe (Actis *et al.* 1999), and has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Erythrocytic inclusion body syndrome (EIBS) (salmon anaemia virus)
The virus affects salmonids only (McAllister 1993), and has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Henneguya salminicola
Henneguya salminicola is a myxozoan parasite of salmonids only (Lom and Dykova 1995), and has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Loma salmonae
Loma salmonae is a microsporidian parasite of salmonids only (Dykova 1995), and has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Lymphocystis
Lymphocystis is a condition of nodular skin lesions which affects a wide variety of freshwater, estuarine and marine fish (Roberts 1989). The condition is caused by an iridovirus. The disease has been recorded from Australian kingfish (Reddacliff and Quartararo 1992) and also from New Zealand in John Dory (*Zeus faber*), imported ornamental fish (P. M. Hine, Fish Pathologist, MAF National Centre for Disease Investigation (NCDI), personal communication), and parore (*Girella tricuspidata*) (B. Diggles, unpublished data). Because the disease agent already occurs in New Zealand, it will not be subject to further consideration in this risk assessment.

Nucleospora salmonis

Nucleospora salmonis (syn. *Enterocytozoon salmonis*) is a microsporidian parasite reported to only affect salmonids (Dykova 1995, Kent and Poppe 1998). The parasite has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Pancreas disease of salmon

The virus causing pancreas disease of salmon has only been recognised in salmonids (Munro *et al.* 1984, Boucher *et al.* 1995), and has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Parvicapsula species

A *Parvicapsula* species (Myxozoa) has caused problems in net-pen reared coho salmon in the United States (Lom and Dykova 1995). The same or a similar parasite, described as *P. minibicornis* by Kent *et al.* (1997), was also detected in wild sockeye salmon in British Columbia (Kent 1992). A member of the genus *Parvicapsula* has also been reported from 2 species of marine finfish (*Chaetodon aureofasciatus* and *Diodon hystrix*) from the Great Barrier Reef in Australia (Lester and Sewell 1989). Members of the genus *Parvicapsula* have not been recorded from kingfish in Australia. There is little evidence for causal association of *Parvicapsula* spp. with significant clinical disease (AQIS 1999), as concurrent infections with *Renibacterium* and *Vibrio* occurred during mortalities of net pen reared salmonids, and the role of the parasite in the mortality events was unclear (Kent and Poppe 1998). Because of these reasons, this disease will not be subject to further consideration in this risk assessment.

Pasteurellosis (pseudotuberculosis)

Pasteurellosis, or pseudotuberculosis, is a serious disease of marine finfish in Japan, USA, Taiwan and Europe caused by the bacterium *Photobacterium damsela* subsp. *piscicida* (formerly known as *Pasteurella piscicida*, reviewed by Daly 1999). Affected fish display granulomatous pseudo-tubercles in the kidney and spleen, and generalised necrosis of internal organs. Pasteurellosis is the second most important bacterial disease of yellowtail (*S. quinqueradiata*) in Japan, where it has affected cultured fish since 1964 (Kusuda and Salati 1993). Losses due to this disease in yellowtail culture often exceed 50% in individual farms (Daly 1999). It has been shown experimentally that *S. quinqueradiata* is more susceptible to *P. damsela piscicida* than kingfish (*S. lalandi aureovittata*), with mortalities of 70% and 10% respectively from a standardised challenge (Kawakami *et al.* 2000). *Photobacterium damsela piscicida* has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Plasmacytoid leukaemia (salmon leukaemia virus)

This disease, probably caused by an oncogenic virus and/or the microsporidian *Nucleospora salmonis* (see Kent and Poppe 1998) has only been reported in chinook salmon in North America (Kent *et al.* 1990, Kent 1992, Kent and Poppe 1998). Lymphosarcoma of Atlantic salmon in Tasmania is unlikely to be the same disease as plasmacytoid leukaemia (AQIS 1999). The disease affects only salmonids, and has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Proliferative kidney disease (PKD)

A myxozoan parasite (*Tetracapsula bryosalmonae*) causes PKD in salmonids only (Canning *et al.* 1999). This disease has not been recorded from Australia (AQIS 1999), hence PKD will not be subject to further consideration in this risk assessment.

Rosette agent

An intracellular protistan parasite of salmonids only (Kent 1992, Kent and Poppe 1998). This disease has not been recorded from Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Sanguinicolid digeneans

Infections by blood flukes of the family Sanguinicolidadae (Digenea) have caused mortality in the culture of yellowtail (*S. quinqueradiata*) and amberjack (*S. dumerili*) in Japan (Ogawa and Fukudome 1994) and *S. dumerili* in the Mediterranean (Crespo *et al.* 1994, Gonzalez *et al.* 1995). The disease is known to affect young of the year fish within 4 months of their introduction into sea cages (Ogawa *et al.* 1993). Sanguinicolid digeneans have not been recorded from kingfish in Australia and New Zealand, though *Cardicola forsteri* has been recorded from cultured southern bluefin tuna in South Australia (Cribb *et al.* 2000), and *Paracardicoloides yamagutii* has been recorded from eels (*Anguilla australis* and *A. dieffenbachii*) in New Zealand (Hine *et al.* 2000). These parasites would not occur in hatchery fish reared in filtered and UV sterilised water supplies due to exclusion of cercarial infective stages from the water supply. The chances of sanguinicolid digeneans occurring in cultured juvenile kingfish which have not left the hatchery are therefore negligible.

Vibriosis: *Vibrio anguillarum* (exotic strains)

Vibrio anguillarum is an opportunistic pathogen which has been recorded as causing disease in a wide variety of marine fish (Edgidus 1987). *V. anguillarum* is ubiquitous in the marine environment and occurs worldwide, including New Zealand (Diggles, unpublished data). Strains of *V. anguillarum* on juvenile kingfish imported from Australia are unlikely to be exotic to New Zealand as Australian strains of the bacterium can already be transferred naturally on ocean currents, migrating kingfish or other fish species, hence *V. anguillarum* will not be subject to further consideration in this risk assessment.

Viral deformity virus of yellowtail (VD)

VD is an important viral disease of yellowtail (*S. quinqueradiata*) in Japan (Nakajima *et al.* 1998b). The disease is characterised by abnormal swimming behaviour, deformity of the body and high mortality rates, with fish less than 10 grams in body weight being most susceptible to the virus (Nakajima *et al.* 1993). The virus is a member of the birnaviridae and is related to, but serologically distinct from IPN (Hosono *et al.* 1996). VD is very closely related to yellowtail ascites virus (YAV), but fish infected with VD have clinical signs and pathology distinct from that of YAV (Nakajima and Sorimachi 1994, Maeno *et al.* 1995b, Nakajima *et al.* 1998b). In Japan, VDV has only been reported from yellowtail, however due to its relatedness with YAV the virus may be infective to other species (AQIS 1999). Kingfish in Australia and New Zealand would be expected to be susceptible to VD (AQIS 1999), however to date there has been no report of VD or VD-like infections of kingfish in either country (AQIS 1999). A more detailed assessment of the risks posed by aquatic birnaviruses has been performed in the risk assessment section.

Viral erythrocytic necrosis (VEN)

VEN is a disease which mainly occurs in the northern hemisphere in Atlantic cod, herring and Pacific salmonids (Meyers *et al.* 1986, Dannevig and Thorud 1999). It is not known to occur in Australia (AQIS 1999), hence it will not be subject to further consideration in this risk assessment.

Yellowtail ascites virus (YAV)

YAV is the most important viral disease of yellowtail (*S. quinqueradiata*) in Japan (Nakajima *et al.* 1998b). Kingfish (*S. lalandi aureovittata*) and amberjack (*S. dumerili*) are also susceptible to the virus (Isshiki and Kusuda 1987, Isshiki *et al.* 2001a). The disease usually occurs during the summer months in fish less than 10 grams in body weight (Nakajima *et al.* 1998b). Moribund juveniles typically exhibit anaemic gills, haemorrhaging in the liver, severe ascites, and pancreatic necrosis. Despite the close serological and genetic relatedness of the two viruses, the clinical signs and the pathology associated with YAV are distinct from those associated with VD (Maeno *et al.* 1995b). YAV is a member of the birnaviridae and is related to, but serologically distinct from IPN (Hosono *et al.* 1996). Kingfish in Australia and New Zealand would be expected to be susceptible to YAV (AQIS 1999), however to date there has been no report of YAV or YAV-like infections of kingfish in either country (AQIS 1999). A more detailed assessment of the risks posed by aquatic birnaviruses has been performed in the risk assessment section.

Kingfish diseases in Spencer Gulf

Benedenia seriolae

Benedenia seriolae (Monogenea: Capsalidae) is an important pathogen of cultured *Seriola* spp. in Japan (Egusa 1983, Ernst *et al.* 2002), where it causes dermal lesions and mortalities due to osmoregulatory dysfunction and secondary bacterial infection. This species is also known to occur on wild kingfish in Australia (Whittington 1996), cultured kingfish in Spencer Gulf (Weaver 2001a, Ernst *et al.* 2002), and has been recorded in cultured kingfish from Spencer Gulf Aquaculture Ltd (Whittington *et al.* 2001a). *Benedenia seriolae* is also known to occur on wild and cultured kingfish in northern New Zealand (Sharp 2001, Diggles *et al.* 2002). This parasite exhibits high host specificity and is known to parasitize only members of the genus *Seriola* (see Whittington *et al.* 2001b). Because *B. seriolae* already occurs in New Zealand, any additional risk of exposure of wild kingfish in New Zealand to *B. seriolae* via importation of cultured juvenile kingfish would be negligible.

Zeuxapta seriolae

Zeuxapta seriolae (Monogenea: Polyopisthocotylea) has been recorded along the east coast of Australia (Rohde 1978) and is known to occur in kingfish cultured in Spencer Gulf (Critchley 2000b, Ernst *et al.* 2002). It also occurs on wild and cultured kingfish in New Zealand (Sharp 2001, Diggles *et al.* 2002). *Zeuxapta seriolae* has high host specificity and occurs naturally only on members of the genus *Seriola*. Because *Z. seriolae* already occurs in New Zealand, any additional risk of exposure of wild kingfish in New Zealand to *Z. seriolae* via importation of cultured juvenile kingfish would be negligible.

5 Risk assessments

The following diseases will be subjected to more detailed qualitative risk assessments.

5.1 Aquatic birnavirus, including infectious pancreatic necrosis virus (IPNV)

5.2 Atypical *Aeromonas salmonicida*

5.3 Epizootic haematopoietic necrosis (EHN) and other iridoviruses

5.4 Epizootic ulcerative syndrome (EUS)

5.5 *Kudoa thyrsites*, *Unicapsula seriolae* and other myxosporea

5.6 Piscirickettsiosis

5.7 Streptococcosis and enterococcal infections

5.8 Viral encephalopathy and retinopathy (VER) (including similar neurological disorders)

5.9 Viral haemorrhagic septicaemia (VHS)

5.1 Aquatic birnavirus, including infectious pancreatic necrosis virus (IPNV)

5.1.1 *Release assessment*

IPN virus and IPN-like viruses are double stranded RNA viruses. Various strains of birnaviruses have been described from at least 65 species of fish in 20 families (McAllister 1993), and also from bivalve molluscs (Reno 1999). These viruses, collectively called aquatic birnaviruses, are considered to be ubiquitous in aquatic environments worldwide (Reno 1999). An IPN-like virus has been recorded in returning chinook salmon in New Zealand, but never has been associated with disease (Tisdall and Phipps 1987). The New Zealand aquatic birnavirus isolate appears to be avirulent in salmonids (Hine 1995), however another birnavirus-like agent has been recently detected in cultured turbot (*Colistium nudipinnis*) in New Zealand (P. M. Hine and B. K. Diggles, unpublished). It is possible that the turbot strain was associated with mortalities in cultured juvenile turbot, however the serological relationship between that isolate and isolates from chinook salmon remain undetermined at this time (P. M. Hine and B. K. Diggles, unpublished data). IPN disease is not known to exist in Australia (Herfort and Rawlin 1999), however an IPN-like birnavirus has been identified in farmed Atlantic salmon (*Salmo salar*), and wild rainbow trout (*Oncorhynchus mykiss*), greenback flounder *(Rhombosolea tapirina*), cod (*Pseudophycis* sp.), spiked dogfish (*Squalus megalops*) and ling (*Genypterus blacodes*) in Macquarie Harbour in western Tasmania (Crane *et al.* 2000). Like the New Zealand chinook salmon isolate, it is related to the IPNV serotype Ab and appears to have low pathogenicity for salmonids (Crane *et al.* 2000).

In Japan, aquatic birnaviruses, namely YAV and VD, cause some of the most important diseases of juvenile yellowtail *(S. quinqueradiata*) (see Nakajima *et al.* 1998b, Muroga 2001). Kingfish (*S. lalandi aureovittata*) and amberjack (*S. dumerili*) in Japan have been shown to be susceptible to YAV (Isshiki and Kusuda 1987, Isshiki *et al.* 2001a), which suggests that kingfish in Australia and New Zealand are also likely to be susceptible to aquatic birnaviruses (AQIS 1999). Although it is known that the Australian and New Zealand salmonid isolates of aquatic birnaviruses are closely related (Crane *et al.* 2000), it is presently unknown whether they are the same strain. Furthermore, aquatic birnaviruses are known to cause disease almost exclusively in juvenile fish, with yellowtail less than 10 grams being particularly susceptible in Japan (Nakajima *et al.* 1998b). The size range of the fish proposed for importation is thus within the range most likely to be susceptible to disease caused by birnavirus infection. Currently it is unknown whether birnavirus occurs in the waters of Spencer Gulf, however there are no barriers to movement of wild fish from areas of Tasmania where birnavirus is known to occur in wild fish, and some fish species do move from these areas into South Australian waters (e.g. Stanley 1978). Because of this, aquatic birnaviruses may occur in the waters of Spencer Gulf. Since there has been no reports of screening of broodstock or juvenile kingfish from Spencer Gulf Aquaculture for birnavirus, their birnavirus status is unknown and there may be a very low to extremely low risk that cultured juvenile kingfish from Spencer Gulf Aquaculture could be exposed to birnavirus.

5.1.2 *Exposure assessment*

Marine teleosts and bivalves in New Zealand are already at risk of exposure to the local strain(s) of aquatic birnavirus. However, there is a very low to extremely low risk of exposure to larger than normal quantities of a possibly exotic strain of birnavirus if large numbers of diseased cultured juvenile kingfish are imported. For this to occur the juvenile kingfish would first have to survive the stresses of transport and quarantine without exhibiting signs of disease, then become diseased once introduced into sea cages. If such an event occurred, the quantities of virus entering New Zealand waters via untreated effluent water from a disease outbreak in seacaged fish, could be greatly increased over natural levels.

5.1.3 *Consequence assessment*

The consequences of introduction of larger than normal quantities of birnavirus into the New Zealand marine environment are likely to be either low or negligible. This is because birnaviruses have already been recorded from New Zealand waters (Tisdall and Phipps 1987, P. M. Hine and B. K. Diggles unpublished data). Even if the birnavirus strains from Australian waters are serologically or genetically different to those from New Zealand waters, given the close relatedness of the two isolates examined to date (Crane *et al.* 2000) the chances of an exotic strain introduced in kingfish causing disease in wild or cultured fish in New Zealand would appear to be negligible.

5.1.4 *Risk estimation*

There may be a very low to extremely low risk of introducing larger than normal quantities of a possibly exotic strain of aquatic birnavirus into New Zealand marine waters via importation of cultured juvenile kingfish.

5.1.5 *Risk management*

To mitigate the risk of introduction of birnaviruses, a testing programme is recommended. An appropriate number of fish (150 fish recommended) from each batch of juvenile kingfish destined for export to New Zealand should be tested for the presence of birnavirus by a competent authority in Australia. The tests used should be the internationally recognised methods for detection of IPNV, preferably cell culture on the BF-2 cell line (OIE 2000). Any batches containing fish which test positive for birnavirus should be rejected.

5.2 Atypical *Aeromonas salmonicida*

5.2.1 *Release assessment*

Atypical strains of *Aeromonas salmonicida* have been isolated from goldfish (*Carassius auratus*), silver perch (*Bidyanus bidyanus*), greenback flounder (*Rhombosolea tapirina*) and Atlantic salmon (*Salmo salar*) in Australia (Trust *et al.* 1980, Humphrey and Ashburner 1993, Whittington *et al.* 1995, Bernoth 2000). The bacterium causes ulcerative dermatitis, or goldfish ulcer disease (GUD) in these species. Most outbreaks of GUD in Australia have been reported from freshwater fishes, particularly those in the Murray-Darling River system, where it is thought the spread of the bacterium has been facilitated by feral populations of goldfish and European carp (*Cyprinus carpio*), in which the bacterium is enzootic (Humphrey and Ashburner 1993, Whittington *et al.* 1995). This suggests that atypical *A. salmonicida* does occur in South Australia freshwaters. Furthermore, during 1993 an atypical *A. salmonicida* strain was recovered from ulcerative dermal lesions and kidney of cultured greenback flounder in Tasmania, which were cultured in seawater (Whittington *et al.* 1995). Atlantic salmon (*Salmo salar*) and striped trumpeter (*Latris lineata*) have been infected by co-habitation with the flounder strain (Herfort and Rawlin 1999), and Atlantic salmon cultured in seacages in Tasmania have also been infected by atypical strains of *Aeromonas salmonicida* (see Bernoth 2000). The bacteria isolated from the greenback flounder differed genetically from isolates from goldfish and silver perch, and probably originated from the marine environment (Whittington *et al.* 1995).

These data indicate that atypical strains of *A. salmonicida* occur in marine areas of Tasmania, but there appears to be no published data to indicate whether they occur in the marine waters of Spencer Gulf. However, there are no barriers to migration of marine fish from Tasmania to South Australia, and some species do migrate between these two areas (Stanley 1978), Because of this, atypical strains of *A. salmonicida* may occur in the waters of Spencer Gulf. Data appear to be lacking on the susceptibility of *Seriola* spp. to atypical *A. salmonicida*. Atypical *A. salmonicida* , historically a disease agent of salmonid and cyprinid fishes in freshwater, has been reported in an increasing number of marine species, mainly flatfish (order Pleuronectiformes), codfish (order Gadiformes) and eels (family Anguillidae) (Wiklund and Dalsgaard 1998). Outbreaks of disease in *Seriola* sp. due to infection by *A. salmonicida* have not been reported in Japan, however, suggesting that kingfish may not be as susceptible to atypical strains of *A. salmonicida* as other species. Hence it is difficult to determine what the risk of infection of cultured juvenile kingfish would be if atypical strains of *A. salmonicida* were present in Spencer Gulf. In the light of this uncertainty, a precautionary approach will be adopted and it will be assumed that there is an extremely low risk that juvenile cultured kingfish could harbour these bacteria as part of their external bacterial flora. However, secondary infection and ulceration of dermal lesions caused by ectoparasites has been reported in cultured kingfish in Spencer Gulf (Weaver 2001a, Whittington *et al.* 2001a). As these bacteria are opportunistic pathogens which manifest in non-salmonids predominately as skin lesions (Wiklund and Dalsgaard 1998), the risks of kingfish with external lesions (such as those caused by ectoparasites) harbouring atypical strains of *A. salmonicida* would be higher, and are assessed as low to moderate.

The risks of introduction of atypical strains of *A. salmonicida* into the New Zealand marine environment appear likely to be increased above natural levels only if injured, unhealthy juvenile kingfish with external lesions and/or ectoparasitic infections were imported. The risk of introducing the disease agent could also be increased above natural levels if large numbers of juvenile kingfish which had survived a previous disease outbreak were imported as asymptomatic carriers of the bacterium.

5.2.2 *Exposure assessment*

Fish carrying *A. salmonicida* usually revert to a diseased state during stressful events ("stress tests" are sometimes used to detect carrier fish (Wiklund and Dalsgaard 1998)). Kingfish carrying atypical strains of *A. salmonicida* would, therefore, have to endure the stress of transport and quarantine without reverting to the diseased state (and being detected) during quarantine, before they could be released into sea cages from where the bacterium could enter New Zealands marine environment. After this, to be spread in the marine environment, the bacterium would then need to be transmitted at an infectious dose to other susceptible wild or cultured fish. The risks of establishment of atypical strains of *A. salmonicida* in New Zealand via importation of asymptomatic carrier fish would therefore appear extremely low.

5.2.3 *Consequence assessment*

The consequences of introduction of atypical strains of *A. salmonicida* into the New Zealand marine environment via healthy juvenile kingfish are likely to be low. Disease caused by atypical strains of *A. salmonicida* in the marine environment are almost exclusively associated with confined or cultured fish when they are injured or held at high densities under poor conditions. A more rigorous approach towards husbandry and farm management practices could minimise any detrimental impacts the disease might have on affected fish culture industries.

5.2.4 *Risk estimation*

The risks of introduction of atypical strains of *A. salmonicida* into New Zealand marine waters via cultured juvenile kingfish appear negligible, provided they are obtained from batches of fish with no history of disease, dermal lesions and/or ectoparasitic infections.

5.2.5 *Risk management*

Risk mitigation measures which are recommended include consideration of only batches of fish with low mortality rates (<5%) for export, and rejection of any batches containing juvenile kingfish with dermal lesions and/or ectoparasitic infections.

5.3 Epizootic haematopoietic necrosis (EHN) and other iridoviruses

5.3.1 *Release assessment*

Epizootic haematopoietic necrosis virus (EHNV) is a member of the Ranavirus genus (Family Iridoviridae) which is apparently confined to freshwater areas in Australia and causes mortalities in wild redfin perch (*Perca fluvatilis*) and farmed rainbow trout (*Oncorhynchus mykiss*) (Langdon *et al.* 1986, 1988, Whittington *et al.* 1996). Experimental bath exposures can also cause mortality in Macquarie perch (*Macquaria australasica*), mosquito fish (*Gambusia affinis*), silver perch (*Bidyanus bidyanus*) and mountain galaxias (*Galaxias olidus*), while Murray cod (*Maccullochella peeli*), and Atlantic salmon (*Salmo salar*) are also susceptible by bath exposure, and may act as asymptomatic carriers (Langdon 1989). Redfin perch, rainbow trout, silver perch and Murray cod all occur in freshwater in South Australia, and outbreaks of EHN have been confirmed from redfin from the Murray River and Mt Bold Reservoir (Pierce *et al.* 1991). Redfin with clinical signs of EHN continue to be detected in reservoirs near Adelaide during the summer months (AQIS 1999).

There is no published information available on the susceptibility of kingfish to EHNV. However it is known that the catadromous species barramundi (*Lates calcarifer*) is refractory to EHNV, while another catadromous species, Australian bass (*Macquaria novemaculeata*) is susceptible to infection only by intra peritoneal inoculation, and hence is probably not a natural carrier of the virus (Langdon 1989). Red sea bream (*Pagrus auratus*) in Japan were also not susceptible to EHNV by intra peritoneal challenge (Nakajima and Maeno 1998), suggesting that obligate marine fishes such as kingfish are not susceptible to EHNV. However, closely related viruses in the family iridoviridae are known to infect the obligate marine turbot (*Scophthalmus maximus*) in Europe (Whittington *et al.* 1996). Furthermore, it has been shown that Bohle iridovirus, an iridovirus which infects amphibians in Australia (Moody and Owens 1994), is pathogenic to barramundi and can cause mortalities in barramundi exposed to virus in seawater (Moody and Owens 1994). In Japan, red sea bream iridovirus (RSIV) has shown pathogenicity to 31 species of marine fish, including yellowtail (*S. quinqueradiata*), kingfish (*S. lalandi aureovittata*) and amberjack (*S. dumerili*) (see Matsuoka *et al.* 1996, Nakajima and Maeno 1998, Kawakami and Nakajima 2002).

Total inactivation of EHNV after 24 hours at 40°C suggests that intestinal transfer by piscivorous birds and mammals is unlikely to be a major route of transmission (Langdon 1989). Transfer of EHNV and amphibian iridoviruses from freshwater rivers into estuarine areas in South Australia with movement of carrier fish species also seems unlikely due to the lack of any dominant catadromous and anadromous fish species in that state (Gommon *et al.* 1994). These data indicate that there are few likely routes by which aquatic and amphibian iridoviruses may be transmitted to susceptible marine teleosts in South Australia. Furthermore, the location of the exporting hatchery at Port Augusta in Spencer Gulf is very remote from the nearest major river or estuary (Nunes and Lennon 1986), therefore the risk of cultured kingfish being exposed to infectious doses of EHNV and amphibian iridoviruses, and becoming carriers of EHNV and/or amphibian iridovirus appears negligible. However, iridoviral diseases are continuing to emerge as significant diseases of cultured marine fish in the Northern Hemisphere, but there has been no effective surveillance of marine fish in Australia for iridoviruses at this time (Dr. B. Jones, Department of Fisheries and Agriculture, Western Australia, personal communication), hence the disease status of kingfish in South Australia with regards to novel or emerging iridoviruses is difficult to determine. Due to the uncertainty regarding the iridovirus status of marine fish in Australia, the possibility that juvenile kingfish from South Australia pose a risk of introduction of an unidentified, novel iridovirus cannot be discounted at this time.

5.3.2 *Exposure assessment*

If cultured juvenile kingfish from South Australia harboured a novel iridovirus, carrier fish would have to endure the stress of transport and quarantine without reverting to the diseased state (and being detected) during quarantine, before they could be released into sea cages from where the virus could enter New Zealands marine environment. After this, to be spread in the marine environment, the virus would then need to be transmitted at an infectious dose to other susceptible wild or cultured fish. The risks of establishment of a novel iridovirus in New Zealand via importation of asymptomatic carrier fish would therefore appear extremely low.

5.3.3 *Consequence assessment*

Because there has been no effective surveillance of marine fish in New Zealand for iridovirus, their status remains undetermined, and hence at this time there is no difference in the current iridovirus disease status (in the marine environment) of either country. Due to the uncertainty involved with the iridovirus status of New Zealand marine fish, it is difficult to assess the magnitude of the consequences which might be associated with the introduction of juvenile kingfish from South Australia infected with a novel iridovirus. However, based on the data available from the northern hemisphere which indicates that iridoviral diseases are continuing to emerge as significant diseases of cultured marine fish, if a novel iridovirus was introduced with cultured juvenile kingfish from South Australia, the consequences for New Zealands marine environment and aquaculture industries are assessed as low to moderate.

5.3.4 *Risk estimation*

As the risk of introduction of EHNV and amphibian iridoviruses appears negligible, no specific safeguards appear warranted for those diseases. However, it has been difficult to determine whether the commodity poses a risk of introducing a novel iridovirus into New Zealand. As the consequences of such an introduction have been categorised as being low to moderate, adoption of a precautionary approach to managing any risks which may be involved appears prudent.

5.3.5 *Risk management*

To mitigate any risk of introduction of a novel iridovirus, it is recommended that iridoviruses should be considered in the virus testing programmes detailed herein. As many iridoviruses can be isolated as primary cultures on the BF-2 cell line recommended by the OIE for detection of birnaviruses, surveillance for novel iridoviruses could be undertaken as part of the routine screening process recommended for birnaviruses. Therefore, kingfish from batches containing fish which have tested positive (e.g. generated a cytopathic effect (CPE) on BF-2 cell culture media) for viruses other than VER and aquatic birnavirus during routine cell culture testing, should not be exported to New Zealand and the identity of the virus(es) responsible for the CPE should be determined.

5.4 Epizootic ulcerative syndrome (EUS)

5.4.1 *Release assessment*

The disease known as epizootic ulcerative syndrome (EUS) has been consistently associated with the presence of the fungus *Aphanomyces invadans* (see Lilley and Roberts 1997). Rhabdoviruses have also been associated with some EUS outbreaks, and secondary gram-negative bacteria invariably infect EUS lesions, however *A. invadans* is recognised as the primary disease agent (OIE 2000). EUS mainly occurs in Asia and the Indo-Pacific region, including Australia (Fraser *et al.* 1992, Callinan *et al.* 1995, Chinabut 1998), however similar ulcerative mycotic diseases have been recorded from estuarine fish along the east coast of the United States (Dykstra *et al.* 1989), and recently *A. invadans* has also been implicated in that region (Blazer *et al.* 2002).

EUS in Australia is primarily a disease of estuarine fish, particularly mullet (*Mugil cephalus*), yellowfin bream (*Acanthopagrus australis*) and whiting (*Sillago* spp.), in which it causes reddened ulcerative dermal lesions known as "red spot disease" (RSD) (Fraser *et al.* 1992). However, EUS has also recently been reported in silver perch (*Bidyanus bidyanus*) cultured in Victoria (Jubb 2001). EUS has been recorded from estuarine fish in Queensland, NSW, the Northern Territory and Western Australia and is restricted mainly to areas of low salinity (2 - 9 ‰), with disease outbreaks usually following heavy rainfall (Fraser *et al.* 1992, Virgona 1992). The waters of Spencer Gulf are classed as a hypersaline inverse estuary, due to the high rate of evaporation and lack of any significant riverine input (Nunes and Lennon 1986). Mean depth-averaged salinities north of Whyalla, in the area where the Spencer Gulf Aquaculture hatchery is based, range between 40 and 48 ‰ (Nunes and Lennon 1986). These data suggest there is negligible risk of juvenile kingfish reared in the Spencer Gulf Aquaculture hatchery being exposed to water of sufficiently low salinity to promote infection with *A. invadans*. Provided that fish are free from ulcerative dermal lesions, and are not exposed to waters of low salinity (<30‰) during transport, no further assessment appears necessary.

5.4.2 *Risk estimation*

There appears to be negligible risk of juvenile kingfish reared in the Spencer Gulf Aquaculture hatchery being exposed to water of sufficiently low salinity to promote infection with *A. invadans*.

5.4.3 *Risk management*

To mitigate any risk of introduction of EUS, juvenile kingfish should be free from ulcerative dermal lesions, and not be exposed to waters of low salinity (<30‰) during rearing and transport.

5.5 *Kudoa thyrsites, Unicapsula seriolae* and other myxosporea

5.5.1 *Release assessment*

Kudoa thyrsites is a myxosporean parasite of a variety of marine fish (Lom and Dykova 1995). The presence of *K. thyrsites* spores in the fillet musculature of infected fish is associated with 'milky flesh' and post-mortem myoliquefaction of the musculature due to excretion of histolytic enzymes (Langdon 1991, St-Hilaire *et al.* 1997). This parasite occurs naturally in the flesh of wild fishes, and also infects cultured fishes reared in sea cages, including mahi mahi (*Coryphaena hippurus*) and Atlantic salmon (*Salmo salar*) (see Langdon 1991, Moran and Kent 1999, Kent 2000). In Australia *K. thyrsites* has been recorded from wild barracouta (*Thyrsites atun*), pilchards (*Sardinops sagax neopilchardus*), southern anchovy (*Engraulis australis*), scaly mackerel (*Sardinella lemuru*), blue sprats (*Spratelloides robustus*), cultured *S. salar* and wild and cultured *C. hippurus* (see Langdon 1991, Munday *et al.* 1998, O'Donoghue and Adlard 2000). An undescribed species of *Kudoa* has also been recorded in the muscle of kingfish in Australia (O'Donoghue and Adlard 2000). In New Zealand, an undescribed species of *Kudoa* has been recorded from the muscle of red cod (*Pseudophycis bachus*), and 65 other species of myxosporeans have been recorded from a variety of fishes (see Hine *et al.* 2000). However, *Kudoa thyrsites* has not been recorded from fishes in New Zealand, including *T. atun* (see Hine *et al.* 2000), but the author was recently presented with anecdotal evidence of post-mortem myoliquefaction in *T. atun* exported from New Zealand (Dr Barry Munday, University of Tasmania, personal communication). The absence of *K. thyrsites* in the parasitological records of New Zealand fishes (Hine *et al.* 2000) could, therefore, be due to a lack of adequate parasitological assessment of New Zealand fishes, rather than the absence of the parasite.

While there is evidence for direct fish to fish transmission for some marine myxosporeans (Diamant 1997, Swearer and Robertson 1999), the lifecycles of the vast majority of marine myxosporeans are unknown (Kent *et al.* 2001). Attempts to transfer *K. thyrsites* infection via feeding spores to Atlantic salmon failed to transmit infection, however Atlantic salmon held in seacages in marine waters where *K. thyrsites* was enzootic became infected within 2 weeks (Moran *et al.* 1999). This suggests that fish in seacages may become infected through either contact with infective stages (actinospores) released by alternate hosts, by eating presporogonic stages in other infected fishes, or even obtaining presporogonic stages via blood transferred by blood feeding vectors such as copepods, leeches or monogeneans.

Baitfish species commonly found in Spencer Gulf may act as reservoir hosts for *K. thyrsites* and other myxosporeans (Langdon *et al.* 1992). *Coryphaena hippurus* is also known to occur in the waters of upper Spencer Gulf (Gommon *et al.* 1994). This suggests that the greatest risk of juvenile kingfish becoming infected with *K. thyrsites* and other myxosporeans would be if they were exposed to infective stages while being maintained in sea cages in Spencer Gulf. However, juvenile kingfish maintained in the Spencer Gulf hatchery in a water supply filtered to 1µm and UV irradiated, and fed hatchery reared or artificial foods, would have an extremely low risk of being exposed to infective stages of *K. thyrsites* and other myxosporeans. Nevertheless, in Canada where *K. thyrsites* is enzootic, Atlantic salmon held in landbased tanks with a water supply filtered to 1 µm can still become infected (Moran *et al.* 1999), hence there may still be an extremely low risk that juvenile kingfish maintained in a filtered hatchery water supply could be exposed to infective stages of *K. thyrsites*.

Unicapsula seriolae is another myxosporean parasite which infects the flesh of kingfish in south east Queensland (Lester 1982). The presence of *U. seriolae* spores in the fillet of infected fish is associated with myodegeneration of the musculature during slow cooking, presumably due to excretion of histolytic enzymes (Lester 1982), which render affected fillets inedible. Rapid

cooking of the flesh, however, does not cause myodegeneration, suggesting that enzyme activity occurs only in a limited temperature range (Lester 1982). *Unicapsula seriolae* occurs naturally in the flesh of wild kingfish in south east Queensland, but has not been recorded in southern Australia, or New Zealand. Because the gross signs of infection by *U. seriolae* are easily discovered by the lay person upon cooking the flesh, the lack of reports of fillet myodegeneration in kingfish from areas other than south east Queensland strongly suggests *U. seriolae* is restricted to that region (Lester 1982). The apparently restricted distribution of *U. seriolae* could be due to a restricted distribution of an as yet unknown alternative host, or perhaps because fish heavily infected by *U. seriolae* do not migrate far. In any case, the absence of reports of myodegeneration of kingfish in South Australian waters suggests there is negligible risk of cultured juvenile kingfish from South Australia becoming infected with *U. seriolae*.

5.5.2 *Exposure assessment*

If cultured juvenile kingfish from South Australia harboured *K. thyrsites* or other myxosporeans, asymptomatic carrier fish would have to survive transport and quarantine before they could be released into sea cages from where the parasites could enter New Zealands marine environment. After this, to be spread in the marine environment, the parasites would then need to be transmitted at an infectious dose to other susceptible wild or cultured fish, and suitable environmental conditions and alternative hosts would need to be present. The risks of establishment of myxozoan parasites in New Zealand via importation of asymptomatic carrier fish are therefore assessed as low.

5.5.3 *Consequence assessment*

Due to the uncertainty involved with the status of New Zealand marine fish regarding *Kudoa thyrsites*, it is difficult to assess the magnitude of the consequences which might be associated with the introduction of juvenile kingfish from South Australia infected with *K. thyrsites*. However, if *K. thyrsites* is absent from the New Zealand environment, based on experiences in the northern hemisphere, it is possible there could be adverse consequences for both salmonid and marine fish sea cage aquaculture in New Zealand. The consequences of such an event occurring would then probably fall into the moderate or low categories.

5.5.4 *Risk estimation*

There appears negligible risk of introduction of *Unicapsula seriolae*, but an extremely low risk of introduction of *Kudoa thyrsites* and other myxosporeans into New Zealand marine waters via cultured juvenile kingfish. There is some uncertainty regarding the status of *K. thyrsites* in New Zealand waters, however if the status quo of "absent" is retained, the consequences of such an introduction have been categorised as being low to moderate. Adoption of a precautionary approach to managing the uncertainties and potential risks involved appears prudent, hence risk management measures are recommended.

5.5.5 *Risk management*

To mitigate any risks of introduction of *Kudoa thyrsites*, juvenile kingfish cultured for export should be maintained within the confines of the hatchery at Spencer Gulf Aquaculture in a water supply filtered to 1 µm and UV sterilised, fed only hatchery reared live food or artificial food, and should not be transferred into sea cages outside the hatchery prior to export.

5.6 Piscirickettsiosis

5.6.1 *Release assessment*

Piscirickettsiosis is a disease caused by an intracellular bacterium, *Piscirickettsia salmonis*, that mainly affects salmonids (Turnbull 1993, OIE 2000). The disease primarily effects coho salmon (*Oncorhynchus kisutch*) but also can occur in rainbow trout (*O. mykiss*), chinook salmon (*O. tshawytscha*) and Atlantic salmon (*Salmo salar*) (Arkush *et al.* 2002). However, closely related intracellular bacteria have recently caused disease in white seabass (*Atractoscion nobilis*) in California (Chen *et al.* 2000a), grouper (*Epinephelus melanostigma*) in Taiwan (Chen *et al.* 2000b), Atlantic salmon in Tasmania (Elliot 2001) and three lined grunt (*Parapristipoma trilineatum*) in Japan (Fukuda *et al.* 2002). The emergence of *Piscirickettsia*-like bacteria (PLB) has had adverse effects on the profitability and productivity of an increasing number of marine fish culture industries (Mauel and Miller 2002). In Tasmania, the discovery of PLB was associated with low level mortalities (<5%) in sea cage cultured Atlantic salmon in south-eastern Tasmania. The source of the disease is unknown, but in other cases it has been suggested that wild marine fish are likely candidates as reservoirs for the bacterium (Johnson 2002, Arkush *et al.* 2002).

These data indicate that PLB may occur in marine areas of Tasmania, but there is no published data to indicate whether they occur in the marine waters of Spencer Gulf. However, there are no barriers to migration of marine fish from Tasmania to South Australia, and some species do migrate between these two areas (Stanley 1978). Because of this, PLB may occur in the waters of Spencer Gulf. Data appear to be lacking on the susceptibility of *Seriola* spp. to PLB. Piscirickettsiosis in cultured *Seriola* sp. has not been reported in Japan after over 40 years of culture, suggesting that kingfish may not be susceptible to PLB. Hence it is difficult to determine what the risk of infection of cultured juvenile kingfish would be if PLB were present in Spencer Gulf. Given that these disease agents are intracellular, they appear less likely to survive for long periods in the marine environment than other heterotrophic bacteria (AQIS 1999). Furthermore, it appears that the minimum infectious dose for *Piscirickettsia salmonis* is relatively high compared to that of the more typical bacterial disease agents (AQIS 1999). This suggests that the chances of juvenile kingfish being exposed to an infective dose of PLB while being maintained in a filtered and UV irradiated water supply would be less than the chances of them being infected by other bacterial pathogens. The risk of healthy juvenile kingfish at Spencer Gulf Aquaculture becoming infected with PLB is, therefore, probably negligible. However, as with other diseases caused by opportunistic bacterial pathogens, the risks of infection are likely to be increased if injured, unhealthy juvenile kingfish with external lesions and/or ectoparasitic infections were imported. The risk of introducing the disease agent could also be increased if large numbers of juvenile kingfish which had survived a previous disease outbreak were imported, as they could act as asymptomatic carriers of the bacterium.

5.6.2 *Risk estimation*

Due to the lack of information on whether *Piscirickettsia* -like bacteria occur in the South Australian marine environment, and also on the susceptibility of *Seriola* sp. to these bacteria, it is difficult to assess the extent of the risk or the potential consequences associated with importation of large numbers of juvenile kingfish. However, given the fact that the disease agent appears less likely to survive for long periods in the marine environment than other heterotrophic bacteria, and its infective dose appears higher than other bacterial pathogens, the chances of healthy cultured juvenile kingfish becoming infected appear negligible. However, cultured kingfish with external lesions or ectoparasite infections, or those sourced from batches of fish with a disease history, may have an increased risk of harbouring *Piscirickettsia* -like bacteria, hence some steps for

minimising risk appear warranted.

5.6.3 *Risk management*

To mitigate potential risks of introduction of *Piscirickettsia* -like bacteria, juvenile kingfish should be exported only from batches of fish which exhibit low mortality rates (<5%), and are free from external lesions and/or ectoparasitic infections.

5.7 Streptococcosis and enterococcal infections

5.7.1 *Release assessment*

Streptococcosis caused by *Streptococcus iniae* and other species of *Streptococcus* is an important bacterial disease of yellowtail (*S. quinqueradiata*) in Japan (Kusuda and Salati 1993, 1999). *S. iniae* and other closely related lactic acid bacteria are ubiquitous opportunistic pathogens of marine and freshwater fish worldwide (Bromage *et al.* 1999, Kusuda and Salati 1999). Zoonotic infection of exposed wounds in humans by *S. iniae* has also been reported (Weinstein *et al.* 1997). In Australia, *S. iniae* is the most important bacterial species affecting sea cage cultured barramundi (*Lates calcarifer*) in northern Queensland (Bromage *et al.* 1999), and also barramundi cultured in landbased aquaculture farms in South Australia (Critchley 2000a). In barramundi the bacterium can be carried in the brain of asymptomatic fish, which can act as reservoirs of infection (Bromage *et al.* 1999). Streptococcal infections of cultured marine fish can be transferred to wild fish (Colorni *et al.* 2002), however to date there have been no reports of streptococcal infections of wild marine fish in Australia (J. Carson, Chief Microbiologist, Fish Health Unit, DPIWE Tasmania, personal communication), or New Zealand (Diggles *et al.* 2002).

Enterococcal infection caused by *Lactococcus garvieae* (syn. *Enterococcus seriolicida*) is the major bacterial disease of yellowtail (*S. quinqueradiata*) in Japan (Kusuda and Salati 1993, 1999). *L. garvieae* is a opportunistic pathogen of marine and freshwater fish worldwide, but has a wide host range which includes both aquatic and terrestrial vertebrates (including humans), and aquatic invertebrates (Eldar *et al.* 1996, Eldar *et al.* 1999). Different strains occur in Japan, Europe and Australia (Eldar *et al.* 1999). One Australian strain was isolated from diseased rainbow trout in freshwater hatcheries in Tasmania and Victoria (Carson *et al.* 1993). In Tasmania, disease associated with *E. garviae* infection was observed in sea run rainbow trout (*Oncorhynchus mykiss*) on two occasions between 1986 and 1990. In both cases the disease episode occurred soon after sea transfer of covertly infected rainbow trout from hatcheries with a known history of disease due to *L. garviae* (J. Carson, Chief Microbiologist, Fish Health Unit, DPIWE Tasmania, personal communication). In both cases, no transfer of disease to other species of marine fish was recorded. Outbreaks of enterococcal disease in cultured yellowtail in Japan occur mainly in summer in seacaged juveniles and are associated with poor water quality and high stocking densities (Kusuda and Salati 1999). *E. garviae* has not been recorded from marine or freshwater fish in New Zealand (Diggles *et al.* 2002).

Lactococcus garviae and *S. iniae* have both been recorded from marine fish cultured in Australia, indicating these bacteria occur in the Australian marine environment. Both disease agents are known opportunistic pathogens of *Seriola* spp. cultured in seacages under suboptimal conditions. However, in Japan, where enterococcal and streptococcal diseases of seacaged *Seriola* spp. are common, both diseases do not feature prominently in hatcheries (Muroga 2001). Hence the risks of these bacteria occurring in healthy hatchery reared kingfish juveniles 1-3 grams in weight would appear negligible. However the opportunistic nature of these bacteria would suggest that the risks of fish from batches with high mortality rates (>5%), or fish with external lesions and/or ectoparasitic infections carrying an infectious dose of either bacterium would be increased over that posed by healthy fish.

5.7.2 *Exposure assessment*

If fish thus affected survived transport and quarantine and were placed in seacages, there could be a low risk that *Lactococcus garviae* or *S. iniae* could be released into the New Zealand marine environment. However, these bacteria would in turn be most likely to affect only other injured, or compromised fish. Injured or compromised fish seldom exist for long in the marine

environment due to predation (AQIS, 1999), therefore the risks of establishment and spread of *Lactococcus garviae* or *S. iniae* to other wild or cultured fish would appear extremely low.

5.7.3 *Consequence assessment*

The consequences of introduction of *Lactococcus garviae* and *S. iniae* into the New Zealand marine environment via healthy juvenile kingfish are likely to be either low or negligible. This is because opportunistic bacterial pathogens rarely, if ever, cause disease in healthy fish. Disease caused by *Lactococcus garviae* and *S. iniae* in the marine environment are almost exclusively associated with cultured fish when they are injured or held at high densities under poor conditions. Avoidance of disease outbreaks due to opportunistic bacteria such as *Lactococcus garviae* and *S. iniae* is, therefore, based mainly on encouragement of sound husbandry practices, and as such is not necessarily related to the simple presence or absence of the disease agent.

5.7.4 *Risk estimation*

Both disease agents are opportunistic and appear to be ubiquitous in the marine environment in Australia, however they have not been recorded from kingfish in Australia or from New Zealand at the present time. Diseases associated with *L. garviae* and *S. iniae* mainly occur in fish reared in seacages under suboptimal conditions, and the risk of introduction of these bacteria with juvenile hatchery reared kingfish is probably negligible. However, kingfish with external lesions or ectoparasite infections, or those sourced from batches of fish with a disease history, would have an increased risk of harbouring *Lactococcus garviae* and *S. iniae,* hence some steps for minimising risk appear warranted.

5.7.5 *Risk management*

To mitigate the risks of introduction of *Lactococcus garviae* and *S. iniae*, juvenile kingfish should be exported only from batches of fish which exhibit low mortality rates (<5%), and are free from external lesions and/or ectoparasitic infections.

5.8 Viral encephalopathy and retinopathy (VER) (including similar neurological disorders)

5.8.1 *Release assessment*

Viral encephalopathy and retinopathy (VER) disease, or viral nervous necrosis (VNN) are serious diseases of larval and juvenile marine fish that occur almost worldwide (OIE 2000). They are associated with necrosis and heavy vacuolation of the central nervous system and retina (Munday and Nakai 1997), and are caused by a group of closely related betanodaviruses in the family Nodaviridae (Nishizawa *et al.* 1997). Fish species reported to be susceptible to nodaviruses include flatfish (turbot (*Scophthalmus maximus*), halibut (*Hippoglossus hippoglossus*), olive flounder (*Paralichthys olivaceus*)), striped jack (or silver trevally, *Pseudocaranx dentex*), groupers (*Epinephelus* spp.), European sea bass (*Dicentrarchus labrax*) and barramundi (*Lates calcarifer*) (OIE 2000). The disease does not appear to affect salmonids reared in the marine environment. The onset of mortalities due to VNN are usually observed between 1 and 40 days post hatching in larval fish, though the latest occurrence of disease outbreaks due to VNN in *D. labrax* was recorded at a body weight of 400-580 grams (OIE 2000). The virus appears to be transmitted vertically through the egg or sexual fluids in *P. dentex* (see OIE 2000). Species which appear not to be susceptible to disease include red sea bream (*Pagrus major*), Japanese yellowtail (*Seriola quinqueradiata*) and kingfish (*Seriola lalandi aureovittata*) (see Arimoto *et al.* 1993). However, some species, such as the gilt head sea bream *Sparus aurata,* become infected, but do not exhibit clinical signs of this disease, and hence can act as asymptomatic carriers (Castric *et al.* 2001).

In Australia it is well established that cultured larvae and juveniles of barramundi are affected by nodaviruses (Glazebrook *et al.* 1990, Munday *et al.* 1992, Munday and Nakai 1997). Barramundi have been farmed in landbased culture facilities in South Australia, and nodaviral disease has been detected in these on a number of occasions (AQIS 1999, Critchley 2001). Broodstock of striped trumpeter (*Latris lineata*) held in Tasmania have also tested positive for nodavirus (Elliot 2001). Seventy day old cultured kingfish from South Australia exhibiting uncoordination and lesions in the brain and spinal chord suggestive of a neurological disease tested negative for nodavirus (Weaver 2001b). In New Zealand hatchery reared juvenile turbot (*Colistium nudipinnis*) sampled during mortality events were negative for nodavirus using the OIE recommended immunoflourescent antibody technique (Diggles *et al.* 2000). A species highly susceptible to nodaviral infection, *P. dentex*, occurs in both Australia (Gommon *et al.* 1994) and New Zealand (Paul 2000). No studies appear to have been conducted to determine whether nodavirus occurs naturally in wild *P. dentex* in either Australia or New Zealand.

It appears that members of the genus *Seriola*, including *S. lalandi aureovittata,* are not affected by nodaviral disease (Arimoto *et al.* 1993). It may be possible, however, that *Seriola* spp. could act as asymptomatic carriers of nodavirus, as shown for *Sparus aurata* (see Castric *et al.* 2001). Samples of a number of kingfish juveniles from Spencer Gulf Aquaculture have been examined for the presence of nodavirus by the Oonoonba Veterinary Laboratory, Townsville, using the RT-PCR method on at least three occasions (A. Tindale, Hatchery Manager, Spencer Gulf Aquaculture, personal communication, M. Deveney, Project Officer, Fish Health, PIRSA, personal communication). Kingfish exhibiting clinical signs of "neurological disease" and lesions in the brain were also examined for nodavirus (Weaver 2001b). In all instances negative results were obtained. This suggests that the broodstock kingfish which supply larvae for ongrowing at Spencer Gulf Aquaculture are free from nodavirus infection. However, no testing of wild kingfish or other fish species such as *P. dentex* has been conducted in South Australian marine waters, and the cause of the neurological disease of 70 day old kingfish (Weaver 2001b) remains uncertain. Because of the lack of data regarding the possibility of kingfish acting as a

carrier species for nodavirus at the present time, and the presence of the "neurological disease" in cultured kingfish in South Australia (Weaver 2001b), the possibility that juvenile cultured kingfish imported from South Australia could present an extremely low risk of introduction of nodavirus cannot be ruled out.

5.8.2 *Exposure assessment*

If cultured juvenile kingfish from South Australia harboured nodavirus in the carrier state, carrier fish would have to endure the stress of transport and quarantine without reverting to the diseased state (and being detected) during quarantine, before they could be released into sea cages from where the virus could enter New Zealands marine environment. After this, to be spread in the marine environment, the virus would then need to be transmitted at an infectious dose to other susceptible wild or cultured fish. The risks of establishment of nodavirus in New Zealand via importation of asymptomatic carrier fish would therefore appear extremely low.

5.8.3 *Consequence assessment*

Nodaviral infections are one of the most important sources of disease in larval marine fish worldwide (OIE 2000), however nodaviral infections have not been demonstrated to cause disease in wild fish. The consequences of the introduction of nodavirus into the marine environment in New Zealand would most likely be negligible to the culture of salmonids, and also kingfish and snapper (the two major non-salmonid species cultured to date), because all are likely to be unaffected by nodavirus (Arimoto *et al.* 1993, Castric *et al.* 2001). However the introduction of large numbers of carrier fish could impede the development of culture of other local marine species which are likely to be susceptible to nodaviral infections, such as striped trumpeter. However, due to the lack of information on the nodavirus status of wild marine fish in New Zealand, particularly *P. dentex*, it is difficult to assess the extent of the consequences associated with importation of large numbers of infected carrier fish.

5.8.4 *Risk estimation*

Due to the presence of an undetermined "neurological disorder" in South Australian kingfish, lack of information on the ability of kingfish to act as asymptomatic carriers of nodavirus, and the lack of knowledge of the nodavirus status of marine fish stocks in New Zealand, it is difficult to assess the extent of the risk or the potential consequences associated with importation of large numbers of juvenile kingfish. Taking into account the limited information available at this time, a precautionary approach to managing any risks which may be involved appears prudent.

5.8.5 *Risk management*

A testing programme is recommended to mitigate any risk of introduction of large numbers of possible carrier fish into New Zealand. An appropriate number of kingfish from each batch of juveniles destined for export to New Zealand (150 fish recommended) should be tested for the presence of nodavirus by a competent authority in Australia. The test used should be either cell culture on SSN-1 cell line, and/or PCR (OIE 2000). Any batches containing fish which test positive for nodavirus should be rejected.

5.9 Viral haemorrhagic septicaemia (VHS)

5.9.1 *Release assessment*

Viral haemorrhagic septicaemia is caused by viral haemorrhagic septicaemia virus (VHSV), a novivirus (Family Rhabdoviridae). In the northern hemisphere, both salmonid and non-salmonid fish species may be naturally infected with VHSV in both freshwater and marine environments (Wolf 1988, McAllister 1993). The occurrence of VHSV in wild marine fish in the northern hemisphere is becoming increasingly evident (Dixon 1999, Meyers *et al.* 1999, Mortensen *et al.* 1999). The host range appears broad, including cod (*Gadus morhua*), turbot (*Scophthalmus maximus*), herring (*Clupea harengus*), haddock (*Melanogramus aeglefinus*), Norway pout (*Trisopterus esmarkii*), sprat (*Sprattus sprattus*), rockling (*Rhinonemus cimbrius*), whiting (*Merlangius merlangius*), and other species in the North Atlantic, and herring (*C. harengus pallasi*), cod (*G. macrocephalus*), chinook salmon (*Oncorhynchus tshawytscha*), coho salmon (*O. kisutch*), pilchard (*Sardinops sagax*) hake (*Merluccius productus*) and other species in the North Pacific (King *et al.* 2001a,b). VHSV has also recently been isolated from wild and cultured Japanese flounder (*Paralichthys olivaceus*) in Asia (Takano *et al.* 2000, Isshiki *et al.* 2001b). Different isolates of VHSV in the marine environment appear to be genetically similar in particular geographic areas, rather than from their host species origin (Stone *et al.* 1997). VHS and VHSV have not been reported from Australia (Herfort and Rawlin 1999). In Western Australia, marine fish submissions to that States Department of Fisheries Animal Health Laboratory have been tested for VHSV since 1995, with all submissions to date being negative (AQIS 1999).

Many tens of thousands of tonnes of frozen mackeral (*Scomber* sp.) and pilchards (*Sardinops* sp.), imported from areas in the Northern Hemisphere where VHS is enzootic in these baitfish, have been introduced into Spencer Gulf over the last two decades as feed for cultured bluefin tuna (AQIS 1999, Gaughan 2002). These imported frozen baitfish have been implicated as a likely source of introduction of fish pathogens, including the pilchard herpes virus which was associated with two separate mass mortalities of pilchards which originated from Spencer Gulf in 1985 and 1988/99 (Harvell *et al.* 1999). The probability of VHSV being introduced into Australia as a consequence of importation of large quantities of whole, round, frozen baitfish from areas in the northern hemisphere where VHS is enzootic was assessed by AQIS as moderate to high (AQIS 1999). Because of this, Biosecurity Australia implemented interim biosecurity measures on 14 May 2002, effective immediately, which prohibited importation of whole round pilchards and mackerel into Australia from all sources other than New Zealand (AFFA 2002).

Water temperature is important in the transmission and production of VHS, with transmission and disease usually occurring in the range of 1-12°C but not above 15°C (Wolf 1988). The virus loses infectivity at temperatures above 20°C. Water temperatures in Spencer Gulf range from a minimum of 10-12°C in winter to 22-25°C in summer (Nunes and Lennon 1986). Even if VHSV was accidentally released into the South Australian marine environment (for example, via frozen imported baitfish) in the past, it is likely that it could only be transmitted to susceptible species and cause disease for a very short period of time during the winter months, and probably could not persist over the summer months. Despite the large volumes of frozen baitfish imported into Spencer Gulf in the last two decades from the Northern Hemisphere, there has been no evidence that this practice resulted in the establishment of VHSV in the Australian marine environment, possibly due to mitigating factors, such as water temperature (AQIS 1999). However there has been little in the way of regular and effective surveillance of marine fish in Spencer Gulf for VHS, hence historically there could have been an extremely low risk of exposure of kingfish broodstock in Spencer Gulf to viable VHSV during the winter months.

5.9.2 *Exposure assessment*

If cultured juvenile kingfish from South Australia were exposed to VHS at some stage, carrier fish would have to endure the stress of transport and quarantine without reverting to the diseased state (and being detected) during quarantine, before they could be released into sea cages from where the virus could enter New Zealands marine environment. After this, to be spread in the marine environment, the water temperatures at the seacage site would need to be below 15°C and the virus would then need to be transmitted at an infectious dose to other susceptible wild or cultured fish. The risks of establishment of VHS in New Zealand via importation of asymptomatic carrier fish would therefore appear negligible during the summer months and extremely low during the winter months.

5.9.3 *Consequence assessment*

The consequences of introduction of VHS with imported cultured juvenile kingfish are assessed as moderate to high. This is because once introduced, the virus may be able to persist year round in some areas of New Zealand if a suitable reservoir of susceptible wild fish was present due to the lower average water temperatures in this part of the world. There could be adverse consequences for fisheries based on wild marine fish, cultured marine fish, and also the aquaculture of salmonids and marine fish if New Zealands VHS free status could not be maintained.

5.9.4 *Risk estimation*

Australia is considered free of VHS and there has been no evidence that this virus has been introduced into Spencer Gulf via importation of large quantities of frozen baitfish from areas in the northern hemisphere where VHS is known to be enzootic. However, the risk of introducing VHS with imported baitfish was assessed by AQIS as moderate to high (AQIS 1999), and theoretically the virus could persist in the region during the winter months. As the consequences of the introduction of VHS via the imported commodity are likely to be moderate to high, adoption of a precautionary approach to managing any risks which may be involved with movements of cultured juvenile kingfish from Spencer Gulf appears prudent.

5.9.5 *Risk management*

To mitigate any potential risks of introduction of VHSV, it is recommended that VHS should be considered in the virus testing programmes detailed herein. VHS can be isolated on the BF-2 cell line (OIE 2000), hence surveillance for VHSV could be undertaken passively, as part of the routine screening process recommended for birnavirus. Therefore, kingfish from batches containing fish which have tested positive (e.g. generated a cytopathic effect (CPE) on BF-2 cell culture media) for viruses other than VER and aquatic birnavirus during routine cell culture testing, should not be exported to New Zealand and the identity of the virus(es) responsible for the CPE should be determined.

Summary

This risk assessment has determined that there may be low to extremely low risks of the following diseases being introduced into New Zealand through importation of cultured juvenile kingfish from Spencer Gulf Aquaculture:

Aquatic birnavirus
Atypical *Aeromonas salmonicida*
Streptococcal and enterococcal infections
Viral encephalopathy and retinopathy (VER)

Furthermore, in light of the limited surveillance of marine fish in Australia for novel iridoviruses and the unique situation in Spencer Gulf regarding recent imports of large quantities of frozen baitfish into the area and the risk of introduction of VHS through these, it is recommended that VHS and novel iridoviruses are also considered in the virus testing programmes detailed herein. As many iridoviruses and VHS can be isolated as primary cultures on the BF-2 cell line recommended by the OIE for detection of birnaviruses, passive surveillance for novel iridoviruses and VHS could be undertaken as part of the screening process required for birnaviruses for minimal extra cost over and above that required for birnavirus screening alone.

Recommended safeguards to mitigate risk of disease introduction

To mitigate the risk of disease introduction associated with importation of the commodity, a number of safeguards and procedures regarding the monitoring and treatment of batches of kingfish destined for export are recommended. These are:

1. That batches of kingfish destined for export are separated as early as possible from other fish reared in the hatchery and are maintained in separate tanks in areas which are physically and spatially separated from other kingfish, particularly broodstock.

2. That detailed records are kept of the mortality rates of each batch of larval/juvenile kingfish and that these data are made available to the competent authority responsible for disease certification of the fish in Australia, the importing company and MAF Biosecurity prior to disease testing in Australia.

3. That any batch of juvenile fish which experiences mortalities greater than 5%, due to unsubstantiated causes, during either the larval or juvenile stages should be classed as suspicious. Juveniles from such batches should not be exported and the cause of the higher than normal mortality rates should be determined.

4. That kingfish destined for export remain in the hatchery water supply (which is to be filtered to 1 µm and UV sterilised at all times), are fed hatchery reared or artificial food at all times and are not placed into Spencer Gulf at any time.

5. That kingfish destined for export are maintained in seawater of at least 30‰ during rearing and transport.

6. That a sample of 150 fish from each batch of kingfish destined for export is tested for VER using OIE approved techniques (cell culture on SSN-1 cell line, or PCR), and also aquatic birnavirus by OIE approved methods for detecting IPNV (cell culture on BF-2 cell line).

7. That this testing is performed by a competent laboratory in Australia approved to undertake such work by AQIS and/or AFFA.

8. That kingfish from batches containing fish which have ulcerative dermal lesions and/or ectoparasitic infections, or from batches which test positive for VER or aquatic birnavirus, should not be exported to New Zealand.

9. That kingfish from batches containing fish which have tested positive (e.g. generated a CPE on BF-2 cell culture media) for viruses other than VER and aquatic birnavirus during routine testing, should not be exported to New Zealand and the identity of the virus(es) should be determined.

10. That after health certification in Australia, the remaining kingfish in each batch are placed in a container sealed with a MAF or AQIS approved seal, so there can be no water exchange during transport.

11. That after clearing customs the kingfish are transported by road directly to an approved transitional facility at NIWA Mahanga Bay for 4 weeks quarantine. Upon reaching the quarantine facility the seal to the transport container should then be broken only by authorised MAF Quarantine officers.

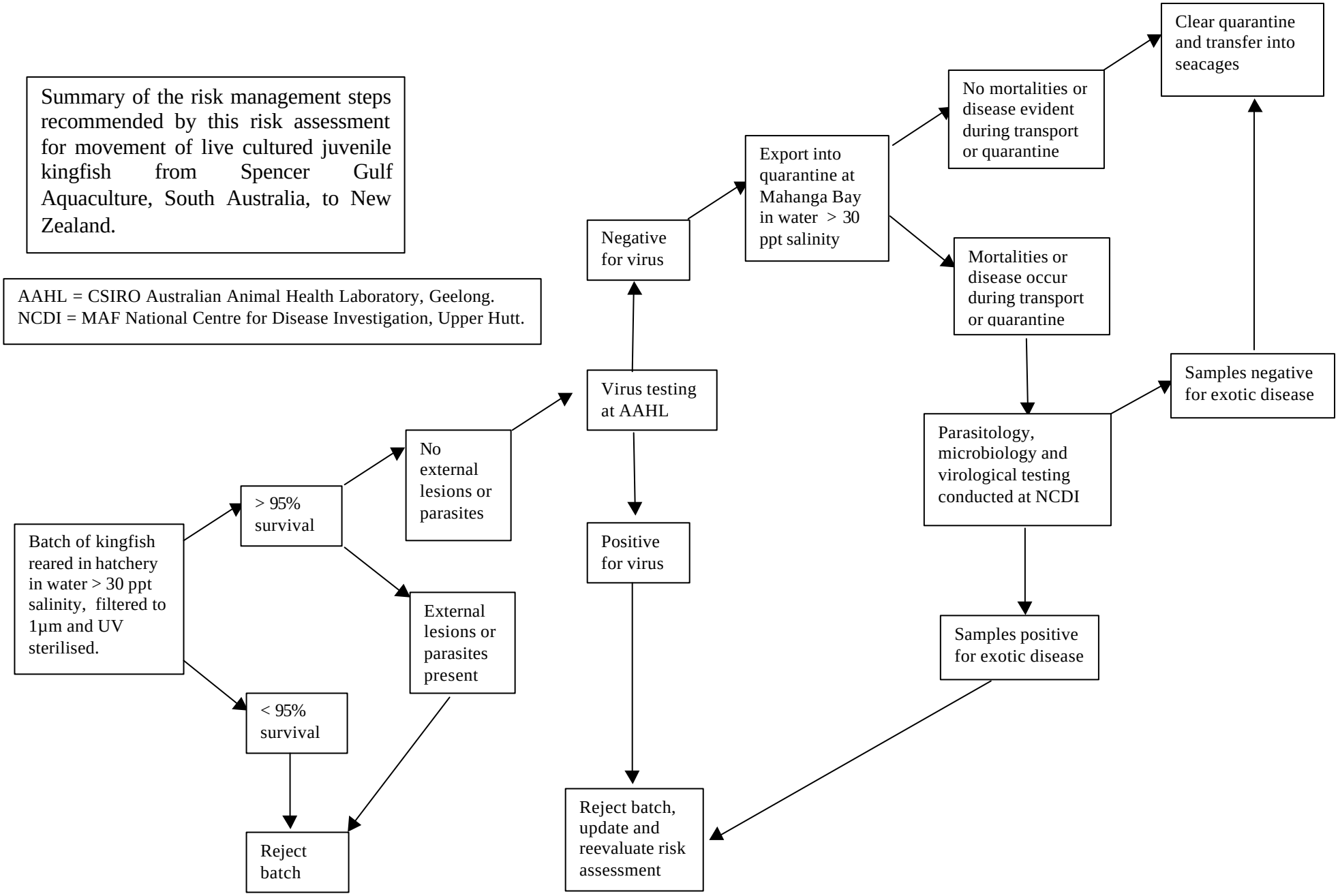
12. That quarantine should be performed as per the standards outlined in MAF Biosecurity Authority Standard 154.02.06, Transitional Facilities for Ornamental Fish and Marine Invertebrates. All wastewater discharged from the quarantine facility shall enter directly into the municipal sewerage system. New Zealand authorities will be immediately informed if a disease outbreak is detected within 4 weeks of the fish being imported into New Zealand.

13. Any mortalities which occur during transport or quarantine should be investigated by the National Centre for Disease Investigation using parasitological, microbiological and virological methods and a diagnosis obtained.

A chart summarising the various risk management steps recommended by this risk assessment is included on the following page.

Summary of the risk management steps recommended by this risk assessment for movement of live cultured juvenile kingfish from Spencer Gulf Aquaculture, South Australia, to New Zealand.

AAHL = CSIRO Australian Animal Health Laboratory, Geelong.
NCDI = MAF National Centre for Disease Investigation, Upper Hutt.



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