Import Risk Analysis on Non-viable Salmonids and Non-salmonid Marine Finfish

AQIS
AUSTRALIAN QUARANTINE AND INSPECTION SERVICE

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Foreword

THIS IMPORT RISK ANALYSIS (IRA) FOR SALMONIDS and non-salmonid marine finfish was conducted in response to findings of the World Trade Organization, in 1997, that Australia's fish quarantine policies were not based on a proper scientific risk analysis.

As an outcome of this IRA, Australia introduced new policies on the importation of non-viable salmonids and non-viable non-salmonid marine finfish. These policies were announced on 19 July 1999.

AQIS acknowledges the contribution of the independent scientists who provided advice on scientific issues and assisted with the analysis (see list on page iv). The assistance of Dr Bernoth with the writing and editing of Part 3 was particularly appreciated.

Australian Quarantine and Inspection Service
July 1999

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Executive summary

IN 1994, FORMAL BILATERAL CONSULTATIONS BEGAN between Canada and Australia on Canada's longstanding market access request for non-viable salmon. AQIS conducted an import risk analysis (IRA) on wild, ocean-caught Pacific salmon from North America and produced draft and final reports in 1995 and 1996, respectively. In the final report, AQIS concluded that the quarantine conditions that applied at that time (ie the prohibition on the importation of uncooked salmon) should be maintained, a position that was contested by Canada under the dispute settlement arrangements of the newly formed World Trade Organization (WTO). The United States also requested and held WTO consultations with Australia in 1995.

Following a request from Canada in 1997, the issue was considered by a WTO dispute settlement panel and Appellate Body. The WTO found that Australia had not complied with its obligations under the WTO Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement) with regard to the measures applying to salmon. The key findings were:

- Australia's IRA on uncooked wild-caught Pacific salmon from Canada did not fulfil all the requirements of the SPS Agreement in relation to an IRA and there was no IRA to support the restrictions on the importation of other uncooked salmon products; and
- there were arbitrary or unjustifiable distinctions in the level of protection applied by Australia in relation to salmon and other fish, and these distinctions resulted in a disguised restriction on international trade.

The WTO Arbitrator gave Australia until 6 July 1999 to address its obligations. In order to meet this deadline, and after consultation with stakeholders, AQIS adopted an accelerated approach to this and a related IRA. The policies arising from the IRAs were published on 19 July 1999 (Animal Quarantine Policy Memorandum 1999/51).[1]

1 Animal Quarantine Policy Memorandum 1999/51. Final reports of import risk analyses on non-viable salmonid products, non-viable marine finfish products and live ornamental finfish and adoption of new policies, 19 July 1999.

This report describes in detail the IRA for non-viable salmonids and non-salmonid marine finfish. It draws on information contained in the previous reports of salmon IRAs by the Australian Government and the New Zealand Government in 1994–97. AQIS has also conducted an accelerated IRA on live, ornamental finfish to address the WTO finding of inconsistency in the quarantine measures applied to live and non-viable finfish. The ornamental fish IRA is described in an accompanying report.

Consultation

AQIS took several steps to ensure the scientific validity of the risk analyses, including considering the reports of consultancies (most of which were commissioned in 1998) on identified gaps in information relating to these risk analyses (see AQPMs 1999/33 and 1999/38). AQIS also contracted 14 independent scientists (in Australia and overseas) to review one or both of the draft reports as they were being prepared and assess the completeness and accuracy of scientific information in the report and the balance and objectivity with which scientific information was treated.

AQIS did not ask the independent reviewers to advise on Australia's appropriate level of protection (ALOP), as this is the responsibility of the Australian Government, having regard to the broad range of quarantine decisions and precedents within AQIS's purview.

To ensure that the process fulfilled the Government's commitment to an open and consultative approach to IRA, AQIS held public meetings in 5 capital cities and 2 meetings of key stakeholders in Canberra. AQIS also made each chapter of the draft reports available to the public for comment by posting them on the AQIS Internet site.

In the course of the risk analyses, AQIS received 35 submissions on scientific issues on the non-viable salmonid and non-salmonid marine finfish IRA and the live ornamental finfish IRA. AQIS also received a large number of representations, most of which restated the importance of maintaining the current prohibition on importation of uncooked salmon, but presented no scientific issues requiring consideration in the risk analyses.

AQIS considered all scientific issues raised in the submissions of respondents and sought the advice of the independent scientific reviewers on significant points in the submissions. All submissions were taken into account in preparing the reports.

The scientific information reviewed in these IRA reports is comprehensive and up-to-date and the independent scientific reviewers have agreed that the scientific analysis is accurate, objective and balanced. On this basis the conclusions in the risk analyses will be incorporated (where appropriate) into legal instruments and procedures for the importation of non-viable salmonid product and non-salmonid marine finfish product and live ornamental finfish in accordance with the recommendations set out in the reports.

Scope of the risk analysis

This IRA considers the quarantine risks associated with the importation to Australia of non-viable salmonid and non-salmonid marine finfish from any source country. The IRA does not cover retorted shelf-stable fish product, live fish or their genetic material. The 'salmonids' covered by this IRA include members of two families: the family Salmonidae and the family Plecoglossidae (of which Ayu, or sweetfish, *Plecoglossus altivelis* is the only member).

The non-salmonid marine finfish covered in this IRA include all finfish species caught in marine or brackish waters, other than species defined above (salmonids). It does not include marine finfish species caught in fresh water as these fish will be the subject of a separate IRA.

The base products considered in this IRA are non-viable fish as follows:

- eviscerated salmonids; and
- whole, round (not eviscerated) non-salmonid marine finfish.

Most product of non-salmonid marine finfish imported into Australia is highly processed (eg consumer ready). However, a significant demand exists for the importation of whole, round product. Non-viable, whole, round non-salmonid marine finfish may be used for human consumption, as feed for fish, as fishing bait or for further processing (eg for pet food). To ensure

consistency in the risk assessment process, non-salmonid marine finfish are assessed from the starting point of whole, round product.

International codes

In preparing this IRA AQIS, has drawn upon principles outlined in the Office International des Epizooties (OIE, or World Organisation for Animal Heath) *International Aquatic Animal Health Code* (the Aquatic Code) and the OIE *International Animal Health Code*.

The Aquatic Code classifies aquatic animal diseases as diseases notifiable to the OIE (transmissible diseases that are important for public health and/or trade reasons); and other significant diseases (diseases that are of current or potential international significance in aquaculture but of less importance than the notifiable diseases, are less widespread, or have less well-defined aetiology).

In making recommendations on the measures that should be applied to trade in non-viable marine finfish, the Aquatic Code identifies evisceration as the recommended risk management strategy for the listed diseases. The Aquatic Code does not make recommendations in relation to unlisted diseases.

Australian quarantine policies

The *Quarantine Act 1908* and subordinate legislation, including Quarantine Proclamation 1998 (QP 1998), are the legislative basis of human, animal and plant quarantine in Australia.

AQIS's objective is to adopt quarantine policies that are, wherever appropriate, based on international standards and that provide the health safeguards required by government policy in the least trade-restrictive way.

Under the Quarantine Act, the importation into Australia of any articles likely to introduce any infectious or contagious disease, or disease or pest affecting persons, animals or plants can be prohibited under proclamation of the Governor General, generally or subject to any specified conditions or restrictions.

The disease risks associated with importations are analysed using IRA, which is a structured, transparent and science-based process that provides the scientific and technical basis for quarantine policies and determines whether an import may be permitted and, if so, the conditions to be applied.

Import risk analysis

AQIS has evaluated the risks associated with individual diseases and disease agents, and has identified measures appropriate to the risks presented by the importation of either non-viable salmonids or non-viable non-salmonid marine finfish. Based on this evaluation, risk management measures for these fish have been proposed, including the means for verifying the health certification provided by exporting countries. The IRA is 'generic' and addresses all relevant pests and diseases, to facilitate assessment of individual access requests according to the health status of the source country.

The IRAs were conducted according to the method previously set out by AQIS in its publication *The AQIS Import Risk Analysis Process: Handbook* (1998). This process, which involves the risk analysis steps of hazard identification and characterisation, risk assessment and risk management, is consistent with Australia's obligations under the SPS Agreement and relevant recommendations of the OIE.

In the light of consultations with independent scientists and risk analysts, AQIS conducted this risk analysis on a qualitative, rather than a quantitative basis. This was due to the complexity of the analysis (the large number of species and disease agents considered), the limited data on some key questions (such as the lack of data on prevalence of many pathogens) and the uncertainty about some important issues, such as the susceptibility of native species to the disease agents under consideration.

AQIS considered all relevant sources of information, including the results of relevant quantitative risk analyses, such as those conducted by the New Zealand Government in 1997 and information submitted to the WTO by the Government of Canada (unpublished). In deciding to use the qualitative approach, AQIS also took

into account the fact that this is consistent with OIE recommendations and the obligations of WTO members.

Hazard characterisation

AQIS used the following criteria to identify the disease agents of quarantine concern that required further consideration in the IRA. A disease agent was given detailed consideration in the IRA if it was assessed to be:

1. infectious; and
2. (a) exotic to Australia, **or**
   (b) present in Australia but subject to official control; **and**
3. (a) OIE listed, **and / or**
   (b) would be expected to cause significant harm in Australia.

Where there were no definitive data relevant to categorisation, AQIS made conservative judgments, drawing upon scientific knowledge and observations made in similar situations, and other appropriate information.

Once the diseases that met the above criteria had been identified, AQIS identified those requiring consideration with higher priority (which were placed in group 1) or lower priority (which were placed in group 2). The disease agents were grouped on the basis of published scientific literature, previous reports of the Australian Government and the New Zealand Government and advice of the independent scientists advising AQIS on the IRAs.

Risk assessment

Quarantine risk is composed of two related factors — the probability of the disease agent entering and becoming established in Australia, and the expected impact or significance (consequences) of such establishment. The IRA method used by AQIS addressed both these factors in a standardised manner to allow consistency in the overall approach to risk management, as follows.

- *Release assessment* — the probability that the agent will enter Australia as a consequence of the importation of eviscerated salmonids.
- *Exposure assessment* — if the disease agent entered Australia in eviscerated salmonids, the probability of susceptible fish being exposed to a dose sufficient to cause infection.
- *Probability of disease establishment* — combining the release and exposure assessments.
- *Consequence assessment* — the consequences of the disease agent establishing in Australia.

These factors were categorised for each disease of concern, using standardised criteria to obtain qualitative measures of the probability of disease establishment and the consequences. These measures were applied to a risk evaluation matrix to determine if for salmonid or non-salmonid marine finfish imports Australia's acceptable level of protection (ALOP) would be met and whether risk management measures were warranted.

Risk management measures

For salmonids, the group 1 priority disease agents that do not meet Australia's ALOP were identified as:

- infectious haematopoietic necrosis virus;
- infectious pancreatic necrosis virus — for juveniles only;
- infectious salmon anaemia virus — for Atlantic salmon only;
- *Aeromonas salmonicida*; typical and atypical strains — all salmonids except for wild ocean-caught Pacific salmon;
- *Renibacterium salmoninarum*;
- *Yersinia ruckeri* (Hagerman strain) — for juveniles only; and
- *Myxobolus cerebralis* — for rainbow trout and for juveniles of all salmonid species.

For non-salmonid marine finfish, group 1 priority disease agents that do not meet Australia's ALOP for susceptible species were identified as:

- aquabirnaviruses (other than infectious pancreatic necrosis virus);
- infectious pancreatic necrosis virus;
- viral haemorrhagic septicaemia virus;
- red sea bream iridovirus;
- *Aeromonas salmonicida* (typical and atypical strains); and
- *Photobacterium damsela piscicida*.

In the case of each disease, AQIS considered risk management measures that would be required if the importation of salmonid or non-salmonid marine finfish was to be permitted while meeting the ALOP. These measures include pre-export requirements for the country of origin and post-import measures that could be imposed in Australia

Finally, the group 2 priority diseases were assessed to ensure that with the implementation of measures required for group 1 disease agents, risks associated with the group 2 disease agents would also meet Australia's ALOP.

POLICIES FOR IMPORT OF NON-VIABLE UNCANNED SALMONIDS

Based on the above procedures, the following risk management measures will apply to the import of non-viable, uncanned salmonid finfish from any country:

- the fish must be eviscerated;
- the fish must be derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority;
- the fish must not be derived from a population slaughtered as an official disease control measure;
- the fish must not be juvenile salmonids;
- the fish must not be sexually mature adults (spawners) (not for New Zealand);
- the fish must be processed in premises approved by and under the control of a competent authority;
- the head and gills must be removed and internal and external surfaces thoroughly washed (not for New Zealand, see below);
- the fish must be subjected to an inspection and grading system supervised by a competent authority;
- the product must be free of visible lesions associated with infectious disease;
- consignments exported to Australia should be accompanied by official certification confirming that the exported fish fully meet Australia's import conditions.
- for countries in which infectious salmon anaemia (ISA) occurs,[2] Atlantic salmon should not come from a farm known or officially suspected of being affected by an outbreak of ISA.

In recognition of the health status of New Zealand, salmonids including Pacific salmon but excluding rainbow trout would be permitted import without head and gills removed. The measures outlined above apply to rainbow trout from New Zealand.

Salmonid product (other than Pacific salmon from New Zealand) meeting these policies will be released from quarantine if imported in consumer-ready form. For the purpose of these policies, the following products are considered to be 'consumer-ready':

- cutlets — including central bone and external skin but excluding fins — of less than 450g in weight;
- skinless fillets — excluding the belly flap and all bone except the pin bones, of any weight;
- skin-on fillets — excluding the belly flap and all bone except the pin bones — of less than 450g in weight;

2 As at July 1999, ISA has been reported from Scotland, Norway and Canada.

- eviscerated, headless 'pan-size' fish of less than 450g in weight; and
- product that is processed further than the stage described above.

Imported head-off, gilled and gutted salmonids of greater than 450g weight (ie, not consumer-ready) should be processed to consumer-ready form in premises approved by AQIS before release from quarantine.

In considering whether to approve commercial processing plants for processing imported salmonid products, AQIS will consider the location of the plant, the type of product processed and other factors. Commercial processing will not be permitted in regions where there are economically significant populations of salmonid fish. This will reduce the probability of susceptible fish being exposed to imported fish or derived waste.

AQIS will also require that premises approved for the further processing of imported salmonids are located to allow quarantine inspectors and auditors ready access and to facilitate regular announced and unannounced inspection. It is likely that most, if not all, approved processing plants would be located in metropolitan centres of mainland Australia.

AQIS is reviewing pre-existing policies for the importation of salmonid roe, smoked salmon and smoked trout; further advice will be provided shortly.

POLICIES FOR IMPORT OF NON-VIABLE, NON-SALMONID MARINE FINFISH

The risk management measures that will be required for import of non-viable, non-salmonid marine finfish from any country, countries other than New Zealand are as follows:

OPTION 1 (no import permit required)

- the fish must be processed in a premises approved by and under the control of a competent authority;
- the fish must be eviscerated;
- the fish must be subjected to an inspection system supervised by a competent authority;
- the head and gills must be removed and internal and external surfaces thoroughly washed;
- the product must be free from visible lesions associated with infectious disease; and
- consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

OPTION 2 (no import permit required)

- AQIS will not require an official health certificate for consumer-ready product that has been processed further than the stage described above. Such product should be packaged to facilitate import inspection.

  (For the purpose of these policies, consumer ready-product is product that is ready for the householder to cook/consume; as for salmonids, above).

OPTION 3 (import permit required)

- if neither option 1 nor option 2 applies, an importer must obtain a permit from AQIS before importing fish.
- the application for the permit should provide details of the finfish species to be imported (scientific and common names), the waters in which the fish were farmed (if applicable) and harvested and the intended end use of the imported fish.
- AQIS will assess the application in light of the quarantine risks it presents; If the delegate concludes that the proposed importation is consistent with Australia's ALOP, a permit for the importation of single or multiple consignments during a specified timeframe would ordinarily be granted.

Under these amended policies, non-salmonid marine finfish may continue to be imported into Australia. For species that are not specified, the most significant change is that importers will be required to obtain an import permit from AQIS. However, AQIS will not require an import permit for consignments of fish that are head-off, gilled, gutted, inspected and accompanied by an official health certificate or for consignments of consumer-ready product (as defined above). Imports of specified marine finfish species (including all farmed

marine finfish) will be subjected to additional controls to address risks associated with certain diseases[3].

For specified finfish species and for farmed marine finfish, AQIS will generally allow the importation of consumer-ready product and fish that are head-off, gilled, gutted, inspected and accompanied by an official health certificate. AQIS will not generally permit the importation of specified species in whole, round form for use as bait or fish feed; rather, AQIS will conduct a case-by-case assessment before deciding whether to grant a permit and under what conditions to allow such importations. For example, delegates would not permit the import of herring for use as bait under conditions which would present an unacceptable risk of the establishment of VHSV.

POLICY FOR IMPORT OF NON-VIABLE, NON-SALMONID MARINE FINFISH PRODUCT FROM NEW ZEALAND

A new condition will apply to the importation of non-viable non-salmonid marine finfish caught in or adjacent to New Zealand's Exclusive Economic Zone (EEZ) by fishers approved/registered under controls administered by a government authority of New Zealand. AQIS will not require an import permit for consignments of such fish, providing they are accompanied by official certification stating that:

- the fish, or fish from which the product was derived, were caught in New Zealand's EEZ or in adjacent international waters; and
- the consignment is product of New Zealand.

The remainder of the policies set out in this report do not apply to non-salmonid marine finfish from New Zealand.

POLICY FOR IMPORT OF ORNAMENTAL FINFISH

In the risk analysis on ornamental finfish (accompanying report), AQIS concluded that importation should continue to be permitted, subject to baseline risk management measures and some additional conditions as warranted by the risk analysis. Additional risk management measures will include official health certification; approval of exporting premises; and treatment, post-arrival quarantine detention and inspection of consignments, to address the risk posed by importation of live ornamental finfish.

3 For whole, round, commercially-harvested, market-size non-salmonid finfish, the disease agents which require specific risk management are: aquabirnaviruses, IPNV, VHSV, iridovirus of red sea bream, *A. salmonicida* and *Photobacterium damsela piscicida*. For *A. salmonicida*, risk management applies to all farmed marine finfish species but not to wild-caught non-salmonid marine finfish. For all other disease agents, risk management applies only to susceptible species (as specified in the IRA).

Part 1
Introduction

Chapter 1
Introduction

1.1 Background to import risk analysis

THIS REPORT CONTAINS THE FINDINGS OF THE Australian Quarantine and Inspection Service (AQIS) from its import risk analysis (IRA) on non-viable salmonids and non-viable non-salmonid marine finfish.

It represents the conclusion of a process that started in 1994, with formal bilateral consultations on Canada's market access request for salmon. AQIS conducted an IRA on non-viable salmon from North America and produced draft and final reports in 1995 (DPIE 1995)[1] and 1996 (DPIE 1996),[2] respectively. In the final report, AQIS concluded that the quarantine conditions that applied at that time (ie the prohibition on the importation of uncooked salmon) should be maintained.

Initially, in 1994, Canada consulted with Australia over the quarantine measures Australia applied to trade in salmon under the General Agreement on Tariffs and Trade (GATT). However, in 1995 the World Trade Organization (WTO) replaced GATT and further consultations between Canada and Australia have been under the new arrangements.

In 1997, following a request from Canada, the dispute with Canada was considered by a WTO dispute settlement panel (WTO 1998a).[3] At the request of Australia and Canada, the WTO Appellate Body considered the dispute settlement panel report (WTO 1998b).[4] In November 1998, the WTO found that Australia had not complied with its obligations under the WTO Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement) with regard to the measures applying to salmon. The key findings were:

1 DPIE (Department of Primary Industries and Energy) (May 1995), *Import Risk Analysis: Disease Risks Associated with the Importation of Uncooked, Wild, Ocean-Caught Pacific Salmon Product from the USA and Canada*, Draft, Commonwealth of Australia.

2 DPIE (Department of Primary Industries and Energy) (December 1996), *Salmon Import Risk Analysis: An Assessment by the Australian Government of Quarantine Controls of Uncooked, Wild, Adult Ocean-Caught Pacific Salmonid Product Sourced from the United States of America and Canada*, Final report, Commonwealth of Australia.

3 WTO (1998a). *Australia — Measures Affecting Importation of Salmon*. Report of the Panel, WT/DS18/R 1998, 12 June 1998.

4 WTO (1998b). *Australia — Measures Affecting Importation of Salmon*. Report of Appellate Body, WT/DS18/AB/R; 20 October 1998.

- Australia's IRA on uncooked wild-caught Pacific salmon from Canada did not fulfil all the requirements of the SPS Agreement in relation to an IRA and there was no IRA to support the restrictions on the importation of other uncooked salmon products; and there were arbitrary or unjustifiable distinctions in the level of protection applied by Australia in relation to salmon and other fish, and these distinctions resulted in a disguised restriction on international trade.
- In December 1998, Canada requested arbitration on the period within which Australia should be required to bring its measures into compliance. On 23 February 1999, the WTO Arbitrator gave Australia until 6 July 1999 to address its obligations.

In a separate dispute over Australia's quarantine restrictions on the importation of salmonid products, the United States also requested and held WTO consultations with Australia in 1995 and a panel was being established in 1999.

In March 1999, AQIS consulted stakeholders on a proposal to conduct IRAs on non-viable salmonids and non-salmonid marine finfish (the subject of the WTO findings) according to a common, accelerated timetable, to meet the WTO deadline. After due consideration of stakeholder comment, AQIS adopted the proposed accelerated approach.[5]

This report describes the IRA for non-viable salmonids and non-salmonid marine finfish, and is in four parts. Part 1 deals with the scope and background to the analysis and methods used to evaluate quarantine risk. Parts 2 and 3 contain the risk assessments for salmonids and non-salmonid marine finfish, respectively. Part 4 contains recommendations on the measures to be applied to the importation of non-viable salmonids and non-salmonid marine finfish.

The report draws on information contained in the reports of salmon IRAs conducted by the Australian Government (DPIE 1995, 1996) and the New Zealand Government[6,7] in 1994–97. Some of the information in this report is a summary of information in the previous reports.

AQIS has also conducted an accelerated IRA on live, ornamental finfish to address the WTO finding of inconsistency in the quarantine measures applied to live and non-viable finfish. This IRA is described in an accompanying report (AQIS 1999).[8]

1.2 Scope of this risk analysis

This IRA considers the quarantine risks potentially associated with the importation to Australia of non-viable salmonid and non-salmonid marine finfish from any source country. The IRA is 'generic' and addresses all relevant pests and diseases, to facilitate assessment of individual access requests according to the health status of the source country.

AQIS has evaluated the risks associated with individual diseases and disease agents, and has identified measures appropriate to the risks presented by the importation of either non-viable salmonids or non-viable non-salmonid marine finfish.[9] Based on this evaluation, risk management measures for these fish have been proposed, including the means for verifying the health certification provided by exporting countries (see Chapter 9).

The base products considered in this IRA are non-viable fish as follows:

- eviscerated salmonids; and
- whole, round (not eviscerated) non-salmonid marine finfish.

Whole, eviscerated salmonids are sold for human consumption internationally, reflecting the

5 AQIS (Australian Quarantine and Inspection Service) (30 March 1999), Animal Quarantine Policy Memorandum 1999/24; and AQIS (23 April 1999), Animal Quarantine Policy Memorandum 1999/27.

6 MacDiarmid SC (1994), The Risk of Introducing Exotic Diseases of Fish into New Zealand Through the Importation of Ocean-Caught Pacific Salmon from Canada, Ministry of Agriculture Regulatory Authority, New Zealand.

7 Stone MAB, MacDiarmid SC and Pharo HJ (1997b), Import Risk Analysis: Salmonids for Human Consumption, Ministry of Agriculture Regulatory Authority, New Zealand, 269 pages.

8 AQIS (Australian Quarantine and Inspection Service) (1999), Import Risk Assessment for Ornamental Fish, AQIS, 1999.

9 'Finfish' includes all bony fish but does not include cartilaginous fish (sharks, rays) or invertebrates.

recommendation of the Office International des Epizooties (OIE, or World Organisation for Animal Health) that there should be no health-related impediment to trade in such fish. Non-viable, whole, round non-salmonid marine finfish may be used for human consumption, as feed for fish, as fishing bait or for further processing (eg for pet food). The IRA does not cover canned or retorted shelf-stable fish product, live fish or their genetic material.

Most product of non-salmonid marine finfish imported into Australia is highly processed (eg consumer ready). However, a significant demand exists for the importation of whole, round product (Factotum 1999). To ensure consistency in the risk assessment process, non-salmonid marine finfish are assessed from the starting point of whole, round product.

1.2.1 SALMONIDS

The members of the family Salmonidae (salmonids) covered by the IRA can be taxonomically classified[10] as follows:

Superorder Protacanthopterygii

Order Salmoniformes

- **Family Salmonidae** (salmonids)
  - **Genus** *Acantholingua*
  - **Genus** *Brachymystax*
  - **Genus** *Coregonus* (whitefishes, ciscoes, vendace)
  - **Genus** *Hucho* (huchen or taimen)
  - **Genus** *Oncorhynchus* (Pacific salmon)
  - **Genus** *Parahucho*
  - **Genus** *Prosopium* (whitefishes)
  - **Genus** *Salmo* (salmon, trout)
  - **Genus** *Salvelinus* (chars)
  - **Genus** *Stenodus*
  - **Genus** *Thymallus* (grayling)

This IRA also covers Ayu or sweetfish, *Plecoglossus altivelis*, the single member of the family Plecoglossidae. In Japan a significant industry including wild-caught and aquaculture operations is based on this fish. Ayu are anadromous, like most members of the family Salmonidae, and are susceptible to infection with many of the same pathogens as the 'true salmonids'.

In this IRA, the term 'salmonid' includes all members of the family Salmonidae and *P. altivelis*. Further taxonomic details of these fish are given in Appendix 1.

1.2.2 NON-SALMONID MARINE FINFISH

This part of the IRA covers all finfish species caught in marine or brackish waters, other than species defined above. This IRA considers anadromous species such as barramundi (*Lates calcarifer*) and catadromous species such as eels (members of the family Anguillidae) that may be caught in marine waters. It does not include marine finfish species caught in fresh water as these fish will be the subject of a separate IRA.

1.3 International framework

1.3.1 WORLD TRADE ORGANIZATION

As a member of the WTO, Australia has certain rights and obligations under the WTO Agreement, including the SPS Agreement. The SPS Agreement recognises the standards, guidelines and recommendations developed by the OIE for animal health and zoonoses as the relevant international benchmark. Under the SPS Agreement, measures put in place by a country must be based on an international standard or upon a scientific risk analysis. A risk analysis must:

- identify the diseases whose entry, establishment or spread a WTO member wants to prevent within its territory, as well as the potential biological and economic consequences associated with the entry, establishment or spread of these diseases;
- evaluate the likelihood of entry, establishment or spread of these diseases, as well as the associated potential biological and economic consequences; and

10 This list was compiled from several sources including the New Zealand salmon IRA (Stone et al 1997b), the NCBI taxonomy browser (http://www.ncbi.nlm.nih.gov/htbin-post/Taxonomy/wgetorg) and Dr Peter Last (CSIRO pers. comm.).

- evaluate the likelihood of entry, establishment or spread of these diseases according to the SPS measures which might be applied.

The SPS Agreement defines 'appropriate level of sanitary or phytosanitary protection' as the level of protection deemed appropriate by the member country establishing a sanitary or phytosanitary measure to protect human, animal or plant life or health within its territory. In Australia, this is called an 'appropriate level of protection' (ALOP). The terms 'acceptable risk' and 'managed risk' are used with similar meaning. Further information on rights and obligations arising from the SPS Agreement may be found in the unpublished report National Risk Management and the SPS Agreement (Wilson and Gascoine 1999).[11] Animal Quarantine Policy Memorandum 1999/26[12] provides an explanation of ALOP and its relationship with quarantine risk management.

1.3.2 OFFICE INTERNATIONAL DES EPIZOOTIES (WORLD ORGANISATION FOR ANIMAL HEALTH)

Australia is a member of the OIE and actively contributes to the development of international animal health standards. The OIE publication relevant to this IRA is the *International Aquatic Animal Health Code* (OIE 1997a)[13] (referred to in this report as 'the Aquatic Code').

> The principal aim of the [Aquatic Code] and its companion volume, the *Diagnostic Manual for Aquatic Animal Diseases*, is to facilitate international trade in aquatic animals and aquatic animal products… by providing detailed definitions of minimum health guarantees to be required of trading partners in order to avoid the risk of spreading aquatic animal diseases' (OIE 1997a).

The Aquatic Code classifies aquatic animal diseases as follows:

> Diseases notifiable to the OIE:
>
> …transmissible diseases that are considered to be of socio-economic and/or public health importance within countries and that are significant in the international trade of aquatic animals and aquatic animal products.
>
> Other significant diseases:
>
> …diseases that are of current or potential international significance in aquaculture but have not been included in the list of diseases notifiable to the OIE, because they are less important than the notifiable diseases; or because their geographical distribution is limited, or is too wide for notification to be meaningful, or is not yet sufficiently defined; or because the aetiology of the diseases is not well enough understood; or approved diagnostic methods are not available.

The Aquatic Code states:

> International trade in aquatic animals and aquatic animal products depends on a combination of factors that should be taken into account to ensure unimpeded trade, without incurring unacceptable risks to human and aquatic animal health.
>
> An exporting country should be prepared to supply the following information to importing countries on request:
>
> 1. information on the aquatic animal health status and national aquatic animal health systems to determine whether that country is free or has free zones of disease notifiable to the OIE, including the regulations in force to maintain its free status;
> 2. regular and prompt information on the occurrence of transmissible diseases;
> 3. details of the country's ability to apply measures to control and prevent diseases notifiable to the OIE and, where appropriate, other diseases;
> 4. information on the structure of the Competent Authority and the authority that it exercises;
> 5. technical information, particularly on biological tests and vaccines used and applied in all or part of the national territory.

11 Available at http://www.aqis.gov.au/docs/qdu/riskmgmtoc.htm

12 Available at http://www.aqis.gov.au/docs/anpolicy/a99-026.htm

13 Available at http://www.oie.int/norms/a_fcode.htm

The OIE *International Animal Health Code* (1999) provides similar guidance in relation to trade in terrestrial animals and their products, including the requirements for an IRA, which are given in the OIE *International Animal Health Code*, Section 1.4. AQIS has structured the analysis along the lines set out in the latest version of Section 1.4 of the *International Animal Health Code*, a copy of which may be viewed on the internet.[14]

In making recommendations on the measures that should be applied to trade in non-viable finfish, the Aquatic Code identifies evisceration as the recommended risk management strategy for the listed diseases. The Aquatic Code does not make recommendations in relation to unlisted diseases.

The OIE-listed diseases relevant to salmonids and non-salmonid marine finfish are shown in Box 1.1.

Several finfish diseases considered significant by Australia are not currently listed by the OIE. Depending on the outcome of this analysis, Australia may recommend that the OIE give consideration to listing additional disease agents.

Box 1.1
OIE-listed diseases relevant to salmonids and non-salmonid marine finfish

Diseases notifiable to the OIE

- Epizootic haematopoietic necrosis
- Infectious haematopoietic necrosis
- *Oncorhynchus masou* virus disease
- Viral haemorrhagic septicaemia

Other significant diseases

- Viral encephalopathy and retinopathy
- Infectious pancreatic necrosis
- Infectious salmon anaemia
- Epizootic ulcerative syndrome
- Bacterial kidney disease
- Piscirickettsiosis
- Gyrodactylosis

Source: International Aquatic Health Code (OIE 1997a)

1.4 Animal quarantine policy framework

1.4.1 LEGISLATION AND CONCEPTUAL FRAMEWORK

AQIS's objective is to adopt quarantine policies that are, wherever appropriate, based on international standards and that provide the health safeguards required by government policy in the least trade-restrictive way. In developing quarantine policies, the disease risks associated with importations are analysed using IRA, a structured, transparent and science-based process.

The *Quarantine Act 1908* and subordinate legislation, including Quarantine Proclamation 1998 (QP 1998), are the legislative basis of human, animal and plant quarantine in Australia. Section 4 of the Act defines the scope of quarantine as follows:

> In this Act, Quarantine has relation to measures for the inspection, exclusion, detention, observation, segregation, isolation, protection, treatment, sanitary regulation, and disinfection of vessels, installations, persons, goods, things, animals, or plants, and having as their object the prevention of the introduction, establishment or spread of diseases or pests affecting human beings, animals, or plants.

Subsection 13(1) of the Quarantine Act provides that the Governor-General in Executive Council may, by proclamation, prohibit the importation into Australia of any articles likely to introduce any infectious or contagious disease, or disease or pest affecting persons, animals or plants. The Governor-General may apply this power of prohibition generally or subject to any specified conditions or restrictions.

For articles prohibited by proclamation, the Director of Animal and Plant Quarantine may permit entry of products on an unrestricted basis or subject to compliance with conditions, which are normally specified on a permit. An IRA provides the scientific and technical basis for quarantine policies that determine whether an import may be permitted and, if so, the conditions to be applied. In practice, specific protocols have been established for a minority of imported aquatic animal

14 Available at http://www.oie.int/norms/mcode/a_summary.htm

products; most enter under standard conditions based on decisions of long standing.

The matters to be considered when deciding whether to issue a permit are set out in section 70 of QP 1998 and include the quarantine risk, whether the imposition of conditions would be necessary to limit the quarantine risk to a level that would be acceptably low and anything else that is considered relevant. Quarantine risk means the likelihood that the importation will lead to the introduction, establishment or spread of a disease or a pest in Australia, the likelihood that harm will result (to humans, animals, plants, the environment or economic activities) and the likely extent of any such harm.

This IRA provides the basis for future consideration of applications for import permits outlined in QP 1998 in relation to the importation of non-viable salmonids and non-salmonid marine finfish. In keeping with the scope of the Quarantine Act, only the factors relevant to the evaluation of quarantine risk (ie the risk associated with the entry, establishment and spread of unwanted pests and diseases) are considered in the IRA. Questions related to the potential economic consequences of importation (other than the economic impact of a disease) are not part of AQIS's process of evaluation.

The actions of the Director of Animal and Plant Quarantine or his delegate in reaching a decision under the Quarantine Act must take into account relevant provisions of other Commonwealth legislation, including the *Endangered Species Protection Act 1992* and the *Environment Protection (Impact of Proposals) Act 1974*.

The Environment Protection (Impact of Proposals) Act and the administrative procedures under that Act require consideration of whether Commonwealth action (such as the granting of an import permit) is an action that will, or is likely to, affect the environment to a significant extent or that will have the effect of permitting or facilitating an action by another person, that will, or is likely to, result in such an effect. Decisions made by AQIS to permit the entry of animal products, made under the Quarantine Act and consistent with Australia's conservative approach to risk, are unlikely to lead to significant adverse effects on the environment. Nevertheless, AQIS would inform the Environment Minister of any intention to make a decision which is likely to result in a significant risk of harm to the environment. Furthermore, Environment Australia (EA) is given the opportunity to comment on proposals to develop new quarantine policies. In consultation with EA, AQIS is also developing guidelines to assist quarantine officers when making decisions to ensure that the likely effects on the environment are taken into account.

1.4.2 DOMESTIC POLICY ENVIRONMENT

In 1992 AQIS commissioned the then Bureau of Rural Resources, later Bureau of Resource Sciences (BRS), to conduct a major review of aquatic animal health and quarantine. The report, released in 1995, was a comprehensive examination of Australia's quarantine policies and practices regarding aquatic animals and their products (Nunn 1995). It considered the review of a consultant, Dr J D Humphrey, and identified concerns in relation to quarantine policy on importation of several aquatic species (Humphrey 1995).[15]

In 1995, the National Task Force on Imported Fish and Fish Products (NTF) was established to examine the BRS report and related issues. The NTF included representatives of relevant Commonwealth, State and Territory government agencies, commercial and recreational fishing groups, importers, aquaculturists, research organisations and environmental groups. It recommended that AQIS review aquatic animal quarantine policies and practices (Higgins 1996).[16]

In 1996, a committee chaired by Professor Nairn conducted a detailed independent review (Nairn et al 1996)[17]. Noting that the IRA process underpins Australia's quarantine policies and procedures, the Nairn

15 Humphrey JD (1995), Australian Quarantine Policies and Practices for Aquatic Animals and their Products: A Review for the Scientific Working Party on Aquatic Animal Quarantine, Bureau of Resource Sciences, Canberra.

16 Higgins RA (Chair) (1996). Report of the National Task Force on Imported Fish and Fish Products: a report into the implications arising from aquatic animal imports. Department of Primary Industries and Energy, Canberra.

17 Nairn ME, Allen PG, Inglis AR and Tanner C (1996), Australian Quarantine: A Shared Responsibility, Department of Primary Industries and Energy, Canberra.

committee identified six principles that should apply. The committee recommended that IRA should be:

- conducted in a consultative framework;
- a scientific process and therefore politically independent;
- a transparent and open process;
- consistent with both government policy and Australia's international obligations (under the SPS Agreement);
- harmonised, by taking account of international standards and guidelines; and
- subject to appeal on the process.

In its response (DPIE 1997)[18] the Australian Government accepted all recommendations of the Nairn report relevant to the IRA process. The AQIS publication *The AQIS Import Risk Analysis Process Handbook* (AQIS 1998) sets out AQIS's approach to IRA, which is consistent with Australia's obligations under the SPS Agreement and with relevant recommendations of the OIE. Copies of the handbook can be obtained from AQIS or viewed on the AQIS homepage.[19]

The Australian Government also supported most of the recommendations in the NTF report and agreed to provide additional resources to AQIS so that it could conduct major reviews of aquatic animal quarantine. A series of policy reviews is being undertaken throughout 1997–2001.[20]

1.4.3 QUARANTINE POLICY ON SALMONIDS AND NON-SALMONID MARINE FINFISH

Quarantine policy on aquatic animals, including marine finfish, has evolved over decades, in response to specific health issues. Major developments included the introduction of quarantine restrictions on oysters in the shell in the 1930s and salmonids in 1975, and the imposition of quarantine on live ornamental fish in 1983.

There are specific and quite detailed requirements for the importation of non-viable salmonid products. In general, importation is prohibited unless the Director of Animal and Plant Quarantine has issued a permit for importation. Canned fish, roe or caviar of salmonid fish are exceptions in that importation is allowed without a permit. The existing quarantine policies on the importation of salmonid products were set out in the Australian Government's IRA final report (DPIE 1996).

With the exception of salmonid fish, prior permission is generally not required to import non-viable marine finfish or their products. Compounded fish feeds and meals derived from aquatic animals (eg fish meal) require prior permission but may be imported subject to compliance with the requirements (including heat processing and inspection) set out in an AQIS import permit.

Prior permission is also required to import live salmonids and their genetic material. AQIS has not approved any such imports since 1975. Live ornamental marine finfish listed in Schedule 6 of the Wildlife Protection (Regulation of Exports and Imports) Act may be imported, subject to inspection on arrival. Other species of marine fish require prior permission, which has only been granted on a case-by-case basis for public display or scientific purposes.

QP 1998 provides details of quarantine legislation on the importation of non-viable marine finfish (including salmonids) and their products.

1.4.4 INTERSTATE QUARANTINE

While the Commonwealth Government is responsible for regulating the movement of animals and their products into and out of Australia, the State and Territory governments have primary responsibility for animal health controls within Australia. Legislation relating to resource management or animal health may be used by State and Territory government agencies to control interstate movement of aquatic animals and their products.

18 DPIE (Department of Primary Industries and Energy) (1997), Australian Quarantine: A Shared Responsibility, The Government Response, Canberra.

19 Available at http://www.aqis.gov.au/docs/anpolicy/risk.pdf

20 Available at http://www.aqis.gov.au/docs/anpolicy/a98-023.htm

Significant finfish diseases/disease agents that have a restricted or regional distribution in Australia include goldfish ulcer disease (found in New South Wales and Victoria), barramundi nodavirus (found in Queensland and the Northern Territory), epizootic haematopoietic necrosis (found in New South Wales, Victoria and South Australia), aquabirnavirus (found in Macquarie Harbour, Tasmania) atypical *Aeromonas salmonicida* (found in Tasmania) and epizootic ulcerative syndrome (found in New South Wales, the Northern Territory, Queensland and Western Australia). In some cases, State and Territory governments impose mandatory control over the movement of live fish and their genetic material within Australia to prevent the spread of these diseases. There are no mandatory controls over the movement of non-viable salmonids or non-viable non-salmonid marine finfish within Australia on account of aquatic pathogens. However, under Tasmanian legislation, salmonids harvested from farms in Macquarie Harbour must be gilled and eviscerated, and the gills and viscera disposed of by burial to prevent the spread of aquabirnavirus.

The *Commonwealth Mutual Recognition Act 1992* has the objective of reducing barriers (including requirements set out in legislation) to the free movement of goods between States and Territories. Quarantine measures enacted by State and Territory governments are exempt from the requirements of the *Commonwealth Mutual Recognition Act 1992*, provided that the measures are required to prevent the entry of diseases that are not present in that region and that would have a long-term and substantially detrimental effect on the State or Territory.

1.5 IRA method

The IRA process described in *The AQIS Import Risk Analysis Process Handbook* provides the scientific underpinning of quarantine policy and practice. QP 1998 states that the Director of Quarantine, when making a decision on whether to permit an import access request, must consider the quarantine risk and the conditions that would be necessary to reduce quarantine risk to an acceptably low level. The IRA documents relevant information for the Director of Quarantine to consider when making a decision on an import access request.

Quarantine risk is composed of two related factors — the probability of the disease agent entering and becoming established in Australia, and the expected impact or significance of such establishment. Describing and addressing both in a standardised manner aids consistency in the management of quarantine risks and consistency in the overall approach to risk management.

In the light of consultations with several independent scientists and risk analysts, AQIS conducted this risk analysis on a qualitative, rather than a quantitative basis. AQIS adopted the qualitative approach due to the complexity of the analysis (the large number of species and disease agents considered) and in recognition of the limited data on some key questions, such as the lack of data on prevalence of many pathogens, and the uncertainty about some important issues, such as the susceptibility of native species to the disease agents under consideration. It was agreed that AQIS would consider all relevant sources of information, including the results of relevant quantitative risk analyses, such as those conducted by the New Zealand Government (Stone et al 1997b) and information submitted to the WTO by the Government of Canada (unpublished). In deciding to use the qualitative approach, AQIS also took into account the fact that this is consistent with OIE recommendations and the obligations of WTO members.

General note on dealing with uncertainty and gaps in data

Many of the observations and assumptions in this risk analysis are generalisations and, as such, stakeholders may challenge them. However, AQIS contends that it is valid to generalise, provided that the nature of factors that may affect the applicability of key assumptions is understood and the implications of such factors for the analysis are properly taken into account. In the absence of definitive, quantitative data on factors relevant to quarantine risk, AQIS applies appropriately conservative professional judgment based on available scientific information and the advice of experts in relevant fields. This is a scientifically valid approach that is adopted by quarantine authorities throughout the world in the face of limited scientific data. Thus, AQIS's approach is consistent with international practice.

1.5.1 HAZARD IDENTIFICATION

AQIS will use the following criteria to identify the disease agents of quarantine concern that require further consideration in the IRA. A disease agent has been given detailed consideration in the IRA if it is:

1. infectious; **and**
2. (a) exotic to Australia, **or**
   (b) present in Australia but subject to official control; **and**
3. (a) OIE listed, **and/or**
   (b) would be expected to cause significant harm in Australia.

Further details of these criteria are shown in Box 1.2. If there are no definitive data relevant to categorisation, AQIS makes conservative judgments that draw upon scientific knowledge and observations made in similar situations and any other appropriate information.

1.5.2 PRIORITY RANKING OF DISEASES/DISEASE AGENTS

AQIS categorised the disease agents according to the criteria set out in Section 1.5.1 and then identified those requiring consideration with higher priority (which were placed in group 1) or lower priority (which were placed in group 2). The disease agents were grouped on the basis of published scientific literature, previous reports of the Australian Government and the New Zealand Government and advice of the independent scientists advising AQIS on the IRAs.

Based on this advice AQIS gave each disease a relative score (expressed as +, ++ or +++, with +++ being the highest score possible) both for the probability of it becoming established in Australia and the consequences of such establishment.

Box 1.2
Criteria for categorising disease agents

1 THE DISEASE AGENT IS INFECTIOUS

A putative disease agent must cause or be causally associated with a recognised disease and the disease must have been shown to have an infectious aetiology.

The disease agent must have been found in association with animals that are the subject of the IRA. The disease agent must be transmissible to susceptible hosts and may have been isolated. Ideally, Koch's[21] or Evans's (Thrusfield 1995)[22] postulates should be satisfied. This criterion excludes diseases of non-infectious aetiology, for example those caused by environmental (eg toxicosis), genetic or nutritional factors.

2(A) THE DISEASE AGENT IS EXOTIC TO AUSTRALIA

The disease agent is considered to be exotic if there is no report of the disease or detection of the causal agent in susceptible animals in Australia. The level of confidence that can be attributed to such a determination depends on factors such as the virulence of the organism, severity of expression of clinical disease and nature of targeted surveillance applied to the disease or disease agent in question.

Where a disease agent is present in Australia, but the strain(s) present in other countries is/are significantly more virulent, these strains will be considered in a similar manner to exotic disease agents.

continued overleaf...

21 Koch's postulates refer to the experimental evidence required to establish a relationship of causation between a microorganism and a disease. The conditions are: 1) the microorganism must be present in every case of the disease, 2) it must be isolated and cultivated in pure culture, 3) inoculation of such culture must produce the disease in susceptible animals, 4) it must be observed in, and recovered from, experimentally diseased animal.

22 Thrusfield MV (1995), *Veterinary Epidemiology*, Blackwell Scientific, Oxford (UK).

Box 1.2 (continued)
Criteria for categorising disease agents

2(B) THE DISEASE AGENT IS PRESENT IN AUSTRALIA BUT SUBJECT TO OFFICIAL CONTROL

If a disease agent or disease occurs in Australia, one or more State/Territory Government(s) must have enacted legislation and be taking action to control or eradicate the disease or disease agent. For the purpose of this process, mandatory control measures would be deemed to exist if such measures relate to products within the scope of this analysis.

3(A) THE DISEASE AGENT IS LISTED BY THE OIE (WORLD ORGANISATION FOR ANIMAL HEALTH)

The disease agent causes a notifiable or other significant disease as listed by the OIE.

3(B) THE DISEASE AGENT WOULD BE EXPECTED TO CAUSE SIGNIFICANT HARM IN AUSTRALIA

The disease agent must satisfy one or more of the following criteria:

- it would be expected to cause a distinct pathological effect in a significant proportion of an infected population;
- it would be expected to cause significant damage to the environment and/or native species;
- it would be expected to cause significant economic harm (eg increased mortality, reduced growth rates, decreased product quality, loss of market access, increased management costs).

The assessment of disease agents in the Humphrey review (1995)[23] was taken into account to identify relative importance. Agents with a score ≥21 in the Humphrey classification (ie the top 1/3 of the Humphrey scale) were placed in group 1 while those with a score <21 were placed in group 2. Then the agents in group 2 that scored ≥++ for probability or for significance of establishment were moved to group 1. The agent erythrocytic necrosis virus (ENV) was moved from group 1 to group 2 because ENV does not characteristically cause high morbidity or significant mortality overseas; hence, the impact of the disease in Australia would not be expected to be significant. Moreover, ENV occurs in many countries, but there is no evidence to suggest that it is actively spreading. *Goussia gadi* was moved from group 1 to group 2 on the basis that members of this genus occur in Australia and the probability and impact of the establishment of new species would be expected to be low.

The grouping (and therefore the priority for assessment) of pathogens affecting salmonids and non-salmonid marine finfish is set out in Table 4.1 and Table 7.1, respectively. The higher priority agents are assessed in Chapters 4 and 7, and the lower priority agents are assessed in Chapters 5 and 8.

1.5.3 RISK ASSESSMENT

Defining the probability of establishment of disease (release and exposure assessments)

The probability of a disease agent entering and becoming established in Australia depends on the factors shown in Box 1.3. Box 1.4 defines the terms used to describe the probability of such an event occurring.

23 The 'Humphrey score' is the sum of seven factors including pathogenic significance, risk of entry, international spread and possible socioeconomic consequences. The scale has a maximum of 30. AQIS consulted Dr Humphrey (one of the independent scientists assisting AQIS with the IRAs) on the application of the Humphrey review to the IRA.

Box 1.3

Factors affecting the probability of a disease agent entering and becoming established in Australia

1. The probability of the disease agent being present in the source country/region of the commodity and, if present, its prevalence.
2. The probability of the disease agent being present in an infective form in the commodity on entering Australia.
3. The probability of the disease agent in an infective form entering the aquatic environment in Australia. This depends on the processing, end-use and disposal of the commodity and the capacity of the disease agent to persist, in an infective form, in the commodity after processing/use/disposal.
4. The probability of the disease agent, having entered the aquatic environment, establishing infection in susceptible hosts, including native species in Australia. This depends on the capacity of the disease agent to survive in the aquatic environment, in an infective form, and the ease of infection of susceptible hosts and subsequent transmission of infection to others within a population.

Note: The OIE describes the factors covered by points 1 and 2 above as the *release assessment* and those covered by 3 and 4 above as the *exposure assessment*. These factors may be evaluated in terms of the probability of key events occurring. The descriptive terms used in this IRA (low, negligible etc) are defined below with a view to clarifying the description of probability in risk analyses.

Box 1.4

Terms used to describe the probability of an event occurring

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 occur rarely
Extremely low:	Event would occur very rarely
Negligible:	Chance of event occurring is so small that it can be ignored in practical terms

Note: These categories are not equidistant from each other; most fall into the range 0<probability<50%.

Defining the consequences of establishment of disease (consequence assessment)

The establishment of a new disease agent may have a biological effect and consequential effects on industry (eg the affected fishery) and the environment. These consequences can be measured in quantitative terms (in relation to their economic impact) and in qualitative terms (in relation to their impact on society and the environment). It is generally the case that the effects of a disease can be ameliorated to various degrees by the adoption of methods for control or eradication — although these measures are associated with costs that must be included in estimates of economic, social and environmental impact.

The biological effect of the establishment of disease is normally evaluated in terms of morbidity and mortality data. In this risk analysis, AQIS took into account the standard epidemiological approach to classification of morbidity and mortality rates. For example, a high mortality rate could be defined as one that is more than two standard deviations (SD) greater than the expected mortality rate for that population over a short period (less than one month) or a rate that is more than 1 SD greater than the expected mortality rate for that

population over the entire production cycle. A high morbidity rate could be defined as one that reduces production (however this is defined) below the normal range by more than 2 SD, over the whole production cycle. As there are limited data on how the establishment of exotic diseases in Australia would affect Australian fish, it is not possible to estimate the biological effect of diseases in such quantitative terms. Accordingly, AQIS evaluated the likely consequences of the establishment of disease by taking into account the effect of the disease agent on commercially significant and non-significant species overseas and the scientific advice of independent experts.

In considering the biological effect of the establishment of disease, AQIS also takes into account direct costs associated with controlling or eradicating the disease, including the pre-emptive destruction of in-contact healthy fish and the effect on productivity in subsequent generations.

The economic effect of the establishment of disease is normally evaluated in terms of the costs arising from the biological effects and the commercial implications for domestic and international marketing of affected animals and their products (which may extend to unaffected animals and products subject to trade restrictions). AQIS does not take into account the economic effects of trade competition when considering the risks associated with importation.

The establishment of disease may also affect the environment in ways that are not readily amenable to evaluation in economic terms. There may be effects that reduce the social amenity of the environment (eg recreational fishing and enjoyment of the ecosystem) or result in environmental harm (eg by reducing biodiversity or upsetting the ecological balance). For example, the ecological balance and/or the quality of the environment could be disturbed by changes to the normal proportions of different native species as a result of the establishment of disease. These effects cannot be quantified in a meaningful way. However, any event that would cause a decline in the number of endangered or threatened species or otherwise damage the environment would be of concern to the Australian community.

In this IRA, the impact or significance of the establishment of disease in Australia is classified into one of five categories, described as catastrophic, high, moderate, low or negligible. The key factors in classifying the significance of a disease are shown in Box 1.5.

Box 1.5

Key factors in classifying the significance of disease

1. The biological effects on aquatic species.
2. The availability, cost and effectiveness of methods for control/eradication.
3. The economic effects at an enterprise/industry/national level, including effects on marketing of the product.
4. The effects on native species and the environment, including any loss of social amenity.

Terms used to describe consequences

The categories defined in Box 1.6 lie within a continuous range of consequences and are indicative of the expected outcomes.

In the face of uncertainty and some data gaps, AQIS makes conservative judgments regarding the expected impact or significance of disease establishment.

Unrestricted estimate of risk (risk evaluation matrix)

AQIS has developed a risk evaluation matrix with the objective of providing a standardised process for evaluating quarantine risk, before and after the implementation of risk management measures. For each disease agent, the combination of probability and consequence (ie risk) can be represented by a cell in the matrix (see Figure 1.1).

The risk determined on the basis of 'no risk management' is the *unrestricted estimate of risk*. If this is in line with Australia's ALOP, the risk would be

considered acceptable without specific management ('yes' in the risk matrix on page 14) and the importation would be permitted.

However, if the unrestricted risk exceeds the ALOP ('no' in Figure 1.1), the next step is to consider whether or how risk management measures may be applied to reduce the quarantine risk to the point where it conforms with Australia's ALOP. If the application of risk management measures cannot reduce the risk to an acceptably low level, the importation would not be permitted. If after applying risk management measures the risk was in line with Australia's ALOP, the risk would be considered manageable ('yes' in the risk matrix below) and the importation would be permitted. It should be noted that, where the probability of establishment of a disease is *negligible*, importation would be permitted regardless of consequences.

Box 1.6
Terms used to describe the severity of the impact (level of significance)

Catastrophic: associated with the establishment of diseases that would be expected to significantly harm economic performance at a national level. Alternatively, or in addition, they may cause serious, irreversible harm to the environment.

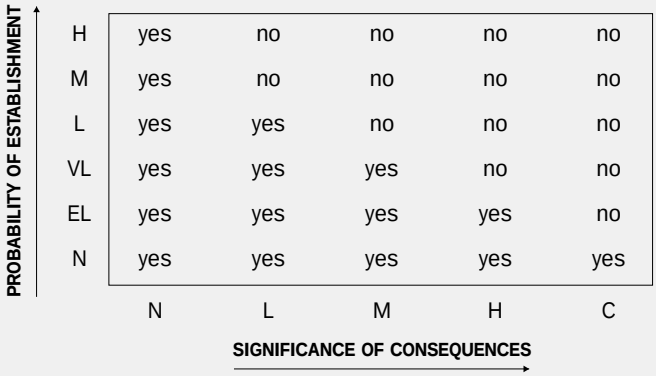
High: associated with the establishment of diseases that would have serious biological consequences (eg high mortality or high morbidity and causing significant pathological changes in affected animals). Such effects would normally be felt for a prolonged period (greater than or equal to a normal production cycle) and would not be amenable to control or eradication. These diseases would be expected to significantly harm economic performance at an industry level. Alternatively or in addition, they may cause serious harm to the environment.

Moderate: associated with the establishment of diseases that have less pronounced biological consequences. These diseases may harm economic performance significantly at an enterprise/regional level, but they would not have a significant economic effect at the 'whole industry' level. These diseases may be amenable to control or eradication at a significant cost, or their effects may be temporary. They may affect the environment, but such harm would not be serious or may be reversible.

Low: associated with the establishment of diseases that have mild biological consequences and would normally be amenable to control or eradication. Such diseases would be expected to harm economic performance at the enterprise or regional level but to have negligible significance at the industry level. Effects on the environment would be minor or, if more pronounced, would be temporary.

Negligible: associated with the establishment of diseases that have no significant biological consequences, may be transient and/or are readily amenable to control or eradication. The economic effects would be expected to be low to moderate at an individual enterprise level and insignificant at a regional level. Effects on the environment would be negligible.

Figure 1.1
Risk evaluation matrix



'Yes' = the risk is acceptable and importation can be permitted.
'No' = the risk is unacceptable and importation cannot be permitted without further risk management.
Level of probability: H=high, M=moderate, L=low, VL=very low, EL=extremely low, N=negligible.
Level of significance: C=catastrophic, H=high, M=moderate, L=low, N=negligible.

Source: AQIS (prepared for this IRA, 1999).

1.6 Release assessment

1.6.1 INTRODUCTION

As discussed in Section 1.5.3 (Box 1.3), in order to construct a scenario whereby a disease might be introduced into and become established in a country, probability factors for the entry into and establishment of a disease in the country must be considered. The factors considered in this IRA are as follows (modified from the OIE Aquatic Code).

1. The probability of the disease agent being present in fish in the waters of origin.
2. The probability of the disease agent being present in the particular fish harvested.
3. The probability of infected or contaminated fish/product passing inspection or grading.
4. The probability of the disease agent surviving processing, transport or storage.
5. The probability of the disease agent being present in the particular tissues imported.

This section discusses the factors relating to the source country and the commodity (eviscerated salmonid fish or whole non-salmonid marine finfish) that together constitute the *release assessment*.

Section 1.7 covers the factors relating to the exposure of susceptible host species in Australia to imported product that may contain infectious organisms (the *exposure assessment*).

Chapters 4 and 7 contain a discussion of all these factors with reference to individual disease agents.

The discussion in this report builds on certain fundamental observations and assumptions that have been presented and discussed in previous Australian Government reports (DPIE 1995, 1996) and in the New Zealand Government report (Stone et al 1997b) on the quarantine risks associated with the importation of salmonid fish.

Previous Australian Government reports (DPIE 1995, 1996) considered the importation of wild, ocean-caught Pacific salmon from Canada and the United States and were therefore narrower in scope than this IRA. They contained extensive reviews of the literature on salmonid diseases, including information on these diseases in non-salmonid finfish. Most of the data available on disease in wild fish originate from observations made during the freshwater part of the life cycle. Moreover,

data on Pacific salmon infections in fresh water generally involve senile fish in hatcheries that have been in fresh water from a few weeks to several months. Their immune system is compromised and the fish are susceptible to infection (DPIE 1996, p 37). However, information presented in DPIE (1996) covered a full range of data on the disease agents affecting salmonid and some non-salmonid fish and the different lifecycle stages of salmon, although these did not form part of the IRA at that time.

As noted by the WTO panel (WTO 1998a),[24] scientific data used in the release assessment for the report prepared by the New Zealand Government (Stone et al 1997b) is also relevant to this IRA in so far as the conditions specified for New Zealand also apply to Australia. AQIS has therefore taken into account information in the New Zealand report, particularly the sections on release and exposure assessment. Where information from previous Australian reports and the New Zealand report has been considered in reaching conclusions, a statement to this effect appears in the text. In most cases, additional information has been taken into account in reaching conclusions on aspects of the release and exposure assessments.

1.6.2 THE PROBABILITY OF THE DISEASE AGENT BEING PRESENT IN FISH IN THE WATERS OF ORIGIN

This IRA has been conducted on a generic basis; thus the prevalence of infectious disease in all potential source countries is considered.

The prevalence (and expression) of infection in aquatic animal populations varies markedly from one country or region to another. For example, according to DPIE (1996), *Piscirickettsia salmonis* occurs in the Pacific rim of North America but is not associated with the severe disease reported in salmonids in South America. *Enterocytozoon salmonis* occurs in Pacific, but not Atlantic, salmon on the Pacific rim of North America, but causes severe disease in South American Atlantic salmon (DPIE 1996). As scientific knowledge increases, and particularly if specific scientific investigations are conducted, information on the prevalence and distribution of disease may change considerably.

The standard of surveillance and reporting of disease in aquatic animals varies from country to country. More information on disease status may be available for countries or regions in which surveillance and reporting are high priorities and are well supported by government and the private sector. Disease may appear to be more prevalent in such countries or regions than in those that do not apply similar effort to surveillance and reporting.

In its submission to the New Zealand Government on its IRA report (Stone et al 1997b), AQIS noted that there is a need for an adequate, documented and ongoing program to investigate the cause of substantial disease incidents in the exporting country that are of concern to the importing country. However, New Zealand has taken the view that where disease surveillance programs are not in place 'it is logical for risk analysis to start with the assumption that the specified disease may be present ("suspected but not confirmed" in OIE terms)' (McVicar 1998). This is consistent with the broad thrust of AQIS's submission to New Zealand and the approach taken in this risk analysis in relation to disease agents such as infectious pancreatic necrosis virus, infectious haematopoietic necrosis virus, *Renibacterium salmoninarum* and *Aeromonas salmonicida*, which have a widespread distribution. However, for disease agents that have a limited, well-defined distribution that has changed little over several years, such as *Gyrodactylus salaris*, *Microsporidium takedai* and *Oncorhynchus masou virus*, AQIS assumes that the agents are only present in those countries in relation to which there are relevant scientific reports or other specific evidence.

The extent and nature of surveillance also affects the speed with which disease epizootics and the emergence of previously unrecorded diseases are detected and reported. As noted in the New Zealand report (McVicar 1998):

24 WTO (1998a); paragraph 6.55.

> OIE lists are recognised as the known diseases of international significance and countries are required to inform OIE of new episodes and new diseases of significance. Ideally, all countries involved in trade would have programmes in place to enable early warnings to be passed to OIE, but this may be considered to be unrealistic. It is not always possible to predict the emergence of new diseases…

An example of the problem is the failure of Canada to diagnose definitively or report the presence of infectious salmon anaemia, which the OIE classified as an 'other significant' disease, to the OIE for some 16 months after the emergence of haemorrhagic kidney syndrome in that country. AQIS acknowledges that there may be initial confusion regarding the identity of a new pathogen, particularly when the pathological or epidemiological presentation is unusual. In this regard, AQIS notes that, despite best scientific efforts, it took almost 12 months to characterise the aquabirnavirus found in Tasmanian waters in 1998 as not being the highly pathogenic strain. In this instance, there being no unusual mortalities, Australia was under no obligation to report to the OIE, yet placed a note on ProMed and informed OIE via the Annual Report. Nonetheless, countries exporting fish product to Australia should report significant disease events promptly and should address reporting requirements in the course of government-to-government negotiations on the certification of fish exported to Australia.

In considering 'minimum requirements' for disease surveillance by exporting countries, Australia has an obligation to consider the principles of equivalence and national treatment in the SPS Agreement. In this regard, it is relevant that most of the baseline information on aquatic disease in Australia is focused on the results of fish health surveillance in Tasmania (see Appendix 6). Aquatic disease surveillance is much less intensive and comprehensive in other Australian States and Territories and for non-salmonid marine finfish. It would be inconsistent with our international obligations if Australia were to require countries to conduct significantly more intensive national surveillance to demonstrate the absence of specified diseases than that deemed sufficient to support Australia's claims to freedom from the same diseases (all other technical issues being equal).

In discussing the requirement for a documented health surveillance program, McVicar (1998) stated:

> …the concept of such surveillance programmes for specified fish diseases…is central to most international (and national) disease control legislation when the risk of transfer of infection is particularly high as with live fish and eggs.

Even for OIE-listed diseases, ongoing disease surveillance is not always necessary. McVicar (1998) notes:

> …such requirements are usually put into place where a commodity carries a particularly high risk from a specified disease and there is limited opportunity to manage that risk to acceptably low levels. For example, the lack of surveillance for the parasite *Gyrodactylus salaris* in many European countries does not prevent the import to approved disease-free areas of non viable fish on which the probability of persistence of the parasite is low. However, for live susceptible hosts, where the risk is particularly high, trade is only permitted from these areas where there is an agreed surveillance program in place demonstrating the continued absence of the parasite.

AQIS agrees with the broad thrust of this statement. AQIS recognises the wide variation in the effectiveness of surveillance of aquatic disease by exporting countries and takes this into account in considering quarantine risks and risk management options. A lack of surveillance for a particular disease would not necessarily demand a ban on importing non-viable fish or fish product, if the risk could be reduced to meet Australia's appropriate level of protection.

The competent authority is defined in the OIE *International Aquatic Animal Health Code* (OIE 1997a) as the National Veterinary Services or other authority of a member country having the responsibility and competence for ensuring or supervising the implementation of the aquatic animal health measures recommended in the code. As an importing country, Australia has the right to evaluate the competent authorities of exporting countries as part of a risk assessment process to determine the measures to be applied to trade in aquatic animals or their products.

AQIS's knowledge of and confidence in information on the health status of fish populations is much greater for those countries that have a competent authority recognised by AQIS than for those that do not. For the countries that have a recognised authority, AQIS would normally accept statements regarding the presence or absence in that country of disease agents considered in this risk analysis.

The effectiveness and timeliness of surveillance will be an important consideration where risk management measures are to be based on the regionalisation of fish diseases. McVicar (1998) states:

> …it is normally recognised that either a prolonged period of intensive testing is required to achieve zone/area disease-free status or that the water supply and farm stocks are protected from outside disease contacts.

This statement is in general agreement with AQIS's view that recognition of an exporting country's competent authority, and detailed understanding of the health of fish populations based on the implementation of ongoing surveillance and monitoring, is required to underpin a claim of disease regionalisation. If disease regionalisation is to be the basis of risk management, such understandings are normally developed through bilateral negotiations between the exporting and the importing country. AQIS would normally preface consideration of a regionalisation proposal on formal recognition of the competent authority and the system for fish health surveillance and monitoring.

In conclusion, AQIS considers the distribution and prevalence of disease in this risk analysis. For those countries that have a competent authority recognised by AQIS, information on the distribution (and, where available, the prevalence) of disease would normally be accepted by AQIS. For those countries that do not have a competent authority or whose authority has not been recognised by AQIS, it is assumed that widely distributed diseases are present, unless a submission is provided to the contrary. However, for disease agents with a limited, well-defined distribution that has changed little over several years, AQIS assumes that the agents are only present in those countries in relation to which there are relevant scientific reports or other specific evidence. The lack of definitive data and the wide variation in the effectiveness of surveillance and reporting around the world means that AQIS cannot recognise subnational regionalisation of diseases, until such time as an exporting country provides advice regarding its competent authority and the system of fish health surveillance and monitoring that underpins a specific claim.

AQIS interprets information on the occurrence, prevalence and expression of disease conservatively, taking into account each country's system of surveillance and monitoring and its record of reporting significant disease events to the OIE. In considering the apparent absence of diseases in countries or regions, AQIS takes into account geographic and temporal trends that suggest that a pathogen has the capability to spread. In the case of serious diseases that have significantly increased their geographic distribution within a short time-frame (such as infectious salmon anaemia), AQIS takes an appropriately conservative approach to risk assessment and management.

1.6.3 THE PROBABILITY OF THE DISEASE AGENT BEING PRESENT IN THE PARTICULAR FISH HARVESTED

Multiple biotic and abiotic factors affect the prevalence of disease in a fish population and the expression of disease in individual fish. Information on factors that affect the prevalence of disease in harvested fish is considered in the IRA under the general categories listed below. Detailed information on factors affecting the prevalence of individual disease agents is in Chapters 4 and 7.

Lifecycle stage

The prevalence of disease varies with the lifecycle stage of the host. For example, in the case of salmonids, juveniles and/or sexually mature fish may display a higher prevalence of infection with pathogens such as infectious haematopoietic necrosis virus and infectious pancreatic necrosis virus than market-size salmonids. Other pathogens may display a predilection for infection of, or clinical expression of disease in, particular lifecycle stages of the host. For example the Australian pilchard mortality event in 1995, thought to be causally associated with a herpes virus, primarily affected adult

fish greater than 10 centimetres long.[25] Significant numbers of dead juvenile fish were not observed at any stage in this extensive mortality event.

The high rate of mortality in adult pilchards in the Australian mortality event of 1995 is unusual in that, for most diseases, commercially harvested adult marine finfish have a lower prevalence of clinical infection than young fish. However, the incidence of subclinical infection may be higher. Subclinically infected fish may harbour pathogenic organisms in their tissues, but the titre of organisms would usually be much lower than in cases of clinical infection. In subclinically infected fish, pathogenic organisms are not usually found throughout the body; rather they are commonly found in particular organs such as the kidney (*Renibacterium salmoninarum*), cartilaginous tissues (*Myxobolus cerebralis*) or intestines (*Yersinia ruckeri*) and may be present at higher titres in particular organs.

Adult fish that have returned to fresh water to spawn may have a higher prevalence of infection and a higher titre of infectious agent for certain pathogens, such as *Renibacterium salmoninarum* and infectious haematopoietic necrosis virus. Previous AQIS reports and the report of the New Zealand Government (Stone et al 1997b) on salmon provide many examples supporting the proposition that there would be a low prevalence of clinical disease in commercially harvested market-size salmonid fish, but prevalence is higher in sexually mature fish. The New Zealand report (Stone et al 1997b) states that many data on the prevalence of infection in mature Pacific salmon were gathered from fish that had already returned from the sea to fresh water to spawn. The data also reflect infections that were acquired by the fish following their return to fresh water. Thus, the reported prevalences probably exaggerate the true prevalence of these pathogens for market-size fish at sea.

Origin of fish

The origin (ie aquaculture or wild fishery) of fish also has a bearing on the expected prevalence of infection. McVicar (1998) stated:

> …it is generally accepted that the level of disease which may be present in aquaculture is likely to be higher than in wild fish. Also, it is in only a few exceptional cases that epizootics of acute disease have been detected in wild marine fish populations (as sick animals are usually removed by predation) but as most aquacultural operations use open waters, they are likely to share diseases with local wild fish populations and so reflect the health status of the local area.

The tendency to observe (hence detect and report) and manage the health of these farmed fish more closely provides information on the status of these fish. McVicar (1998), quoting K H Amos, Department of Fish and Wildlife, Washington (US) stated that 'aquaculture products could be considered (to be safe) for import on the basis of there normally being better knowledge of which diseases are present.'

In addition to the general lack of surveillance for disease in wild fish, the accuracy of information on the prevalence of disease agents is further confounded by uncertainty as to the extent to which populations commingle or overlap within geographic regions (such as the coastal waters of continents). In species that have migratory patterns of behaviour, disease agents may be more widely distributed than in fish populations that remain within well-circumscribed waters. However, for most species there is little information on the extent to which fish populations overlap and intermingle. In the pilchard mortality event in Australia in 1995, the rapid spread of the kill through the Australian pilchard fishery may have been due to fish movements, the activities of predators or other causes.

25 While it has been hypothesised that the pilchard herpes virus entered Australia via imported pilchards, this has not been proven. The original source of the virus has not been determined.

Local dispersal of disease agent

The presence of a disease agent in certain parts of a fishery does not mean that fish within the entire geographical range of that fishery would necessarily be exposed to or become infected with the agent. For fisheries that are geographically extensive, local environmental conditions may vary greatly and will have an effect on the ease of transmission of the disease agent and its capacity to persist in regional populations. For example, viral haemorrhagic septicaemia virus (VHSV) causes disease in herring and cod in north Pacific waters. The New Zealand report (Stone et al 1997b), quoting Wolf (1988), noted that transmission of and disease due to VHSV occurs at a water temperature range of 1–12°C but not above 15°C. Pilchards are known to be susceptible to infection with VHSV. Under normal circumstances, large populations of pilchards are not found in colder northern waters; rather they occur in the warmer southern waters off North America. Thus, VHSV is not considered to be endemic in these pilchards. However, under exceptional circumstances, as occurred in Canada in November 1998–March 1999, pilchards were present at an unusually high population density in a system of bays, and a serious mortality event was associated with VHSV infection.

Seasonality

Season, or time of year, can also affect the prevalence of disease. For example, DPIE (1996) report notes 'furunculosis has long been regarded as a disease mainly occurring at relatively high water temperatures. Clinical outbreaks are often precipitated by an increase in water temperature' (p 137). The report further notes 'furunculosis occurs in chinook and coho in the summer months, usually at temperatures above 10°C in fresh water. There is no clinical disease in returning broodstock except in very hot summers' (p 135). In the United Kingdom, the seasonality of bacterial kidney disease is so well recognised that the code of practice for the removal of infection from a fish farm requires only spring and autumn sampling (A McVicar pers. comm.).

Most of the diseases have a seasonal character due to the heavy influence of water temperature on the course of disease. This is very much the case for proliferative kidney disease, viral haemorrhagic septicaemia and infectious salmon anaemia, the agents of which are at undetectable levels when the water temperature is much above or below the limit for occurrence of the clinical disease. During certain times of the year the risk of introducing these agents will be substantially less (B Hill pers. comm.).

1.6.4 THE PROBABILITY OF INFECTED OR CONTAMINATED FISH/PRODUCT PASSING INSPECTION OR GRADING

Fish may be treated and/or inspected by industry or government employees before sale to domestic users or export. Fish that are used for fish feed, pet food or as bait are not normally inspected. The processing of such fish would normally be limited to freezing in blocks or individually soon after harvest and shipment direct from the catching vessel.

Fish for human consumption are normally inspected by industry or government employees to verify that they are fit for human consumption. Inspectors conduct an organoleptic assessment (touch, smell, visual), which allows abnormal fish (eg those affected by generalised disease or visible lesions, or that are damaged, for example by seal bites) to be identified and rejected, or diverted for further processing. Fish product that is downgraded for aesthetic reasons may be further processed, often by cooking, to ensure consumer acceptance.

AQIS acknowledges that processing lines may operate at high speed, so there is little time for detailed inspection. However, under normal commercial arrangements, inspection and grading decisions are made at multiple points along the processing line. Employees are trained to detect fish/product that does not meet specified criteria, which are usually simple and clear-cut (eg absence of visible lesions, cleanliness of the interior of the fish). Thus, inspection and grading can contribute to the reduction of quarantine risk overall.

Fish processing plants in many countries, including Canada, the United States and New Zealand, are required by government regulation to use systems based on hazard analysis critical control point (HACCP) to control their operations. HACCP systems are based on the monitoring of key (critical control) points in the

process to verify whether the system is under control, and taking action to correct deficiencies (and reprocess non-complying product) on detection. This system has largely replaced the traditional approach, which relied on inspection of the end-product for compliance with product safety and quality parameters. It provides a structured system of control over key processes, such as operational hygiene and refrigeration, that minimises problems caused by biofilms and bacterial replication in fish product (see Section 1.6.5).

HACCP systems emphasise not only the detection but also the prevention of undesirable practices (such as cross-contamination between cooked and raw product) that may be relevant to the IRA.

Government inspection agencies normally supervise the implementation of HACCP-based food processing systems by audit, which demands that the operator maintain complete and accurate records of monitoring results. This may support certification that the importing country's requirements have been satisfied and should also provide for traceback of product if required. However, the utility of records depends on the information that is stored. As inspection based on HACCP tends to focus on the protection of public health, much of the information collected may be of little significance from an animal quarantine perspective.

Higher-value fish, such as salmonids and tuna, are normally graded as to quality. Fish are graded according to commercial criteria that vary between plants, regions and countries. The grading criteria usually include size, species, flesh colour and appearance. Undersized fish, sexually mature adult fish (in the case of salmonids) and fish with external lesions (eg due to trauma) would be downgraded from 'first quality' and, depending on commercial policy, may be sold only on the domestic market or further processed, including by cooking.

In summary, inspection and grading of fish and fish product would provide for the detection and removal from the human food chain of fish affected by generalised disease and visible lesions associated with infectious diseases. Covertly infected fish would not be detected by inspection and grading. Fish that are rejected for aesthetic reasons (eg external damage, blood spots) but are considered to be fit for human consumption would normally remain in the human food chain but would be further processed, including by cooking. Information on the inspection and grading systems of the United States, Canada and New Zealand is provided in Appendix 2, 3 and 4.

1.6.5 THE PROBABILITY OF THE DISEASE AGENT SURVIVING PROCESSING, TRANSPORT OR STORAGE

The factors relevant to the persistence of disease agents in the course of processing, transport and storage include factors intrinsic to the agent (that allow it to persist in an infective form) and the conditions of processing, transport and storage.

Processing of fish may include the following steps: evisceration, removal of the head, washing of internal and external surfaces, removal of the skin, filleting, and preservation processes that may include freezing or chilling, cooking, smoking or pickling.

Processing and preservation treatments may significantly reduce the titre of pathogens that may be present in fish.

Evisceration of the fish and removal of the head and gills remove a large proportion of the pathogens associated with the bone, cartilage and retrobulbar tissues of the head. This would reduce the titre of pathogens such as *Myxobolus cerebralis*, which are associated with cartilaginous structures. Deheading also removes the brain, which may contain a significant titre of pathogens such as infectious haematopoeitic necrosis virus and *Renibacterium salmoninarum* (B Munday pers. comm.). Removal of the skin and filleting (ie removal of the bones) would be expected to reduce the titre of organisms that are preferentially located on or in these parts of the fish.

Washing

Washing would be expected to reduce the titre of organisms located on the skin and those associated with residual visceral tissues and blood remaining in the carcase after evisceration. Many factory trawlers wash fish in seawater, which has some virucidal and bactericidal properties (B Jones pers. comm., quoting Yamamoto et al 1982). It should be noted that washing alone would not remove any residue of the kidney remaining on the backbone and ribs after evisceration.

However, scrubbing or high pressure water sprays can be used to substantially remove residual kidney tissues.

Washing would be expected to reduce the amount of mucus on the surface of the fish, to remove blood, faeces or other contaminants from the product surface and to kill organisms that are sensitive to the levels of chlorine present in the wash water. The removal of mucus reduces the load of pathogens such as *Aeromonas salmonicida* and infectious salmon anaemia virus; however, some pathogens would be expected to remain in association with residual mucus on the skin.

Chlorine is effective in inactivating some pathogens, including infectious haematopoietic necrosis virus and *Renibacterium salmoninarum*. However, it is less effective when there are high levels of organic material (Stone et al 1997b). In most developed countries, health authorities require the use of potable water in land-based food processing plants, which normally means that the water would contain a minimum residual level of 0.2 to 0.5 milligrams per litre of free chlorine. This concentration of chlorine would not have a lethal effect on many viruses but would have a lethal effect on some bacteria and viruses on the surface of the fish's tissues.

Biofilms[26]

Chlorine can be less effective if microorganisms are embedded in biofilms, which occur on many surfaces in aquatic environments. Microorganisms embedded in biofilms are better protected from lethal conditions than those free in water or sediment. Thus, pathogens in biofilms can survive treatment by chlorine in wastewater treatment facilities (Ford 1993). In particular, there has been concern about *A. salmonicida* persisting in biofilms in the same way as *Aeromonas hydrophila* can. However, AQIS is unaware of evidence of *A. salmonicida* persistence in biofilms or its ability to multiply under such conditions; unlike the ubiquitous and free-living *A. hydrophila*, *A. salmonicida* is associated with its fish hosts and its persistence in the environment is related to heavy contamination from outbreaks of furunculosis (DPIE 1996).

AQIS has concluded that the possible presence of biofilms in processing plants would have negligible quarantine significance. This takes into account advice provided by BRS in 1996 (DPIE 1996, p 365):

The bacteria under consideration[27] are unlikely to be associated with biofilms because they cannot compete with other microorganisms under the conditions present — and if they could exist in biofilms they were unlikely to be present because of the type of fish processed.

BRS further advised:

The growth characteristics of many of the bacteria in this study do not lend themselves to establishment of biofilms…[on processing machinery due to] …their unlikely presence in marine fish …and their slow growth characteristics.

This IRA considers a wider range of bacteria than did BRS, and some may be more suited to survival in biofilms, but significant bacterial pathogens are not as well adapted as environmental organisms to life outside the host and are not expected to feature significantly in biofilms. Additionally, if processing plants do not adequately control biofilms, product quality problems, such as excessive product spoilage and reduced shelf life, will become apparent well before fish pathogens build up to a significant problem.

Cold storage

Fish are normally transported and stored in a frozen state. More valuable fish and fish product may be chilled and packed in a vacuum or a modified atmosphere pack. Under commercial conditions, fish are typically frozen at a temperature lower than -18°C or chilled at a temperature of 0°C to 7°C (ADVS 1999).[28] Frozen fish are usually transported by sea container, taking weeks or months depending on shipping schedules (DPIE 1996).

26 Biofilms are thin films of bacteria that are difficult to remove

27 The bacteria under consideration in the 1996 IRA were *Aeromonas salmonicida, Edwardsiella tarda, Piscirickettsia salmonis, Renibacterium salmoninarum, Vibrio anguillarum, V. ordalii, V. salmonicida* and *Yersinia ruckeri*.

28 ADVS (Aquaculture Development and Veterinary Services) (1999), *Consultancy on Routes for Exposure of Aquatic Animals to Aquatic Animal Products Intended for Human Consumption*, A report prepared for AQIS by ADVS, Allens Rivulet, Tasmania, 222 pp.

Frozen product is usually stored as head off, gilled and eviscerated, glazed and frozen fish (AQIS 1996). Fresh product is stored at temperatures below 4°C and must reach the consumer within a few days (DPIE 1996).

ADVS (1999) reviewed the effect of several food processing techniques, including cooking, cold preservation, brining, salting, smoking, fermentation and marination, on the persistence of aquatic animal pathogens. Chilling and freezing generally reduce the rate of inactivation of microorganisms (ADVS 1999); however, storage at freezing temperature kills many food-borne pathogenic protozoa, cestodes and nematodes (Kim 1997). Furthermore, most viruses persist at chill temperatures for hours to days and are quite stable at freezing temperatures (ADVS 1999), while bacteria that are pathogenic or potentially pathogenic to aquatic species are inactivated to some degree by chilled or frozen storage (ADVS 1999). AQIS notes that laboratories commonly freeze samples in order to ensure the preservation of viruses. However, under laboratory conditions frozen storage is normally at very low temperatures (-70°C or lower) and a specially prepared high protein solution is used to protect the viability of viruses.

A freeze–thaw cycle would be expected to decrease titres of agents such as *Aeromonas salmonicida* and infectious haematopoietic necrosis virus (DPIE 1996). The persistence of some pathogens, such as *Piscirickettsia salmonis* and *Enterocytozoon salmonis*, would be adversely affected by freezing. Other pathogens, such as infectious pancreatic necrosis virus, could persist in frozen fish for long periods.

A McVicar (pers. comm.) noted that 'many of the survival figures quoted in the ADVS report are not necessarily extremes but often represent the longest survival which has been observed in limited studies'.

Viable but non-culturable

Results of survival studies of vibrios and other organisms at temperatures of less than -15°C require careful interpretation because of the 'viable but non-culturable' (VBNC) phenomenon (ADVS 1999). Further, the data reported for vibrio inactivation in food products may represent not inactivation *per se* but, rather, the inability to culture any remaining pathogens on normal culture media (ADVS 1999).

Many bacteria, especially the enteric bacteria and vibrios, enter the VBNC state under adverse physical conditions (ADVS 1999). This phenomenon could occur with some salmonid pathogens (eg *A. salmonicida* and *R. salmoninarum*) where surveys of fish during the seawater phase of the life cycle demonstrate few, if any pathogens, but where serious disease outbreaks can occur when the fish return to fresh water for spawning (ADVS 1999). These authors noted that it is difficult to distinguish between recrudescence of covert infection and reinfection of the fish when they return to fresh water. AQIS has previously noted that the existence and significance of VBNC forms of *A. salmonicida* are very contentious issues and that there is still debate over their infectivity for salmon (DPIE 1996, p 39). AQIS has taken into account the quarantine implications of the possibility that the survival of *A. salmonicida* could be significantly extended if it entered a 'dormant' phase (DPIE 1996, p 138).

Heating

Of the processes that seafood products are likely to be subjected to during normal processing, only thorough cooking (canning, hot smoking, pasteurisation) and processes that lead to shelf-stable products offer a high level of probability of inactivation of any aquatic animal pathogens present (ADVS 1999). Most aquatic pathogens would be inactivated at typical cooking temperatures (those resulting in an internal temperature of 55–70°C, the surface temperature often being significantly higher). Some organisms (eg birnaviruses) are relatively resistant to inactivation by thermal treatment, but even for the more resistant pathogens a significant proportion of the population is likely to be inactivated if food is held at an internal temperature of 55–70°C for several minutes.

Multiplication during storage

In considering the effect of refrigeration on microorganisms present in or on food, it is important to note that viruses, metazoans and most protozoal pathogens do not multiply in the tissues of a dead host. There are some exceptions; for example, the protistan *Ichthyophonus* sp undergoes extensive proliferation by subdivision after death of the host, to the extent that infected marine fish may become inedible (A McVicar pers. comm.).

Most information on the multiplication of pathogens in foods relates to bacteria such as *Listeria monocytogenes* and *Salmonella* spp that have public health significance. The ability of *A. salmonicida* to multiply in muscle tissue postmortem is unknown (DPIE 1996). The quarantine significance of bacterial replication in non-viable fish is unclear, as commensal organisms and environmental bacteria are likely to multiply much more rapidly and would effectively overgrow any aquatic pathogens present in the tissues. However, the proliferation of infective agents under certain conditions during processing, storage and transport of the product is an important element that must be considered (A McVicar pers. comm.).

1.6.6 THE PROBABILITY OF THE DISEASE AGENT BEING PRESENT IN THE PARTICULAR TISSUES IMPORTED

Infectious agents display characteristic tissue preferences that are largely determined by the mode of infection and pathogenic characteristics of the agent in a particular host species. The nature and distribution of host cellular receptors largely determine the tissue tropisms of viruses and other intracellular agents. Some agents are highly specific, while others use cell surface receptors that occur on many tissues of the body (or at many lifecycle stages of the host).

This risk analysis is limited to non-viable fish (whole, eviscerated salmonids and whole, round non-salmonid marine finfish) (see Section 1.2). Evisceration removes many pathogens, including metazoan endoparasites of the gastrointestinal tract and pathogens that are largely confined to the blood-rich visceral organs.

For diseases such as bacterial kidney disease, proliferative kidney disease and infectious pancreatic necrosis, evisceration would therefore reduce (but may not eliminate) the probability of pathogens being present in product. However, some blood-rich organs, such as remnants of the anterior kidney and the retrobulbar tissues of the head, would remain in the eviscerated carcase and certain pathogens are preferentially located at these sites.

Other pathogens prefer different or additional sites in the body. *Myxobolus cerebralis*, which causes whirling disease of trout, infests cartilaginous tissues of young fish, in which it can cause high mortality. It is mainly found in cartilaginous tissues, especially of the head. Older fish are more resistant to infection and show little clinical effect. In adult salmonid fish, the removal of the head, fins and tail would remove nearly all the remaining cartilaginous tissues, but residual *M. cerebralis* spores could be found in bone. Evisceration would have little effect on the load of spores in the fish because the spores are not localised in the viscera.

Some pathogens are distributed throughout the tissues of the body (eg infectious salmon anaemia virus and *Enterocytozoon salmonis*, which infect red blood cells). Moreover, systemic infection with many different pathogenic organisms may result in bacteraemia or viraemia, with the infectious agent occurring at high titre throughout the body. In such cases, the removal of blood-rich organs would reduce the load of pathogens present, but a significant proportion can remain in somatic muscle.

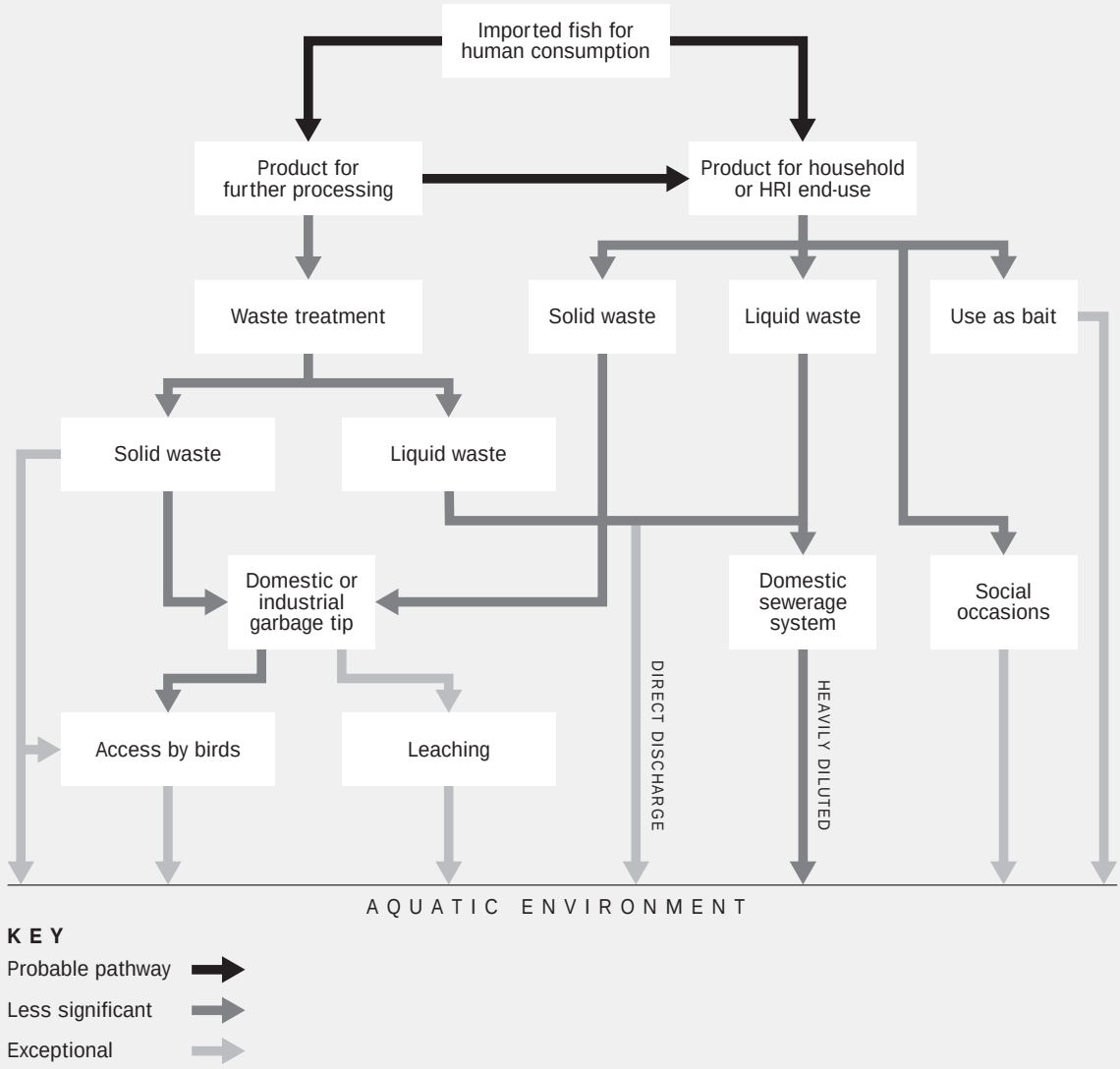
Fish affected by generalised disease are usually visibly abnormal, showing signs such as discolouration, exophthalmos, skin lesions and external haemorrhage. These signs would be detected during inspection and grading, and the fish excluded from human consumption. In the case of agents that are associated with the blood, the practice of bleeding out fish at slaughter would reduce the load of pathogens remaining in the fish. However, this practice is generally limited to farmed, higher value fish, such as Atlantic salmon and tuna.

1.7 Exposure assessment

1.7.1 INTRODUCTION

This section discusses the quarantine implications of exposure of susceptible aquatic animals in Australia to imported non-viable finfish and their products. Live fish are not considered in this risk analysis. An understanding of these matters is central to the IRA, because the exposure of susceptible fish to imported product (and infectious organisms that may be present in such product) is a major determinant of quarantine risk. In considering the quarantine significance of the various pathways of exposure, AQIS takes into account the probability with which product would be expected to follow a particular pathway and other factors relevant to the quarantine significance of such exposure. The pathways relevant to product imported for human consumption are discussed in the ADVS (1999) report to AQIS and are shown in Figure 1.2.

Figure 1.2
Pathways followed by product imported for human consumption



Source: prepared by AQIS

In constructing a scenario whereby a disease might be introduced into and become established in a country, the following factors must be considered in relation to the exposure assessment (modified from the OIE Aquatic Code).

1. The probability of imported product entering the aquatic environment.
2. The probability that imported product entering the aquatic environment contains a disease agent at a dose[29] sufficient to infect susceptible hosts in the importing country, and the probability that the agent initiates infection (an index case).
3. The probability of disease spreading from the index case and becoming established in host populations in the importing country.

Some pathways occur commonly (eg product imported for human consumption would commonly be consumed by the human population) while others occur uncommonly, rarely or exceptionally. AQIS takes into account the extremely low probability of imported product following rare or exceptional pathways in considering their quarantine significance. This is consistent with Australia's international obligations. The WTO requires the risk evaluated in a risk assessment to be an ascertainable risk; it is necessary to look at the probability of particular consequences, not just the possibility.[30,31]

The probability and nature of exposure of susceptible species to imported product are important factors in assessing quarantine risk; others include the likelihood of pathogens being present in the product, the titre and condition (infectivity) of such organisms, and the minimum infective dose required to initiate an index case of infection. AQIS has taken into account relevant data that are available.

For most agents, the infectious dose cannot be quantified and it is only possible to conclude that the minimum infective dose is likely to be high or low, relative to the range of disease agents under consideration.

The first case of disease in a susceptible population is the index case (in practice, the index case in an individual fish in a susceptible population would be expected to pass unrecognised). The IRA is concerned with the establishment of new diseases in a population. Thus, the risk analysis considers not only the probability of an index case occurring but also the probability of infection being transmitted from the index case to other aquatic animals, resulting in the establishment of disease in the population. Disease transmission is causally associated with many factors relating to the epidemiology of the disease and the characteristics of the disease agent that enable it to persist in a form that is infectious to other hosts in the aquatic environment.

1.7.2 RELATIVE PROBABILITIES OF IMPORTED PRODUCT ENTERING THE AQUATIC ENVIRONMENT: LIVE FISH COMPARED WITH NON-VIABLE FISH AND FISH PRODUCTS

Importing live fish and their gonadal material presents greater quarantine risk than importing non-viable finfish and their products, because infectious agents are more likely to persist in live fish. Furthermore, live fish are introduced into an aquatic environment (albeit in a closed system, such as an aquarium) where the pathogen may multiply and spread to fish of a similar kind.

Non-viable products imported for human consumption would not generally be introduced into the aquatic environment, so the opportunity for transmission of infectious pathogens would be greatly reduced. However, imported non-viable fish or products that are used for fishing bait, or for feeding to farmed fish, would enter the aquatic environment. For certain pathogens, the quarantine risks associated with this practice may be at least as high as those associated with the importing of live fish and gonadal products.

The primary role of live fish and their gonadal material in disseminating infectious organisms is generally recognised, and is reflected in the emphasis the OIE Aquatic Code places on measures to ensure the health

29 The infectious dose of a particular agent for a particular host will vary with the strain of the agent, the route of infection, the environmental conditions and the host factors.

30 WTO (1998a); paragraphs 6.37 et seq.

31 WTO (1998b); paragraph 127 et seq.

of traded live fish and their products. The code sets out measures such as health certification, inspection and quarantine (pre-export or post-arrival); testing for specific diseases may be applied to manage any risks associated with trade in live fish and gonadal products.

In the course of evolution, pathogens develop mechanisms that facilitate long-term persistence in a live, infected host. Such mechanisms include the establishment of subclinical infection, latency and mechanisms to avoid or frustrate the host's immune response. Most pathogens (including viral, metazoan and most protozoan agents) do not replicate or persist long term in the tissues of dead fish. Some bacteria may replicate in non-viable product, but these are usually fast growing and non-fastidious in their culture requirements (eg *Pseudomonas* spp and the Enterobacteriaceae). Bacteria also grow most readily on non-viable product at ambient temperatures, to a lesser extent in chilled product and not at all in frozen product. Growth in product at colder temperatures is normally much more significant for commensal bacteria than for bacteria that are primary pathogens of aquatic animals, such as *Aeromonas salmonicida* and *Renibacterium salmoninarum*.

Similarly, pathogenic bacteria may in theory replicate in the aquatic environment; however, they must compete for nutrients with commensal and environmental organisms, many of which have developed mechanisms that enable them to successfully compete for nutrients with the microorganisms that normally obtain these inputs from their hosts. Pathogenic bacteria such as *A. salmonicida*, *Y. ruckeri* and certain *Vibrio* spp can persist in the aquatic environment in the absence of infected hosts. The capacity for such organisms to establish free-living populations in the environment or lower-order hosts is relevant to the discussion of exposure pathways and is considered in detail under individual disease agents.

AQIS's import risk analysis on live ornamental fish (AQIS 1999) contains detailed information on the quarantine issues associated with their importation into Australia.

The rest of this section deals with the quarantine issues relating to the entry of imported non-viable finfish into the aquatic environment.

1.7.3 PROBABILITY OF IMPORTED PRODUCT ENTERING THE AQUATIC ENVIRONMENT

In this section, the use and disposal of non-viable fish imports and their products is assessed in terms of the probability of their entry into the aquatic environment.

Probable pathways

(a) Human consumption

This section addresses the quarantine risks associated with human consumption of salmonids and marine finfish and their products.

Non-viable fish and product imported for human consumption will generally be consumed, in cooked or raw form, by people in households, hotels, restaurants or institutions. Most potential exotic fish pathogens are unlikely to survive food processing, the gastrointestinal tract and effluent treatment processes, and are therefore unlikely to reach the aquatic environment. Only the most resistant organisms are likely to persist in an infective form through these routes. However, uncooked or unprocessed products that bypass these routes are of greater concern (DPIE 1996; ADVS 1999).

The risk analysis must therefore take into account what parts of product are discarded, and in what form. In preparing salmonid fish and the larger non-salmonid finfish for human consumption, the head, skin and tail would normally be trimmed and discarded raw. Moreover, the fish may be filleted before cooking, leaving the frame to be discarded. Whole salmon may be cooked in hotels or restaurants but Australian householders are much more likely to cook smaller ('pan-size') fish such as rainbow trout and consumer-ready portions (such as salmon cutlets or steaks) whole, discarding any waste, such as the head, bones or skin, after cooking.

32 WTO WT/DS18/R 1998, 12 June 1998. Australia — measures affecting importation of salmon: report of the panel, paragraph 6.40.

Cooking procedures typically result in the internal temperature of the food reaching 55–70°C, with significantly higher temperatures at the surface. The internal temperature is usually held for seconds to minutes. Cooking is the most reliable method of killing parasites contaminating animal tissues, and normal, thorough cooking temperatures and times are usually sufficient. Thorough cooking (canning, hot smoking, pasteurisation) gives a high certainty of inactivation of aquatic animal pathogens (ADVS 1999).

However, numerous studies have reported species that are pathogenic to aquatic species both on raw and processed seafood product at the point of retail sale. Pathogens of marine finfish that are relatively resistant to heating include infectious pancreatic necrosis virus, *Mycobacterium cheloni* and *Renibacterium salmoninarum*. The detection of *Vibrio* spp on cooked product may be due to thermal resistance or post-processing contamination, but either way this suggests a route for exposure of aquatic animals to pathogens via cooked product (ADVS 1999).

However, AQIS considers that consumer preparation of finfish by cooking would be expected to inactivate most organisms of quarantine concern, including most viruses, vegetative bacteria, protozoa and metazoa. Some viruses (such as infectious pancreatic necrosis virus) and spore stages of bacteria and protozoa may be relatively resistant to thermal inactivation. The DPIE (1996) report noted that heat treatment does not affect bacterial endospores but, as none of the bacteria reviewed in the IRA produce endospores, heat-treated material (such as to 70°C) poses a lower risk than fresh or frozen tissue, as many of the disease agents discussed are heat-labile (DPIE 1996, p 344). AQIS considers that cooking, whether followed by human consumption or not, would greatly reduce the probability of pathogens entering the aquatic environment.

Most marine finfish are cooked before being eaten, but some (especially salmon, tuna and herring) are consumed raw. According to DPIE (1996), up to 30% of salmon may be eaten raw as sashimi or cold smoked salmon. However, an expert advising the WTO commented that this was probably an incorrect assumption.[32] There is no definitive information on this point.

The question of how much fish is consumed raw by the human population is only relevant to the risk analysis if such consumption would result in an increased probability of pathogens entering the aquatic environment by this route. For reasons set out below, AQIS considers that this makes no significant difference to the probability.

Most infectious organisms will be destroyed in the human gastrointestinal tract and it is unlikely that passage through the human gut will increase their number (ADVS 1999). However, there might be exceptions to this finding, such as *Aeromonas hydrophila*, which may multiply and be present in large numbers in wastewater containing human faeces (ADVS 1999). Infectious pancreatic necrosis virus is also an aquatic pathogen that could survive passage through the mammalian intestine (cattle); consequently, use of processing wastes in animal feeds may present risks unless the feed has been further processed to inactivate the agent (A McVicar pers. comm.).

AQIS has taken into account the extremely low proportion of imported marine finfish (including salmonids and non-salmonid finfish) consumed raw. Moreover, in Australia as a whole, human faecal wastes are in almost all cases disposed of via the domestic sewerage system, where any aquatic animal pathogens would be a negligible proportion of the total liquid waste (see discussion below). AQIS considers that the incidence of pathogens entering the aquatic environment via human consumption of imported salmonids or marine finfish would be extremely low.

(b) Waste disposal pathways — wastewater

ADVS (1999) considered in detail the processes used for waste disposal in Australia and reached the following conclusions on wastewater:

> Primary treatment essentially removes particulate materials and microorganisms attached to them… providing a 1 log removal of bacteria and substantially lower removals of viruses and protozoan parasites. Primary effluent may be discharged at ocean outfalls, provided adequate dilution is achieved… Due to the high organic

content of this effluent, many aquatic species often frequent primary ocean outfall discharge points.

Secondary wastewater treatment reduces the BOD and suspended solids loadings by about 90%. Pathogen numbers can be reduced by up to 2 log orders…Virus removal mostly results due to the association of viruses with particulate matter. While secondary treatment removes most particles >100 mm, most viruses remain associated with particles <100 mm in the effluent. Some secondary effluents are discharged into the ocean without further treatment, whereas discharge to rivers usually includes [sic] additional treatment in the form of nutrient reduction and disinfection. Where secondary effluent is discharged to the ocean fewer aquatic species congregate due to its lower organic content. The sludge from secondary treatment can still maintain a high number of pathogens.

Tertiary treatment further reduces suspended solids. While tertiary treatment also further reduces pathogen numbers, it will not eliminate pathogens. In particular, many protozoa can survive outside their host in an encysted form for weeks/months.

ADVS (1999) advised that wastewater effluent was reused and that sludge was further treated before use (eg for landfill). Although some aquatic pathogens may survive in sludge, the strict guidelines applied to reuse schemes would reduce the quarantine significance of these pathways. However, the discharge of undisinfected effluent into a waterway could be important, particularly if primary effluent or raw sewage were discharged where there were congregations of aquatic species. Wastewater derived from imported seafood product was likely to be considerably diluted by wastewater from other sources (ADVS 1999).

AQIS notes that the processing of wastewater in the domestic sewerage system would not completely inactivate any aquatic animal pathogens present in imported product. However, the physical conditions in the sewerage system, including the presence of chlorine and other chemicals inimical to the survival of microorganisms, and competition from other microorganisms for nutrients would be expected to limit the survival of many of the aquatic pathogens considered in this risk analysis. AQIS has also considered how the dilution of wastewater from imported product by wastewater from other sources would affect the concentration of pathogens entering the aquatic environment.

Table 1.1 shows the volume of wastewater discharged to the ocean (average dry weather flow) through three major sewerage treatment plants (STPs) in Sydney in 1998.

Table 1.1
Wastewater treatment through sewage treatment plants in Sydney, 1998

	WASTEWATER PRODUCTION	
SEWERAGE TREATMENT PLANTS	MEGALITRES/DAY	MEGALITRES/YEAR
Cronulla STP	52	18,720
Malabar STP	480	172,800
Bondi STP	130	46,800
Total	**662**	**238,320**

Source: G Allen, Australian Water Technologies (AWT).

Hypothetical scenario

In considering the quarantine significance of wastewater associated with imported product, AQIS has examined a hypothetical scenario based on the importing of non-viable salmonids for human consumption. Salmonids could carry more aquatic pathogens of quarantine significance than other marine finfish species covered by this risk analysis. In considering this scenario, AQIS made the following conservative assumptions:

- **A total of up to 5000 tonnes of eviscerated whole salmonids would be imported annually.** This is based on a domestic consumption of Atlantic salmon in Australia of about 4400 tonnes per year, excluding imported smoked salmon (ADVS 1999). As imports would in reality only partially displace the domestic product, this assumption is at the upper limit of what could occur.

- **This volume of imported product (as whole eviscerated fish) would generate approximately 3000 tonnes of waste product.** This is based on advice in ADVS (1999) that the edible portion of most aquatic animals comprises <50% of their weight. Again this assumption is very high, given that the starting point of the risk analysis is eviscerated fish. If imported product was eviscerated, waste would be much less than 50% of the total weight of imported product.
- **Processing of 5000 tonnes of imported salmonid product would generate approximately 10 megalitres of wastewater.** This assumes that 2 litres of water are required to process each kilogram of product. This estimate is high, based on commercial advice to AQIS that less than 2 litres per kilogram is used in fish processing.
- **All imported product would be processed and consumed in Sydney in the October to December quarter, to supply Christmas demand.** This is a conservative assumption for the Sydney area as imported product would in reality be distributed throughout Australia in a pattern largely determined by the distribution of consumers. Thus, the volume of product consumed and processed in Sydney would be less than the total imported into Australia. Although the peak period of demand may coincide with the Christmas holidays, product would probably be imported all year round.

  In practice, the proportion of imported product directed to other parts of Australia would be expected to be far less than that processed and/or consumed in Sydney, given the concentration of importers, food processors and distributors, restaurants and tourism ventures in that city. Thus, although smaller population centres would produce less liquid effluent, the lower volume of product processed and consumed means that the dilution factor would be large.

In this scenario, wastewater associated with imported salmonid product would be less than 0.02% of the wastewater discharged to the ocean through these three STPs. This equates to a 4 log dilution in the concentration of pathogens, if any were present, in addition to reduction in concentration due to the effect of physical conditions. The use of these assumptions provides for an extremely conservative assessment; in practice the dilution factor would be several orders of magnitude higher. Thus, the processing of effluent through the domestic sewerage system would provide for substantial dilution of wastewater associated with imported aquatic product.

Another way of considering the dilution factor is to compare the daily processing of imported product through a processing plant with the production of wastewater through a single STP. Assuming that a plant processing 10 tonnes of head off, gilled and gutted fish in a day would produce 20 kilolitres of wastewater, this would represent 0.04% of the daily wastewater discharge from the Cronulla STP to the ocean.

Moreover, the processing of wastewater, even if limited to primary level processing, would reduce the load of bacterial pathogens, if present, by up to 90% (for viruses, this reduction would be less). Processing to secondary or tertiary level would reduce the concentration of microorganisms by 99% or more (for viruses, this reduction would be less). AQIS has taken these findings into account in concluding that the treatment and dilution of wastewater in the domestic sewerage system would reduce the concentration of pathogens (if present) entering marine waters by several orders of magnitude.

Availability of susceptible species

As indicated by ADVS (1999), the density of susceptible species in the vicinity of sewerage outflows is also relevant to the quarantine risk presented by the discharge of commercially treated wastewater. In considering the discharge of wastewater off the coastline of mainland Australia, it is relevant that any pathogens present would potentially come into contact with various marine finfish species. However, significant populations of salmonids occur mainly (but not exclusively) in the fresh waters and marine waters of Tasmania, and in fresh water in Victoria (Lake Eildon and neighbouring shires) and the highlands of New South Wales. For

diseases that are relatively host-specific, there would be minimal probability of susceptible finfish consuming scraps containing infectious organisms, as non-susceptible aquatic species (including invertebrates, cartilaginous fish and mammals) would compete with the susceptible species for the nutrients.

For example, bream, whiting, flathead and yellowtail scad are common in the marine waters of New South Wales. These species may be susceptible to infection by introduced pathogens that are not highly host-specific, such as infectious pancreatic necrosis virus and VHSV. These disease agents could cause an index case of disease if susceptible hosts consumed material containing a high titre of infectious organisms. However, the considerable dilution of effluent containing imported aquatic product and the effect of physical factors on the condition and infectivity of these pathogens would reduce to an extremely low level the probability that an index case of disease would occur.

Notwithstanding this finding, AQIS notes that the siting of fish processing plants in the vicinity of susceptible hosts, such as occur in a fish farming area or in streams containing populations of wild trout, may present a disease risk unless the disposal of waste and effluent water is rigorously controlled (A McVicar pers. comm.).

Significant populations of salmonids are found in the marine waters in and around Hobart. Salmonids in Macquarie Harbour would have a greater probability of exposure than those in other waters, because Macquarie Harbour contains several salmonid sea farms and a large run of brown trout in proximity to many restaurants and other sources of seafood (B Munday pers. comm.). The discharge of treated wastewater into Tasmanian waters containing significant salmonid populations may pose a higher risk than such discharge into the marine waters of mainland Australia.

Significant populations of salmonids are also found in freshwater environments, particularly in Tasmania, Victoria and the New South Wales highlands. The discharge of effluent into fresh water is usually controlled by local authorities, who normally require processing to a secondary or tertiary level to protect public health and the environment. Such processing would reduce the concentration of pathogens entering freshwater systems by several orders of magnitude. However, the siting of fish processing plants in the vicinity of fresh water containing significant trout populations (eg trout-smoking premises on the Goulburn River) would present a particular risk factor.

(c) Waste disposal pathways — solid waste

In its report to AQIS, ADVS (1999) considered in detail the processes used for waste disposal in Australia and reported that most solid wastes result from seafood processing (eg frames, gills, guts or shells) or from wastewater treatment processes. If this material is moved to properly designed and controlled sanitary landfills,[33] the risk of pathogens entering the aquatic environment is extremely low but in other cases the risks may be more significant.

ADVS (1999) discussed the pathways that imported product may follow and the physical processes used in commercial disposal systems for solid waste. It also described the consequences for pathogens present in imported product. The report concluded:

- Where costs for collection… and disposal are low, … illegal dumping into waterways or areas not designated for solids waste collection (would) be lower.
- Poorly maintained facilities can lead to wide variation in reuse water or solids quality and this could lead to impacts on groundwater and receiving water quality.
- Effective and reliable control of leachate must be employed to ensure limited impacts on groundwaters and receiving surface waters.
- Burial of putrescible wastes must be performed as soon as possible and in accordance with best management practices to ensure minimal exposure

33 In the context of the ADVS report, a properly designed and controlled sanitary landfill is taken to be one that is designed and constructed to contain putrescible materials, such as fish processing waste, and is managed to prevent accidental leakage to the aquatic environment and to minimise the opportunity for scavengers, including birds, to remove material from the site.

of contaminated materials with vectors such as seabirds and rodents.

- Non-sludge wastes are generally higher in solids content and as such are more readily accepted by landfill management authorities. Nevertheless these materials… require special burial in dedicated landfill cells… engineered to provide full containment or… with leachate control mechanisms for reduction of groundwater contamination.
- Stivers (1998) [C. Stivers, Parametrix, pers. comm.] found that in well-managed landfills, risk potential…lies essentially with chemical and nutrient species contamination.
- Some pathogens may degrade during storage and transportation to landfill sites. Increased storage time may result in limited viral attenuation; these organisms require a living host to replicate, hence replication opportunities are not likely. Many may nevertheless remain viable in stable storage conditions.
- The risk to humans resulting from exposure to microbial pathogens of faecal origin deposited in designed and adequately operated sanitary landfills is below that currently considered to be acceptable …a minimum 4 log (>99.99%) reduction/inactivation in enteric viruses during treatment of surface waters for human consumption.
- Beneficial reuse schemes are sometimes employed whereby the waste is generally mixed with (material)…such as sewage sludges and composted under aerobic conditions. The process is exothermic and the resulting temperatures… (may be) 60°–70°C. These temperatures are usually adequate for significant pathogen reduction; however, poor maintenance of the process may mean that some portions of the pile are not exposed for sufficiently long periods of time to ensure pathogen destruction.
- It is not always the case that seafood waste will enter traditional waste streams.

AQIS notes that processing solid waste in the commercial waste management system would not completely inactivate any aquatic animal pathogens present in imported product. However, the extent to which waste derived from imported product would be diluted by waste from other sources (household or commercial) is an important consideration.

The production of solid waste in Melbourne in 1998 is shown in Table 1.2.

Table 1.2
Production of solid waste in Melbourne in 1998

	VOLUME OF WASTE	
TYPE OF WASTE	MEGATONNES/YEAR	TONNES/DAY
Municipal	1.189	3257
Other	2.297	6293
Total	**3.486**	**9550**

Source: EcoRecycle, Victoria.

In the hypothetical scenario discussed above for wastewater disposal, AQIS conservatively assumed that a total of 5000 tonnes of salmonid product was imported annually, resulting in the production of 3000 tonnes of solid waste. If all imported product was processed and consumed in Melbourne, the waste from imported product would constitute about 0.13% of the non-municipal solid waste produced and processed in a year. The processing of 10 tonnes of product in a day would generate 6 tonnes of solid waste, representing about 0.10% of the daily production of such waste.

Although this is an oversimplification of the situation that would apply, the use of such conservative assumptions means that, in practice, imported aquatic product would represent an even smaller proportion of total solid waste produced in a major Australian city. Solid waste derived from imported product would constitute a minuscule proportion of the solid waste processed through controlled systems. It is also the case that aquatic pathogens may not survive at waste disposal sites (because of desiccation, ultraviolet radiation, low oxygen potential, daily variations in temperature and competition from other microorganisms for nutrients). In combination with dilution, such physical conditions would significantly reduce the concentration of pathogens in waste at commercial processing sites and would greatly reduce

the probability of susceptible species being exposed to a significant concentration of pathogens via such pathways as uncontrolled run-off, leachate or the activities of scavenging seabirds.

AQIS has taken into account information presented by ADVS (1999) in concluding that the probability of pathogens entering the aquatic environment via the disposal of imported seafood-derived solid waste from household and commercial sources would be very low. This results mainly from the reduced survival of aquatic pathogens under the physical conditions that prevail at waste disposal sites, and the dilution of waste containing imported product in the total volume of waste at such sites.

Quarantine issues associated with the entry of imported seafood waste to other-than-traditional waste streams are discussed below.

(d) Fish feed and fishing bait

A number of species are imported into Australia for use as fish feed or bait, including for commercial purposes (eg for use on long-line tuna boats). These include so-called 'trash fish', such as pilchards and herring, which are usually quick-frozen as whole, round fish and shipped soon after capture. These fish are used extensively to feed farmed fish such as southern bluefin tuna. Species imported for use as bait in the Western Australian rock lobster fishery include North Sea herring (from Holland), blue mackerel (from the United States and Holland), and kawahai (Australian salmon, *Arripis trutta*) from New Zealand, as well as hoki, trevally and orange roughy fish-heads from New Zealand (ADVS 1999).

Fish used for feed or bait currently enter Australian waters with minimal prior treatment (primarily limited to freezing) or quarantine restriction. However, there is no definite evidence that the importation of bait fish into Australia has resulted in the establishment of any exotic disease to date. In this risk analysis, AQIS has taken into account information presented in the Western Australian Fishing Industry Council report *Risk Analysis for the Practice of Importing Frozen Fish as Bait* (Jones and Gibson 1997). It has been proposed that risks associated with the use of imported frozen fish as aquaculture feed (eg by the South Australian tuna industry) may be greater than those associated with their use for bait (eg by the Western Australian rock lobster industry) because the bait is used in a more restricted area in the former case (B Jones pers. comm.). In considering this proposition, AQIS also takes into account the species used as bait and the expected prevalence and nature of pathogens in these species, as well as environmental factors and the density of susceptible host species, relative to non-susceptible species, in the receiving waters.

Less significant pathways

DPIE (1996) stated that product reaching consumers (household, hotel, restaurant or institution) will mostly be consumed. However, the report also commented that there are many possible routes, many of which are complex and generally considered unlikely. Similarly, ADVS (1999) reported that there are many potential pathways by which imported fish products can ultimately enter the Australian aquatic environment.

This section discusses the pathways considered to be less significant routes for imported non-viable fish and product to enter the aquatic environment.

(a) Use of product imported for human consumption as fishing bait

Imported fish products may be used for bait by recreational and commercial fishers. However, the nature and extent of such usage is more difficult to ascertain (ADVS 1999). ADVS (1999) discussed the factors that affect the probability of imported fish and product being used for fishing bait or berley. A series of examples illustrated the diverse range of products used for this purpose, including whole animals (prawns, pilchards and herrings) and derived parts (heads, flaps and tails of Australian salmon *Arripis trutta*, tuna heads and abalone gut).

Many fishers purchase whole or processed finfish from seafood outlets for use as bait and/or berley (ADVS 1999). Species commonly purchased include mullet, garfish, pilchards, tommy rough (Australian herring), tuna and bonito. Furthermore, parts of many finfish purchased primarily for human consumption are used as bait and/or berley or to breed maggots for bait. Pilchards/herrings/sardines, prawns and squid/octopus

are the baits generally favoured by recreational fishers; pilchards are the individual species most frequently used for recreational bait (ADVS 1999).

A lot of seafood is imported and consumed in the Easter and Christmas holidays and some is used as bait (ADVS 1999). Householders tend to purchase higher value items such as smoked salmon and prawns in the Christmas period and fish fillets and shellfish in the Easter period. Smoked salmon and 'consumer ready' products such as fish fillets are unlikely to be used in recreational fishing unless they are considered to be unfit for human consumption (eg due to spoilage). It could be argued that aquatic pathogens would be less likely to multiply under the conditions of heightened microbial competition in spoiled product.

ADVS (1999) noted that although heads and frames of Australian salmon (*Arripis trutta*) are not the most favoured bait for catching lobsters, they are used in significant quantities for this purpose. Salmonid flesh or byproducts are not favoured for catching finfish by either commercial or recreational fishers and therefore would be rarely used for this purpose.

(b) The role of birds

The report of the National Task Force (Higgins 1996) suggested that seabirds may provide a pathway of exposure by scavenging at rubbish tips and moving waste derived from imported aquatic product to the aquatic environment. ADVS (1999) noted that seagulls occur in large numbers at certain landfill sites and are also common inhabitants of salmon farms in those areas, with anecdotal evidence suggesting that these populations can travel over significant distances. There is also some opportunity for these seabirds to obtain salmonid waste from processing, wholesaler and retail sites. The risk posed by this pathway (ie seabirds at landfill and salmonid aquaculture sites) depends largely on how effectively seafood is buried at landfill sites immediately upon its arrival.

Some scientific reports suggest that seabirds may play a part in the dissemination of aquatic pathogens; however, the significance of this route remains unclear. Lightner et al (1997) reported that seagulls (*Larus atricilla*) were shown to serve as potential vectors of Taura syndrome virus (TSV) and that gulls and other shrimp-eating seabirds could transmit TSV to farms within their flight path. However, these authors noted that it is not known how long TSV remains in the gut contents of gulls or other seabirds and, therefore, how important these birds might be in spreading this disease beyond a given region. They also noted that an aquatic insect, *Trichocorixa reticulata* (Corixidae), was shown to be infected with TSV. Available data suggest that if the insect feeds on shrimp that have died from TSV, the winged adults can then transmit the virus within or between farms.

AQIS considers that the role of seabirds and other physical vectors in disseminating disease may be significant in circumstances where a high concentration and/or volume of the infectious agent is present (eg in animals dead or dying as a consequence of disease). This is the situation cited by Lightner et al (1997). When disease agents are present at low titre (as would be the case with apparently healthy fish imported for human consumption and then discarded at rubbish tips), seabirds and other mechanical vectors are much less likely to transmit disease. In any case, seabirds and other mechanical vectors appear to be less significant in the spread of aquatic pathogens than live fish or gonadal material, or untreated effluent from seafood processing plants.

Exceptional pathways

This category includes events whereby imported non-viable fish and product may enter the aquatic environment, although only with extremely low frequency. Such events cannot be completely discounted, as there are instances where the spread of disease may have been associated with, for example, the disposal of unwanted food from vessels (A McVicar pers. comm.).

There are many possible pathways for the disposal of imported fish and product, but many are complex and are considered unlikely. Product that reaches the consumer (household, hotel, restaurant or institution) is most likely to be consumed, but the use of salmon product as fish feed and bait and the disposal of product into waterways during picnic and pleasure boat outings may be a more significant risk factor (DPIE 1996, pp 25–31).

Salmonid products are commonly found on the menu for catered boat cruises and aboard yachts. The product would mainly be in a ready-to-eat form, but discarded scraps could provide a direct pathway to the aquatic environment. Events associated with the Sydney to Hobart Yacht Race may contribute to this pathway, but the amount of imported salmonid and marine fish products used (ie as a feature of Tasmanian cuisine and the tourist experience) is open to debate (ADVS 1999).

In considering the exposure of aquatic species to pathogens in food discarded from boats or other sources, a distinction should be made between wild fish populations in the sea and in large freshwater bodies as opposed to river populations and farmed populations, because of greater dilution in the former (A McVicar pers. comm.). The probability of disease being established from an index case is highest in farmed populations because of the high population density.

Imported fish product could be discarded into the aquatic environment but this is likely to occur, at most, infrequently. In any case, susceptible fish would be very unlikely to become infected because the food scraps would be rapidly diluted in water and they would be unlikely to contain pathogens in infective form and at high titre (particularly as most product would have been prepared and cooked for human consumption). Moreover, discarded scraps (whether they contained infective pathogens or not) would be more likely to be consumed by non-susceptible than susceptible species.

Significant salmonid populations are not present in marine waters off mainland Australia; rather, they occur in marine waters around Tasmania. However, most of the Atlantic salmon and rainbow trout are in net pens. A small number of farmed fish have escaped into the wild but these populations are not self-sustaining (ADVS 1999). In contrast, no brown trout are kept in sea cages but brown trout occur in significant numbers in Tasmanian estuaries and near-shore marine waters. There are also significant freshwater populations of salmonids in the inland waters of Tasmania and mainland Australia. The possibility of aquatic pathogens entering these waters via imported seafood discarded from boats or other sources cannot be discounted, but it is very unlikely that such an event would cause an index case of disease. This is because it is unlikely that the food would contain pathogens in infective form at a significant titre (because of the dilution of material entering the aquatic environment) and because of the likelihood of non-susceptible species consuming the scraps.

AQIS has taken these factors into account and has concluded that the quarantine significance of this exposure pathway would be negligible.

1.7.4 THE PROBABILITY THAT IMPORTED PRODUCT INITIATES AN INFECTION (INDEX CASE)

Information in previous Australian government reports (DPIE 1995 and 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b) is directly relevant and should be read in conjunction with this discussion. These reports contain referenced reviews of relevant literature.

The infectious dose of a particular agent for a particular host will vary with the strain of the agent, the route of infection, environmental conditions and host factors. Factors relevant to this probability include the titre of organisms likely to be present in imported product entering the aquatic environment and the capability of organisms to survive in the aquatic environment long enough for a susceptible host to be exposed to them. If the route of exposure involves fish consuming imported product that contains pathogens, the likelihood of a susceptible fish rather than a non-susceptible host consuming the product is also relevant.

Most of the factors affecting the level of pathogens that may be present in imported fish and aquatic product are discussed in Section 1.6. However, the concept of minimum infective dose has been considered in previous risk analyses and should be discussed at this point.

DPIE (1996) reported that the relationship between the titre of viable pathogens likely to occur in muscle tissue and the number of organisms needed to initiate infection if consumed by a susceptible host is an important consideration. Key data on infectious dose in the natural environment are, in the main, unavailable and a conservative approach is therefore warranted. In general, experimental infectivity studies have required either relatively high doses of the pathogen or exposure to live infected fish to produce infection. The applicability of

some of these data to the infection of Australian fish should be interpreted with caution (DPIE 1996).

Similarly, ADVS (1999) noted that it could be misleading to recommend a minimum infective dose of any pathogen, and that such a recommendation would need to take into account the capability of a laboratory to determine accurately the number of cells or viruses present when they may be in a cryptic state. The environmental conditions at the time of infection or release from a carrier and the health and immunological status of the recipient host animal would also have to be taken into account. For some diseases (such as furunculosis), infection may be initiated by a very low dose of organisms (ADVS 1999). This report further suggested that advances in knowledge on issues such as transfer of virulence genes, genetic recombination and quorum-sensing or auto-induction will change the concept of infective dose.

AQIS has taken into account relevant data that are available. For most agents, data are not available to provide a meaningful quantification of infectious dose, and it is only possible to conclude that the minimum infective dose is likely to be high or low, relative to the range of disease agents under consideration.

1.7.5 THE PROBABILITY OF DISEASE SPREADING FROM THE INDEX CASE AND BECOMING ESTABLISHED IN HOST POPULATIONS IN THE IMPORTING COUNTRY

For most disease agents, infection is most readily transmitted via the introduction of a live, infected host into a naive (and susceptible) population. Some agents may cause subclinical infection, so apparently normal infected fish (ie carriers) may still be a source of infectious organisms. However, new disease is most likely to be introduced into a population by introduced fish that are clinically diseased and actively disseminating infectious organisms into an environment that contains susceptible host fish. The greater the population density of susceptible fish, the more readily disease may be transmitted and the greater the rate of morbidity in the susceptible population.

The dynamics of transmission of disease have been studied extensively in farmed livestock and birds, but there is much less information in relation to aquatic animals. Nonetheless, it is well recognised that most pathogens are transmitted (and disease is expressed) far more readily in aquaculture than in wild fisheries. Density of susceptible species is an important factor but other factors that affect the susceptibility of the host to infection (eg lifecycle stage, environmental conditions and intercurrent stress) may be equally important.

Susceptibility of Australian salmonids and non-salmonid species to infection

In considering the susceptibility of Australian species to exotic pathogens, AQIS has taken into account information in previous reports (DPIE 1995 1996). In particular, DPIE (1996) reported concerns that Australian salmonids may be highly susceptible to exotic diseases because they have been in Australia for many generations without further introductions of genetic material and have not been exposed to selection pressures for resistance to exotic diseases. These previous IRAs were based on an assumption that Australian salmonids would be fully susceptible to all the organisms shown to be pathogenic for salmonid fish overseas. AQIS concluded that there is no reason to change this assessment.

In the draft of this report AQIS stated:

> In addition to Australian salmonids lacking acquired immunity, stakeholders have postulated that, in Australia, these fish are farmed and live in relatively warm, slow-flowing waters that represent the environmental limits of distribution of these species. It has been argued that this stresses the fish, rendering them more susceptible to infection with pathogens. This may be correct. On the other hand, most Australian salmonids are farmed in pristine environmental conditions. Moreover, Tasmanian salmonid farms have low stocking rates, compared with international practice. For example, stocking rates for 2kg Atlantic salmon in Tasmania are approximately 8–12kg per cubic metre, compared with about 18kg per cubic metre in Scotland and 25kg per cubic metre in Norway (B Munday pers. comm. and A McVicar pers. comm.). The absence of many pathogens found elsewhere in the world and the reduced need to implement therapeutic practices, such as

vaccination, may mean that Australian salmonids are relatively free from stress and have a generally high level of immune competence.

Stakeholders commented that this statement was inaccurate, on the basis that, in comparison with most northern hemisphere situations, Australian salmonids live in relatively warm and slow-moving waters. For example, water temperatures in Tasmania regularly rise above 20°C, and in winter rarely fall below 12°C, whereas in Scotland, water temperatures seldom rise above 12°C.

AQIS acknowledges that the relatively high water temperatures have two major implications: first, salmonids in Australia are farmed under conditions reaching the upper limit of those species' temperature tolerance ranges, and, second, inherently associated with higher water temperatures, is that the maximum levels of available (solubilised) oxygen are lower. The dissolved oxygen levels in Tasmanian waters are still adequate for salmonids in well-managed pens in all but the extreme maximum temperatures (B Munday pers. comm.). The period when water temperatures are at the upper limit for Atlantic salmon in Tasmania varies between a few weeks and a few months depending on the year and the site of particular farms (B Munday pers. comm.).

Additional problems due to increased temperatures include a higher level of the toxic ammonia (compared to ammonium ions), potentially leading to decreased excretion of metabolic wastes via the gills of fish, rapid net fouling and early maturation. Other stressors encountered in the Australian environment include jellyfish, seals and other predators. Management measures such as net changing, and freshwater bathing to treat gill amoeba, also present a stressful situation to the fish.

Increased stress associated with water conditions and management practices as described above is alleviated in part by the unpolluted Tasmanian waters. Tasmanian salmon do not have to deal with the effects of pollutants such as human or terrestrial animal effluent, heavy metals or pesticide run-off from agriculture (just to mention a few), all of which would — if present — add to the baseline level of 'stress'. Australian farmers also compensate for many of these problems by a variety of management tools (eg operating with low stocking densities to adjust to the carrying capacity of the water and using air-lifts for the transfer of fish from cage to cage instead of fish-pumps, which reduces stress and handling abrasions) (E Bernoth pers. comm.).

A McVicar (pers. comm.) advised that it would be misleading to imply that the difficulties in Tasmania are unique, as salmon farmers in all cultivation areas have to adapt their husbandry practices to the local set of conditions, even to the extent that markedly different practices often have to be employed to optimise performance and survival even in closely adjacent farms. These differences are likely to become greater, the greater the distance between farms; there are certainly major differences evident between Norway, Scotland and Ireland.

AQIS noted that farmed Australian salmonids grow to market size in approximately 12 months, compared to an average of 18 months to 2 years in most other salmonid-producing countries. Such high growth rates would not occur if the fish were constantly suffering severe stress. Furthermore, the lower stocking densities would be expected to reduce transmission of disease by simply keeping fish further apart.

It is also relevant that although there are no published data on the effect of high water temperature on the immune competence of Atlantic salmon, it is well established that the immunological response is directly proportional to the water temperature. The closer a fish is to its physiological temperature maximum, the better the immunological response; however, once the fish are exposed to unphysiologically high water temperatures, the resultant release of cortisol, heat shock proteins and other stress-related products leads to compromisation of the immune system (B Munday pers. comm.).

Without evidence from experimental infections, it cannot be said with any degree of certainty that the salmonids in Australia are any more or any less susceptible to any of the diseases than are stocks of those species anywhere else in the world. The genetic lineage, gradual selection for certain traits and environmental conditions could well have a marked influence on the inherent susceptibility to any given disease, making them either more susceptible or more resistant (B Hill pers. comm.).

DPIE (1996) reported that, because it has been assumed that native salmoniform fish may be at least as susceptible to disease as the most susceptible salmonid species, an appropriately conservative approach needs to be taken. In considering the broader scope of this risk analysis, AQIS notes that there are native counterparts to most of the species considered. As in DPIE (1996), it has been assumed that the risk to native species would not be significant for most of the pathogens, which are (from overseas experience) characteristically specific in their host range. However, AQIS has assumed that pathogens that have a wide host range (such as some *Vibrio* species and *Aeromonas salmonicida*) may well pose a more significant risk to native species.

AQIS has applied conservative judgments regarding the susceptibility of Australian salmonids and non-salmonid species to infection with exotic pathogens in this risk analysis.

1.7.6 CONCLUSIONS

AQIS concludes that most salmonids and non-salmonid marine finfish imported for human consumption would be consumed (in raw or cooked form) by people, and that this would generally present an extremely low probability of pathogens, if present, entering the aquatic environment. However, people may also dispose of such product by pathways that would result in a higher probability of pathogens, if present, entering the aquatic environment. Such pathways include the use of imported product for fishing bait and the disposal of scraps into waterways. The 'fishing bait' pathway would occur with a very low frequency in the case of most products imported for human consumption. For aquatic pathogens that are highly host-specific (including many salmonid pathogens) there would be a high probability of scraps being consumed by aquatic species that are not susceptible to infection. The concentration of pathogens entering the aquatic environment via scraps of product would also be rapidly reduced through dilution.

In contrast, most marine finfish imported for use as fish feed or fish bait would enter the aquatic environment. The concentration of pathogens entering the aquatic environment by this pathway would be reduced by dilution. The probability of disease resulting from this pathway also depends on the species used as bait and the expected prevalence and nature of pathogens in these species, as well as environmental factors and the density of susceptible host species, relative to non-susceptible species, in the receiving waters. For most fish used as bait, fewer pathogens have been reported than for salmonid fish.

For domestic sewerage, factors associated with dilution and physical conditions would greatly reduce the probability that pathogens (in an infective state) would enter the aquatic environment by the pathway of wastewater treated in the domestic sewerage system.

Wastewater from fish processing plants may contain a higher concentration of aquatic pathogens. If such water bypassed the domestic sewerage system or were discharged into waterways without treatment or disinfection, aquatic pathogens could enter the aquatic environment in significant quantity.

There may be a higher concentration of pathogens at the point of discharge of untreated/undisinfected wastewater. However, the concentration would be reduced by dilution further away from the point of discharge. Aquatic species may congregate at wastewater discharge points. For aquatic pathogens that are highly host-specific (including many salmonid pathogens) there would be a very high probability of pathogens being consumed by aquatic species that are not susceptible to infection. For aquatic pathogens that have a wider host range, the probability of susceptible species consuming pathogens would be greater, but non-susceptible species would still compete with these species for scraps and this would reduce the probability of an index case of disease occurring.

Due to the effect of dilution and exposure to physical conditions, there would be a very low probability of pathogens entering the aquatic environment via solid waste in the commercial waste management system. Although the role of seabirds and other scavengers in moving solid waste around cannot be discounted, this would not significantly increase the probability in the risk analysis overall.

AQIS has considered other pathways and the probability of aquatic pathogens entering the aquatic environment by such routes. These pathways would be followed rarely

or exceptionally. Moreover, in the light of other factors discussed, AQIS concludes that these pathways would not significantly increase the probability in the risk analysis overall.

1.8 Consequence assessment

1.8.1 INTRODUCTION

This section discusses the factors considered by AQIS in assessing the significance, or impact, of the establishment of exotic disease. As outlined in Section 1.5.3, Box 1.6, AQIS considers all relevant factors and classifies the significance of each disease according to categories that have been defined in qualitative terms ('negligible', 'low', 'moderate', 'high' or 'catastrophic'). The significance and the probability of establishment are considered together in estimating the risk.

AQIS has initially considered the pathogens expected, on the basis of current scientific knowledge, to have a moderate (or greater) probability of entering and becoming established in Australia or, if established, to cause moderate (or more significant) consequences. Factors relating to the probability of entry and establishment are discussed in Sections 1.6 and 1.7.

The key points relevant to the consequences of establishment of individual diseases are set out in Chapters 4 and 7. This section describes the general considerations relevant to this assessment.

1.8.2 FACTORS RELEVANT TO THE IMPACT OF DISEASE

Biological effects

In Section 1.5.3, the effect of establishment of a disease was defined in biological terms (with reference to mortality, morbidity and the pathogenic effects of the agent) and in terms of economic or environmental impact. Most of the disease agents considered in the risk analysis have the capacity to cause marked pathological effects in a significant proportion of hosts in a susceptible population and to cause significant economic effect, if they were to become established.

The biological effect of disease depends on the interaction of the environment, pathogen and host. The nature of this interaction reflects factors intrinsic to the pathogen (such as virulence and infectivity), the host (such as immune competence and population density) and the environment (such as availability of habitat for susceptible hosts).

The biological effect of disease is normally evaluated in terms of morbidity and mortality. Standard epidemiological parameters such as case fatality rate and cumulative mortality rate can be used to describe changes in the mortality rate within an infected population. Changes in the mortality rate may be graphed against time (eg for a production cycle). Morbidity can be evaluated in terms of reduced production, and described by parameters such as food conversion efficiency and fecundity that are relevant to the population under study. Diseases that reduce the efficiency of production without causing large increases in mortality are more likely to be significant in farmed fish than wild-caught fish.

Epidemiology

The epidemiology of disease in aquatic populations is generally poorly understood. In farmed fish, 'normal' or baseline values for production and mortality are often highly variable, reflecting husbandry practices, stocking rates and stress. Many economically significant diseases of farmed fish are caused by commensal organisms that are opportunistic pathogens (ie they cause disease only when environmental or other conditions predispose fish to infection).

It is likely that in wild fish, as well, the effect of pathogens is influenced by environmental factors that predispose the host population to infection and the expression of disease. This would be consistent with the generally higher prevalence of disease in farmed than in wild fish and the apparently greater rate of emergence of new pathogens in farmed fish. However, as discussed in Section 1.6.3, this may reflect closer monitoring of production and mortality in farmed fish.

The underlying or 'normal' rate of mortality in wild populations may be statistically estimated, based on data collected in studies of population density, age

structure and catch rates. Normally, this type of modelling becomes more accurate over time and the natural rate of mortality can be estimated with increasing accuracy. Population fluctuations can be linked quite closely to other factors, such as fishing pressure, using these sorts of data. However, it is usually the case that only major epizootics involving significant mortalities are detected in wild fish.

The complex interaction between host factors, environmental factors (including husbandry in farmed fish) and agent factors makes it difficult to predict accurately the effect of the establishment of exotic disease. Stakeholders have commented that Australian fish would be more susceptible to disease due to the historical absence of many pathogens. AQIS has made a series of conservative assumptions, including that farmed and wild fish (including native species) in Australia would be at least as susceptible to infection as fish of the same, or closely related, species reported as susceptible under similar conditions in other countries.

AQIS has also assumed that the consequences of disease becoming established would be at least as serious as those reported overseas, having regard to the paucity of methods for treatment and control in Australia.

Australia's capacity to respond to disease incursions

There are few diagnostic tests or other measures, including specific contingency plans, available in Australia for exotic diseases of fish. This would limit Australia's capacity to deal with entry and establishment of new diseases.

This deficiency has been recognised and contingency planning for aquaculture disease emergencies is now well advanced at the national level. AQUAPLAN is a strategic aquatic animal health plan developed by Agriculture, Fisheries and Forestry — Australia (AFFA) in consultation with aquatic industries and State agencies with responsibility for fisheries and aquaculture. Since the inception of AQUAPLAN in 1998, significant progress has been made on preparedness and response plans to deal with aquatic animal disease emergencies. The components of an emergency aquatic animal disease plan have been drafted in consultation with industry, and a field handbook for recognition of aquatic animal diseases has been commissioned.

There have recently been coordinated responses to aquatic disease emergencies, including the 1998–99 pilchard mortalities, the invasion of black striped mussel in Darwin Harbour and the detection, during routine diagnostic submissions, of a disease of freshwater crayfish in Western Australia that was previously unreported in that State.

AQIS has taken an appropriately conservative approach to the IRA, in the light of the high cost associated with attempts to eradicate new diseases and the low likelihood of success.

In the case of pathogens shown by overseas experience to be highly pathogenic (eg furunculosis, infectious salmon anaemia and infectious haematopoietic necrosis), AQIS has assumed that rates of morbidity and mortality in Australia would be comparable to those overseas. AQIS has also assumed that diseases that have been shown by overseas experience to be difficult or impossible to eradicate (eg infectious salmon anaemia, bacterial kidney disease and whirling disease) would present similar difficulties in Australia.

For the diseases that are routinely controlled overseas by husbandry measures (eg reduced stocking rate) or veterinary intervention (eg vaccination or antimicrobial treatment), AQIS has generally assumed that a similar approach may be applicable in Australia.

For some diseases there are clear parallels. For example, Australian salmonids are routinely vaccinated to prevent disease due to *Vibrio anguillarum*. Coldwater vibriosis (due to *Vibrio salmonicida*) is controlled in Europe and North America by vaccination. Thus, in the event that *Vibrio salmonicida* were to become established in Australia, it is likely that this disease could be controlled by similar means and the consequences of establishment on farmed fish thereby mitigated.

However, environmental conditions (including husbandry) clearly influence the expression of clinical disease and the amenability of introduced disease to prevention and control. Thus, methods used with success overseas may not be feasible or similarly effective in Australia.

AQIS notes that there would be a need for regulatory approval of any drug or vaccine required that is not currently available in Australia if drugs or vaccines were to be used to control an introduced disease. Moreover, the implementation of a control strategy would introduce new costs and, potentially, adverse implications for product quality and image.

AQIS also notes that the application of measures for the control, prevention or eradication of disease is generally more difficult for viral diseases (particularly those that are transmitted vertically) than for disease due to bacterial, protozoal or metazoan agents. Most bacterial diseases are amenable to treatment and/or prevention. The impact of diseases due to protozoal and metazoan agents may, in many cases, be substantially mitigated by the application of chemotherapeutants and/or improvements in husbandry and environmental conditions. For pathogens that have an indirect life cycle (such as *Myxobolus cerebralis* and proliferative kidney disease agent) it may be possible to prevent transmission of infection in farmed fish by excluding intermediate or alternative hosts. This method has been used to prevent the spread of *M. cerebralis* outside its limited area of distribution in New Zealand. However, for some pathogens, the implementation of measures for control or eradication would be so costly as to be unfeasible in practice.

Economic effects

Increased morbidity and mortality cause direct economic losses due to decreased production. Production is usually measured in terms of output in number or weight of fish/product in relation to the cost or volume of inputs.

For fish farmed in Australia, the most significant inputs are normally labour, feed and access to water of suitable quality and volume. There may also be significant costs associated with handling fish (eg for sorting or treatment) and with catching, transporting and processing them for human consumption.

If a disease is characterised by significant pathological effects, its establishment can have a moderate (or greater) impact due to increased mortality (as a peak, or on a cumulative basis) and/or reduced growth, feed conversion efficiency, reproductive performance or product quality. Control, prevention or eradication of the disease would add to the cost of production. Even vaccination may result in failure (eg in fish that are stressed at the time of vaccine administration), and this would be reflected in higher costs; for example, insurance premiums may be increased or it may be necessary to increase stocking rates in case of unexpected increases in mortality. AQIS takes into account the costs associated with increased morbidity and mortality and costs associated with the implementation of control measures.

If a disease becomes established, economic performance can also be harmed indirectly (eg through reduction of value of domestic or export markets for live fish, gonadal products or product for human consumption). For example, the establishment of infectious pancreatic necrosis in a salmonid farm that produces and exports salmonid genetic material would cause the loss of domestic and export markets for genetic materials, pending clarification of the disease situation. Moreover, farms on the same waterway as the affected farm would be subjected to similar restrictions and costs as those of the affected farm. The total economic losses associated with loss of market access can be reduced if the disease were contained in a circumscribed area (regionalised) and markets reopened for farms outside the affected area. Markets might be reopened if disease were eradicated or the affected farm implemented a testing program (at additional cost) to underpin health certification to the effect that fish used to produce genetic material were free from disease.

Effects of disease in wild capture fisheries

The economic effects of disease in wild capture fisheries have been well illustrated by the pilchard mortalities of 1995–96 and 1998–99 in South Australia and Western Australia. These extended well beyond the value of the dead pilchards. Fisheries were closed as the mortality event occurred and were not reopened until some weeks after deaths had stopped. When reopened, catch rates were poor. This caused direct economic loss to fishers, crews, processors and factory staff and extended to those industries supporting the fishing infrastructure.

Since this was the second time a disease epizootic had occurred, some companies had to re-evaluate the extent of their future financial exposure to pilchard fishery fluctuations. This affected plans for expansion and equipment replacement. There were also considerable costs in investigating the cause of the mortalities.

Zoning or regionalisation of disease

WTO member countries recognise the concept of zoning, or regionalising, disease to minimise negative effects on trade. However, there are currently few international aquatic disease restrictions on international trade in eviscerated product for human consumption. Thus, in practice, zoning of aquatic diseases normally only applies to trade in live fish and genetic material. Similarly, interstate movement restrictions in Australia cover live fish and genetic material, but do not normally apply to movement of non-viable fish products. Exceptions to this include the closure of pilchard fisheries affected by mortalities and controls on salmonids harvested in Macquarie Harbour. If an exotic disease became established, Australia would use zoning to maintain access to international markets for live fish, genetic material and, if required, non-viable product. This would require additional specific regulatory measures such as movement controls, testing and certification, with attendant costs.

Public health and perceptions of quality

The establishment of aquatic diseases considered in this risk analysis would have no public health significance. With the exception of certain metazoan diseases, organisms that are primary pathogens of aquatic species normally do not cause disease in humans. However, pathogens such as *Aeromonas salmonicida* (which causes furunculosis in salmon) or *Henneguya salminicola* can cause the formation of visible lesions in tissues, and affected product would be unacceptable to the consumer.

Public perception can also significantly affect markets for product for human consumption. For example, despite the virus being inactivated at temperatures at or below mammalian body temperature, public concern about infectious salmon anaemia in Scotland caused affected farms to lose access to some domestic markets, resulting in major economic losses (A McVicar pers. comm.). Similarly, the establishment of disease may affect the quality of non-viable product, causing a reduction in price and competitiveness. This may occur with or without the implementation of a regime of treatment or prevention. The use of chemical treatments or the occurrence of lesions/blemishes on the product can also affect any price premiums paid for high-quality products. This may occur regardless of whether the effect on quality was real or perceived.

RECREATIONAL FISHERIES

Recreational fisheries present a special case, in that 'production' is not easily quantified. However, numerous studies have estimated the value of the Australian recreational fishing industry in terms of direct and indirect expenditure. McIlgorm and Pepperell (1999) reviewed these studies and valued the industry in 1998 terms. National expenditure was conservatively estimated to be A$2926 million, of which 20% was direct expenditure (rods, reels, tackle, club membership); almost 50% was indirect expenditure (travel, accommodation, boat fuel and hire and other costs); and 30% was capital expenditure (boat purchase, maintenance, insurance and registration). These authors also considered the impact of the establishment of disease on recreational amenity. Disease introduction could affect a species or group of species considered important by recreational anglers. The immediate impact may be evaluated in terms of freshwater or marine species and the impact of disease on a specific subsector of the recreational fishing sector.

The immediate concerns of recreational fishers and associated industries would include:

- a reduced catch rate for a given species — a short-term, immediate problem;
- the health of fish stocks — long-term implications;
- the availability of substitute species that would maintain the recreational amenity;
- sources of the affected species in other areas;
- impact of disease on other species; and

- reduced direct and indirect expenditure associated with recreational fishing; for some regions, expenditure associated with recreational fishing would be a significant proportion of the total economy.

In considering the consequences or impact of the establishment of disease, AQIS takes into account the likely host range for each pathogen and how the disease would affect recreational fisheries based on single and multiple species, as well as factors listed above.

ECOLOGICAL AND ENVIRONMENTAL EFFECTS

In considering the significance, or impact, of the establishment of disease, AQIS also takes into account effects on the environment. The establishment of disease can have biological or ecological effects that could affect the survival of native species that are not farmed or otherwise commercially exploited. For example, the ecological balance of freshwater systems and the quality of the environment could be disturbed if the normal proportions of different native species were significantly altered by the selective loss of one or more particularly disease-sensitive species. Equally, such effects may result from the introduction of an exotic species that becomes a pest, such as *Cyprinus carpio*. These effects cannot be quantified. However, if they cause a decline in the number of endangered or threatened species, the potential loss of biodiversity would be of concern to the Australian community.

AQIS takes a conservative approach when considering the susceptibility of native species, particularly those that are endangered or threatened, to infection with exotic pathogens. In considering the consequences of establishment of an exotic disease, the establishment of any disease that is likely to result in the extinction of a species (which equates to having a serious, irreversible effect on the environment) would be classified as 'catastrophic'. In most cases there is limited information on the effect of exotic pathogens in Australian conditions. However, in drawing conclusions on the likely impact of exotic disease on the environment, AQIS considers overseas data on the species of fish that are susceptible to infection, the effect of infection on those fish populations and the influence of the physical environment on the outcome of infection.

In considering the likely effect of exotic pathogens on Australian native species, AQIS takes account of evidence that the pathogens could infect a wide range of species or families, including any that are related to or occupy similar ecological niches to Australian native species. In the case of pathogens that infect a narrow/specific range of hosts that are unrelated to Australian species, AQIS assumes that effects on native species would be negligible. However, for exotic pathogens that have a wide/non-specific host range, including species that are related or similar to Australian species, AQIS assumes that native species would be susceptible to infection and that the establishment of disease could have consequences as least as severe as those reported overseas.

Scientific reviewers commented that species other than fish (in particular, amphibians) may be susceptible to exotic disease agents of finfish. In response, AQIS reviewed scientific information on diseases of amphibians in Australia and overseas and took this information into account in this risk analysis. This scientific review may be found in AQIS's report on the risk analysis of live ornamental finfish (AQIS 1999).

Chapter 2
Salmonids and non-salmonid marine finfish in Australia

2.1 The Australian fishing industry

AUSTRALIA HAS ONE OF THE MOST DIVERSE marine faunas in the world (Kailola et al 1993). This fauna includes more than 3600 species of fish in 303 families, reflecting the diversity of the Australian marine environment. Environment Australia's *Interim Marine and Coastal Regionalisation for Australia* (IMCRA Technical Group 1997) describes and classifies the diverse marine environments around Australia, the transition zones (biotones) between them, and the adjacent oceanic regions. Protecting this diversity is an important consideration in the current import risk analysis (IRA).

Despite this diversity and despite being the world's third largest fishing zone, Australia ranks only about 50th in world fisheries production in terms of tonnes of fish landed. This lower productivity is a result of lower nutrient levels, as well as continental shelf and slope size and topography. Nevertheless, many of Australia's fisheries are based on high-value products, such as prawns, lobsters, abalone and sashimi-grade tuna. The gross value of Australia's fishery production in 1997–98, including aquaculture, was approximately A$1860 million. The total weight of production for this period was 222,837 tonnes. Australia's wild fisheries are not expected to expand production significantly in the near future, but substantial increases in aquaculture production are anticipated. The value of production of the aquaculture industry was estimated at A$491 million in 1997–98 (ABARE 1998).

Fisheries and aquaculture based in inland waters or State waters produced 82% of total fisheries production for 1997–98. Management of these industries is the responsibility of the various State and Territory governments under the relevant State and Territory fisheries legislation.

Under the Commonwealth *Fisheries Management Act 1991*, the Australian Fisheries Management Authority is responsible for the development and administration of management plans for marine fisheries in Commonwealth waters.

In 1979, Australia declared an Australian Fishing Zone extending 200 nautical miles out from the coast. Australian-flagged vessels can fish in this zone, as can

some foreign vessels through various bilateral access and joint venture agreements. These arrangements are consistent with the United Nations Convention on the Law of the Sea and generally involve the payment of an access fee or levy.

2.2 Australian salmonid and marine finfish industries

Aquaculture, mainly involving Atlantic salmon, trout, tuna and ornamental fish, is a growing sector of the finfish industry. The culture of Atlantic salmon and rainbow trout is described in Section 2.2.1. There is a substantial tuna aquaculture industry in South Australia based on the grow-out of wild-caught juvenile southern bluefin tuna in inshore cages.

The most lucrative export markets for Australian fisheries are Japan, China (including Hong Kong) and the United States. Japan is the main buyer of tuna, while the United States takes more than 90% of fish fillets. Australia's domestic market is supplied with fresh and frozen fish caught domestically and imported from several countries, a major part coming from New Zealand.

2.2.1 SALMONIDS

Salmonid fish are not native to the southern hemisphere. They have been introduced into Australia in the last 150 years. The five species of salmonid fish found in Australia are:

- Atlantic salmon — *Salmo salar*;
- brook trout — *Salvelinus fontinalis;*
- brown trout — *Salmo trutta;*
- chinook (quinnat) salmon — *Oncorhynchus tshawytscha; and*
- rainbow trout — *Oncorhynchus mykiss.*

Of these species, brown, brook and rainbow trout have established self-sustaining populations. Wild populations of Atlantic and chinook salmon are supported by regular release of hatchery-bred fish. The distribution of salmonids in Australia is summarised in Table 2.1.

The Australian salmonid industry includes commercial farming, hatcheries, tourism and recreational fishing. The industry started with the introduction of trout into Australia's inland waters for sporting use. Since then it

Table 2.1
Distribution of salmonids in Australia

	ATLANTIC SALMON	BROOK TROUT	BROWN TROUT	CHINOOK SALMON	RAINBOW TROUT
Australian Capital Territory	W	W	W		W
New South Wales	W/A	W/A	W/A		W/A
Northern Territory					
Queensland					A
South Australia	A	W/A	W/A		W/A
Tasmania	W/A	W/A	W/A		W/A
Victoria	W/A	W/A	W/A	W/A	W/A
Western Australia	W/A		W/A		W/A

W — wild population
A — aquaculture populations
Source: AQIS

has grown into the second most highly valued aquaculture industry in Australia (Industry Commission 1996), and the most important aquaculture industry in Tasmania (Industry Commission 1996, McKelvie et al 1996, Brown et al 1997). The gross value of salmonid production in 1997–98 was approximately A$76 million (ABARE 1998).

The most important farmed species are Atlantic salmon and rainbow trout, with smaller amounts of brown trout, brook trout and chinook salmon (Brown et al 1997). The following sections discuss Atlantic salmon and rainbow trout.

Atlantic salmon

Production

From its inception the Australian Atlantic salmon industry has focused on producing a high-quality product. The farming techniques used in the Australian industry have been adapted from those successfully employed for many years in Norway and Scotland. The farming methods used in Australia involve the production of early-stage salmon in freshwater facilities. After 12–18 months they smolt (become acclimatised to seawater) and are ready for transfer to cages located in estuaries or sheltered coastal waters. Some pre-smolts are transferred to brackish water as a prelude to transfer to full seawater.

Most Australian salmon producers harvest fish all year round. They achieve this through a number of strategies, such as the use of spring and autumn smolts, pre-smolts and triploid fish. In Australia, Atlantic salmon grow from around 80 grams to a marketable size of 3.4–4.5 kilograms in 12–15 months after introduction to saltwater. This is partly attributable to the relatively warm waters in which Australian salmon are farmed. The warmer waters can also cause increased stress (see Section 1.7 for a description of Australian farming conditions and the susceptibility of Australian salmonids to disease). The development of new techniques and equipment has, in part, helped to reduce production costs while improving quality (Industry Commission 1996).

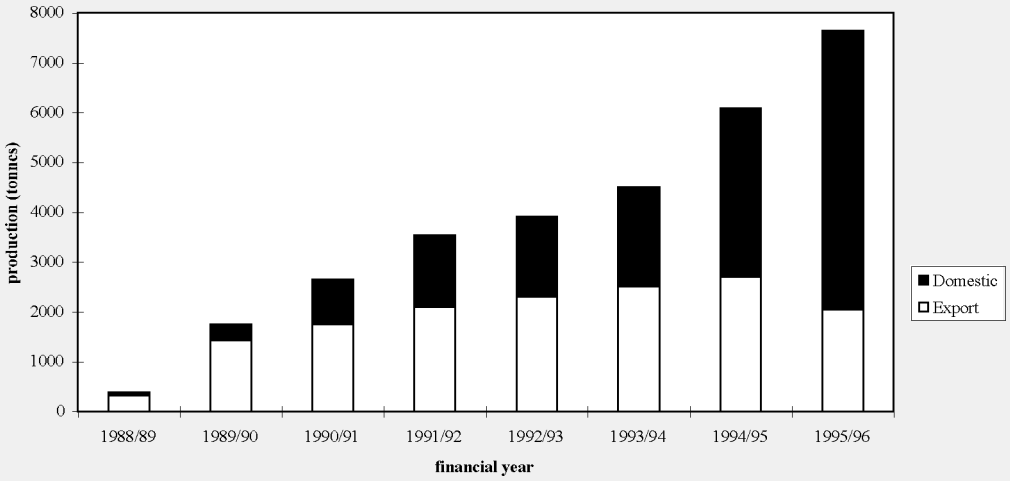
There are several small Atlantic salmon producers in Victoria and South Australia, but most of the Atlantic salmon industry is based in Tasmania, where it was established in 1985 as a joint venture between the State government, the Norwegian Company Noraqua, and a group of private Australian companies (Deck 1997, Industry Commission 1996). This also led to the formation of Salmon Enterprises of Tasmania (Saltas) which, under the Tasmanian *Salt Water Salmon Act 1985*, had a monopoly on salmon smolt production for 10 years.

Currently there are seven Tasmanian Atlantic salmon farming companies, of which three dominate the market. The largest producer is Tassal, which supplies the majority of Australian Atlantic salmon to the Asian market, though in recent years much of its product has been sold on the domestic market. The second largest producer, Aquatas, which is wholly Japanese owned, exports most of its product. Nortas Aquaculture also exports product but, like Tassal, sells most of its salmon on the domestic market. Smaller farms grow fish under contract or for sale to domestic processors.

The Tasmanian industry has grown steadily over more than a decade, from producing 20 tonnes in 1986–87 to just over 7000 tonnes in 1997–98 (Industry Commission 1996, ABARE 1998).

This rapid rise can be attributed to several factors. These include an initial high level of government support and involvement, including financial assistance; regulatory arrangements to ensure the industry functioned at a sustainable rate; and the provision of research and advice to the industry. Strict quarantine conditions (prohibiting the importation of uncooked salmon for human consumption) and the absence of serious diseases of salmon which occur overseas are other factors that have helped the industry.

Figure 2.1
Growth in the Tasmanian Atlantic salmon industry, 1988–96



Source: Industry Commission 1996; Brown et al 1997.

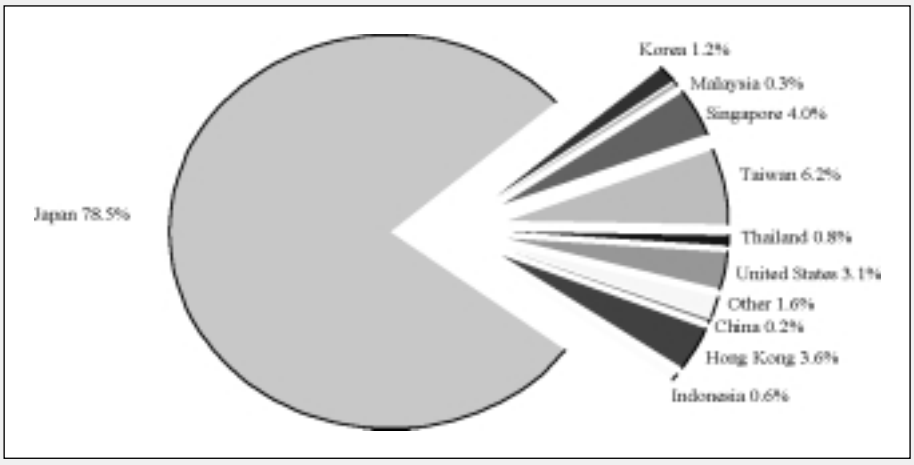
Markets

While the Australian Atlantic salmon industry was initially export-orientated, domestic sales now account for around two-thirds of the total production (see Figure 2.1).

This change reflects lower prices, as supplies of Atlantic salmon to the domestic market have increased, and improved marketing and distribution. Fresh, frozen and smoked product is marketed to supermarket chains, restaurants, hotels and airlines. Little use is made of the wholesale fish markets.

In export markets, Australian Atlantic salmon competes with farmed Atlantic and Pacific salmon from other countries, and high-quality, wild-caught Pacific salmon and other premium foods (Doyle et al 1996). Australia annually exports salmon product worth around A$20 million, most of which is destined for Japan.

Figure 2.2
Atlantic salmon exports



Source: Industry Commission 1996, Brown et al 1997.

Although Australia provides only a small percentage of total salmon imports to Japan, it has a marketing advantage as a supplier of high-quality product. Australian fresh Atlantic salmon has consistently sold at premium prices on the Japanese market since the late 1980s. In 1996–97, the average price premium was around 20% (ABARE 1998).

Hong Kong, Taiwan, the United States and Singapore are other important export markets for Australian product. Figure 2.2 displays export markets for Australian Atlantic salmon.

Rainbow trout

Production

Rainbow trout are farmed in fresh water and seawater. Techniques used in the freshwater farming process have changed very little since the establishment of the first commercial farm in the 1960s (Treadwell et al 1991). When rainbow trout are raised in brackish and ocean waters they are marketed as ocean trout. Initially a small number of operators in Tasmania produced only ocean trout (McKelvie et al 1996) and most of the companies farming Atlantic salmon also farmed ocean trout (Brown et al 1997). However, this situation is changing as the industry develops, and fewer farms now produce ocean trout.

Most current commercial production involves freshwater production in the temperate areas of Victoria and New South Wales, as well as smaller operations in Tasmania, South Australia and Western Australia (Brown et al 1997).

Victoria is the largest commercial producer of rainbow trout in Australia, with approximately 40 farms, 10 of which produce trout commercially for export or domestic markets (Brown et al 1997). The rest are tourist farms, with some commercial production. Trout farms are the oldest and largest aquaculture industry in the State (Industry Commission 1996).

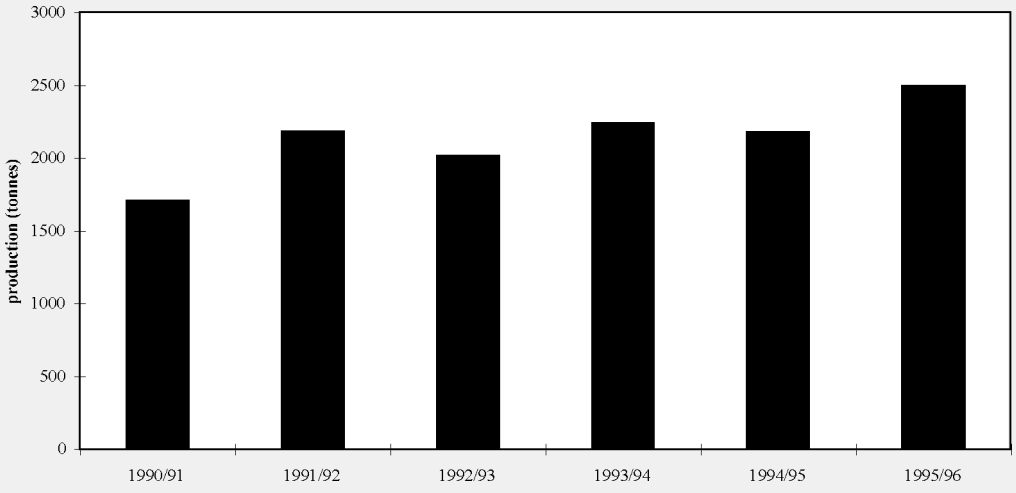
Export of eyed ova is undertaken from trout farms in Tasmania.

In New South Wales, trout farms may concentrate on producing fingerlings for stocking farm dams, rivers or tourist farms. In 1995–96, 18 trout farms were in commercial operation (Brown et al 1997).

The South Australian rainbow trout industry targets the recreational fishing market. In 1995–96, six commercial farms were operating (Brown et al 1997).

Trout aquaculture in Western Australia takes place mostly in fresh water. There was some attempt to farm ocean trout in sea cages on the south coast, but the success of these ventures was limited by high water temperatures and lack of sheltered sites (Lawrence 1996).

Figure 2.3
Trout production in Australia, 1990–96



Source: Brown et al 1997, ABARE 1998.

In 1997–98, 2118 tonnes of rainbow trout were produced throughout Australia, with a value of around A$13 million. Trout production figures since 1990–91 are presented in Figure 2.3.

Production has not increased greatly in the last five years. One limiting factor seems to be the supply of fresh, cold water. However, there is scope for expansion of this sector and improvements in efficiency and productivity through research on types of rainbow trout that tolerate higher water temperatures, and research on improved feeds, development of mid-season supplies of eyed ova and other methods of culturing ocean trout. There are many sites off the Australian coast with the right water temperature range and there is great potential for export of ocean trout, because the larger market size (1–3 kilograms) allows more options for 'value adding' to the product.

Markets

The majority of rainbow trout produced in Australia are sold domestically in wholesale fish markets or directly to restaurants, hotels and clubs (Treadwell et al 1991). Farmed trout are mainly marketed as whole fresh, frozen or smoked product.

The rainbow trout produced in Australia are of a high quality, and there is a small but stable export market in Hong Kong, Singapore, Malaysia and Taiwan. The export volume of fresh-chilled and frozen trout has risen from 17 tonnes in 1990–91 to 176 tonnes in 1995–96, with a value of A$2 million. Exports represent about 3–4 % of total trout production. The superior health status of Australian trout and the availability of eyed ova 'out of season' have assisted the development of export markets in the northern hemisphere for eyed ova.

2.2.2 NON-SALMONID MARINE FINFISH

The non-salmonid marine finfish industry is made up of a number of commercial fisheries that are managed by the Commonwealth Government and State/Territory governments, in many cases through cooperative arrangements. The status of the Commonwealth government managed fish stocks is reported each year by the Bureau of Rural Sciences in *Fishery Status Reports* (BRS 1998).

The **South-east Fishery** lies off the south-east coast, from Sydney to Kangaroo Island (South Australia). This is the main source of fresh fish domestically. It is a multispecies fishery, harvesting more than 100 species, although the bulk of the catch comes from 17 species, including blue grenadier, orange roughy, school whiting, tiger flathead, spotted warehou, redfish, Jackass morwong, ling and gemfish. In 1997, the landed value of the fishery was approximately A$64.8 million at point of sale.

The global **Southern Bluefin Tuna Fishery** is managed by the Commission for the Conservation of Southern Bluefin Tuna. The members of the commission did not agree on a total allowable catch for 1998. However, the members agreed to be bound by their previous national allocations of the total allowable catch, set in 1997 at 11,750 tonnes. Australia's national allocation totals 5265 tonnes annually. In 1997, around half of this was placed in grow-out cages off Port Lincoln, with the remainder harvested by pole and longline vessels. For 1997, the value of this fishery was A$40 million.

The **Eastern Tuna and Billfish Fishery** lies off the east coast, from north Queensland to eastern Tasmania. The main species targeted in this fishery include yellowfin tuna, bigeye tuna, skipjack, striped marlin and broadbill swordfish. In 1997–98 the total landed value of this fishery was A$32.4 million.

The **Great Australian Bight Trawl Fishery** is located off the coast of South Australia and Western Australia. The catch is predominantly deepwater flathead and Bight redfish, orange roughy and jackass morwong. In 1997–98 the total landed value was A$6 million.

Other fisheries such as the pilchard, jack mackeral and tommy rough fisheries are not as significant in terms of the production of fish for human consumption, but have a strategic importance to other fisheries. For example, approximately 30% of the bait used in the A$0.5 billion per annum rock lobster fishery is locally sourced (Jones and Gibson 1997). Similarly, these fisheries provide a major input to the southern bluefin tuna aquaculture industry and are a significant source of product for recreational fishing bait and the production of pet food and fishmeal.

2.3 Employment in Australian fisheries and aquaculture

Employment within the fishing industry has remained stable during the last 25 years. In 1964–65 approximately 13,000 people worked full time in the catching sector. In 1990, 14,000 people fished full time and a further 4000 were involved with processing the catch (Kailola et al 1993). There are no employment figures for related industries, such as vessel construction, maintenance services, and fishing gear and electronic equipment suppliers.

There are 1525 boats with licences to fish in Commonwealth waters and a further 7950 licences issued for State and Territory waters (ABARE 1998).

In 1994, a Tasmanian Salmonid Growers Association (TSGA) survey indicated that 450 people were employed in the Tasmanian salmonid aquaculture industry, most in rural areas that had high unemployment.

The salmon industry has also contributed to job growth in other industries as a result of its need for new equipment and mechanical maintenance, including for boats, outboard motors, diving equipment, packing materials, processing equipment, refrigeration and feed. Construction companies have erected new offices, factories and jetties, eg Nortas's new processing facility at Mornington, Aquatas's smokehouse expansion and the relocation of Tassal's administration into expanded office space in Hobart (TSGA 1994). Tassal has recently constructed a second processing plant at Huonville.

The rainbow trout industry has played an important role in tourism in Australia in many areas that have few other attractions. There are no figures on employment indirectly associated with salmonid recreational fishing, but in rural areas the proportion of people involved with the supply of goods and services is likely to be high. The tourism industry has also benefited from the growth of the Atlantic salmon industry. For example, Tasmania has become well known for its fine foods, and tourists visit to experience the local produce. Small trout farms gain much of their business as sources of local produce and operate in collaboration with other local producers such as wineries and cheese makers.

In 1998 an economic impact study[1] on the South Australian tuna industry showed the extent of employment associated with this industry. The study included the fishing activity associated with tuna farming but excluded the contribution associated with the export of tuna direct to Japan.

The study showed that a significant number of jobs are created as a result of direct and flow-on business activity. Tuna farms were responsible for the direct employment of over 800 people in the Port Lincoln area in 1998. In 1996–97 flow-on business activity was estimated to generate a further 760 jobs. The sectors of the economy with employment gains from tuna farming include fishing (205 jobs), trade (117), other manufacturing (74), property and business services (68), transport (68) and finance (47). For 1996–97 the study showed that, for each job generated directly in tuna farming, an additional 3.2 jobs were created elsewhere in the State. Thus, the flow-on figures quoted above probably underestimate the extent to which the tuna industry would generate indirect employment.

2.4 Recreational fishing

Recreational fishing is a popular pastime in Australia, with an estimated 25–32% of persons aged from 13 years and above participating and nearly 50% of people aged 5 to 15 participating (McIlgorm and Pepperell 1999). It has been estimated that more than 4 million people nationally participate in recreational fishing, with more than 50 million 'fishing days' annually (McIlgorm and Pepperell 1999).

McIlgorm and Pepperell (1999) examined previous studies of expenditure on recreational fishing in Australia and used the consumer price index to estimate the national expenditure on recreational fishing in 1998 terms. They conservatively estimated that the national expenditure was A$2926 million, of which 20% was direct expenditure (rods, reels, tackle, club membership);

1 EconSearch (1998), The economic impact of aquaculture in the Eyre Peninsula region and South Australia, 1996–97. Unpublished report prepared for Aquaculture Group, Primary Industries and Resources South Australia by EconSearch Pty Ltd, April 1988.

almost 50% was indirect expenditure (travel, accommodation, boat fuel, boat hire and other costs); and 30% was capital expenditure (boat purchase, maintenance, insurance and registration). This estimated expenditure should not be confused with the economic value of the recreational fishing sector.

Recreational fishing activities may be divided into freshwater and marine activities. In New South Wales, Queensland, South Australia and Western Australia, more than 72% of activity is in marine water. Victoria, the Northern Territory and Tasmania have the highest percentage (40–45%) of freshwater recreational activity.

Relevant expenditure estimates for finfish and other species in Australia are as follows:

- marine finfish, A$1603 million;
- freshwater finfish, A$584 million;
- marine other species, A$556 million; and
- freshwater crustaceans, A$184 million.

McIlgorm and Pepperell (1999) estimated that salmonids accounted for 41% of freshwater finfish expenditure nationally, making this sector worth approximately A$234 million. In Tasmania, New South Wales and Victoria salmonid fishing activity is significant. Trout fishing is important in Western Australia and South Australia.

McIlgorm and Pepperell (1999) discussed the potential impact of disease on recreational fishing in Australia. In estimating the economic and social impacts of disease, they considered the barramundi fishery in the Northern Territory and the national salmonid fishery. For the barramundi fishery, an outbreak of disease could reduce expenditure by as much as A$9.8 million. Should this last for five years, there would be a present value of A$42 million, assuming a 6% discount rate. For the salmonid recreational fishing sector, McIlgorm and Pepperell estimated a present value of A$1025 million (highest estimate) arising from the total collapse of expenditure nationally due to disease over a five-year period, assuming a 6% discount rate. These estimates are 'worst case scenarios'. It is not possible to predict how fishermen may change activities and redirect expenditure on recreational activity in the face of a disease outbreak. It is probable that anglers would turn to other fishing or recreational activities and that the net effect on expenditure, even in the event of a major outbreak of disease, would be minimal.

The Australian Quarantine and Inspection Service (AQIS) recognises that the benefits of recreational fishing to the welfare of Australians cannot be fully accounted for in economic terms. The pleasure derived by many Australians in pursuing this pastime is real, but difficult to measure. When AQIS conducts import risk analyses, it takes into account the social amenity of relevant sectors in considering the effect of the introduction and establishment of diseases.

2.5 Australian native fish and the environment

In conducting IRAs, AQIS takes into account the importance of maintaining biodiversity in considering the effect of the introduction and establishment of diseases.

Australia has approximately 217 recognised species and subspecies of native freshwater fish (Wager and Jackson 1993). Of these, 26 species from five distinct families had previously been placed in the order Salmoniformes (which then encompassed the Salmonidae and related families). However, this taxonomic relationship was unclear and it is now generally agreed that the Salmoniformes (Salmonidae) are not as closely related to the Osmeriformes (the Osmeroidea and Galaxoidea) as previously supposed. The order Salmoniformes now contains only one family, Salmonidae, to which no Australian native species belongs. Information on the native fish that are related to the salmoniforms may be found in Appendix 5.

The Commonwealth *Endangered Species Protection Act 1992* prescribes protection for species listed as vulnerable or endangered. Four of the 26 species discussed above as previously belonging to the order Salmoniformes are listed as endangered, and a further three are listed as vulnerable. Information on these species and the endangered species that occur in the same waters as introduced salmonids may be found in Appendix 5.

Australian marine waters contain representatives of most marine finfish families. Many of the species covered in these IRAs are closely related to species present in Australia.

2.6 Health status of finfish

Throughout the world, there is much less scientific information on the diseases of marine finfish than on livestock and avian species. One reason is that it is difficult to investigate disease in aquatic animals because the marine environment is extensive and variable and because a large number of species are involved. However, information on the health status of commonly cultured species, such as the salmonids, tends to be more comprehensive because disease events are more likely to be recognised in aquaculture enterprises than in wild fisheries.

In many cases, the presence of a disease agent is only recognised after an outbreak of clinical disease occurs. Diagnosis is usually based on evaluation of clinical, pathological, virological, bacteriological and parasitological findings, the interpretation of which may be confounded if the quality of specimens is poor. Diagnostic methods based on the detection and identification of viruses are often not available. While technology is improving, definitive diagnostic methods are generally limited to specialised laboratories and do not lend themselves to low-cost, large-scale testing as required in routine health screening programs.

2.6.1 SALMONIDS

There is limited information on the health status of salmonids when they were first introduced to Australia. Early records do not refer to health precautions (Clements 1988). However, by 1963, health measures, including inspection and treatment for disease, had been imposed by the Commonwealth–State Advisory Committee on the Importation of Exotic Food and Sport Fish (Francois 1963). The introduction of Atlantic salmon into Tasmania in the 1980s for the establishment of the salmonid industry was undertaken under strict health controls. The New South Wales government hatchery at Gaden was chosen as the source of the fish because the health status of its stocks was assured. The process took three years and cost A$0.5 million to successfully introduce high-quality fish.

Australian salmonid populations are free of many of the significant diseases that affect salmonid populations in other parts of the world. This claim is supported by the results of passive surveillance (long-term observation of these species in Australia and the absence of epizootics of infectious disease) and testing programs that monitor the occurrence of specific diseases and that support export certification.

The Australian salmonid industry conducts surveillance and monitoring programs to detect disease in hatchery-bred fish used in aquaculture and for augmenting wild populations. Additionally, hatcheries that export live fish or eggs conduct testing programs to support export certification that attests to the absence of diseases of concern to importers. While the intensity of surveillance and the sophistication of testing methods used differ between States, on a national basis Australia has a high level of confidence that many significant salmonid diseases are not present.

The following disease agents and diseases of salmonids have not been reported in Australian salmonid fish in the field:

- *Aeromonas salmonicida* (typical and some atypical strains)
- *Piscirickettsia salmonis* (piscirickettsiosis)
- *Renibacterium salmoninarum* (bacterial kidney disease)
- *Vibrio salmonicida* (Hitra disease)
- *Yersinia ruckeri* (virulent Hagerman strains)
- erythrocytic necrosis virus
- *Herpesvirus salmonis* type 1
- infectious haematopoietic necrosis virus[2]
- infectious pancreatic necrosis virus
- Pacific salmon anaemia virus (erythrocytic inclusion body syndrome)

2 A single episode of disease in the early 1960s was eradicated.

- salmon leukaemia virus (plasmacytoid leukaemia)
- salmon pancreas disease virus
- viral haemorrhagic septicaemia virus
- *Enterocytozoon salmonis*
- *Loma salmonae*
- *Ceratomyxa shasta* (ceratomyxosis)
- *Henneguya salminicola*
- *Myxobolus cerebralis* (whirling disease)
- *Parvicapsula* sp
- proliferative kidney disease agent
- Rosette agent
- *Oncorhynchus masou* virus
- infectious salmon anaemia virus
- *Hexamita salmonis*
- *Microsporidium takedai*
- nervous mortality syndrome
- Japanese new virus
- *Lepeophtheirus salmonis*
- *Gyrodactylus salaris* (Gyrodactylosis)

There is evidence that the following disease agents and diseases may be present in salmonids that occur in Australia. Some of these have been isolated from salmonid and non-salmonid species; others have been isolated from salmonids in the absence of disease; and some are represented by only a limited number of strains.

- *Aeromonas salmonicida* (some atypical strains — the causal agent of goldfish ulcer disease has a restricted distribution)
- *Edwardsiella tarda*
- *Yersinia ruckeri* (some strains)
- *Vibrio anguillarum* (some strains)
- *Kudoa thyrsites*
- *Paramoeba pemiquidensis* (amoebic gill disease)
- *Vibrio ordalii*
- Epizootic haematopoietic necrosis virus (limited to south-eastern mainland Australia)
- A condition similar to plasmacytoid leukaemia has been identified in Atlantic salmon in Tasmania
- Aquabirnavirus[3] (not identified as a cause of disease)

2.6.2 NON-SALMONID MARINE FINFISH

The following information is based on published scientific literature, reports provided by Commonwealth and State/Territory government agencies, and official notifications to regional and international organisations. Additionally, AQIS has drawn upon published and unpublished information held by Commonwealth and State and Territory government agencies, universities, industries and research organisations.

The following salmonid and/or non-salmonid marine finfish disease agents and diseases listed by the Office International des Epizooties (OIE, or World Organisation for Animal Health; see Section 1.3.2, Box 1.1) have not been identified in Australia.

3 In a personal communication, Dr M Crane advised AQIS that in 1998 an aquabirnavirus had been isolated in Australia from farmed Atlantic salmon (in apparently healthy fish and in 'pinheads'), rainbow trout, wild flounder, cod, spiked dogfish and ling on the west coast of Tasmania. This virus is currently being characterised and its precise relationship to other aquabirnaviruses is not yet known. Experimental transmission of this virus to young salmonid species indicated that the virus is of low pathogenicity to brook trout and Atlantic salmon and hence should not be described as IPNV (M Crane pers. comm.).

Diseases notifiable to the OIE	Other significant diseases
Infectious haematopoietic necrosis	Infectious pancreatic necrosis[4]
Oncorhynchus masou virus disease	Piscirickettsiosis
Viral haemorrhagic septicaemia	Gyrodactylosis

The following finfish disease agents and diseases listed by the OIE occur in Australia.

Diseases notifiable to the OIE	Other significant diseases
Epizootic haematopoietic necrosis	Viral encephalopathy and retinopathy
	Epizootic ulcerative syndrome

Many native species, especially those that are not commercially important or being examined for their aquaculture potential, have not been sampled or tested for the presence of disease agents. Very few disease epizootics in native fish are attributed to infectious disease on the basis of laboratory investigation of 'fish kills' or clinical disease events.

According to C Rodgers (pers. comm. 1999), uncertainty regarding the susceptibility of native species to exotic disease agents (due to the unique taxonomic status of some species in Australia and the lack of experimental challenge studies) could be used as an argument against the conclusion that virulent strains recorded overseas are different from those found in Australia. In other words, it is possible that endemic counterparts of exotic pathogens occur in Australia, but have not been recognised.

Notwithstanding this advice, AQIS has assessed the issue of susceptibility in a conservative manner, taking into account the advice of relevant experts.

Details of Australia's regulatory control system for the health of salmonids and non-salmonid marine finfish, including arrangements for surveillance and monitoring, are shown in Appendix 6.

4 See footnote 3

Part 2
Salmonid fish

Chapter 3
Hazard identification: salmonids

3.1 Method

MANY DISEASE AGENTS HAVE BEEN REPORTED in association with salmonids or salmonid product. The disease agents considered in this section include those identified by the Australian Quarantine and Inspection Service (AQIS) in the course of a comprehensive scientific review as well as the diseases nominated in the course of AQIS's previous consultations with stakeholders. This section also includes salmonid diseases listed in the New Zealand Government's salmonid import health risk analysis (Stone et al 1997b) and the aquatic animal pathogens listed by the World Organisation for Animal Health (Office International des Epizooties, OIE 1997a).

In identifying hazards that may be associated with products derived from salmonids, AQIS is aware that the availability of scientific data reflects the research effort committed to the investigation of disease in relevant species. Moreover, there is more information on disease in aquacultured species than in wild fish. Keeping fish in the artificial environment of a farm or aquarium makes it easier to detect disease, because these fish can be more closely observed and because suboptimal environmental conditions or husbandry may result in the clinical expression of otherwise unapparent infections.

In this section, AQIS considers diseases and disease agents against several criteria to determine whether they should be given detailed consideration in the import risk analysis (IRA). A disease or disease agent was given detailed consideration in the IRA if it was:

1. infectious; **and**
2. (a) exotic to Australia, **or**
   (b) present in Australia but subject to official control; **and**
3. (a) OIE listed, **and / or**
   (b) would be expected to cause significant harm in Australia.

Box 1.2 in Section 1.5 gives further details of these criteria.

Table 3.1 shows the classification of salmonid diseases and disease agents according to these criteria.

Table 3.1
Salmonid disease agents

DISEASE AGENT/PEST	1 DISEASE AGENT IS INFECTIOUS	2A AGENT OR STRAIN EXOTIC TO AUSTRALIA	2B CONTROL PROGRAM IN AUSTRALIA	3A OIE-LISTED	3B SIGNIFICANT DISEASE	FURTHER CONSIDERATION OF DISEASE AGENT IS REQUIRED
Viruses						
Erythrocytic necrosis virus	Y	Y	N	N	Y	Y
Herpes virus salmonis type 1	Y	Y	N	N	N	N
Infectious haematopoietic necrosis virus	Y	Y	N	Y	Y	Y
Infectious pancreatic necrosis virus	Y	Y[a]	N	Y	Y	Y
Infectious salmon anaemia virus	Y	Y	N	Y	Y	Y
New Japan virus	Y	Y	N	N	Y	Y
Oncorhynchus masou virus	Y	Y	N	Y	Y	Y
Pacific salmon anaemia virus — erythrocytic inclusion body syndrome	Y	Y	N	N	Y	Y
Salmon leukaemia virus — plasmacytoid leukaemia	Y	Y	N	N	Y	Y
Salmon pancreas disease virus/sleeping disease of rainbow trout	Y	Y	N	N	Y	Y
Viral haemorrhagic septicaemia virus	Y	Y	N	Y	Y	Y
Bacteria						
Aeromonas salmonicida — atypical	Y	Y[b]	N[e]	N	Y	Y
Aeromonas salmonicida — typical	Y	Y	N	N	Y	Y
Edwardsiella tarda	Y	N	N	N	Y	N
Piscrickettsia salmonis	Y	Y	N	Y	Y	Y
Renibacterium salmoninarum	Y	Y	N	Y	Y	Y
Vibrio anguillarum	Y	Y[b]	N	N	Y	N[d]
Vibrio ordalii	Y	N	N	N	Y	N
Vibrio salmonicida	Y	Y	N	N	Y	Y
Yersinia ruckeri (Hagerman strain)	Y	Y	N	N	Y	Y
Protozoans						
Ceratomyxa shasta	Y	Y	N	N	Y	Y
Dermocystidium spp	Y	Y	N	N	N	N
Enterocytozoon salmonis	Y	Y	N	N	Y	Y
Henneguya salminicola	Y	Y	N	N	Y	Y
Hexamita salmonis	Y	Y	N	N	Y	Y
Kudoa thyrsites	Y	N	N	N	Y	N
Loma salmonae	Y	Y	N	N	Y	Y
Microsporidium takedai	Y	Y	N	N	Y	Y
Myxobolus cerebralis	Y	Y	N	N	Y	Y
Parvicapsula spp	Y	Y	N	N	N	N
Proliferative kidney disease agent	Y	Y	N	N	Y	Y
Idiopathic diseases						
Nervous mortality syndrome	Y	Y	N	N	Y	Y
Rosette agent	Y	Y	N	N	Y	Y
Metazoans						
Gyrodactylus salaris	Y	Y	N	Y	Y	Y
Lepeophtheirus salmonis	Y	Y	N	N	Y	Y
Caligus elongatus	Y	N	N	N	Y	N
Other metazoans	Y	N[c]	N	N	N	N

Y = yes; N = no

a A non-IPNV aquabirnavirus has been reported.

b Some strains occur.

c Numerous species have been reported but few identified at species leve.

d This pathogen was rated 'Y' — for further consideration — in the draft of this report. The rationale for changing this rating is set out in the text.

e No movement controls apply to non-viable fish/fish products.

3.1.1 DISEASES DUE TO INFECTION WITH VIRUSES OR BACTERIA

In a personal communication to AQIS, A McVicar stated that there is increasing evidence that several 'species' of virus include substantially different organisms under the same name, and that the inadequacy of the diagnostic methods currently available prevents their distinction. A McVicar stated that infectious pancreatic necrosis virus (IPNV) and viral haemorrhagic septicaemia virus (VHSV) are known to include quite diverse infective agents, as indicated by their host ranges and pathogenicity. Similarly, the range of diseases associated with nodaviruses is assuming much greater significance in fish farmed in seawater. Fish farmed in both warm water and cold water appear to be susceptible to infection with nodaviruses, and there may be several virus species involved.

Although scientific knowledge on many strains of viruses and other pathogens is not sufficiently well developed to provide a clear basis for legislative controls, AQIS acknowledges that there is evidence that different pathogens are present in foreign countries, and takes this into account in the IRA.

For pathogens that have been recorded in Australia, AQIS has carefully considered the evidence for the presence of exotic strains overseas. In the case of agents that have been reported sporadically or exceptionally in Australia and for which there are few data, it may be difficult to determine if strains reported overseas are more pathogenic and should be considered in the IRA. This is particularly the case for agents that have not been identified to species level. Many pathogenic bacteria (eg *Mycobacterium* spp, *Nocardia* spp *Edwardsiella tarda* and *Vibrio ordalii*) have been excluded from consideration in the IRA on the basis that strains of similar virulence to, or greater virulence than, those that occur overseas are found in Australia. However, where there is evidence for the existence of significantly more pathogenic strains overseas, these agents have been included in the IRA for further consideration (eg *Yersinia ruckeri, Aeromonas salmonidica* (atypical forms) and aquatic birnaviruses, known as aquabirnaviruses).

3.1.2 DISEASES DUE TO INFESTATION WITH PROTOZOAN AND METAZOAN PATHOGENS

In a personal communication to AQIS, A McVicar stated that, in general, diseases due to parasitic infestations are not considered to present the same level of risk of introduction or establishment in a new area as those caused by bacteria and viruses. Many parasites (protozoans and metazoans) have a sufficiently discontinuous distribution to be used as natural indicators of host stock history (this pattern is sufficiently strong for a scientific discipline to be established around the phenomenon).

For diseases due to protozoan parasites, many agents have been shown to cause significant pathology in individual fish, but there are few data on the effects on fish populations. Protozoan infestations can cause serious diseases and the species listed in this section for further consideration are recognised as among the most significant pathogens in this group. For most protozoans, it is unlikely that free-living stages would survive for any significant period in a dead fish (A McVicar pers. comm.).

AQIS has considered the parasitic metazoans associated with salmonids. This is a very large group of organisms and for many species/genera there is little information on distribution (including in Australia), host range and pathogenic significance. There are very few records of serious disease epizootics due to metazoan infestations in wild fish. With certain exceptions (specified below), AQIS will not give metazoans further consideration in this IRA, for the following reasons.

While there are some exceptions, it is generally the case that infestation with metazoan organisms, in the absence of additional stressors such as overcrowding, insanitary environmental conditions or intercurrent disease, is of minor significance to the vertebrate host.

Most of the metazoa are obligatory parasites that display varying degrees of host-specificity. Many (but not all) have life cycles that involve several host animals. Although some species have free-living stages, generally speaking parasites would not survive beyond about 48 hours in a dead host that has been removed from the aquatic environment. Moreover, freezing the product would rapidly kill any metazoan parasites that may be

present (this is an important step in treating fish for consumption in raw form that may contain metazoan parasites of public health concern).

Many metazoan parasites are big enough to be seen on the fish and removed during inspection of the product. Most of the metazoans that infest the internal organs and the gastrointestinal tract would be removed from the product at the time of evisceration.

In a personal communication to AQIS, B Jones (1999) stated that many genera of metazoan parasites have been recorded in fish in Australian waters and in most cases these species have not been defined. There is a growing literature on the taxonomic relationships of the Australian aquatic parasite fauna with the parasite fauna of neighbouring regions. These studies show that the relationships are complex and often reflect the faunal groupings of the host animals and historical migration and movement patterns. There are no mandatory controls in Australia to address endemic diseases due to metazoan parasites.

3.1.3 USE OF CONSERVATIVE JUDGMENT

Where definitive data relevant to this process of classification are lacking, AQIS makes conservative judgments based on current scientific information and the advice of experts in relevant fields.

3.2 Classification of diseases and disease agents

3.2.1 VIRUSES

This section describes the distribution and effects of 11 viruses known to affect salmonids.

Erythrocytic necrosis virus or viral erythrocytic necrosis

In this IRA, erythrocytic necrosis virus (ENV) is defined as the iridovirus that causes viral erythrocytic necrosis (VEN). Other viruses that can cause erythrocytic necrosis in salmonids, such as the togavirus, which causes erythrocytic inclusion body syndrome (EIBS), are considered in appropriate sections of this chapter.

VEN is a viral infection causing erythrocytic abnormalities in at least 17 families of marine and anadromous fish, including Atlantic cod, Atlantic and Pacific herring and Pacific salmonids (review by Humphrey 1995).

VEN does not cause high mortalities in salmonids, but it impairs fish health and production (Nicholson and Reno 1981). Detrimental effects on the health of infected salmonids include a decreased capacity to regulate sodium and potassium, a significantly decreased tolerance to oxygen depletion and a threefold greater mortality from vibriosis (MacMillan et al 1980).

Postmortem findings ascribed to ENV are pale gills, pale internal organs and hyperactive haematopoietic tissue (Dannevig and Thorud 1999). VEN has been reported from Europe, the United States, Canada and Greenland. It is suspected to be present off the coast of Portugal (review by Humphrey 1995, Dannevig and Thorud 1999).

While the impact of ENV on salmon populations is not clearly understood, recent studies suggest that the disease has a greater effect on fish populations than previously believed (Haney et al 1992).

ENV has not been recorded in Australia. This agent may cause significant disease. Accordingly, it will be the subject of further consideration in this IRA.

Herpes virus salmonis — herpes virus type 1

There has been only one record of herpes virus salmonis (HPV) in association with fish mortalities. It was detected in a group of salmonid broodstock fish affected by post-spawning mortality in a Washington hatchery in 1975 (Eaton et al 1991). HPV has not been reported to cause clinical disease in farm or wild salmonids, although disease may be induced by experimental infection. Experimental studies suggest that the virus is of low virulence for rainbow trout and chinook salmon (Eaton et al 1989). Coho salmon and brown trout appear to be refractory to infection (Eaton et al 1989).

There is little evidence for a causal association of HPV with significant clinical disease. Accordingly, this agent will not be the subject of further consideration in this IRA.

Infectious haematopoietic necrosis virus

Infectious haematopoietic necrosis virus (IHNV) causes a serious, systemic disease associated with anaemia, ascites and haemorrhage. Spinal deformities may be

seen in salmonids that have survived infection (Bruno and Poppe 1996). The disease has been recorded in central, eastern and northern America, Canada, Japan and southern Europe, and in wild and farmed fish.

Disease epizootics with mortality rates as high as 100% have occurred in farmed juvenile salmonids. Significant losses have been recorded in chinook and sockeye salmon and steelhead trout (Follet and Burton 1995, Wolf 1988). Atlantic salmon are particularly susceptible to infection, which may be transmitted by bath and cohabitation exposure (Traxler et al 1993). The impact of disease due to IHNV has increased to the point where effective prevention of this disease has become central to the successful rearing of many salmonid species (Fryer 1986). There are no effective treatments or prophylactic measures for IHNV (Bruno and Poppe 1996).

IHN is listed by the OIE as a notifiable disease. It does not occur in Australia. Accordingly, IHNV will be the subject of further consideration in this IRA.

Infectious pancreatic necrosis virus

In this IRA, 'infectious pancreatic necrosis' (IPN) describes the acute disease of juvenile salmonids caused by infection with an aquabirnavirus. The various strains of virus that cause infectious pancreatic necrosis — referred to as infectious pancreatic necrosis virus (IPNV) — differ in virulence and serological characteristics.

Hill and Way (1995) reviewed the serological classification of aquabirnaviruses, many of which are serologically related to reference strains (Ab, Sp and VR299) of IPNV. Some of these viruses were isolated from non-salmonid fish and can be called IPNV as they produce IPN in salmonid fry. There is no evidence that many of the aquabirnaviruses that are serologically related to IPNV are pathogenic in salmonids; they should therefore not be described as IPNV (Hill and Way 1995).

IPNV is widespread in Europe (including the United Kingdom), North America and Asia and it continues to be the main viral problem in both farmed Atlantic salmon smolts following transfer to seawater and in many freshwater salmon hatcheries in Norway (OIE 1999). It has also been reported from Chile after being undetected for over 10 years (OIE 1999).

Disease due to infection with IPNV is characterised by severe damage to the pancreas and other organs of farmed salmonids, particularly fry and fingerlings. Severe disease epizootics with mortality rates as high as 100% have been recorded (Kimura and Yoshimizu 1991, Reno 1999). Fish that survive infection with IPNV develop immunity but may become asymptomatic carriers of infection for the rest of their lives (Wolf 1988).

IPNV has not been reported in Australia or New Zealand. Routine histopathological diagnosis and surveys in Australia have not detected IPNV (DPIE 1996). However, while sea-caged salmon have been extensively tested, free-ranging anadromous salmon from Australia have not, and it is possible that these fish, which would have access to marine molluscs and crustaceans, would pick up birnavirus (Jones and Gibson 1997).

In a personal communication, Dr M Crane advised AQIS that in 1998 an aquabirnavirus had been isolated in Australia from farmed Atlantic salmon (in apparently healthy fish and in 'pinheads'), rainbow trout, wild flounder, cod, spiked dogfish and ling on the west coast of Tasmania. This virus is currently being characterised and its precise relationship to other aquabirnaviruses is not yet known. Polymerase chain reaction analysis of viral nucleic acid indicates that the virus appears to be more closely related to IPNV fr21 and N1 isolates than other birnavirus isolates available for comparison. The Australian isolate is neutralised by an antiserum raised against IPNV Ab strain and by a commercial IPNV monoclonal antibody. Further analysis is required to confirm this relationship. Experimental transmission of this virus to young salmonid species indicated that the virus is of low pathogenicity to brook trout and Atlantic salmon and hence should not be described as IPNV (M Crane pers. comm.).

As a result of the discovery of the aquabirnavirus in Macquarie Harbour on the west coast of Tasmania, this area has been proclaimed a disease control zone. Restrictions on movement of live farmed salmonids from the zone and protocols for treatment of nets and processed fish have been developed; that is, all harvested fish from the area must be appropriately gilled and eviscerated, and gills and viscera are to be buried. There are no restrictions on wild-caught fish from the area.

A marine aquabirnavirus with characteristics of the IPNV group has been detected a number of times in healthy sea-run quinnat salmon in New Zealand. Clinical signs of disease due to birnavirus infection have never been observed in New Zealand and the virus has had no impact on salmon farming (Anderson 1996).

IPN is listed by the OIE as an 'other significant' disease. Disease due to IPNV infection has not been reported in Australia. Accordingly, IPNV will be the subject of further consideration in this IRA.

*Infectious salmon anaemia (***orthomyxo-*like virus)***

Infectious salmon anaemia (ISA) is a significant disease of salmonid fish. It has been identified in Norway, Canada and Scotland, and is associated with severe mortality rates in affected stock (ISA Workshop 1997, Binde 1997, Anon 1998). Disease due to systemic infection with ISA is characterised by anaemia, ascites, congestion and enlargement of the liver and spleen, and generalised haemorrhage (Hovland et al 1994). With the exception of one outbreak in a freshwater hatchery, where the source of the virus was not defined, disease is only seen in fish that have been exposed to seawater. This virus is considered to be of low virulence, although disease epizootics may be associated with high mortality rates (Hastein 1997).

Atlantic salmon are the only species known to be naturally susceptible to ISA. Brown trout and rainbow trout may be experimentally infected with the virus, although these species do not succumb to clinical disease (Totland et al 1996, Nylund et al 1997). Infection may be transmitted between fish via direct contact or by exposure to large volumes of organic material, such as mucus, blood and viscera, for example via effluent from slaughterhouses and processing plants (Hastein 1997).

ISA is listed by the OIE as an 'other significant' disease. Disease due to ISAV infection has not been reported in Australia. Accordingly, ISAV will be the subject of further consideration in this IRA.

New Japan virus

A newly described retro-like virus has been isolated from salmonids in northern Japan. It affects the brain tissue of coho salmon, rainbow trout, char and ayu, and the ovarian fluid of masou salmon (Oh et al 1995). Disease is characterised by abnormal swimming behaviour, exophthalmia and anorexia. Mortality rates may reach 26%.

The New Japan virus may cause significant disease. It has not been reported in Australia; accordingly it will be the subject of further consideration in this IRA.

Oncorhynchus masou *virus — herpes virus type 2*

Oncorhynchus masou virus (OMV) causes skin ulceration in salmonid fish. Systemic infection may lead to oedema and haemorrhage and may result in death (Bruno and Poppe 1996). Natural infection of sockeye and masou salmon has been recorded. Rainbow trout, chum and coho salmon have been shown to be susceptible to infection (Wolf 1988). This disease has been reported only in Japan.

The OIE classifies disease due to OMV infection as a notifiable disease. Disease due to OMV has not been reported in Australia. Accordingly OMV will be the subject of further consideration in this IRA.

Pacific salmon anaemia virus

Infection with Pacific salmon anaemia virus (PSAV) is associated with erythrocytic inclusion body syndrome (EIBS). This recently described syndrome is associated with severe anaemia and mortality in farmed freshwater and saltwater salmonids, and frequently with other infectious diseases such as bacterial kidney disease, bacterial coldwater disease and fungal infection.

In North American Pacific salmon, anaemia and secondary bacterial or fungal infections were consistently associated with EIBS infection. In Japan, significant anaemia and resultant mortality were found in association with EIBS (Rodger and Richards 1998). EIBS-like virus infection has recently been described in Atlantic salmon, in association with intercurrent disease and in otherwise healthy fish in Norway and Ireland (Rodger et al 1991).

Infection with PSAV appears to cause significant disease. Disease due to PSAV has not been reported in Australia. Accordingly, PSAV will be the subject of further consideration in this IRA.

Salmon leukaemia virus (plasmacytoid leukaemia)

Plasmacytoid leukaemia due to infection with salmon leukaemia virus (SLV) has caused extensive mortality in chinook salmon in British Columbia (Eaton and Kent 1992). Fish affected with this disease have darkened skin, are lethargic and show abnormal swimming behaviour. Clinical signs include anaemia, exophthalmia, petechial haemorrhage and enlargement of the spleen and kidney (Kent et al 1990). While clinical infection of Atlantic salmon and sockeye salmon has been induced experimentally, disease has not been reported in these species under natural conditions.

Plasmacytoid leukaemia is a potentially significant disease. Disease due to SLV has not been reported in Australia. Accordingly, SLV will be the subject of further consideration in this IRA.

Salmon pancreas disease virus

Infection with salmon pancreas disease virus (SPDV), a togavirus, causes one of the most serious diseases affecting the farmed salmon industry. Pancreas disease has been recorded in Scotland (McVicar 1987), Ireland (McLoughlin 1995), Norway, the western states of the United States, France and Spain (Houghton 1994). Mortality rates as high as 60% have been reported (McLoughlin 1995). The major economic effect of the disease results from substantial retardation of growth in the critical growing season and the need to cull affected fish (McVicar 1986). Atlantic salmon are thought to be the only salmonid species susceptible to infection with SPDV.

In France, a disease known as 'sleeping disease', caused by a togavirus, occurs in freshwater rainbow trout. Affected fish are lethargic, do not feed properly and fail to thrive. It is thought that sleeping disease is related to SPDV; however, the association between the two conditions is unclear (Boucher and Baudin-Laurencin 1996).

Subacute to chronic infection with SPDV may cause necrosis of the pancreas and skeletal and cardiac muscle (Houghton 1994). Such infections are associated with clinical signs of lethargy, anorexia and abnormal swimming behaviour. Some cases result in death.

Infection with SPDV may cause significant disease. Disease due to SPDV has not been reported in Australia. Accordingly, SPDV will be the subject of further consideration in this IRA.

Viral haemorrhagic septicaemia virus

VHSV typically causes profuse haemorrhage and the rapid onset of mortality (Wolf 1988). Rainbow trout are the most susceptible salmonids, although other salmonids and non-salmonids may become infected in fresh water and saltwater (Meier et al 1994, Bruno and Poppe 1996). Mortality rates may be as high as 100% in fresh water and 80% in seawater (Bruno and Poppe 1996).

VHS is considered to be the most widespread and contagious viral disease affecting rainbow trout production in Europe. Disease outbreaks have occurred in Europe, Japan and North America (Wolf 1988, Smail 1999).

A number of biotypes of VHSV have been identified by genomic analysis, which has shown that isolates from Europe and North America are genotypically heterogeneous. Isolates from non-salmonid finfish have been identified as related to, but distinct from, those found in salmonids (Oshima et al 1993).

VHS is listed by the OIE as a notifiable disease. Disease due to VHSV has not been reported in Australia. Accordingly VHSV will be the subject of further consideration in the IRA.

3.2.2 BACTERIA

Aeromonas salmonicida — *'atypical' and 'typical' strains*

Aeromonas salmonicida causes a number of acute to chronic disease syndromes of fish including furunculosis, goldfish ulcer disease, carp erythrodermatitis and ulcer disease of flounder. There are currently four recognised subspecies of *Aeromonas salmonicida: A. salmonicida salmonicida*; *A. salmonicida masoucida*, *A. salmonicida achromogenes* and *A. salmonicida smithia* (Whittington et al 1995). Subspecies *salmonicida* includes isolates also described as 'typical'. Other subspecies are described as 'atypical'. The terms 'typical' and 'atypical' relate to growth and biochemical characteristics of isolates in culture.

Infection with *A. salmonicida* may develop into septicaemia (usually associated with typical isolates) or may be restricted to cutaneous ulcerative lesions (often associated with atypical isolates).

Infection with *A. salmonicida* subsp *salmonicida* has been recorded from salmonids and other fish (Hammel 1995), not always in association with clinical disease (Bricknell et al 1996). Other affected species include wrasse (*Labridae* spp) (Treasurer and Laidler 1994), turbot (*Scophthalmus maximus* (L.) (Nougayrede et al 1990), Atlantic cod (*Gadus morhua* (L.) (Willumsen 1990) and coalfish (*Pollachius viriens* (L.) (Willumsen 1990). Infection with this bacterium causes disease epizootics and major losses in wild and cultured salmonids. It is considered one of the most serious diseases of salmonids cultured in Canada, Norway and Scotland (Inglis et al 1993, review by Humphrey 1995, Husevag and Lunestad 1995). Typical *Aeromonas salmonicida* has not been reported in Australia.

The expression 'atypical *Aeromonas salmonicida'* was initially used to describe bacterial strains belonging to the species *A. salmonicida* that show biochemical characteristics different from those described for *A. salmonicida* subsp *salmonicida.* Although several subspecies of *A. salmonicida* have been described, many reports of atypical isolates have not been identified to subspecies level (Wahli et al 1992). As such, the following information on atypical *Aeromonas salmonicida* refers only to the disease caused, the host species involved and the geographical area in which the disease agent was isolated. Distinctions will be made only between typical and atypical isolates.

The number of published reports of disease outbreaks associated with atypical strains has increased significantly during the last decade, and these strains have been isolated from an increasing number of fish species and geographical areas (Wahli et al 1992). Atypical strains of *A. salmonicida* have been associated with high cumulative mortality in sea trout in Sweden (Wichardt 1983) and Atlantic salmon in Canada (Paterson et al 1980). They have caused losses of 15–25% of total production in Iceland (Güomundsüttir et al 1995) and have led to salmonid mortality in Japan (Bruno and Poppe 1996). Disease due to atypical *A. salmonicida* is a major economic constraint to salmonid culture in Newfoundland, with mortality rates of up to 29% (Groman et al 1992).

An atypical strain of *A. salmonicida* that causes goldfish ulcer disease is usually reported from non-salmonid species, but may cause disease in salmonids under experimental conditions (Carson and Handlinger 1988, Whittington and Cullis 1988). This pathogen has been isolated from goldfish and koi carp and is endemic in some regions of Australia. Accordingly, some Australian States have adopted internal quarantine measures for live fish to prevent the spread of this disease. However, there are no restrictions on the movement of non-viable fish (Carson and Handlinger 1988). Atypical infection with a marine strain distinct to the goldfish ulcer disease isolate has also been reported in flounder in Australia (Whittington et al 1995).

Exotic atypical strains and typical *A. salmonicida* may cause significant disease. As they are not present in Australia, they will be the subject of further consideration in this IRA.

Edwardsiella tarda

Infection with *Edwardsiella tarda* may cause septicaemia and abscessation of muscle tissues, skin, gills and internal organs. *E. tarda* has been isolated from catfish, eels, salmonids, whales, waterfowl and reptiles (J Carson pers. comm.). Infections have also been reported in Australia (Humphrey et al 1986), including in Australian eels (Eaves et al 1990) and diseased rainbow trout (Reddacliff et al 1996).

Edwardsiella tarda occurs widely, including in the United States, Asia and Africa. This organism is present in Australia, where there are no statutory control measures applied to control or limit its distribution. *E. tarda* will not be the subject of further consideration in the IRA.

Piscirickettsia salmonis

Infection due to *Piscirickettsia salmonis* (Piscirickettsiosis) is a newly recognised disease of salmonids farmed in marine and fresh water (Bravo and Campos 1989; Bravo 1994). This disease has caused high mortality rates and significant economic losses in the salmon farming industry of Chile. It has been described in Canada (Kent 1992; Brocklebank et al

1992), Norway (Olsen et al 1997) and Ireland (Palmer et al 1997).

Infection with *P. salmonis* has been reported in Atlantic, coho, pink, chinook and masou salmon, and rainbow trout (Cvitanich et al 1991, Kent 1992). Clinical signs of disease include lethargy, anaemia, necrosis of the haematopoietic tissue of the spleen and kidney, and haemorrhages throughout skeletal muscle tissues (Bravo 1994).

Infection with *P. salmonis* is listed by the OIE as an 'other significant' disease. This disease has not been identified in Australia and will be the subject of further consideration in this IRA.

Renibacterium salmoninarum

Bacterial kidney disease (BKD), caused by infection with *Renibacterium salmoninarum*, is a serious, slowly progressive, frequently fatal disease of cultured and wild salmonids in fresh and marine waters. The disease is endemic in wild salmonid populations of the Pacific coast of North America and has been reported from Western Europe, North America, Japan and Chile (Fryer and Saunders 1981, Hoffmann et al 1984, Sanders and Barros 1986).

Clinical signs of BKD may not be evident until the disease is well established. External signs typically include exophthalmos and skin lesions. Skin lesions may take the form of unruptured cysts containing blood cells and necrotic tissue, with large numbers of *R. salmoninarum*. In advanced cases, lesions may take the form of large shallow ulcers (Bullock and Herman 1988). Internal lesions include necrosis of the kidney and haemorrhages in the body wall and hind gut.

Fish are most commonly infected with *R. salmoninarum* in the freshwater stage of their life cycle. Disease may be carried through to the marine phase, impairing the adaptation of juvenile fish to seawater and causing death (Bullock and Herman 1988). The organism may be transmitted vertically. The disease is not readily prevented or treated.

The OIE lists infection with *R. salmoninarum* as an 'other significant' disease. This disease has not been identified in Australia and will be the subject of further consideration in this IRA.

Vibrio anguillarum, V. ordalii and V. salmonicida

Vibriosis is a disease caused by infection with bacteria belonging to the genus *Vibrio*. Members of the genus are ubiquitous in marine environments and include significant pathogens such as *Vibrio anguillarum, V. ordalii*, and *V. salmonicida*.

V. anguillarum is the most common and widespread of the pathogenic *Vibrio* species affecting fish (Egidius 1987). It is associated with systemic infection and localised infection of skin, resulting in ulceration. Mortality rates of up to 100% have been recorded in infected salmonids (Ransom et al 1984). Sixteen different serotypes of *V. anguillarum* have been reported. Most disease outbreaks have been ascribed to serotypes 1 and 2 (Grisez and Ollevier 1995). Disease due to infection with *V. anguillarum* occurs in all major fish-rearing countries in the northern hemisphere, including the United States, Japan, Canada, Norway, Denmark and Scotland.

V. anguillarum serotype 1 occurs in Australia (Carson 1990). Disease due to this pathogen is controlled by immersion vaccination of juvenile salmonids before stocking to sea pens. There are no mandatory controls in relation to this disease in Australia. Accordingly, it will not be further considered in the IRA. *V. anguillarum* serotype 2 has not been reported in Australia. This strain is no more pathogenic than the strain present in Australia. Because strains of the agent are present in Australia and the disease is under effective management, it is expected that, if any new strain of *V. anguillarum* became established, it could be controlled with similar methods. Accordingly, *V. anguillarum* is not further considered in this IRA.

In salmonids, infection with *V. ordalii* may cause a haemorrhagic septicaemia similar to, but less severe than, the disease caused by infection with *V. anguillarum* (Austin and Austin 1993). Although this pathogen is usually isolated from salmonids, it may cause disease in other marine fish species (Wards et al 1991). *V. ordalii* has been isolated from the water column and sediment in Tasmania (Cameron et al 1988) and it is not uncommonly isolated from diseased fish in Western Australia (B Jones pers. comm.). Accordingly, *V. ordalli* will not be the subject of further consideration in this IRA.

V. salmonicida may cause coldwater vibriosis or 'Hitra disease'. It has been recorded from, and is widespread in, North America, Norway and Scotland (Actis et al 1999). Infections have been recorded in salmonids and gadoids. Disease due to *V. salmonicida* is characterised by severe haemorrhage and necrosis of the internal organs (Nielsen and Dalsgaard 1991, Jorgensen et al 1989).

Infection with *V. salmonicida* may cause serious disease. This disease has not been recorded in Australia; accordingly, it will be the subject of further consideration in the IRA.

Yersinia ruckeri *(Hagerman strain)*

Infection with *Yersinia ruckeri* may cause a systemic disease known as 'enteric redmouth' in salmonids, the severity of which varies with the biotype of pathogen and the age and species of salmonid host. At least five serotypes of *Y. ruckeri* have been identified. The three most virulent serotypes may be grouped into type 1 (Inglis et al 1993, Austin and Austin 1993), also referred to as the 'Hagerman strain'. The other serotypes are considered to be relatively avirulent.

Although enteric redmouth occurs most commonly in rainbow trout, it has also been reported in Atlantic salmon, cutthroat trout, coho salmon, chinook salmon (Bullock and Snieszko 1979), brook trout (Cipriano et al 1987), brown trout and sockeye salmon (McDaniel 1971). Disease-related losses of up to 40% of fish infected with type 1 *Y. ruckeri* have been reported (Cornick 1990).

Two clonal types of *Y. ruckeri* occur in Australia, one of which shares characteristics with isolates from Europe and the United States, and one of which appears to be unique to Australia. Neither has been classified as Hagerman strain *Y. ruckeri* (Davies 1991).

Infection with Hagerman strain *Y. ruckeri* may cause significant disease. It does not occur in Australia and will be the subject of further consideration in this IRA.

3.2.3 PROTOZOANS

Ceratomyxa shasta

Ceratomyxosis is a disease of salmonid fish caused by infection with *Ceratomyxa shasta*. This parasite infests the intestinal tissues, causing high rates of mortality in diseased salmonids (Bartholomew et al 1992). Initial signs of infection may include darkening, lethargy and loss of appetite. As the disease progresses, the descending intestine and anus become swollen and the abdomen distended as a result of ascites (Bartholomew et al 1989).

C. shasta has been identified in salmonids from marine and freshwater environments from northern California, the north-west Pacific region of the United States and Canada (Bartholomew et al 1989).

Infection with *C. shasta* may cause serious disease. This disease has not been recorded in Australia and will be the subject of further consideration in this IRA.

Dermocystidium spp

Dermocystidium spp principally infect the gill lamellae, oral cavity and skin of a wide range of marine fish. Infected fish have a swollen abdomen with small, round, white cysts visible within the abdomen and on the gills. Heavy infestations may cause structural changes in the gills, resulting in anoxia and mortality. Cysts may also protrude through the body wall and cause a marked inflammatory response with haemorrhage and hyperplasia (Bruno and Poppe 1996).

Infections and, in some cases, mortalities have been recorded in Atlantic salmon, brown and rainbow trout in Europe and in chinook salmon in north-west America (Bruno and Poppe 1996, Olson and Holt 1995).

A *Dermocystidium*-like sp has been recorded in Australia (Langdon 1988, cited in Humphrey 1995). There is little evidence for a causal association of *Dermocystidium* spp with significant clinical disease. AQIS considers that the pathogenic potential of species that may be exotic to Australia is not sufficient to warrant further assessment of this agent in the IRA.

Enterocytozoon salmonis
(= Nucleospora salmonis)

Infection with this microsporidian parasite has been associated with a syndrome characterised by anaemia and mortality in chinook salmon and rainbow trout in California and Washington, and yearling steelhead trout in Idaho (Baxa-Antonio et al 1992). More recently, *E. salmonis* has been isolated from Atlantic salmon imported as eggs from the United States and reared in seawater in Chile (Bravo 1996). In this report the cumulative mortality rate reached 64%.

The pathological changes associated with *E. salmonis* infections are similar to those reported for plasmacytoid leukaemia. However, the nature of any association between *E. salmonis* and plasmacytoid leukaemia is unclear, as leukaemia can occur without infection with *E. salmonis* (Kent et al 1990). Infected fish show clinical signs including anaemia, lethargy, exophthalmos and swelling of the kidneys, spleen and intestinal tissues (Morrison et al 1990, Bravo 1996).

Infection with *E. salmonis* may cause significant disease. This disease has not been recorded in Australia and will be the subject of further consideration in this IRA.

Henneguya salminicola

This myxosporean parasite has been reported in five species of Pacific salmon common to the North American and Asian coasts (Boyce et al 1985). Infestation with *Henneguya salminicola* is potentially significant because it may cause cysts and soft flesh in muscle tissue, which reduces the commercial value of the fish (Garden 1992). There are marked differences in the prevalence of infection in salmon species and stocks, the order of decreasing prevalence by species being coho, sockeye, chinook, chum and pink salmon (Boyce et al 1985).

Henneguya spp are found in Australia in a number of fish species but are distinct from *H. salminicola* and have not been associated with disease in Australian salmonids.

Infestation with *H. salminicola* may cause significant disease. This disease has not been recorded in Australia and will be the subject of further consideration in this IRA.

Hexamita salmonis

Infestation with *Hexamita salmonis* (hexamitosis) primarily occurs under conditions of poor husbandry. These flagellates are opportunistic parasites of the upper intestine, pyloric caeca and gall bladder of salmonid species, including brown trout, rainbow trout, brook trout, lake trout and grayling (Lom and Dykova 1992). Symptoms of infection include swimming disorders, emaciation and lethargy.

More recently, disease characterised by significant mortality has been reported in farmed chinook salmon in British Columbia and in farmed Atlantic salmon in Norway (Bruno and Poppe 1996). In these cases, disease was characterised by swelling of the kidney and liver, abdominal distension, severe exophthalmos and abscessation, with mortality rates as high as 75%.

Four ornamental species of fish have been recorded as hosts of *Hexamita* spp in Australia. These *Hexamita* species are considered to be distinct from *H. salmonis* and have not been associated with disease in Australian salmonids.

Infestation with *H. salmonis* may cause significant disease. This disease has not been recorded in Australia and will be the subject of further consideration in this IRA.

Kudoa thyrsites

This parasite infests the muscle tissue of many species of marine fish, including salmonids. Disease results in liquefactive necrosis of the muscle tissues postmortem, significantly reducing the commercial value of diseased fish. The disease occurs in many countries of the world, including Australia, where it is not the subject of mandatory control.

Due to the presence of *Kudoa thyrsites* in Australia and the absence of mandatory controls, this disease will not be the subject of further consideration in this IRA.

Loma salmonae

Loma salmonae is a microsporidian parasite that causes the formation of xenoparasitic cysts (xenomas) in the gills, arteries, bulbous arteriosis, pseudobranch, choroid gland and kidney. The clinical signs of disease include

exophthalmos, ascites, haemorrhages of the caeca and fins, and petechial haemorrhages on the opercula and skin (Hauck 1984).

L. salmonae has been recorded from California (Wales and Wolf 1955), Washington State (Kent et al 1989) and British Columbia (Magor 1987). Infections have been recorded in chinook salmon, rainbow trout, coho salmon, sockeye salmon and brook trout (Morrison and Sprague 1983, Hauck 1984; Wales and Wolf 1955). Infection of non-salmonid fish has not been reported.

Infestation with *L. salmonae* may cause significant disease. This disease has not been recorded in Australia and will be the subject of further consideration in this IRA.

Microsporidium takedai

Microsporidium takedai is highly pathogenic and specific to salmonids. Several salmonid species are susceptible to infection, including sockeye, pink, chum and masou salmon; rainbow and brown trout; and Japanese char. Most reports of infestation with this microsporidium are in freshwater salmonids from Japan (Bruno and Poppe 1996). Infection prevalences of 100% have been recorded. Acute infections frequently cause high rates of mortality (Lom and Dykova 1992).

Infestation with *M. takedai* is characterised by the formation of whitish, spindle-shaped, cyst-like lesions in the musculature, including the heart. The lesions may be visible through the skin. In chronic cases, the heart becomes hypertrophic and deformed and there is inflammatory oedema.

Infestation with *M. takedai* may cause significant disease. This disease has not been recorded in Australia and will be the subject of further consideration in this IRA.

Myxobolus cerebralis

Infestation with *Myxobolus cerebralis* causes 'whirling disease' in salmonids. The spores of this parasite invade the cartilaginous tissues of the head and body, harming the nervous system. The clinical effects depend on the age of the fish at the time of infestation. In newly hatched fry, mortality rates may reach 100%, while fish infected at ages greater than six months show few or no clinical effects.

Whirling disease has been reported in many regions of the world, including Europe, Asia, and North and South America. The disease occurs with a very restricted distribution in New Zealand (Noga 1996). All salmonids, especially rainbow trout, are susceptible to infestation to varying degrees. Infestation with *M. cerebralis* has been reported in non-salmonids (Noga 1996), but the basis of this report is contentious.

The life cycle of *M. cerebralis* involves parasitic and free-living stages, with the development of infectivity for the salmonid host depending on the ingestion of spores by the intermediate host, a tubificid worm *Tubifex tubifex*. The spores develop into the infective triactinomyxon stage within the worm, then a susceptible salmonid fish may be infected via the skin or buccal cavity (Bruno and Poppe 1996). The intermediate host, required for completion of the life cycle, occurs in Australia.

Infestation with *M. cerebralis* may cause significant disease. This disease has not been recorded in Australia and will be the subject of further consideration in this IRA.

Parvicapsula spp

A *Parvicapsula* species found in the kidney of pen-reared marine coho salmon and other salmonid species was reported to cause severe disease in the early 1980s on the northern Pacific coast of the United States (Hoffman 1984). However, the pathogenic significance of this parasite was unclear because concurrent infection with BKD and *Vibrio* was frequently reported. Johnstone (1984) also reported infection with *Parvicapsula* spp in chinook, Atlantic and masou salmon and cutthroat trout.

More recently, Kent et al (1997) described *Parvicapsula minibicornis* from the kidney of wild sockeye salmon in British Columbia. No lesions were associated with this infection. It is difficult to ascertain whether *P. minibicornis* is the same species that was recorded in the studies by Hoffman (1984) and Johnstone (1984), because descriptions of the latter were taken from preserved material. Nevertheless, these descriptions suggest differences between the two *Parvicapsula* species. Furthermore, there are no records of disease

caused by *Parvicapsula* spp beyond those in 1984. Kent et al (1994) do not consider *Parvicapsula* spp to be an important pathogen in salmonids in British Columbia.

Infestation with *Parvicapsula* spp has been reported from two species of marine finfish in Australia. Like the *Parvicapsula* spp recorded above, the species recorded in Australia have not been identified at species level.

There is little evidence for a causal association of *Parvicapsula* spp with significant clinical disease. Accordingly, these agents will not be the subject of further consideration in this IRA.

Proliferative kidney disease agent

Proliferative kidney disease (PKD) is one of the most economically harmful diseases of salmonids in the United Kingdom, Canada and the United States. The causative agent is probably a *Sphaerospora* spp. Natural infestations have been reported in brown trout (Wootten and McVicar 1982), grayling, sockeye salmon (Kent et al 1993), chinook salmon (Hedrick et al 1993) and various salmonids (Bucke et al 1991). Infestations have been reported in cultured Atlantic salmon, brown trout and char (Bucke et al 1991). PKD has also been recorded in non-salmonid fish species (Bucke et al 1991).

Clinically, PKD is characterised by lesions in the kidney and in the red muscle of the lateral lines. Grossly, there are protuberances in the kidney and on the surface of the skin (Fernandez-de-Luco et al 1997). The disease is not usually associated with a sudden onset of significant mortality. Rather, the normal clinical picture is one of decreased production and increased mortality rates thought to be associated with immunosuppression. High mortality rates are frequently reported in association with concurrent bacterial infection.

Sphaerospora spp have been found in four species of marine finfish in Australia, but PKD has not been reported in salmonids or non-salmonids.

PKD is a potentially serious condition affecting salmonid and non-salmonid fish. This disease has not been recorded in Australia and will be the subject of further consideration in this IRA.

3.2.4 IDIOPATHIC DISEASES

Nervous mortality syndrome

Nervous mortality syndrome (NMS) is an economically significant condition affecting Atlantic salmon in Ireland. Clinical signs include lethargy, abnormal swimming behaviour, loss of balance and apparent unconsciousness. Mortality rates in post-smolt stock may be as high as 90% within four weeks of the onset of clinical signs (Rodger et al 1995). Although the aetiology of NMS has not been definitively described, small extrasporogenic stages of a myxosporean organism have been observed in association with clinical signs. Clinical and pathological evidence would suggest that NMS is an infectious disease caused by infestation with a myxosporean organism.

NMS is a potentially serious condition affecting Atlantic salmon. This disease is probably of infectious aetiology. It has not been recorded in Australia and will be the subject of further consideration in this IRA.

Rosette agent

The Rosette agent has not yet been defined; however, it has some similarities to a *Dermocystidium* sp.

The Rosette agent has been reported to cause serious disease in chinook salmon of the North American north-west Pacific (Kerk et al 1995). This disease has also been reported in freshwater rainbow trout in California (Hedrick et al 1989) and in marine-farmed Atlantic salmon on the Atlantic coast of Canada (Cawthorn et al 1990). Mortality rates as high as 95% have been recorded (Elston et al 1986).

A similar syndrome has been reported in rainbow trout in France, and in brown trout and Atlantic salmon in Ireland (Nash and Nash 1989).

Infections with the Rosette agent appear to be systemic. Clinical signs include anaemia, enlargement of the kidney and spleen, and granulomatous infection.

Although the Rosette agent has not yet been characterised, infection of salmonid fish may cause serious disease. The disease is probably of infectious aetiology. It has not been recorded in Australia and will be the subject of further consideration in this IRA.

3.2.5 METAZOANS

Other than as set out below, AQIS considers that the pathogenic significance of metazoan species that may be exotic is not sufficient to warrant further assessment in this IRA (see Section 3.1.2).

Gyrodactylus salaris

Gyrodactylus salaris is a highly contagious, parasitic freshwater monogenean of Atlantic salmon and rainbow trout in Scandinavia, Russia and a few southern European countries. It is thought that this parasite was introduced into Norway with smolts imported from Sweden, and that it has since decimated the Atlantic salmon stocks in 38 rivers (Lux 1991).

G. salaris occurs on the body surface and fins, where it attaches via hooks. The hooks apparently cause only superficial damage, but the wounds allow infection by opportunistic fungal and bacterial pathogens. Large ulcers often form as a result, sometimes with impairment of osmotic regulation and death (Bruno and Poppe 1996).

G. salaris causes a potentially serious infection that has not been reported in Australia. It will be given further consideration in this IRA.

Lepeophtheirus salmonis and Caligus elongatus (sea lice)

Lepeophtheirus salmonis and *Caligus elongatus* (sea lice) are significant pathogens of Atlantic and Pacific salmon in the northern hemisphere. These parasites are common in the wild but are rarely reported in large numbers on individual fish. In culture, however, sea lice can rapidly multiply and seriously threaten commercial production.

Initial infection with the copepod *L. salmonis* produces small white patches around the head, along the base of the dorsal fins and in the perianal areas. Progressive hyperplasia in these areas and the invasion of secondary pathogens is associated with severe ulceration, haemorrhage, oedema and exposure of the cranium and other areas of supporting tissue (Bruno and Poppe 1996). If infestation with large numbers of lice on salmonids is left untreated, high mortality is very likely (Bjordal 1994). This copepod has not been recorded in Australia and will be given further consideration in this IRA.

C. elongatus is a non-specific marine parasite of many species of fish including salmonids. Greyish patches on the back, particularly near the dorsal fin, indicate the first signs of infestation. The lice may erode the epidermal and subepidermal layers down to the basement membrane (Bruno and Poppe 1996). The ulcerative lesions are further extended by necrotising bacteria (Cusack 1995). In general, moribund fish can be expected to have high lice burdens. *C. elongatus* has been recorded in Australia on the leatherjacket, *Eubalicthyes mosiacus* (Hewitt 1971, cited in Humphrey 1995) and there are no movement restrictions relating to this agent. No further consideration will be given to this copepod in this IRA.

Chapter 4
Risk assessment: salmonids

4.1 Methods

IN CHAPTER 3, THE AUSTRALIAN QUARANTINE AND Inspection Service (AQIS) identified the disease agents that would be the subject of further consideration in the risk analysis, based on defined criteria. The criteria include the absence of the agent from Australia and features of the disease agent, including its ability to cause serious disease and its status according to the Office International des Epizooties (OIE, or World Organisation for Animal Health).

4.1.1 PRIORITY RANKING OF DISEASES

As a next step, AQIS identified the disease agents to be considered with higher priority, based on the probability of the disease becoming established in Australia, the consequences that would arise from such establishment and the assessment of disease agents in the Humphrey review (1995) (see Section 1.5.2). Disease agents for consideration with high priority were placed in group 1 and those for consideration with lower priority were placed in group 2. The priority rating of each pathogen is shown in Table 4.1.

This chapter covers all disease agents in group 1. Chapter 5 contains an assessment of disease agents in group 2 (see Section 5.5).

4.1.2 RISK ASSESSMENT

The risk assessment covers the following factors.

- *Release assessment* — the probability that the agent will enter Australia as a consequence of the importation of eviscerated salmonids.
- *Exposure assessment* — if the disease agent entered Australia in eviscerated salmonids, the probability of susceptible fish being exposed to a dose sufficient to cause infection.
- *Probability of disease establishment* — combining release and exposure assessment.
- *Consequence assessment* — the consequences of the disease agent becoming established in Australia.

Each of the above assessments is defined and described in qualitative terms in Section 1.5.3.

Table 4.1
Salmonid disease agents — priority in import risk assessment (IRA)

DISEASE AGENT	PROBABILITY OF ESTAB.	IMPACT OF ESTAB.	HUMPHREY SCORE[a]	PRIORITY	COMMENT
Viruses					
Erythrocytic necrosis virus	+	+	24	2	Reason for score 24 is not clear. ENV does not characteristically cause high morbidity/mortality overseas. It occurs in many many countries, but there is no evidence of active international spread.
Infectious haematopoietic necrosis virus	++	++/+++	27	1	
Infectious pancreatic necrosis virus	+	++/+++	26	1	
Infectious salmon anaemia virus	++	+++	NA	1	Serious disease with moderate probability of entry and establishment.
New Japan virus	+	+	NA	2	Disease occurs only in Japan; has not shown the propensity for international spread.
Oncorhynchus masou virus	+	++	21	1	
Pacific salmon anaemia virus — Erythrocytic inclusion body syndrome	+	+/++	18	2	Humphrey score <21
Salmon leukaemia virus — Plasmacytoid leukaemia	+	+/++	NA	2	Probability of establishment and impact of disease would be expected to be low.
Salmon pancreas disease virus/sleeping disease of rainbow trout	+	++/+++	15	1	Disease may have serious impact if it became established.
Viral haemorrhagic septicaemia virus	+/++	+++	26	1	
Bacteria					
Aeromonas salmonicida– atypical	+/++	++	28	1	
Aeromonas salmonicida– typical	++/+++	+++	28	1	
Piscirickettsia salmonis	+	++	24	1	
Renibacterium salmoninarum	++	++/+++	29	1	
Vibrio salmonicida	+	+/++	19	2	Humphrey score <21
Yersinia ruckeri[b] (Hagerman strain)	+	++	23	1	
Protozoa[c]					
Ceratomyxa shasta	+	+/++	19	2	Humphrey score <21
Enterocytozoon salmonis	+	+/++	18	2	Humphrey score <21
Henneguya salminicola	+	+/++	19	2	Humphrey score <21
Hexamita salmonis	+	+	15	2	Humphrey score <21
Loma salmonae	+/++	+/++	17	2	Humphrey score <21
Microsporidium takedai	+	++	21	1	
Myxobolus cerebralis	++	++/+++	24	1	
Proliferative kidney disease/ proliferative kidney disease agent	+	++	20	1	Disease may have serious impact if it became established.
Idiopathic diseases					
Nervous mortality syndrome	+	+/++	NA	2	Probability of establishment and impact of disease would be expected to be low.
Rosette agent	+	+	15	2	Humphrey score <21
Metazoans[c]					
Gyrodactylus salaris	+	+/++	24	1	
Lepeophtheirus salmonis	+	++/+++	19	1	Disease may have serious impact if it became established

a Disease score according to Humphrey (1995); NA = not scored.

b For *Yersinia ruckeri*, only the Hagerman strain is further considered.

c The myxosporeans are now classified as metazoans, not as protozoans. However, *M. cerebralis* is considered with the protozoa in this IRA.

4.1.3 UNRESTRICTED RISK ESTIMATE

The combined probability and consequences of disease establishment represent the unrestricted risk assessment (ie the risk if no management measures are applied). As presented in the risk evaluation matrix in Section 1.5.3, the unrestricted risk estimate either exceeds or meets the appropriate level of protection (ALOP). Risk management measures would be required (in the former case) or would not be justified (in the latter case).

The conclusions are summarised in a box at the end of the assessment for each disease agent.

4.2 Risk assessments for high priority diseases

4.2.1 INFECTIOUS HAEMATOPOIETIC NECROSIS VIRUS (INFECTIOUS HAEMATOPOIETIC NECROSIS)

Release assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Infectious haematopoietic necrosis (IHN) is listed under 'diseases notifiable to the OIE' and is included in List II of the European Union Directive 91/67/EEC.
- Infectious haematopoietic necrosis virus (IHNV) was confined to the Pacific coast of North America (from California to Alaska) until the early 1970s. The disease subsequently spread[1] to Japan, Taiwan, Korea, France, Belgium, Germany, Austria and Italy. The virus also spread to eastern North America, but has since been eradicated from this region.
- IHNV can infect many species of salmonids including sockeye salmon, sea-run cutthroat salmon, Atlantic salmon, chinook salmon, chum salmon and rainbow trout. Sockeye salmon and Atlantic salmon are considered the most susceptible species. The severity of disease outbreaks varies in fish populations from different sources.
- Challenge experiments on marine fish commonly found in and around salmonid net pens in British Columbia showed that tubesnouts (*Aulorhynchus falvidus),* shiner perch (*Cymatogaster aggregata*) and Pacific herring (*Clupea harengus pallasi*) are all susceptible to intraperitoneal inoculation with IHNV, with losses exceeding 50%. Herring species were the most susceptible to immersion challenge, with losses of 25% reported.
- In Alaska, surveillance data for the period 1980–1994 indicated an IHNV prevalence of 21.6% (2379 of 11,004) in Pacific salmon. In British Columbia, IHNV was detected in 2% (48 of 2331) of samples from wild, adult Pacific salmon submitted to the Pacific Biological Station in the period 1985–94. In Washington state, viral testing of salmon in the period 1991–95 showed 0.8% (399 of 51,947) to be infected. Japanese surveillance data for the period 1976–91 detected IHNV in 0.07% (11 of 15,432) mature females of five species of salmonid fish sampled in northern Japan.
- Clinical disease is most common in juvenile salmonid fish, especially fry and fingerlings. Disease outbreaks in fry or fingerlings may lead to mortality approaching 100%. Outbreaks in smolt normally result in low mortality. Severity of disease tends to decrease with age.
- Infection may occur but clinical disease is generally not seen at water temperatures higher than 15°C.
- In acutely ill fish, virus can be isolated from all major organs, though it is accepted that the virus is most abundant in the kidney, spleen, encephalon and digestive tract and virus is shed via the faeces, urine, sexual fluids and external mucus.

1 In a personal communication to AQIS, Dr B Hill noted that there is no firm evidence for the spread of IHNV from North America to other countries in the world. Rather, it has been assumed by some people that the first-time occurrence of this disease in a country, particularly in a different continent, must have been due to importation of salmonid eyed-ova from North America. Dr Hill stated that this is an assumption that is not supported by hard scientific evidence. It is quite possible that the virus had been naturally present for a long time in some affected countries but only recently detected by chance or due to increasing skills and facilities for fish disease investigation.

- Fish may survive infection to become chronic carriers. The location of virus in carrier fish is unknown, and virus can only be isolated immediately before, during or after spawning. In pre-spawning female salmon, viral titres were highest in the gills (10^2–10^5 plaque forming units [PFU]/g) and lower (10^0–10^4 PFU/g) in kidney, spleen, pyloric caecae, brain and eggs. In spawning fish, high titres of virus (10^6–10^9 PFU/g tissue) can be detected in most major organs including gill, kidney, spleen, pyloric caeca. In spawning females, titres as high as 10^8 PFU/mL and 10^6 PFU/mL have been reported in ovarian fluids and mucus respectively.
- Viral titres in gonadal fluid were consistently highest in one study of sexually mature carrier fish (Mulcahy et al 1982, cited in Stone et al 1997), which concluded that gonadal fluid was the sample of choice for detection.
- IHNV has been isolated from wild marine salmonid fish but this is unusual.
- Clinically infected fish are unlikely to pass inspection and grading. However, carrier fish would appear normal and would pass inspection.
- The titre of IHNV was reduced by three orders of magnitude after more than 20 weeks storage at 4°C. A single freeze-thaw cycle reduced the virus titre by four orders of magnitude (from 10^6 to 10^2). Under specialised laboratory conditions, virus preparations undergo several freeze-thaw cycles with little effect on infectivity for cell cultures when high concentrations of dissolved protein are present. However, in fish product, the effect of freezing is likely to be significant.

AQIS considered more recent information on IHNV, summarised below.

IHN was not described from Europe until 1987, when reports were presented to OIE of the occurrence of the disease in France (Baudin-Laurencin 1987) and Italy (Bovo et al 1987). In subsequent years infection was detected in other mainland Europe countries. This spread to other countries was discussed by International Council for the Exploration of the Seas, although not in great detail as outbreaks occurred in fresh water. It is widely accepted that IHNV has been spread by anthropogenic means, particularly through the movement of live rainbow trout. In continental Europe, the disease had already spread extensively in several countries before it was recognised that it had been introduced. It is subject to control at the national level in the UK, Ireland, Denmark, Sweden and Finland and at local or farm level in several other countries (A McVicar pers. comm.).

Outbreaks of clinical disease caused by IHNV associated with high mortality have occurred in farmed Atlantic salmon of market size in British Columbia in recent years (OIE 1999).

Recently, IHNV was reported for the first time from adult sockeye salmon in marine waters during their return migration to spawn (Traxler et al 1997). Natural infection has been reported in tubesnouts and shiner perch in net pens with salmonids experiencing an IHNV outbreak, and from Pacific herring associated with salmon farms (Kent et al 1998).

Under experimental conditions larval white sturgeon (*Acipenser transmontanus*) were challenged with large doses of IHNV. Viral replication and limited mortality were reported; however, juvenile fish and older stock were resistant to infection. These authors considered that white sturgeon could be a potential source of IHNV (LaPatra et al 1995).

Key findings

IHN is primarily a disease of young, farmed salmonids in fresh water, although outbreaks in market-sized Atlantic salmon in British Columbia are of increasing concern. Infection has been reported rarely in wild-caught marine salmonids. In salmonids, infection typically causes acute systemic disease in juvenile fish, especially fry and fingerlings, whereas infection of adult fish is usually covert.

Juvenile salmonid fish (the lifecycle stage most likely to have clinical disease) are not usually harvested for human consumption. Adult fish are less likely than juvenile fish to have clinical disease. Clinically infected fish would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. Adult fish surviving infection with IHNV may be inapparently infected. Such fish would appear normal

and would not be detected at inspection. In apparently healthy, eviscerated adult salmonids, the titre of virus, if any were present, would be extremely low (probably undetectable by traditional diagnostic methods).

In sexually mature fish returning to spawn, IHNV may be present in visceral organs and the brain.

In apparently healthy, infected fish of market size the location of virus is unknown, but most would presumably be located in visceral organs and the brain. In such fish, evisceration would substantially reduce the titre of virus present; however, virus may remain in other parts of the body, particularly the head. Unlike pathogens, such as *A. salmonicida* and infectious salmon anaemia virus, that may be widely dispersed in tissues of chronically infected fish, there is no evidence to suggest that IHNV would be in the somatic musculature of apparently healthy, market-size fish.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Susceptible salmonids in Australia include Atlantic salmon, chinook salmon, rainbow trout, brown trout and brook trout.

The life cycle of IHNV is direct and infection may be transmitted horizontally. The gills, gastrointestinal tract and possibly skin may be routes for infection. Pseudo-vertical transmission may occur as a result of external contamination of eggs by virus.

- Transmission of infection is known to occur at water temperatures below 15°C. Some inland and coastal waters of southern Australia would regularly be cooler than 15°C, providing conditions amenable to the establishment of disease.
- The minimum infective dose under natural conditions is not known, but would vary with several factors, including the age and species of fish, route of infection and strain of virus. Immersion of 1188 juvenile sockeye salmon in water containing 10^3 $TCID_{50}$ (median tissue culture infective dose) per mL for one hour was shown to initiate severe disease. Under experimental conditions, waterborne infection of yearling sockeye salmon with IHNV induced infection of gills, but infection did not become systemic except when viral titres in the gills exceeded 10^5 PFU/g.
- IHNV is a stable virus that may survive for weeks in fresh water, seawater and estuarine water at 15°C and for months in river sediment. IHNV is sensitive to dehydration, mildly sensitive to extremes of pH, readily inactivated by lipid solvents and disinfectants such as chlorine and iodine and is susceptible to ultraviolet irradiation.

AQIS considered the following additional information.

Rainbow trout with a 'neurotropic' form of IHN had high virus concentrations localised in brain tissue. In fish with the 'hematopoietic' form of IHN, the highest concentrations of virus were detected in kidney-spleen tissue. IHNV isolates obtained from brain or kidney-spleen tissue were tested for serological relatedness and virulence and pathogenicity differences in two sizes of rainbow trout. Virulence and pathogenicity differences were not evident (LaPatra et al 1995).

Key findings

IHN is primarily a disease of farmed, juvenile salmonids in fresh water. The minimum infective dose of IHNV would be higher in adult salmonids than in juvenile salmonids and may be high in juvenile salmonids relative to pathogens such as *Aeromonas salmonicida* for which the minimum infectious dose appears to be very low. Infection of non-salmonid fish with IHNV is unlikely to occur, except in exceptional circumstances, for example in fish penned with farmed salmonids affected by an outbreak of IHN.

All salmonids farmed in Australia would be susceptible to infection with IHNV. Salmonid species such as Atlantic salmon would be particularly susceptible to infection. Non-salmonid finfish are relatively resistant to infection with IHNV and infection would be unlikely to occur in non-salmonid species in Australia. If infection did occur in such fish, it would probably be in species penned with, or living in close proximity to, farmed salmonids affected by an outbreak of IHN.

IHNV may be transmitted horizontally, via exposure to a significant titre of virus in the freshwater environment. Exposure to a higher titre of virus would be required to initiate infection in adult fish or in the marine environment. Exposure to a low titre of virus would need to be maintained for a prolonged period for infection to result.

IHNV would be expected to be susceptible to inactivation under the physical conditions occurring at sites for disposal of solid waste but would be expected to persist in the aquatic environment, especially in river sediment. IHNV is less resistant to inactivation than infectious pancreatic necrosis virus (IPNV). IHNV does not replicate outside a fish host. IHNV would not be expected to persist in the environment at a significant titre for as long as infectious pancreatic necrosis virus or *Aeromonas salmonicida*. Thus, IHNV would need to enter the aquatic environment continuously and/or at high levels for infection to result.

For susceptible fish to become infected with IHNV, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts for the disease to establish in the population. IHNV would be expected to spread between fish under conditions in the Australian aquatic environment, except in waters at a temperature greater than 15°C.

Repeated high level exposure of susceptible fish to a significant titre of IHNV (for example, from regular discharge of untreated effluent from a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of IHNV into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on salmonids and commercially significant finfish species

IHN is a severe disease affecting many species of salmonid fish including Atlantic and chinook salmon and rainbow trout. The virus has caused substantial losses of salmonids along the Pacific coast of North America and has caused large disease outbreaks in salmonids in Japan and Taiwan and in several European countries. In recent years outbreaks in Atlantic salmon in British Columbia have caused mortality and led to loss of production.

IHNV typically causes systemic disease in juvenile fish with mortalities up to 100% being recorded. Older fish are generally refractory to infection. IHN has historically had little significance in market-size fish; however, outbreaks of clinical disease with high mortality have occurred in farmed Atlantic salmon of market size in British Columbia in recent years.

Once established in a susceptible salmonid population, IHN is not readily amenable to control. No commercial vaccines are available to date. Overseas, the spread of IHNV is controlled by management measures such as disinfection and quarantine. Disinfection, screening and rejection of infected batches of eggs are routinely used to reduce the prevalence of IHNV in fry. Elevating hatchery water temperatures to above 15°C has been advocated as a method of controlling disease outbreaks, but is only effective for some strains of the pathogen.

It is expected that all salmonid species in Australia would be susceptible to infection with IHNV. Of the species present in Australia, young, farmed freshwater salmonids would be most susceptible to infection. If IHNV were to become established, it would be expected to primarily affect hatchery stocks, especially of Atlantic salmon and rainbow trout. Based on the occurrence of clinical IHN in market-size fish in British Columbia, it is possible that the establishment of IHNV in Australia could also affect the production of salmonids for market.

It is expected that the establishment of IHNV in Australia would cause significant mortality in young rainbow trout, which would cause economic losses in the farmed rainbow trout industry and may affect the recreational

trout-fishing sector. IHNV could cause significant mortality in individual batches of Atlantic salmon smolts. If a similar situation to that in British Columbia occurred, the establishment of IHNV may affect regional production and the availability of marketable salmonids. IHNV would not be expected to cause major losses in production or profitability in the Atlantic salmon industry nationally.

The establishment of IHNV would affect farms exporting eyed ova, as they may be required to implement additional testing and certification to preserve their export markets. However, the effects of establishment of IHNV would primarily be felt regionally and at the level of individual premises rather than at the whole industry or national level. Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

There is limited information on the effect of IHNV on wild salmonid populations. It is likely that there would be some impact on trout populations and, therefore, the recreational sector. The establishment of IHNV would be expected to reduce wild populations of rainbow trout, but may have a less pronounced effect on populations of brown trout (which is considered to be relatively refractory to infection with IHNV). Effects on the recreational salmonid sector may be significant locally or regionally, but not nationally.

Ecological and environmental effects

Natural infection of non-salmonid fish with IHNV is unusual. If such infection does occur, it is usually in fish penned with, or living in close proximity to, salmonids affected by an outbreak of IHN. IHNV has not been reported to cause disease in wild, non-salmonid finfish under natural conditions overseas. Based on the literature, infection with IHNV is of little pathogenic or economic significance in wild salmonids or non-salmonid finfish overseas. There is little evidence to suggest that the establishment of IHNV would significantly affect wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated salmonids, including juveniles and sexually mature fish, the probability of IHNV establishing would be very low. The consequences of establishment would be of moderate to high significance.

From the risk management matrix presented in Section 1.5.3, for IHNV, the risk associated with the unrestricted importation of eviscerated salmonids, including juveniles and sexually mature fish, does not meet Australia's ALOP and the implementation of risk management measures are warranted.

A summary of the risk assessment is shown in Box 4.1. Appropriate risk management measures are discussed in Chapter 5.

Box 4.1
Risk assessment — infectious haematopoietic necrosis virus

RELEASE ASSESSMENT (R)

The probability of infectious haematopoietic necrosis virus (IHNV) entering Australia as a consequence of the unrestricted importation of eviscerated salmonids would be low.

Because IHNV is primarily clinically expressed in juvenile salmonids, and there is a greater probability of a significant viral titre in juvenile salmonids and sexually mature salmonids, the probability associated with the unrestricted importation of these lifecycle stages would be moderate.

EXPOSURE ASSESSMENT (E)

If IHNV entered Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be very low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of IHNV becoming established in Australia because of unrestricted importation of eviscerated salmonids, including juveniles and sexually mature fish, would be very low (VL).

CONSEQUENCE ASSESSMENT

Due primarily to the reduced supply of juvenile Atlantic salmon and juvenile rainbow trout, and the effect of a reduced population of trout on the recreational sector, the consequences of the establishment of IHNV in Australia would be moderate (M) to high (H).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of IHNV would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL
- significance of consequences = M–H
- importation risk for IHNV = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated salmonids, including juveniles and sexually mature fish, does not meet Australia's ALOP; and
- risk management measures are warranted.

4.2.2 INFECTIOUS PANCREATIC NECROSIS VIRUS (INFECTIOUS PANCREATIC NECROSIS)

In this IRA, 'infectious pancreatic necrosis' describes the acute disease of juvenile salmonids caused by infection with an aquabirnavirus. The various strains of virus that cause infectious pancreatic necrosis (IPN) — referred to as infectious pancreatic necrosis virus — differ in virulence and serological characteristics.

Hill and Way (1995) reviewed the serological classification of aquatic birnaviruses (aquabirnaviruses), many of which are serologically related to reference strains (Ab, Sp and VR299) of infectious pancreatic necrosis virus (IPNV). Some of these viruses were isolated from non-salmonid fish and can be called IPNV as they produce IPN in salmonid fry. There is no evidence that many of the aquabirnaviruses that are serologically related to IPNV are pathogenic in salmonids; they should not therefore be described as IPNV (Hill and Way 1995).

In reviewing the scientific literature on aquabirnaviruses, Reno (1999) noted that it was difficult to evaluate the virulence of non-salmonid isolates for salmonid fish as many different experimental protocols had been used.

Water-borne infectivity trials demonstrated that IPN occurred in brook trout downstream from striped bass (*Morone saxatilis*) infected with an aquabirnavirus (McAllister and McAllister 1988). Immersion challenge of juvenile brook trout with aquabirnaviruses isolated from various aquatic hosts gave clear evidence of the presence of IPNV in non-salmonid fish and other aquatic hosts (McAllister and Owens 1995).

IPNV and aquabirnaviruses pathogenic for non-salmonid marine finfish are covered in Chapter 7.

Release assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- IPN is listed by the OIE as an 'other significant disease' and is included in List III of the European Union Directive 93/54.
- The geographic distribution of IPNV is essentially worldwide, covering continental Europe, Scandinavia, the United Kingdom, North America, South America and North Asia, but not Australia.
- Classical IPN is most commonly associated with farmed freshwater salmonids, especially rainbow trout and brook trout. Pacific salmon appear to be relatively resistant to infection. There have been no reports of disease in wild, marine Pacific salmon.
- Clinical infection with classical IPN is most common in fish less than four months of age. Signs of clinical infection include exophthalmia, abdominal distension, haemorrhage on the surface of the skin and erratic swimming behaviour.
- Clinically infected fish would be visibly abnormal and it would be expected that such fish would be detected and rejected in the course of inspection for human consumption. Carrier fish would not be visibly abnormal and would not be detected at inspection.
- Older fish are less susceptible to infection with IPNV; at approximately six months of age most fish are resistant to this pathogen. Fish infected at an older age generally survive infection.
- Fish that survive infection may become chronic carriers and shed virus via faeces and reproductive fluids for the rest of their lives. In an endemically infected population, the prevalence of covertly infected fish may be high.
- In clinically diseased fish, IPNV may be found in many organs, with the highest viral titres being reported in the kidney.
- Carrier fish of market size may contain viral titres as high as $10^{6.7}$ $TCID_{50}$/g in the viscera, especially the kidney. Virus may also be in muscle tissue, at a lower titre ($10^{0.3}$ $TCID_{50}$/g).
- Evisceration would reduce, but not eliminate, the probability of the pathogen being present in salmonids of market size.
- In Atlantic salmon, IPN may manifest as 'failed-smolt syndrome', showing a peak of mortality in smolts approximately eight weeks after transfer to seawater, with clinical signs similar to those of classical IPN. It has been hypothesised that covertly infected Atlantic salmon smolts in which infection is reactivated during acclimatisation to seawater become 'failed smolts'.

AQIS considered more recent information on IPNV, summarised below.

The geographic range of IPN includes South Africa and East Asia (B Hill pers. comm.). IPNV has recently been reported from Chile after being undetected for more than 10 years (OIE 1999). 'Classical' IPNV of salmonids is not reported in Australia or New Zealand.

A marine aquabirnavirus with some characteristics in common with the infectious pancreatic necrosis virus group has been detected a number of times in healthy sea-run quinnat salmon in New Zealand. Clinical disease due to aquabirnavirus infection has never been observed in New Zealand and the virus has had no impact on salmon farming (Anderson 1996).

Classical IPN is primarily a disease of young fish, being most common in fish younger than four months. Older fish are generally less susceptible to infection; by 6 months of age, fish are generally resistant to infection. However, significant pathology can occur in Atlantic

salmon in Norway and Scotland in the first few months after transfer to seawater, when the fish are normally older than one year (A McVicar pers. comm.).

In a personal communication to AQIS, A McVicar advised 'it has not been shown that failed-smolt syndrome is associated with recrudescence of disease in covertly infected fish. Although this may be speculated, it is possible that a locally endemic strain in the marine environment infects fish soon after seawater transfer'. McVicar further advised that, in Scotland, failed-smolt syndrome is a complex condition that may or may not be associated with IPNV infection — the condition can occur without detectable infection. Differentiation of failed-smolt syndrome from salmon pancreas disease may be problematic (B Munday pers. comm.). IPNV has been linked with serious pathology, morbidity and mortality problems in the immediate post-smolt period and, as a consequence, IPN is considered to be one of the most economically significant diseases in salmon farming in Norway. The Norwegian strain is N1 and the Scottish strains are similar but not identical — both are Sp variants (A McVicar pers. comm.).

Key findings

Classical IPN mainly affects farmed rainbow and brook trout. The prevalence of infection is greatest in freshwater salmonids and is very low in wild-caught fish. Classical IPN normally affects young fish (younger than one year); however, disease has been reported in fish older than one year.

Clinical disease most commonly affects juvenile fish that are not normally harvested for human consumption. It is expected that clinically infected fish would be detected and removed in the course of inspection for human consumption.

Adult and market-size fish that have survived infection may be apparently healthy carriers of IPNV. Such fish would not be detected during inspection. In carrier fish, the highest viral titre would be in the viscera, but virus may also occur in muscle at very low titre. In adult carrier fish, IPNV is mostly localised in visceral organs that would be removed from eviscerated fish.

These findings would also apply to Atlantic salmon affected by failed-smolt syndrome.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- All salmonid species present in Australia would be susceptible to infection with IPNV.
- IPNV has a direct life cycle. It may be transmitted horizontally via ingestion and across the gills and vertically, via sperm.
- The minimum infective dose of IPNV is unknown. Infection has been initiated by feeding brook trout fry a dose of 10^3 $TCID_{50}$ virus/mL per 100 fish in a two-day period. This resulted in greater than 70% mortality; hence, the infective dose was somewhat less than this.
- IPNV is a relatively robust virus and would be expected to survive for considerable periods in the environment. IPNV can survive for several years at -70°C and for several months at 4°C. It is highly resistant to low pH and can survive for 22 hours at 50°C. In municipal tap water, IPNV survived for seven months at 10°C. At chlorine concentrations of 200 mg/L, IPNV was inactivated in 10 minutes in soft water; in hard water at a concentration of 0.7 mg/mL inactivation occurred in two minutes. 90mg/L ozone inactivated IPNV in 10 minutes in hard water and 30 seconds in soft water.
- The isolation of 'non-IPNV' aquabirnavirus in Tasmania indicates that conditions exist in Australia for the transfer and establishment of pathogens in the aquabirnavirus family.
- Carrier fish may shed IPNV intermittently for a prolonged period, providing an enhanced opportunity for the spread of infection.

AQIS also considered more recent information on IPN, summarised below.

While Wolf (1988) suggested that farmed salmonids may be at risk of infection if exposed to IPNV in the water supply, McAllister and Bebak (1997) reported that chronic, low-level exposure to IPNV in stream water does

not pose a significant risk of infection of wild salmonid and non-salmonid fish. McAllister and Bebak (1997) reported that exposure to a lower level (about 10^2 PFU/L) of virus compared to higher levels (about 10^4 PFU/L) in hatchery effluent did not result in infection in downstream adult salmonid and non salmonid fish, although one out of nine salmonid fingerlings was virus positive. These authors commented that laboratory immersion challenges generally use high levels of virus (about 10^5 PFU/mL) with short exposure times (about five hours) to assure consistent levels of mortality. The virus levels found in stream water were about 10^7-fold lower than the levels used in immersion challenge. Therefore, even though stream fish were exposed continuously to IPNV, infection might not have occurred because virus concentration in the water was too low or because natural defence mechanisms of the fish effectively controlled low-level virus exposure.

Key findings

Freshwater salmonids, in particular juvenile fish, are susceptible to infection with IPNV, whereas marine fish and fish older than six months of age are relatively resistant to infection. Infection may be transmitted horizontally via exposure to a relatively high titre of virus in the aquatic environment. An even higher titre of virus would be required to initiate infection in adult fish or in the marine environment. Exposure to lower titres of virus would need to be maintained for a prolonged period to initiate an index case of infection.

IPNV is relatively resistant to inactivation. If IPNV entered the aquatic environment, it would be expected to survive in infective form for a prolonged period.

For susceptible fish to become infected with IPNV, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. IPNV would be expected to readily spread between infected fish under conditions in the Australian aquatic environment.

Repeated high level exposure of susceptible fish to a significant titre of IPNV (for example, from regular discharge of untreated effluent of a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of IPNV into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen.

Consequence assessment

Effects on salmonids and commercially significant species

Disease due to IPNV causes substantial loss of young salmonids in northern Europe and North America, especially under conditions of stress or high temperature (relative to the low water temperature occurring in most salmon farms in the northern hemisphere). In countries where infection with IPNV is endemic, mortality rates of up 70% have been reported among fry and fingerlings up to 20 weeks of age. Under experimental conditions, highly virulent strains of IPNV have been reported to cause mortality rates in excess of 90%.

IPNV has also been linked with serious pathology, morbidity and mortality problems in the immediate post-smolt period and, as a consequence, IPN is considered to be one of the most economically significant diseases in salmon farming in Norway (A McVicar pers. comm.). Failed-smolt syndrome may cause substantial loss of Atlantic salmon post-smolts.

There are no effective chemotherapeutic agents or proven vaccines available for the treatment or control of IPN. Moreover, there is no evidence that maternally transferred immunity or heritable resistance is protective against disease. Overseas, the disease is controlled by maintaining strict hatchery hygiene, screening broodstock and minimising stress.

It is expected that the establishment of IPNV in Australia would cause significant mortality in young rainbow trout, which would cause economic losses in the farmed rainbow trout industry and may affect the recreational trout-fishing sector. Based on experience overseas, effects on the recreational salmonid sector may be significant locally or regionally, but not at a national level.

The occurrence of 'failed-smolt syndrome' could cause significant mortality in individual batches of Atlantic salmon smolts but would not be expected to cause major losses in production or profitability in the Atlantic salmon industry nationally.

The establishment of IPNV would affect farms exporting eyed ova, as they may be required to implement additional testing and certification to preserve their export markets. However, the effects of establishment of IPNV would primarily be felt at an individual premises or regional level rather than a whole industry or national level. Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

IPNV has occasionally been recovered from non-salmonid hosts (eg menhaden, striped bass and southern flounder) during disease epizootics but its causative role in disease in these hosts has not been established. Experimental transmission studies, including intra-peritoneal inoculation with a high titre of IPNV, have failed to produce disease in juvenile striped bass (*Morone saxatilis*) (Wechsler et al 1987b). IPNV isolated from Japanese eel appeared to be avirulent for that species (McAllister and Owens 1995). Generally the detection of IPNV in non-salmonid finfish is an incidental finding and is not associated with disease.

Ecological and environmental effects

Based on the literature, infection with IPNV is of little pathogenic or economic significance in wild salmonids or non-salmonid finfish overseas. There is little evidence to suggest that the establishment of IPNV would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated salmonids, the probability of the establishment of IPNV would be extremely low. For juvenile fish the probability would be low. The consequences of establishment would be of moderate to high significance.

From the risk management matrix presented in Section 1.5.3, for IPNV, the risk associated with the unrestricted importation of eviscerated adult salmonids meets Australia's ALOP and the implementation of risk management measures are not warranted.

For juvenile salmonids, the risk does not meet Australia's ALOP and the implementation of risk management measures are warranted.

A summary of the risk assessment is shown in Box 4.2. Appropriate risk management measures are discussed in Chapter 5.

Box 4.2
Risk assessment — infectious pancreatic necrosis virus

RELEASE ASSESSMENT (R)

The probability of infectious pancreatic necrosis virus (IPNV) entering Australia as a consequence of the unrestricted importation of eviscerated salmonids would be extremely low.

Because IPNV is clinically expressed in juvenile salmonids and there is a greater probability of a significant viral titre in young fish, the probability associated with the unrestricted importation of juvenile salmonids would be low.

EXPOSURE ASSESSMENT (E)

If IPNV entered Australia, the probability of a susceptible fish being exposed to a dose sufficient to cause infection would be low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of IPNV becoming established in Australia as a consequence of the unrestricted importation of eviscerated salmonids would be extremely low (EL).

Because IPN is clinically expressed in juvenile salmonids and there is a greater probability of a significant viral titre in fish of this lifecycle stage, the probability of IPNV becoming established in Australia as a consequence of unrestricted importation of juvenile salmonids would be low (L).

CONSEQUENCE ASSESSMENT

Due primarily to effects on the farmed and recreational freshwater salmonid sectors, the consequences of establishment of IPNV in Australia would be moderate (M) to high (H).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of IPNV would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

For adults

- probability of establishment = EL
- significance of consequences = M–H
- importation risk for IPNV = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated adult salmonids meets Australia's ALOP; and
- risk management measures are not warranted.

For juveniles

- probability of establishment = L
- significance of consequences = M–H
- importation risk for IPNV = unacceptable ('no' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of eviscerated juvenile salmonids does not meet Australia's ALOP; and
- risk management measures are warranted.

4.2.3 INFECTIOUS SALMON ANAEMIA VIRUS (INFECTIOUS SALMON ANAEMIA)

In view of the significance of this disease, AQIS has undertaken a review of the literature (see Appendix 8) as a basis for this section of the risk analysis. The following review also draws upon information in the 1997 report of the New Zealand Government (Stone et al 1997b).

Release assessment

Key findings

The prevalence of infection with infectious salmon anaemia virus (ISAV) in Atlantic salmon sourced from farms affected by an outbreak of infectious salmon anaemia (ISA) would be high. There is little evidence to suggest that salmonids (other than Atlantic salmon), or non-salmonid fish from ISA-infected areas, would be infected with ISAV.

ISAV is listed as an 'other significant' disease by the OIE and is the subject of European Union legislation. This pathogen is the subject of concern and a focus of scientific research, surveillance and monitoring in countries of Europe and North America that have a significant farmed Atlantic salmon industry. Accordingly, it is expected that the emergence of disease suggestive of ISA or haemorrhagic kidney syndrome (HKS) would attract official attention and would be the subject of intense investigation. If the presence of ISA was confirmed, it is expected that this would be reported to the OIE without delay. AQIS acknowledges that the emergence of ISA in Canada was only reported to the OIE after many months of investigation. However, scientific knowledge on ISA and particularly diagnostic methods for it have improved greatly since this time. Therefore, AQIS expects that OIE Member countries would make a definitive diagnosis faster and report disease more promptly in future.

Accordingly, there would be a negligible probability of salmonids (or other finfish) from areas that have not reported the presence of ISA or HKS being infected with ISAV.

Because of the pathological changes associated with ISA, clinically diseased Atlantic salmon would be visibly abnormal. These fish are unlikely to be harvested for human consumption (under European Union regulations, market-size fish from ISA-infected farms may be harvested if they show no signs of clinical disease). If harvested, clinically infected fish would be detected and removed in the course of inspection for human consumption. Salmonids that were inapparently infected with ISAV (which could include fish in a population affected by an outbreak of ISA) would not be visibly abnormal and would not be detected at inspection.

ISAV may be present in visceral organs, gills, mucus, somatic muscle and blood of infected fish. For both clinically and covertly infected fish, evisceration would substantially reduce the titre of virus present; however, virus may remain in other parts of the body, particularly the gills. ISAV may occur in the somatic musculature of clinically infected fish. The titre of virus in muscle tissue would be lower than in internal organs and material from the head. In subclinically infected fish, the titre of ISAV in muscle tissue would be expected to be low.

Were routine vaccination to be introduced in ISAV-infected countries, it is possible that there would be increased numbers of salmonid fish that were inapparently infected carriers of ISAV. The titre of virus in such fish would be expected to be low relative to that in clinically infected fish. It is probable that most ISAV in apparently healthy vaccinated carrier fish would be located in the viscera and other blood-rich organs. While these fish would not be detected at inspection, evisceration would substantially reduce the titre of virus present.

Exposure assessment

Key findings

On current knowledge, Atlantic salmon in Australia would be susceptible to infection under natural conditions. Brown trout and rainbow trout were shown to be susceptible to infection under experimental conditions. It is possible that brown trout in waters where Atlantic salmon are farmed (such as in the Huon/Esperance region of Tasmania) could become infected with ISAV if the pathogen were to become established in Atlantic salmon. It is not expected that species in Australia, other than Atlantic salmon and possibly brown and rainbow trout, would be susceptible to infection with ISAV.

ISAV may be transmitted horizontally from fish to fish and via exposure to infected waste material at relatively high volume/concentration. Published information states that viscera and trimmings[2] from the slaughter process are highly contagious, including when the fish has no clinical or macroscopic signs of disease. This would be mainly due to the lag phase in the development of disease and the fact that infection may occur in the absence of clinical signs. Muscle 'filet' is less infective than internal organs and material from the head. Most infective material is removed by the bleeding and primary processing of fish; a fact that is acknowledged in European Union regulations that permit free movement from ISAV-infected farms of eviscerated salmon for human consumption.

The Joint Working Group (JWG) on ISA in Scotland prepared an interim report on the key risk factors for ISA and the measures that should be introduced on an urgent basis to deal with ISA. In the report, key risk factors were associated with eggs, transmission of live fish between sea sites, well-boats and equipment and harvesting/processing operations. The movement of eviscerated salmonids for human consumption was not identified as a risk factor for the spread of ISA. Moreover, there is no evidence that outbreaks of ISA in Canada and Scotland were associated with the importation of eviscerated salmon (A McVicar pers. comm.). Further, all counties in the European Union have agreed via a European Commission Decision to accept imports of uneviscerated non-viable salmonids from Norway if they are accompanied by a certificate from the competent authorities in Norway confirming they originate from a farm which is not under official control for ISA (B Hill pers. comm.).

ISA is reported to spread slowly in an infected population. This could mean that the minimum infective dose is relatively high. If so, exposure to a low titre of virus would need to be maintained for a prolonged period for infection to result.

In order for ISAV to infect salmonids in Australia, susceptible host fish (probably Atlantic salmon) would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Atlantic salmon in Australia are mostly in farms and there is only a low number (mainly comprising fish that have escaped from farms) in the wild. The introduction of infective material directly into a cage containing Atlantic salmon could bring about exposure of susceptible fish to sufficient virus to cause infection; however, this would be very unlikely to occur. Exposure of brown trout to ISA-infective material could theoretically result in infection. However, brown trout were shown to be infected only when cohabiting with ISA-infected Atlantic salmon, which suggests that ISA would be unlikely to infect brown trout in Australia unless the disease first became established in Atlantic salmon.

Infection would need to be transmitted from the index case of infection to other susceptible hosts to establish disease in the population. ISAV would be expected to spread readily between fish under conditions in the Australian aquatic environment.

In studies on host range conducted to date, ISAV has not been shown to infect non-salmonid fish under natural or experimental conditions. While this possibility cannot be discounted, current knowledge suggests that there would be an extremely low probability of the entry of ISAV into the aquatic environment causing the establishment of ISA in non-salmonid finfish. Any infective material entering the aquatic environment and being consumed by fish would most probably be consumed by non-susceptible species, reducing the probability of Atlantic salmon being exposed to and becoming infected with ISAV.

High level exposure of susceptible fish to a significant titre of ISAV (for example, from discharge of untreated effluent from a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of ISAV into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species being present. Moreover, if susceptible hosts were present, there would be a very low probability of the virus being

2 The term trimmings, as used in the report of Torgerson (1997) may include material from the head and gills.

present in sufficient titre to induce infection unless such exposure was maintained for a prolonged period.

Consequence assessment

Effects on salmonids and commercially significant finfish species

Under conditions reported overseas, ISAV infects Atlantic salmon only. If this also applied under Australian conditions, the consequences of disease establishment would arise from commercial losses in the Atlantic salmon industry. In infected populations mortality rates range from 15%–100% (Thorud 1991 as cited in Falk and Dannevig 1995) potentially resulting in serious production losses.

The presence of ISAV in farmed Atlantic salmon has resulted in loss of markets and significant reductions in profitability in affected countries. In Scotland, the emergence of ISA contributed to the closure of many Atlantic salmon farms (Weir, cited by the Tasmanian Salmon Growers Association). The loss of markets primarily relates to live fish and uneviscerated fish for human consumption. Import restrictions on the importation of salmonids for human consumption, initially applied by several countries to Norway, were subsequently lifted for eviscerated fish. Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

Significant costs are also associated with compensation for, control and eradication of ISAV. The British government planned to provide UK£9 million over three years to producers affected by the 1998 outbreak of ISA in Scotland.

In an effort to assist the farmers, the Province of New Brunswick provided a US$5 million stock replacement package in compensation for the apparently healthy fish that were destroyed in the Canadian outbreak. In Canada, US$3 million was also allotted to monitor ISAV. It was estimated that the ISA outbreak cost the New Brunswick salmon industry US$20 million through reduced fish growth and increased mortality.

Based on overseas experience, the establishment of ISAV in Australia would seriously damage profitability and could threaten the long-term sustainability of the Atlantic salmon industry. If vaccination against ISA were shown to be effective, the effect of establishment of ISAV on sustainability might be substantially reduced; however, the industry would still experience cost increases and a reduction in profitability that would be significant at a national level.

Ecological and environmental effects

As natural infection has not been reported in fish species other than Atlantic salmon, it is extremely unlikely that infection with ISAV would have a significant effect on non-salmonid fish or native fish species in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated Atlantic salmon from areas infected with ISAV or affected by HKS, the probability of establishment of ISAV would be low. The consequences of establishment would be of high significance.

From the risk management matrix presented in Section 1.5.3, for ISAV, the risk associated with the unrestricted importation of eviscerated Atlantic salmon from ISAV-infected and HKS-affected areas does not meet Australia's ALOP and the implementation of risk management measures are warranted.

For salmonids other than Atlantic salmon (and for non-salmonid fish) from areas infected with ISAV or affected by HKS the probability would be negligible. For salmonids (and non-salmonid fish) from areas that have not reported the presence of ISAV or HKS the probability would be negligible. No risk management is warranted in these cases.

A summary of the risk assessment is shown in Box 4.3. Appropriate risk management measures are discussed in Chapter 5.

Box 4.3
Risk assessment — infectious salmon anaemia virus

RELEASE ASSESSMENT (R)

The probability of infectious salmon anaemia virus (ISAV) entering Australia from the unrestricted importation of eviscerated Atlantic salmon from areas infected with ISAV or affected by haemorrhagic kidney syndrome (HKS) would be high.

For salmonids other than Atlantic salmon (and for all non-salmonid fish) from areas infected with ISAV or affected by HKS the probability would be negligible.

For salmonids (and other finfish) from areas that have not reported the presence of ISAV or HKS the probability would be negligible.

EXPOSURE ASSESSMENT (E)

If ISAV entered Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of ISA becoming established in Australia as a consequence of the unrestricted importation of eviscerated Atlantic salmon from areas infected with ISAV or affected by HKS would be low (L).

For salmonids other than Atlantic salmon (and for non-salmonid fish) from ISAV-infected and HKS-affected areas the probability would be negligible (N).

For salmonids (and non-salmonid fish) from areas that have not reported the presence of ISAV or HKS the probability would be negligible (N).

CONSEQUENCE ASSESSMENT

Due to the effects on the commercial Atlantic salmon industry, the consequences of the establishment of ISAV in Australia would be high (H).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of ISAV would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

For eviscerated Atlantic salmon from areas infected with ISAV or affected by HKS

- probability of establishment = L
- significance of consequences = H
- importation risk for ISAV = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated Atlantic salmon from areas infected with ISAV or affected by HKS does not meet Australia's ALOP; and
- risk management measures are warranted.

For salmonids other than Atlantic salmon (and for non-salmonid fish) from areas infected with ISAV or affected by HKS

- probability of establishment = N
- significance of consequences = H
- importation risk for ISAV = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of salmonids other than Atlantic salmon (and for non-salmonid fish) from areas infected with ISAV or affected by HKS meets Australia's ALOP; and
- risk management measures are not warranted.

For salmonids (and non-salmonid fish) from areas that have not reported the presence of ISAV or HKS

- probability of establishment = N
- significance of consequences = H
- importation risk for ISAV = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of salmonids (and non-salmonid fish) from areas that have not reported the presence of ISAV or HKS meets Australia's ALOP; and
- risk management measures are not warranted.

4.2.4 *ONCORHYNCHUS MASOU* VIRUS

Release assessment

The following key points are based on information in the 1997 report of the New Zealand Government (Stone et al 1997b). This report contains referenced reviews of relevant literature.

- Infection with *Oncorhynchus masou* virus is listed by the OIE as a notifiable disease.
- *Oncorhynchus masou* virus (OMV) has only been reported in Japan, where it is widespread in the northern regions.
- Under natural conditions, outbreaks of disease due to OMV only affect *Oncorhynchus* spp. The most significant losses are recorded in coho salmon.
- The disease is more common in the freshwater phase of the salmonid lifecycle. However, larger fish (up to 1 kg in weight) may also be affected by disease.
- In subclinically and chronically infected fish, evisceration is likely to significantly reduce the titre of OMV. Ovarian fluids and tumours are the only documented sources of OMV under natural conditions.

AQIS considered the following information from the 1997 OIE Diagnostic Manual for Aquatic Animal Diseases.

Infection with OMV presents as a systemic and frequently lethal condition that is associated with oedema and multiple haemorrhage. The virus multiplies in endothelial cells, haematopoietic tissue and hepatocytes, giving rise to typical clinical signs. About 4 months after infection, surviving fish may develop tumours around the mouth and on the caudal fin, operculum and body surface. Post-infection, tumours may be found for up to a year.

Salmonid species differ in susceptibility to infection with OMV. The most susceptible species is sockeye salmon, followed by masou salmon, chum salmon, coho salmon and rainbow trout in decreasing order of susceptibility. The effect of infection depends on the age of fish at the time of infection. Alevins one month old are the most susceptible to infection. In one-year-old coho salmon, infected fish developed ulcers on the skin, lesions in the liver and tumours on the mouth and surface of the body. Infected rainbow trout showed few external symptoms, mainly limited to ulcerative lesions of the skin, intestinal haemorrhage and lesions in the liver.

Fish surviving the septicaemic phase of infection frequently become carriers of infection and may shed OMV in the faeces, urine, sexual products and, probably, in the skin mucus. In clinically infected fish, the highest titre of OMV occurs in the kidney, liver, spleen and in tumours.

Key findings

OMV is a disease of salmonids of the genus *Oncorhynchus,* and particularly affects juvenile fish in fresh water. Infection is reported in wild and farmed fish and only occurs in Japan. Infection typically causes acute systemic infection in juvenile fish, especially alevins. Fish surviving infection frequently become subclinical carriers of infection.

Juvenile salmonids are not usually harvested for human consumption. Adult fish are less likely to have clinical disease than juvenile fish. Clinically infected fish would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. Inapparently infected adult salmonids would not be visibly abnormal and would not be detected at inspection. In eviscerated carrier fish, the titre of OMV would be very low.

The location of OMV in carrier fish is not known, but most virus would probably be located in visceral organs. In such fish, evisceration would substantially reduce the titre of the virus present. If present in muscle of covertly infected fish, the titre of OMV would be expected to be very low.

Exposure assessment

The following key points are based on information in the 1997 report of the New Zealand Government (Stone et al 1997b). This report contains referenced reviews of relevant literature.

- Horizontal and vertical infection may play a role in the transmission of OMV.

- OMV is an obligate pathogen, which has limited survival outside the host. As a member of the Herpesviridae, OMV would be expected to be labile to ether, heat and acid. At –20°C OMV lost 99.9% infectivity within 17 days. No virus survived at 15°C.

AQIS considered further information on OMV, summarised below.

Experiments on the susceptibility of salmonid fry to infection with OMV by immersion in water containing 100 $TCID_{50}$/mL OMV at 10°C for 1 hour showed that this dose caused 100% mortality of sockeye salmon. The same experimental challenge caused mortality of 87% and 83% of masou and chum salmon respectively. Coho salmon and rainbow trout were not as susceptible to infection with OMV, the same challenge causing 39% and 29% mortality respectively. Immersion of 8-month-old chum salmon fingerings in a suspension of OMV followed by intraperitoneal inoculation of 200 $TCID_{50}$ per fish did not cause mortality (Kimura and Yoshimizu 1989).

Fish-to-fish transmission of OMV was effected by holding 5-month-old fry with fry infected by immersion. The resulting rate of mortality was similar to that observed as a result of infection by immersion (Kimura and Yoshimizu 1988).

The most important environmental factor favouring OMV infection is low (<14°C) water temperature (OIE Manual).

Key findings

OMV is primarily a disease of farmed, juvenile *Oncorhynchus* salmonids in fresh water. The minimum infective dose of OMV would be higher for adult salmonids than juvenile salmonids. The minimum infective dose of OMV may be high in juvenile salmonids relative to pathogens such as *Aeromonas salmonicida* for which the minimum infectious dose appears to be very low. Salmonid species in Australia (particularly rainbow trout and chinook salmon) may be susceptible to infection with OMV, but other Australian salmonid species would be relatively resistant to infection. OMV has not been reported in non-salmonid finfish species.

OMV may be transmitted horizontally, via exposure to a significant titre of virus in the fresh water environment. Exposure to a higher titre of virus would be required to initiate infection in adult fish. Transmission has not been reported to occur in the marine environment so it is unlikely that the entry of infective material into the marine environment would result in infection.

OMV would be expected to be susceptible to inactivation under physical conditions occurring at sites for disposal of solid waste and would not be expected to survive outside a live host for any significant period. OMV has not been shown to replicate outside a fish host. Thus, OMV would need to enter the aquatic environment continuously and/or at high levels for infection to result.

In order for susceptible fish to become infected with OMV, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts for the disease to be established in the population. OMV would be expected to spread between fish under conditions in the Australian aquatic environment, except in waters at a temperature greater than 14°C.

Repeated high level exposure of susceptible fish to a significant titre of OMV (for example, from regular discharge of untreated effluent from a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of OMV into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on salmonids and commercially significant finfish species

There is limited information on the impact of OMV. Mortality rates as high as 31% have been recorded in epidemics of disease, which occur in waters at a temperature <14°C. The spread and prevalence of infection of OMV may be managed by disinfecting eyed ova and treating hatchery water with UV radiation.

Given that OMV is listed by the OIE, it is expected that the establishment of disease in Australia would have some effect on trade in live fish and gonadal material. The establishment of OMV would affect farms exporting eyed ova, as they may be required to implement additional testing and certification to preserve their export markets. However, the effects of establishment of OMV would primarily be felt at an individual premises or regional level rather than a whole industry or national level. Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

The establishment of OMV in Australia would be expected to cause clinical disease primarily in farmed rainbow trout and chinook salmon. Based on scientific literature, it is likely that effects would be significant at an individual premises or regional level but not at a national level.

There is limited information on the effect of OMV on wild salmonid populations. However, it is likely that there would be some impact on trout populations and, therefore, the recreational sector. The establishment of OMV would be expected to reduce wild populations of rainbow trout but to have little effect on populations of brown trout (considered to be relatively refractory to infection with OMV). Effects on the recreational salmonid sector may be significant locally or regionally, but not at a national level.

Ecological and environmental effects

Infection with OMV has not been reported in non-salmonid finfish. There is no evidence to suggest that the establishment of OMV would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated *Oncorhynchus* spp, including juveniles, from Japan the probability of the establishment of OMV would be very low. For salmonids other than *Oncorhynchus* spp from Japan and for all salmonids from other countries, the probability would be negligible. The consequences of establishment would be of moderate significance.

From the risk management matrix presented in Section 1.5.3, for OMV the risk associated with the unrestricted importation of eviscerated salmonids, including juveniles, meets Australia's ALOP and the implementation of risk management measures are not warranted.

A summary of the risk assessment is shown in Box 4.4.

Box 4.4
Risk assessment — *Oncorhynchus masou* virus

RELEASE ASSESSMENT (R)

The probability of *Oncorhynchus masou* virus (OMV) entering Australia as a consequence of the unrestricted importation of eviscerated *Oncorhynchus* spp from Japan would be low. For salmonids other than *Oncorhynchus* spp from Japan and for all salmonids from other countries, the probability would be negligible.

Because OMV is primarily clinically expressed in juvenile salmonids, and there is a greater probability of a significant viral titre in juvenile salmonids, the probability associated with the unrestricted importation of this lifecycle stage of *Oncorhynchus* spp from Japan would be moderate.

EXPOSURE ASSESSMENT (E)

If OMV entered Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be very low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of OMV becoming established in Australia as a consequence of the unrestricted importation of eviscerated *Oncorhynchus* spp from Japan, including juvenile salmonids of this species, would be very low (VL). For salmonids other than *Oncorhynchus* spp from Japan and for all salmonids from other countries the probability would be negligible (N).

CONSEQUENCE ASSESSMENT

Due primarily to the reduced supply of rainbow trout smolts and the effect of a reduced population of trout on the recreational sector, the consequences of the establishment of OMV in Australia would be moderate (M).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of OMV would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL (eviscerated *Oncorhynchus* spp, including juveniles, from Japan) to N (salmonids other than *Oncorhynchus* spp from Japan)
- significance of consequences = M
- importation risk for *Oncorhynchus masou* virus = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated salmonids, including juveniles, meets Australia's ALOP; and
- risk management measures are not warranted.

4.2.5 SALMON PANCREAS DISEASE VIRUS (SALMON PANCREAS DISEASE)

Release assessment

The following key points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- Salmon pancreas disease (SPD) was first recognised in Scotland, and is now reported in farmed Atlantic salmon in Ireland, Norway, Spain, France, United States (Washington State) and Canada (British Columbia).
- Salmon pancreas disease is a subacute to chronic infectious disease of farmed Atlantic salmon caused by a togavirus.
- Under natural conditions, salmon pancreas disease virus (SPDV) infection has been reported in brown trout. Experimentally, infection can be transmitted to rainbow trout.
- In France, a disease known as 'sleeping disease', caused by a togavirus, occurs in freshwater rainbow trout. Affected fish are lethargic, do not feed properly and fail to thrive. It is thought that sleeping disease is related to SPDV; however, the association between the two conditions is unclear.
- Experimentally, Atlantic salmon is the salmonid species most susceptible to infection with SPDV. Only Atlantic salmon developed pancreatic lesions indicative of SPD. Rainbow trout are less susceptible and brown trout the least susceptible salmonid species.
- SPD has not been reported in wild marine salmonids. However, the detection of SPDV in such stocks would be difficult.
- Under natural conditions, SPD has only been reported in farmed fish in seawater. Up to 100% of post-smolts can be infected. The fish are lethargic and anorexic with mortality rates of 10–50%. Affected stocks normally recover from a disease outbreak after a period of two weeks to three months. Up to 10% of survivors may develop chronic pancreatitis and become runts. These fish often die or are culled.
- SPD principally affects fish in their first year in seawater, although older fish may also be affected when a previously SPD-free farm is first infected. For salmon farms in which SPDV is endemic, disease normally affects post-smolts only.
- SPDV typically causes necrotic lesions in the exocrine pancreas. It can also cause lesions in skeletal and cardiac muscle, gills, eyes and gut. Blood, spleen and kidney tissues from infected animals have all been shown to be infective.
- It is not known if fish infected with SPDV may become carriers. It is reported that fish that fully recovered from experimentally induced pancreas disease developed immunity and showed no evidence of becoming carriers of SPDV. However, the potential for fish with chronic pancreatitis to carry and shed SPDV is unknown.

AQIS considered more recent information, as follows.

Sleeping disease has been described in France (Boucher and Baudin Laurencin 1996) and Italy (Ghittino 1987). Studies on sleeping disease in rainbow trout in France show the virus has a characteristic envelope with an external diameter 55–65 nm (Castric et al 1997). The similarity between the sleeping disease virus and SPDV and the pathology (McLoughlin et al 1996) affecting marine-farmed Atlantic salmon has promoted the idea that these agents might be similar. An acquired cross-protection against sleeping disease and SPDV in laboratory studies supports this hypothesis (Boucher and Baudin Laurencin 1996).

Over the past few years there has been a decline in the number of fish diagnosed with this disease in Scotland and it is no longer considered as one of the most serious diseases affecting the farmed salmon industry (D Bruno pers. comm.). Mortality rates are invariably low in Scotland and the other lesions of heart and skeletal muscle are not associated with the primary disease but are probably secondary (Bell et al 1987). These are major differences from findings in Ireland and may be associated with the different environment and rearing

conditions, the presence of other diseases or other unknown factors (A McVicar pers. comm.).

Classical SPD can be induced in salmon parr in fresh water by cohabitation with infected fish and by injecting kidney homogenate from infected fish. The absence of natural occurrences of the disease in freshwater salmon farms, including broodstock farms, in Norway, Scotland, Ireland and North America when the disease is widespread in the sea, is good evidence for the absence of vertical transmission (A McVicar pers. comm.).

The origin of the infection is unknown, but the widespread distribution of SPD and sporadic recurrence after fallowing, indicate that SPD is probably endemic in seawater in both the North Atlantic and North Pacific (A McVicar pers. comm.).

Key findings

SPD is primarily a disease of young, farmed salmonids in seawater, although disease may occur in older stocks in the initial stages of SPDV infection in a previously uninfected salmon farm. Infection with SPDV has not been reported in wild-caught salmonids. In Atlantic salmon, infection typically causes necrotic lesions of the pancreas and, in some cases, the skeletal and cardiac muscle of juvenile fish. There is little evidence that surviving fish become asymptomatic carriers, however fish with chronic pancreatitis have the potential to carry and shed SPDV.

Juvenile salmonids, the lifecycle stage most likely to have clinical disease, are not usually harvested for human consumption. Adult fish are much less likely than juvenile fish to have clinical disease. Clinically infected fish would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. It is unlikely that adult fish surviving infection with SPDV would become carriers of infection; however, if there is a carrier state, such fish would not be visibly abnormal and would not be detected at inspection.

In apparently healthy eviscerated adult salmonids, the titre of virus, if any were present, would be extremely low (probably undetectable by traditional diagnostic methods). Unlike pathogens that may be widely dispersed in tissues of chronically infected fish, like *Aeromonas salmonicida* and infectious salmon anaemia virus, there is no evidence to suggest that SPDV would be in the somatic musculature in apparently healthy eviscerated adult salmonids.

These key findings also apply to sleeping disease of rainbow trout.

Exposure assessment

The following key points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- The most susceptible species, Atlantic salmon, occurs in Australia. Rainbow trout and brown trout are also present, but would be less susceptible to infection with SPDV.
- SPDV has been shown to be transmissible in Atlantic salmon by injection, cohabitation and via effluent waters from pre-clinically and clinically affected fish.
- The minimum infectious dose is unknown. The infectious agent has only recently been isolated and cannot be easily quantified. Atlantic salmon may be experimentally infected by intraperitoneal inoculation of 10^7 $TCID_{50}$/mL in a volume of 0.1 mL.
- There is limited information on the thermostability of SPDV, but the virus appears to be of moderate stability at 4°C and may survive in chilled or frozen product for some weeks. Infectivity was reduced after 30 minutes at 37°C and 45°C, and lost after 30 minutes at 50°C. The virus was sensitive to exposure to chloroform and pH 3.

Key findings

Atlantic salmon, brown trout and possibly rainbow trout in Australia would be susceptible to infection with SPDV. Atlantic salmon would be particularly susceptible to infection. SPD principally affects fish in their first year in seawater, although older fish may also be affected during initial infection of a previously free farm. For salmon farms in which SPDV is endemic, disease normally affects post-smolts only. SPDV has not been reported in non-salmonid fish.

SPDV may be transmitted horizontally, via exposure to a significant titre of virus in the seawater environment. It is expected that an exposure to a higher titre of virus would be required to initiate infection in adult fish. Exposure to a low titre of virus would need to be maintained for a prolonged period for infection to result.

SPDV would be expected to be susceptible to inactivation under the physical conditions occurring at sites for disposal of solid waste. The period for which SPDV would survive in the aquatic environment is unknown, but is likely to be limited. SPDV has not been reported to replicate outside a fish host. SPDV would not be expected to persist in the environment at a significant titre for as long as infectious pancreatic necrosis virus or *A. salmonicida*. Thus, SPDV would need to enter the aquatic environment continuously and/or at high levels for infection to result.

For susceptible fish to become infected with SPDV, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. SPDV would be expected to spread between fish under conditions in the Australian aquatic environment.

SPDV is not known to infect non-salmonid fish under natural or experimental conditions. There would be a negligible probability of the entry of SPDV into the aquatic environment causing the establishment of SPD in non-salmonid finfish. Any infective material entering the aquatic environment and being consumed by fish would most probably be consumed by non-susceptible species, reducing the probability of Atlantic salmon or trout being exposed to and becoming infected with SPDV.

Repeated high level exposure of susceptible fish to a significant titre of SPDV (for example, from regular discharge of untreated effluent from a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of SPDV into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on salmonids and commercially significant finfish species

Susceptible species in Australia are Atlantic salmon, brown trout and possibly rainbow trout. Based on reports from overseas, losses on affected farms can be significant. Mortality of Atlantic salmon post-smolts may be as high as 60% in exceptional circumstances. However, the main impact of SPD is through decreased food conversion rate, increased time to market, reduced uniformity of carcass weight and the resulting effects on marketing (eg the flow of product to market may be seriously disrupted). Based on overseas reports, if SPDV became established in Australia, the impact could be significant locally or regionally, but not at a national level.

It is expected that in Australia the effects of SPD would be significant because the salmon industry currently benefits from the rapid growth of fish to a uniform harvest size. Measures to manage stress and other factors to minimise mortality may reduce the impact of SPD; however, these measures would increase the cost of producing salmon for market. The effectiveness of recently introduced strategies to extend the harvest season by using out-of-season smolts and the introduction of pre-smolts into brackish water sites would be compromised. The establishment of sleeping disease in farmed rainbow trout in Australia would have similar effects.

Neither infection with SPDV or sleeping sickness has been reported in wild salmonid populations overseas. Thus, it is unlikely that the establishment of either condition would have a significant impact on wild trout populations in Australia. The establishment of SPDV may affect trout hatcheries producing fish for restocking; hence, there may be effects on the recreational salmonid sector locally or regionally, but not at a national level.

Ecological and environmental effects

Infection with SPDV has not been reported in non-salmonid finfish. There is no evidence to suggest that

the establishment of SPDV would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated Atlantic salmon, brown trout and rainbow trout (including juveniles), the probability of establishment of SPDV would be very low. The consequences of establishment would be of moderate significance.

From the risk management matrix presented in Section 1.5.3, for SPDV, the risk associated with the unrestricted importation of eviscerated Atlantic salmon, brown trout and rainbow trout meets Australia's ALOP and the implementation of risk management measures is not warranted.

For the unrestricted importation of all other eviscerated salmonids, the probability of establishment of SPDV would be negligible. Thus, for SPDV, the risk associated with the unrestricted importation of all other eviscerated salmonids meets Australia's ALOP and the implementation of risk management measures is not warranted.

A summary of the risk assessment is shown in Box 4.5.

Box 4.5
Risk assessment — salmon pancreas disease virus

RELEASE ASSESSMENT (R)

The probability of salmon pancreas disease virus (SPDV) entering Australia as a consequence of the unrestricted importation of eviscerated Atlantic salmon, brown trout and rainbow trout would be very low.

Because SPDV is primarily clinically expressed and there is a greater probability of a significant viral titre in juveniles, the probability associated with the unrestricted importation of juvenile Atlantic salmon, brown trout and rainbow trout would be low.

For eviscerated salmonids of other species the probability would be negligible.

EXPOSURE ASSESSMENT (E)

If SPDV entered Australia, the probability of a susceptible fish being exposed to a dose sufficient to cause infection would be very low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of SPDV becoming established in Australia as a consequence of the unrestricted importation of eviscerated Atlantic salmon, brown trout and rainbow trout would be very low (VL).

For the unrestricted importation of eviscerated juvenile salmonids of these species, the probability would be higher but still very low (VL).

For other salmonid species the probability would be negligible (N).

CONSEQUENCE ASSESSMENT

Due primarily to reduced production of Atlantic salmon and the need to change marketing strategies, the consequences of the establishment of SPDV in Australia would be moderate (M).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of SPDV would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL (eviscerated Atlantic salmon, brown trout and rainbow trout, including juveniles) to N (all other eviscerated salmonids)
- significance of consequences = M
- importation risk for SPDV = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated salmonids meets Australia's ALOP; and
- risk management measures are not warranted.

4.2.6 VIRAL HAEMORRHAGIC SEPTICAEMIA VIRUS (VIRAL HAEMORRHAGIC SEPTICAEMIA)

Release assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Viral haemorrhagic septicaemia (VHS) is listed under 'diseases notifiable to the OIE' and is included in List II of the European Union Directive 93/54.
- There is a wide genetic diversity of strains of viral haemorrhagic septicaemia virus (VHSV). The strains of VHSV that occur in salmonids in Europe and North America appear to be distinct, suggesting that there has been little if any sharing of VHSV between those salmonid populations. VHSV has a wide distribution including Continental Europe, Scandinavia and North America.
- The freshwater strains of VHSV found in Europe are highly pathogenic for salmonids. The marine strains of VHSV found in Europe and North America are substantially less virulent.
- In Europe, disease epizootics occur primarily in farmed rainbow and brown trout in fresh water. In the United States, VHS occurs naturally in chinook salmon, coho salmon and steelhead trout. An isolated incident of VHSV infection associated with undiagnosed mortality has been reported in market-size Atlantic salmon in British Columbia.[3] Clinical disease has not been recorded in wild salmonids infected with VHSV in North America.
- In salmonids, VHSV is more common in farmed fish. Based on limited testing of wild salmonid populations, the prevalence of VHSV infection in wild salmonids is considered to be extremely low.
- In Europe, rainbow trout is the species most susceptible to infection. Clinical infection is most common in rainbow trout reared in fresh water.
- VHSV can infect fish of all ages; however, clinical infection is more severe and the mortality rate is higher in young fish. Where infection occurs in wild Pacific salmon, it is most often in sexually mature fish in fresh water.
- Disease may be transmitted horizontally and it is thought that the primary route of infection is via the gills. Live carrier fish are regarded as a significant factor in the transmission of VHSV, presumably because they shed virus into the water. Infection of salmonids via the ingestion of infective material has not been demonstrated.
- Vertical transmission of VHSV has not been demonstrated.
- In clinically infected rainbow trout, the highest titre of virus is in the kidney and spleen. Virus is also found in milt, ovarian fluid, liver, heart and muscle. A neurological form of VHS is associated with a high titre of virus in the brain, and possibly the spinal cord.
- In salmonids, signs of clinical infection may include lethargy, darkening of the skin, exophthalmia, anaemia, and haemorrhage in the eyes, skin, gills and at the base of the fins.
- Clinically infected fish would be visibly abnormal and it would be expected that such fish would be detected and rejected in the course of inspection for human consumption. Carrier fish would not be visibly abnormal and would not be detected at inspection.
- Salmonids that survive infection may become apparently healthy carriers of VHSV. The carrier state is less prevalent in fish in water at a higher temperature. Virus can be isolated from the kidney,

3 In a personal communication, G. Traxler advised that the North Amercian strain of VHSV isolated from farmed Atlantic salmon was not associated with clinical disease or significant losses. Based on previous testing the North AMerican strain of VHSV is not signoficantly pathogenic to salmonids.

spleen, brain and ovarian fluid of carrier fish. While the titre of virus in carrier fish is not known, it is expected that, as for other viruses, the titre would be lower and the tissue distribution would be relatively limited as compared with fish affected by clinical disease.

- If VHSV is present in market-size salmonid fish processed for human consumption, evisceration would significantly reduce the titre of the virus. However parts of the kidney commonly remain attached to the backbone and ribs of fish eviscerated under commercial conditions.

AQIS considered more recent information on VHSV, summarised below.

In advice to the Tasmanian Salmonid Growers Association (TSGA), Dr A Munro reported the isolation of VHSV from 12 marine species out of more than 24 species tested from the Baltic Sea and North Sea, and Atlantic ocean west of Scotland. He stated that the highest prevalence was in herring and sprats in the Baltic off the coast of Denmark. All marine isolates were of negligible virulence for salmon and rainbow trout but several were virulent for turbot. The freshwater strains of VHS that were virulent for rainbow trout were not virulent for salmon (via bath exposure).

There is currently considerable uncertainty about the status and homogeneity of VHSV as a consequence of the discovery of VHSV-like rhabdoviruses in a wide range of marine fish. Using current international standards for diagnosis, all strains were identified as VHSV. However, it is probable that current methods fail to discriminate between viruses in the group. It is now being shown that isolates differ in infectivity and pathogenicity for various fish species (A McVicar pers. comm.).

Key findings

In salmonids, VHSV primarily causes disease in fish in fresh water. The prevalence of VHSV is higher in farmed than in wild salmonids. Rainbow trout are the salmonid species most susceptible to infection. There has been only one instance of VHSV infection (associated with mortality) in Atlantic salmon.

Based on limited studies, the prevalence of infection with VHSV in wild-caught salmonids is extremely low. In wild salmonids the prevalence is highest in fish returning to spawn.

Clinical disease most commonly affects juvenile salmonids that are not normally harvested for human consumption. Because of the pathological changes associated with VHS, salmonids with clinical disease would be visibly abnormal and would be detected in the course of inspection and grading for human consumption.

In apparently healthy infected fish, VHSV may occur in visceral organs and in the brain and ovarian fluid; however, the virus would be at lower titres than in clinically infected fish. Covertly infected fish would not be visibly abnormal and would not be detected at inspection. In such fish, evisceration would substantially reduce the titre of virus present; however, virus may remain in other parts of the body, particularly the head. Unlike pathogens that may be widely dispersed in tissues of chronically infected fish, like *A. salmonicida* and infectious salmon anaemia virus, there is no evidence to suggest that VHSV would be in the somatic musculature of carrier fish of apparently healthy, market-size fish.

Infection of salmonids via the ingestion of infective material has not been demonstrated, suggesting that the minimum infectious dose by the oral route may be high.

There is evidence that strains of VHSV are host specific. For example, the strain isolated from freshwater rainbow trout in Europe was not virulent for Atlantic salmon. The strains of VHSV isolated from non-salmonid marine finfish have been shown to have low to negligible pathogenicity for salmonids.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Susceptible species in Australia include rainbow trout, brown trout, Atlantic salmon, chinook salmon and brook trout as well as a range of non-salmonids. Salmonid populations are found in the cooler southern waters of Australia.

- Under experimental conditions, VHSV is transmitted in water at a temperature of 1–12°C. It is not transmitted at temperatures above 15°C.
- Under natural conditions, outbreaks of VHS occur at water temperatures of 3–12°C, especially 3–5°C. The rate of mortality and prevalence of carrier fish is reduced at higher water temperatures.
- The minimum infective dose is unknown. Infection of salmonids via the ingestion of infective material has not been demonstrated, suggesting that the minimum infectious dose by the oral route may be high. Infection has been achieved by immersing salmonid fish in 5 x 10^4 PFU/mL for three hours.
- The North American strain of VHSV replicates poorly, if at all, in gill and epidermal tissues of rainbow trout and chinook salmon while the virulent European strain replicates effectively.
- VHSV is readily inactivated by several common disinfectants. It is heat labile and survives for 3–10 days at temperatures of 17–22°C; it is totally inactivated at 45°C for one hour. If environmental conditions are favourable, VHSV may persist in the aquatic environment for weeks.

AQIS considered additional information on VHSV, summarised below.

Environmental conditions in many parts of Australia would not be suitable for the establishment of VHSV. Temperatures of marine waters do not fall below 9°C in the coldest regions and for most of the year are too high for VHSV to become established. In areas of Australia where there are populations of freshwater salmonids, water temperatures are low enough for a large part of the year for VHSV to become established.

Key findings

Freshwater salmonids, particularly juveniles, are more susceptible to infection than marine fish. Transmission of VHSV infection is generally limited to waters at a temperature of 1–12°C and does not occur above 15°C. The temperature of marine waters in most parts of Australia is too high for transmission and establishment of VHSV.

It is extremely unlikely that eviscerated market-size fish would contain a titre of VHSV sufficient to initiate infection in susceptible species. Transmission has not been shown to occur after ingestion of infected material, further reducing the probability of infection.

Overseas, the normal hosts for marine strains of VHSV are herring (*Clupea harengus*) and members of the Family Gadidae, neither of which occurs in Australia. The potential for marine finfish in Australian waters, including other members of the Family Clupeidae (such as the bony bream *Nematolosa come*), southern herring (*Harengula abbreviata*), southern sprat (*Sprattus novaehollandiae*) and pilchards (*Sardinops sagax*), to become infected and provide a reservoir for VHSV is uncertain.

VHSV may persist in the aquatic environment for weeks, however it is heat labile and survives for only 3–10 days at temperatures of 17–22°C. Several common disinfectants also readily inactivate it.

For susceptible fish to become infected with VHSV, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. VHSV would be expected to spread between fish under conditions in the Australian aquatic environment, except in waters at a temperature greater than 12°C.

Repeated high level exposure of susceptible fish to a significant titre of VHSV (for example, from regular discharge of untreated effluent of a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of VHSV into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on salmonids and commercially significant finfish species

Based on overseas experience, the effect of the establishment of VHSV would depend on the strain and its characteristics, particularly its pathogenicity and host specificity. The most significant consequences would be expected to arise if a freshwater strain of VHSV virulent for salmonids were to become established in Australia.

The establishment in Australia of European strains of VHSV isolated from non-salmonid marine finfish, which have been shown to be of low virulence, would have little consequence for salmonids or other finfish in Australia.

The establishment of North American strains of VHSV would also be of low significance for salmonids, as these strains appear to be of low virulence for these species, although the potential for the virus to mutate and become more virulent cannot be dismissed (Meyers and Winton 1995). Inapparent natural infection with VHSV has been recorded in coho and chinook salmon in North America. Mortality rates of 0–7% were recorded in eight species of salmonids challenged by immersion with four North American isolates (Stone et al 1997b citing Winton, pers. comm.). However, the establishment of North American strains of VHSV in Australia could have significant consequences for commercially significant non-salmonid species.

In Europe, infection with the freshwater salmonid strains of VHSV causes mortality rates up to 80–100% in rainbow trout fry. Fingerlings and growers are also susceptible to VHSV and virulent strains produce mortality rates of 10–50%. Significant commercial losses (US$40 million per year) are associated with VHS in freshwater salmonids (cited in Humphrey 1995).

In the freshwater environment, husbandry measures such as destocking and disinfection of hatcheries, followed by restocking from pathogen free sources, can be used to prevent and control VHSV infection. Surviving fish are resistant to reinfection (Wolf 1988). Immunisation with a DNA-based vaccine has been shown to confer protective immunity to rainbow trout (Lorenzen et al 1998).

The establishment of freshwater European strains of VHSV in Australia would be expected to cause significant mortality in young rainbow and brown trout, which would cause economic losses in the farmed rainbow trout industry and may affect the recreational trout-fishing sector. Based on the low virulence of freshwater European strains of VHSV for Atlantic salmon, the establishment in Australia of these strains of VHSV would be of very low significance for the Atlantic salmon industry.

The establishment of any strain of VHSV would affect farms exporting eyed ova, as they may be required to implement additional testing and certification to preserve their export markets. However, the effects of establishment of VHSV would primarily be felt regionally or at the level of individual premises rather than nationally or at the level of the whole industry. Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

There is limited information on the effect of VHSV on wild salmonid populations. The establishment of VHSV would be expected to cause some reduction in wild populations of rainbow and brown trout and to affect the recreational fishing industry at a local or regional rather than a national level.

Ecological and environmental effects

There is no evidence to suggest that the establishment of strains of VHSV virulent to salmonids would lead to disease or mortality in native or other fish species in Australia.

Overseas, the normal hosts for marine strains of VHSV are herring (*Clupea harengus*) and members of the Family Gadidae, neither of which occurs in Australia. The potential for marine finfish in Australian waters, including other members of the Family Clupeidae, (such as the bony bream (*Nematolosa come*), southern herring (*Harengula abbreviata*), southern sprat (*Sprattus novaehollandiae*) and pilchards (*Sardinops sagax*) to become infected and provide a reservoir for VHSV is uncertain.

As VHSV has a wide host range and has shown the potential to adapt to new hosts under overseas

conditions, it is expected that some marine finfish in Australia would be susceptible to infection. Given that marine strains of VHSV are not virulent to freshwater salmonids, and there have been no records of these strains causing disease in other freshwater species, it appears unlikely that there would be significant effects on freshwater finfish species, including native fish, in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated salmonids, the probability of establishment of VHSV would be very low. The consequences of establishment of freshwater European strains of VHSV would be of moderate significance. The consequences of establishment of other strains of VHSV would be of low significance.

From the risk management matrix presented in Section 1.5.3, for VHSV, the risk associated with the unrestricted importation of eviscerated salmonids is consistent with Australia's ALOP and the implementation of risk management measures is not warranted.

A summary of the risk assessment is shown in Box 4.6.

Box 4.6
Risk assessment — viral haemorrhagic septicaemia virus

RELEASE ASSESSMENT (R)

The probability of viral haemorrhagic septicaemia virus (VHSV) (all strains) entering Australia as a consequence of the unrestricted importation of eviscerated seawater salmonids would be very low.

Because VHSV is primarily clinically expressed in juvenile salmonids and there is a greater probability of a significant viral titre in juvenile salmonids, freshwater salmonids and sexually mature salmonids, the probability associated with the unrestricted importation of these fish would be low.

EXPOSURE ASSESSMENT (E)

If VHSV (all strains) entered Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be very low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of VHSV (all strains) becoming established in Australia as a consequence of the unrestricted importation of eviscerated salmonids, including freshwater salmonids and all juveniles and sexually mature salmonids, would be very low (VL).

CONSEQUENCE ASSESSMENT

Due primarily to effects on commercial and recreational trout stocks in Australia, the consequences freshwater European strains of VHSV establishing in Australia would be moderate (M).

The effect on the Atlantic salmon industry would not be significant. Effects on the recreational salmonid sector may be significant locally or regionally, but not at a national level.

The consequences of the establishment of marine European strains of VHSV and all strains of VHSV from North America would be low (L), due primarily to the limited impact that these strains of VHSV would have on salmonids and other finfish species in Australia.

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of VHSV would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL
- significance of consequences = M (European strains) to L (other strains)
- importation risk for VHSV = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated salmonids meets Australia's ALOP; and
- risk management measures are not warranted.

4.2.7 *AEROMONAS SALMONICIDA,* TYPICAL (FURUNCULOSIS) AND ATYPICAL STRAINS

In view of the significance of this disease, AQIS has undertaken a review of the literature (see Appendix 7) as a basis for this section of the risk analysis, which also draws upon information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b).

Release assessment

Key findings

The prevalence of infection with typical *A. salmonicida* in wild-caught marine Pacific salmon of market size is normally low (0–10%), higher values applying to sexually mature adult fish in freshwater. Quantitative data are not generally available for other salmonids. In those limited studies that have been conducted — in Scotland (in Atlantic salmon returning to spawn) and Ireland (in sea trout) — infection is not usually found in wild marine salmonids (A McVicar pers. comm.) and this suggests a low prevalence. In other countries where typical *A. salmonicida* occurs, a higher prevalence of infection has been recorded in juvenile salmonids and sexually mature marine salmonids that have returned to fresh water to spawn. There is little evidence to suggest that mature wild fish are covertly infected while in seawater; rather it is thought that exposure and disease occur in fresh water. The probability of *A. salmonicida* infection of product derived from wild-caught, susceptible species would be low, but the likelihood would be greater in species with a high tolerance to infection (where carriers are more common).

In farmed salmonids the prevalence of infection with typical *A. salmonicida* has been high historically. However, the use of effective vaccines and other methods of control have greatly reduced the prevalence of disease and in many countries furunculosis is now much less important than previously. Clinical disease most commonly occurs in smolts after transfer to sea.

Because of the pathological changes associated with this disease, salmonids with clinical furunculosis would be visibly abnormal and would be detected and rejected in the course of inspection and grading of fish for human consumption. Infection of salmonids with atypical strains of *A. salmonicida* causes ulceration of the skin, which could also be detected during inspection. Salmonids that were inapparently infected with typical or atypical strains of *A. salmonicida* would be visibly normal and would not be detected at inspection.

In clinically diseased salmonids, *A salmonicida* is predominantly found in visceral organs but may also be found in muscle tissues; a factor distinguishing this agent from many other significant pathogens that are almost exclusively located in the visceral organs. In covertly infected fish, the pathogen may be found in kidney tissues, gills and skin mucus, generally at low titre. The titre may be higher in inapparently infected fish sourced from a population experiencing an acute furunculosis epizootic or just before an outbreak.

For both clinically and covertly infected fish, evisceration would substantially reduce the titre of *A. salmonicida* present. However, the pathogen may remain in other parts of the body, including the somatic musculature. The titre of *A. salmonicida* in muscle of covertly infected fish would normally be very low. The titre may be higher in muscle of salmonids affected by clinical disease and in sexually mature spawners.

Clinically infected salmonids are likely to be detected and rejected in the course of inspection for human consumption. Adult carrier fish would not be visibly abnormal and would not be detected at inspection. However, the bacterial titre in eviscerated, adult, carrier fish would be extremely low, unless these fish had been derived from a population affected by an acute disease epizootic.

Exposure assessment

AQIS has taken into account the following information.

Short-term exposure to high titres (10^6 colony forming units [CFU]/mL) of *A. salmonicida* initiated infection while long-term exposure (three weeks) was required to initiate infection with a low titre (10^2 CFU/mL) of the pathogen.

While it is difficult to draw conclusions about differences in susceptibility of salmonid fish in the marine and freshwater phases of the lifecycle, the period of stress around the time of smolting, particularly on entry into seawater, could be expected to be a time of higher vulnerability.

Typical *A. salmonicida* may persist for an extended period in the environment, particularly in fresh water and at low temperature (a greater bacterial count was reported at 4°C than at 15°C). Typical *A. salmonicida* bacteria outside fish hosts may enter the viable but non-culturable state; however, the epidemiological significance of such organisms and their capability to cause disease has not been established (Hiney and Olivier 1999). The ability of atypical strains of *A. salmonicida* to persist outside a fish host is unknown, but cannot be discounted.

A. salmonicida (all strains) has a direct lifecycle and conditions in Australian waters would be suitable for the establishment of disease. Clinically infected fish may shed *A. salmonicida* in exudate from lesions, the skin mucus, urine and possibly faeces, contributing to the spread of *A. salmonicida* through water. Disease may also be transferred via contaminated equipment and feed. In natural outbreaks, fish may be exposed to bacteria from infected live fish or decomposing, infected carcases. The skin mucus of covertly infected fish may be a source of infectious pathogens. Under Australian conditions, it is necessary to regularly change nets and dip salmonids to prevent and treat amoebic gill disease. This could predispose Australian salmonids to skin damage, making them more susceptible to infection with *A. salmonicida*.

Non-salmonid marine fish are unlikely to contract infection with typical *A. salmonicida* unless in close proximity to farmed salmonids affected by a disease epizootic. For example, *Ctenolabrus* spp in net pens with salmonids affected by *A. salmonicida* have been infected overseas (*Ctenolabrus* spp do not occur in Australia). Very few of the non-salmonid finfish species recorded with typical *A. salmonicida* infections occur in Australia. While this possibility cannot be discounted, it is unlikely that the entry of typical *A. salmonicida* into the marine environment would lead to the establishment of disease in non-salmonid marine finfish.

It is noted that, for infection with atypical strains of *A. salmonicida* in non-salmonid fish in fresh water, most of the non-salmonid freshwater species infected overseas do not occur in Australia. Non-salmonid finfish in fresh water would be expected to be less susceptible than trout to infection with typical *A. salmonicida*, therefore AQIS considers that there would be a very low probability of disease becoming established in these fish in Australia.

Key findings

All salmonids farmed in Australia would be susceptible to infection with typical, and some strains of atypical, *A. salmonicida*. Non-salmonid freshwater and marine finfish may be susceptible to infection with typical, and some strains of atypical, *A. salmonicida*. However, there is generally little or no evidence that non-salmonid species would be more susceptible to infection than trout. The one exception reported is turbot. It was shown in one experimental study to be more susceptible than rainbow trout to becoming infected by a typical strain of *A. salmonicida* (lower infectious doses). Non-salmonid finfish in Australia would be more likely to become infected with atypical strains than with typical *A. salmonicida*, should these pathogens enter Australia.

Infection may be transmitted horizontally, via exposure to a significant titre of the pathogen in the aquatic environment. A higher titre of typical *A. salmonicida* would generally be required to initiate infection in non-salmonid fish than in salmonid fish. Exposure to a low titre of the pathogen would need to be maintained for a prolonged period for infection to result.

Were typical *A. salmonicida* organisms to enter a freshwater or brackish aquatic environment, they would be expected to survive for a prolonged period in organic material and sediment; however, they would not be expected to survive in the marine environment for a significant period. While there is little definite evidence that atypical strains of *A. salmonicida* would persist to the same extent as the typical strain, this possibility cannot be discounted.

For susceptible fish to become infected with typical or atypical *A. salmonicida*, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficiently high dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts for the disease to establish in the population. Typical or atypical *A. salmonicida* would be expected to spread between fish under conditions in the Australian aquatic environment.

Repeated high-level exposure of susceptible fish to a significant titre of typical or atypical *A. salmonicida* (for example, from regular discharge of untreated effluent of a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of *A. salmonicida* into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have lesser significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on salmonids and commercially significant finfish species

Infection with typical *A. salmonicida* may cause serious disease in farmed salmonids, but is of little pathogenic or economic significance in other finfish. The significance of this disease in salmonids has decreased greatly in recent years with the adoption of effective management strategies that directly address the aspect of lateral/horizontal spread of furunculosis between fish populations both wild and farmed. However, furunculosis caused by typical *A. salmonicida* is still one of the economically significant diseases of farmed salmonids in northern Europe and North America.

Experience in Europe shows that management and veterinary strategies can be used to prevent clinical disease but infection will still occur. The use of antibiotics has decreased significantly because of improvement in farming practices and effective vaccination. Oil adjuvant vaccines are effective in controlling outbreaks of disease, however they also have adverse effects, including the development of lesions in the carcase, increased cost of production and reduced growth rate (Lillehaug et al 1996, Midtlyng 1996). The use of vaccines may also mask the presence of infection. It is possible to vaccinate hatchery fish to be used for the replenishment of wild stocks. However, vaccination provides a limited period of protection, and there is current research interest in developing an oral vaccine (A McVicar pers. comm.).

If disease due to *A. salmonicida* became established in Australia, control measures similar to those used overseas could be implemented. This would necessitate the use of antibiotics that would have a direct cost and could also harm the product image of Australian salmon. The establishment of antibiotic resistant strains of *A. salmonicida* would add to costs and limit the effectiveness of control measures. The introduction of practices, such as 'all in-all out' management, would add to the cost of production, especially for Atlantic salmon farms using out-of-season smolts. Attempts could be made to eradicate disease if it was detected in an isolated locality; however, it is unlikely that disease in wild fish or at multiple sites could be eradicated.

ABARE (1994) reported that reduced fish survival, loss of product and increased costs could threaten the viability of the Australian salmonid industry in the event that furunculosis became established. Effective strategies for the management and prevention of furunculosis have been adopted in countries affected by this disease since ABARE conducted this study. A. McVicar (pers. comm.) advised, 'because of the success of control, furunculosis has now dropped well down the ranking in importance of diseasesÖcurrently affecting the Scottish salmon farming industry'. If disease due to typical *A. salmonicida* was to become established in Australia, it is likely that similar management measures would be adopted. The impact of establishment may be lower than that predicted by ABARE, but it is likely that establishment would result in increased costs and reduced profitability for the salmonid farming industry. Australia's 'disease and chemical residue free' image could also be harmed, reducing the price premium that Australian salmon attracts.

The establishment of disease due to typical *A. salmonicida* in wild freshwater salmonids would be expected to affect the recreational fishery (primarily trout angling) at a local/regional level as infection caused mortality in young and adult fish in naive populations. Although it is likely that the disease could not be eradicated from wild salmonids, experience in the UK suggests that the initial high impact would eventually be reduced as salmonids developed resistance to the pathogen (A McVicar pers. comm.). The adoption of management strategies to prevent the spread of disease to additional freshwater catchments would be expected

to prevent the disease having a significant impact on the recreational sector at the national level.

Infection with atypical strains of *A. salmonicida* has caused significant disease in farmed Atlantic salmon in some cases, but is of little economic significance in other finfish.

Based on experience overseas, the establishment of typical or atypical *A. salmonicida* in non-salmonid fish would not be expected to have significant consequences at a regional or national level. Perhaps the most significant aspect of the establishment of infection in non-salmonids would be the potential for these fish to serve as a reservoir of the pathogen for freshwater salmonids.

Taking into account the expected effects on the farmed and the recreational salmonid sectors, AQIS concludes that the establishment of disease due to typical *A. salmonicida* in Australia would have moderate to high consequences. Taking into account the capability of some atypical strains of *A. salmonicida* to cause disease and mortality in farmed salmonids overseas, the consequences of the establishment of additional atypical strains of *A. salmonicida* in Australia would be moderate.

Ecological and environmental effects

Based on the literature, infection with typical *A. salmonicida* is of little pathogenic or economic significance in non-salmonid finfish, including native fish, overseas. Non-salmonid fish in fresh water would be more likely to be infected with atypical strains than with the typical strain of *A. salmonicida* (A McVicar pers. comm.).

It has been suggested that the establishment of *A. salmonicida* (typical or atypical strains) would threaten the survival of native freshwater species in Australia. For non-salmonid freshwater species, the most common hosts of *A. salmonicida* infection overseas are members of the Family Cyprinidae. There is little evidence that Australian native fish, none of which are closely related to the Family Cyprinidae would be particularly susceptible to infection with typical or atypical strains of *A. salmonicida*. While Australian experience of infection with *A. salmonicida* is limited, atypical strains occur, including the GUD biovar and *A. salmonicida* in greenback flounder. An atypical strain of *A. salmonicida* was detected by an indirect fluorescent antibody test (IFAT), but not isolated, in roach with ulcerative dermatitis in a Victorian lake (cited by Whittington et al 1995). This finding is tentative as the IFAT used was not specific for *A. salmonicida*; it cross-reacted with other *Aeromonas* spp (B Jones pers. comm.). A single case of disease due to the goldfish ulcer disease (GUD) biovar of *A. salmonicida* was reported in native fish (silver perch) at a farm where goldfish had been infected. The following conclusions can be drawn from the behaviour of these pathogens under Australian conditions. The presence of the GUD variant of *A. salmonicida* in Australia has had little consequence other than for the specific premises affected. It has had no discernible effect on wild fish or the environment and has not significantly affected the status of vulnerable or endangered native fish. Similarly, the presence of other atypical strains of *A. salmonicida* in Tasmania and in Victoria has not been associated with disease under natural conditions and has had little consequence for farmed or wild salmonids or native finfish.

AQIS has considered how the entry and establishment of typical *A. salmonicida* or more virulent atypical strains might affect the environment and native fish. The finfish species listed by Environment Australia as vulnerable and/or endangered under the *Endangered Species Protection Act 1992* belong to 13 genera, as listed in Appendix 5. Several factors have led to the current status of these species. The more important contributing factors include predation (including by introduced salmonid species such as brown trout) and degradation of habitat. Equally, it is important to prevent the establishment of exotic diseases that could affect the survival of native species. On the other hand, it could be argued that the establishment of a pathogen that had its main pathogenic effects on introduced salmonid species (such as brown trout) could have positive consequences for vulnerable species such as the galaxids, through a reduction in the population of key predators.

Overseas experience shows that the presence of *A. salmonicida* has had no significant effect on populations of wild non-salmonid fish. Therefore, while the effect of establishment of additional, more virulent strains of *A. salmonicida* cannot be discounted, there is no reason to expect that this would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

Unrestricted risk estimate for importation of salmonids

Unrestricted importing of eviscerated salmonids would result in a low probability of typical *A. salmonicida* establishing. The consequences of establishment would be of moderate to high significance.

From the risk management matrix presented in Section 1.5.3, for typical *A. salmonicida*, the risk associated with the unrestricted importation of eviscerated salmonids does not meet Australia's ALOP and the implementation of risk management measures is warranted.

For the unrestricted importation of eviscerated salmonids, the probability of establishment of additional strains of atypical *A. salmonicida* would be low. The consequences of establishment would be of moderate significance.

Thus, for atypical *A. salmonicida*, the risk associated with the unrestricted importation of eviscerated salmonids does not meet Australia's ALOP and the implementation of risk management measures is warranted.

A summary of the risk assessment is shown in Box 4.7. Appropriate risk management measures are discussed in Chapter 5.

Box 4.7
Risk assessment — *Aeromonas salmonicida* (typical and atypical strains)

RELEASE ASSESSMENT (R)

The probability of typical or additional atypical strains of *A. salmonicida* entering Australia as a consequence of the unrestricted importation of eviscerated wild ocean-caught Pacific salmon would be extremely low.

The probability of typical or additional atypical strains of *A. salmonicida* entering Australia as a consequence of the unrestricted importation of other eviscerated salmonids would be moderate. Because the prevalence of infection and the titre of pathogens may be higher in juvenile salmonids and in sexually mature salmonids, this probability would be higher but still moderate.

Given the very low prevalence of infection of salmonid fish with atypical strains of *A. salmonicida*, the probability of additional strains of atypical *A. salmonicida* entering Australia as a consequence of the unrestricted importation of eviscerated salmonids (including juvenile salmonids and sexually mature salmonids) would be very low.

EXPOSURE ASSESSMENT (E)

If typical *A. salmonicida* entered Australia, the probability of susceptible fish in the marine environment being exposed to a dose sufficient to cause infection would be low. The probability of susceptible fish in the brackish or freshwater environment being exposed to a dose sufficient to cause infection would be higher but still low.

If additional atypical strains of *A. salmonicida* entered Australia, the probability of susceptible fish in the marine or other aquatic environment being exposed to a dose sufficient to cause infection would be low.

Probability of disease establishment (R + E)

The probability of typical or additional atypical strains of *A. salmonicida* becoming established in Australia as a consequence of the unrestricted importation of eviscerated wild ocean-caught Pacific salmon would be extremely low (EL). The probability of typical or additional atypical strains of *A. salmonicida* becoming established in Australia as a consequence of the unrestricted importation of other eviscerated salmonids would be low (L).

Box 4.7 (continued)
Risk assessment — *Aeromonas salmonicida* (typical and atypical strains)

CONSEQUENCE ASSESSMENT

The consequences of the establishment of typical *A. salmonicida* in Australia would be moderate (M) to high (H), due primarily to effects on the farmed and the recreational salmonid sectors. Taking into account the capability of some atypical strains of *A. salmonicida* to cause disease and mortality in farmed salmonids overseas, the consequences of the establishment of additional atypical strains of *A. salmonicida* in Australia would be moderate (M).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of typical or atypical strains of *A. salmonicida* would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

Wild ocean-caught Pacific salmon

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = EL
- significance of consequences = M–H (typical *A. salmonicida*); M (additional strains of atypical *A. salmonicida*)
- importation risk for *A. salmonicida* = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of wild ocean-caught Pacific salmon meets Australia's ALOP; and
- risk management measures are not warranted.

Other salmonids

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L
- significance of consequences = M–H (typical *A. salmonicida*); M (additional strains of atypical *A. salmonicida*)
- importation risk for *A. salmonicida* = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of other eviscerated salmonids does not meet Australia's ALOP; and
- risk management measures are warranted.

4.2.8 *PISCIRICKETTSIA SALMONIS* (PISCIRICKETTSIOSIS)

Some disease agents causing outbreaks of piscirickettsiosis in countries other than Chile may not be identical to *Piscirickettsia salmonis* and are typically reported as a related rickettsia-like organism (RLO). It is unknown, or at least not proven, that the isolates of *Piscirickettsia* from salmonids around the world are the same species (D Bruno pers. comm.). In this risk analysis *Piscirickettsia* isolates from salmonids are all referred to as *P. salmonis*.

RLO reported from Canada and Ireland react positively with a polyclonal antibody against *P. salmonis*, demonstrating their relatedness (Brockelbank et al 1992). Significant differences in virulence between the Chilean (LF-89), a Canadian (ATL-4-91) and a Norwegian (NOR-92) type strain have also been demonstrated. However, phylogenetic analyses demonstrate that strains from different geographic locations form a tight monophyletic cluster with 16S rDNA similarities ranging from 99.7–98.5% (Mauel et al 1999).

Release assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Piscirickettsiosis is listed by the OIE as an 'other significant' disease.
- The geographical distribution of *P. salmonis* includes the major salmonid-producing countries of Chile, Ireland and Norway and the Canadian province of British Columbia. The significance of infection with *P. salmonis* in Chile far exceeds that reported in other countries.
- Piscirickettsiosis has been recorded in coho, pink, chinook and Atlantic salmon and rainbow trout. The highest mortalities are recorded in members of the genus *Oncorhynchus*, particularly coho salmon.
- Piscirickettsiosis normally affects salmonids reared in seawater although it has, on occasion, been isolated from apparently healthy rainbow trout and coho salmon pre-smolts in fresh water.
- There is a single confirmed report of piscirickettsiosis occurring in fish in fresh water. The authors reported concurrent infection of *Renibacterium salmoninarum* and a 2–3°C rise in water temperature which may have contributed to the disease observed.
- Clinical disease is most common in smolts 10–12 weeks after transfer to sea.
- In cases of clinical infection, *P. salmonis* occur in most tissues including the spleen, liver, kidney, heart, brain, ovaries, ovarian fluid, testes, intestine, visceral fat, gills, skin and muscle. Evisceration would substantially reduce the titre of the pathogen in infected fish.
- The clinical signs of infection include scale loss, raised areas of skin in the dorso-lateral regions and patchy haemorrhage on the ventral surfaces. Clinically infected fish would be visibly abnormal and would be detected in the course of inspection and grading for human consumption.
- *P. salmonis* is readily inactivated at freezer temperatures. There was a >99% decline in the $TCID_{50}$ after a single freeze–thaw cycle at –70°C.

Key findings

Piscirickettsiosis is primarily a disease of young, marine-farmed salmonids, although outbreaks were seen in market-size coho salmon (marine-farmed) in Chile in 1989 (these fish were introduced as eyed ova from North America). Infection has not been reported in wild-caught salmonids or in non-salmonid finfish (under natural conditions). In salmonids, gross pathology and clinical signs are most common after transfer to seawater and consistently include pale gills, swollen kidneys and enlarged spleens. Moribund fish are lethargic, dark in colour and swim near the surface.

In Chile, piscirickettsiosis is associated with serious clinical disease and high mortality while in other salmonid farming countries it occurs as a chronic condition with low mortality.

Juvenile salmonid fish (which is the lifecycle stage most likely to have clinical disease) are not usually harvested for human consumption. Adult fish are less likely than juvenile fish to have clinical disease. Clinically infected fish would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption.

Fish with chronic infection commonly have few if any clinical signs. There would be a high likelihood that inapparently infected fish would be harvested for human consumption. Such fish would not be visibly abnormal and would not be detected at inspection.

In chronically infected fish and carriers, many *P. salmonis* cells would be in the visceral tissues, particularly the kidney. In such fish, evisceration would substantially reduce the number of *P. salmonis* cells present but the pathogen may remain in other tissues, including those of the head. Like other pathogens that may be widely dispersed in tissues of chronically infected fish (for example, *Aeromonas salmonicida* and infectious salmon anaemia virus), *P. salmonis* may be in the somatic musculature at a low level in apparently healthy market-size fish.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Salmonids are the only finfish considered to be naturally susceptible to infection with *P. salmonis*. In Australia, susceptible salmonid species may include populations of rainbow trout, chinook salmon and Atlantic salmon, which occur in the cooler southern waters of Australia.
- Data on the source and mode of transmission of *P. salmonis* in the natural environment are limited. However, experimental studies indicated that *P. salmonis* may be transmitted horizontally and that the life cycle is direct. If an intermediate host is required for completion of the life cycle under natural conditions, the availability of a suitable intermediate host in Australia would affect the probability of completion of the life cycle of the pathogen.
- Some studies suggest that infection could be transmitted vertically, however there is no direct evidence to support this hypothesis.

The minimum infective dose is not known. In experimental studies, 100% mortality occurred in fish that received intraperitoneal doses of $10^{3.3}$ to $10^{5.3}$ $TCID_{50}$. Fish held in flow-through tanks with fish that were inoculated intraperitoneal also died from piscirickettsiosis. Given the extremely high doses used in these experimental studies it is likely that the minimum infective dose would be relatively high.

- *P. salmonis* is rapidly inactivated in fresh water but may survive for at least two weeks in seawater.
- The pathogen appears to be sensitive to high temperatures, as it did not infect fish or cause CPE in cell culture when held at 37°C. In culture *P. salmonis* grows optimally at 15–18°C. Replication is greatly retarded above 20°C and below 10°C and does not occur at or above 25°C.

AQIS considered more recent information, summarised below.

Smith et al (1996) reported that the ID_{50} (intraperitoneal route) was $10^{1.9}$ $TCID_{50}$ for coho salmon and $10^{2.1}$ $TCID_{50}$ for rainbow trout. Rainbow trout infected with *P. salmonis* exhibited a lower titre of the pathogen than that reported in smears of kidney from coho salmon. These authors produced piscirickettsiosis in rainbow trout under experimental conditions. However, they reported that trout cleared *P. salmonis* from tissues more efficiently than coho salmon.

Key findings

Piscirickettsiosis is primarily a disease of farmed, juvenile salmonids in seawater. Information on the epidemiology suggests that the minimum infective dose of *P. salmonis* would be higher in adult salmonids than in juvenile salmonids—and may be high in juvenile salmonids relative to pathogens such as *Aeromonas salmonicida,* for which the minimum infectious dose appears to be very low.

Chinook salmon, rainbow trout and Atlantic salmon in Australia would be susceptible to infection with *P. salmonis*. There are no records of *P. salmonis* infections in brown trout or brook trout. Salmonid species such as Atlantic salmon would be particularly susceptible to infection. Natural infections have not been recorded in non-salmonid finfish; infection was induced in flounder after intraperitoneal inoculation. Infection would be unlikely to occur in non-salmonid species in Australia.

P. salmonis may be transmitted horizontally, via exposure to a significant titre of the agent in the marine environment. Exposure to a higher titre of pathogen would be required to initiate infection in fish in the freshwater environment. Exposure to a low titre of rickettsia in the marine environment would need to be maintained for a prolonged period for infection to result. Based on the scientific literature, the presence of *P. salmonis* at a low titre in the freshwater environment would be unlikely to induce infection, regardless of the duration of exposure.

P. salmonis would be expected to be susceptible to inactivation under the physical conditions occurring at sites for disposal of solid waste and in fresh water but would be expected to persist in the marine aquatic environment for a limited period. *P. salmonis* is much more readily inactivated than IPNV. *P. salmonis* has not been reported to replicate outside a fish host and would not be expected to persist in the environment at a significant titre for as long as infectious pancreatic necrosis virus or *Aeromonas salmonicida*. Thus, *P. salmonis* would need to enter the aquatic environment continuously, and/or at high levels, for infection to result.

For fish to become infected with *P. salmonis*, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts for disease to establish in the population. *P. salmonis* would be expected to spread between fish under conditions in the Australian aquatic environment.

Repeated high level exposure of susceptible fish to a significant titre of *P. salmonis* (for example, from regular discharge of untreated effluent from a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of *P. salmonis* into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on salmonids and commercially significant finfish species

In Chile, economic losses due to piscirickettsiosis are highest in spring and autumn. Mortality rates of 30% in coho salmon, 20% in rainbow trout and 10% in Atlantic salmon have been recorded and piscirickettsiosis was estimated to have caused losses greater than US$50 million in 1994.

The high rate of mortality reported in Chile has not been reported in Ireland, British Columbia or Norway.

Atlantic salmon and rainbow trout are the most commercially significant salmonid species farmed in Australia. Although marine-farmed, these species are more resistant to infection with *P. salmonis* than coho salmon. The nature of the Australian salmonid industry (lack of coho salmon and low stocking densities) is such that the establishment of *P. salmonis* in Australia would have limited consequences, similar to the situation in

Norway, Ireland and British Columbia. In culture, *P. salmonis* has optimal growth at 15–18°C, thus the impact of establishment of the agent in Australia may be higher than in the northern hemisphere. If *P. salmonis* caused similar effects in Australia as in Chile, the consequences would be significant.

In many cases, the effects of piscirickettsiosis wane without treatment. In Chile, fish are screened before being selected as broodstock and eggs are disinfected with iodophor. These treatments effectively reduce the prevalence of disease in freshwater salmonid hatcheries. There is no known method for preventing the spread of piscirickettsiosis in salmonids in the seawater phase.

The establishment of *P. salmonis* in Australia would affect farms exporting eyed ova, as they may be required to implement additional testing and certification to preserve their export markets. However, the effects of establishment of *P. salmonis* would primarily be felt at an individual premises or regional level rather than a whole industry or national level. Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

There is no record of *P. salmonis* causing significant disease in wild salmonids. Accordingly the establishment of piscirickettsiosis would have minimal consequence for the salmonid recreational fishing sector.

Ecological and environmental effects

P. salmonis has not been reported in non-salmonid finfish under natural conditions. Thus, there is little evidence to suggest that the establishment of *P. salmonis* would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of salmonids

The probability of *P. salmonis* becoming established in Australia as a consequence of the unrestricted importation of eviscerated marine-farmed salmonids, including juveniles, would be very low. For the unrestricted importation of eviscerated, freshwater-farmed, adult salmonids the probability of establishment of *P. salmonis* would be extremely low. For freshwater-farmed, juvenile salmonids the probability would be very low. For wild-caught salmonids the probability of establishment of *P. salmonis* would be negligible. The consequences of establishment would be of low to moderate significance.

From the risk management matrix presented in Section 1.5.3, for *P. salmonis*, the risk associated with the unrestricted importation of eviscerated salmonid fish, wild and marine and freshwater farmed, including juveniles, meets Australia's ALOP and the implementation of risk management measures is not warranted. A summary of the risk assessment is shown in Box 4.8.

Box 4.8
Risk assessment — *Piscirickettsia salmonis*

RELEASE ASSESSMENT (R)

The unrestricted probability of *P. salmonis* entering Australia as a consequence of the importation of eviscerated, marine-farmed, adult salmonids would be very low.

Because piscirickettsiosis is primarily clinically expressed and there is a greater probability of a significant viral titre in juvenile salmonids, the probability associated with the unrestricted importation of this lifecycle stage of eviscerated, marine-farmed salmonids would be low.

For eviscerated, freshwater-farmed, adult salmonids the probability would be extremely low, while for freshwater-farmed, juvenile salmonids the probability would be very low.

For eviscerated, wild-caught salmonids the probability would be negligible.

EXPOSURE ASSESSMENT (E)

If *P. salmonis* entered Australia, the probability of susceptible fish in the marine environment being exposed to a dose sufficient to cause infection would be very low. In the freshwater environment, the probability would be extremely low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of *P. salmonis* becoming established in Australia as a consequence of the unrestricted importation of eviscerated, marine-farmed salmonids, including juveniles, would be very low (VL).

For the unrestricted importation of eviscerated, freshwater-farmed, adult salmonids the probability of establishment of *P. salmonis* would be extremely low (EL). For freshwater-farmed, juvenile salmonids the probability would be very low (VL).

For wild-caught salmonids the probability of establishment of *P. salmonis* would be negligible (N).

CONSEQUENCE ASSESSMENT

The consequences of the establishment of *P. salmonis* in Australia would be low (L), due to the absence of coho salmon and the limited effects of piscirickettsiosis on Atlantic salmon and rainbow trout in *P. salmonis*-infected countries. However, if piscirickettsiosis caused similar effects in Australia to those reported in Chile, the consequences would be moderate (M), due to the effect on the commercial rainbow trout and Atlantic salmon industry.

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of *P. salmonis* would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL (eviscerated, marine-farmed salmonids, including juveniles and freshwater-farmed, juvenile salmonids) to EL (freshwater-farmed adult salmonids) to N (wild-caught salmonids).
- significance of consequences = L–M
- importation risk for *P. salmonis* = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated salmonid fish, wild and marine and freshwater farmed, including juveniles, meets Australia's ALOP; and
- risk management measures are not warranted.

4.2.9 *RENIBACTERIUM SALMONINARUM* (BACTERIAL KIDNEY DISEASE)

Release assessment

The following key points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- Infection with *R. salmoninarum* is listed by the OIE as an 'other significant' disease.
- BKD is recognised internationally as one of the most prevalent diseases of cultured salmonids. It has a wide geographical distribution that includes most salmon producing countries.
- Natural outbreaks of BKD are restricted to members of the family Salmonidae and the greatest losses are recorded in fish of the genus *Oncorhynchus.*
- Clinical disease is most common in farmed fish but wild fish may also be affected.
- Clinical disease is most likely to occur when smolts are transferred to sea; infections hinder adaptation to seawater and death commonly follows.
- In most circumstances there would be a low prevalence of clinical infection in adult fish. Most infections would be subclinical, but these may become clinical in stressed fish.
- The prevalence of infection in salmonid fish can vary widely between stocks, and at times of stress the prevalence can increase. During the freshwater stages (ie from fry to pre-smolts and again as spawning adults), prevalence is higher than during the marine phase.
- Over a nine-year period, 369 of 2331 (15.8%) wild Pacific salmon from the Pacific northwest of North America tested for *R. salmoninarum* were positive. In a 21-year period, 25,984 fish from the same source were tested and the prevalence was recorded as 4.6%. Prevalence in sockeye salmon and chum salmon in British Columbia was 3.3% and 4.7% respectively. An average prevalence of 20% was recorded in pre-smolt chinook and coho salmon and steelhead trout in downstream migration in the Columbia River.
- Information on distribution and prevalence needs to be assessed with caution because of difficulties in diagnosing infection in subclinically infected fish, however, more recent testing by enzyme-linked immunosorbent assay (ELISA) and indirect fluorescent antibody test (IFAT) may have improved diagnostic accuracy.
- In fish with clinical and subclinical infection with BKD, *R. salmoninarum* localises in the anterior kidney. Evisceration would remove most bacteria but a residue of the anterior kidney would remain attached to the backbone and ribs of fish eviscerated under commercial conditions.

AQIS considered more recent information on the geographical distribution and prevalence of BKD, summarised below.

The Fish Diseases Commission (March 1999) reported that clinical BKD was not reported in Iceland in 1998, but examination of migratory wild Atlantic salmon showed that 1–3% were carriers. BKD was also diagnosed for the first time in wild stocks of Baltic salmon in Sweden. In 1998–99, seven new infected farms were identified in Finland.

BKD was reported for the first time in Denmark in 1997 (Lorenzen et al 1997), but the disease does not appear to have spread since the first outbreaks. A voluntary surveillance program comprising two annual inspections and sampling of approximately 30 broodstock fish farms has been initiated. *R. salmoninarum* has not been detected on any of the farms so far tested (OIE 1999). The source of infection is not known.

Results of immunological testing performed 10 years ago in the United States suggested that some New Zealand salmon were infected with *R. salmoninarum*. Culture tests did not confirm the presumptive diagnosis. The results of specific surveillance provided no evidence that BKD is present in salmonids in New Zealand. Surveillance testing of 2163 freshwater, sea-cage and sea-run salmon in New Zealand in 1986–97 using ELISA,

Gram stain and culture gave negative results. New Zealand reports freedom from BKD (MAF Regulatory Authority 1999; Boustead et al 1999).

Key findings

There is a high likelihood that populations of *Oncorhynchus* spp would be infected with *R. salmoninarum.* The likelihood of infection of other salmonid species would be moderate. Clinical infection is most common in smolts after transfer to sea, however the prevalence of infection is highest during the freshwater phase of the salmonid lifecycle (fry to pre-smolts and in spawning adults).

Infection with *R. salmoninarum* may become systemic, particularly in young fish and spawning adults, and cause the development of visible lesions in the tissues and/or skin. Fish with systemic infection may show several non-specific signs such as exophthalmos, darkening of the skin, ascites, meningitis and haemorrhage around the base of the fins. Clinically infected fish would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption.

Fish with chronic infection commonly have few if any clinical signs. There would be a high likelihood that inapparently infected fish would be harvested for human consumption. Such fish would not be visibly abnormal and would not be detected at inspection.

In chronically infected fish and carriers, most *R. salmoninarum* cells would be in the visceral tissues, particular the kidney. In such fish, evisceration would substantially reduce the number of *R. salmoninarum* cells present but the pathogen may remain in other tissues, including those of the head. Like other pathogens that may be widely dispersed in tissues of chronically infected fish (for example, *A. salmonicida* and infectious salmon anaemia virus), *R. salmoninarum* may be in the somatic musculature, at a low level. The titre of bacteria in the muscle of carrier fish would be much lower than that in visceral tissues, particularly kidney.

Exposure assessment

The following key points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- In Australia, susceptible host species include Atlantic salmon, brook trout, brown trout, chinook salmon and rainbow trout, which are found in the cooler southern waters of Australia.
- *R. salmoninarum* may be transmitted horizontally and vertically. In aquaculture the horizontal route is of greater epidemiological significance.
- The minimum infective dose is unknown. Infection has been achieved by continuously feeding infected raw viscera to juvenile chinook salmon for a period of 41 to 52 days. Experimentally, high doses of *R. salmoninarum* are required to infect fish by bath exposure. Thus, available evidence suggests that the minimum infective dose by either route may be relatively high.
- *R. salmoninarum* is an obligate intracellular organism, with limited capacity for survival in fresh or seawater outside the host. *R. salmoninarum* is readily inactivated by exposure to chlorine.
- *R. salmoninarum* has a direct lifecycle.
- Conditions in parts of the Australian aquatic environment would be suitable for infection of fish, providing for the establishment of disease.
- Subclinically and chronically infected fish may shed *R. salmoninarum* in their faeces, thus disseminating the pathogen.

Key findings

The minimum infective dose of *R. salmoninarum* by the horizontal route may be high in juvenile salmonids relative to pathogens such as *Aeromonas salmonicida*, for which the minimum infectious dose appears to be very low. The minimum infective dose would be higher in adult salmonids than in juvenile salmonids.

R. salmoninarum would have limited capacity to survive outside a fish host, reducing the probability of susceptible salmonids being exposed to the pathogen. Furthermore, the agent is readily inactivated by chlorine.

Thus, washing and processing may help to reduce the probability of susceptible salmonids being exposed to the pathogen. *R. salmoninarum* would not be expected to persist in the environment at a significant titre for as long as hardy pathogens, such as infectious pancreatic necrosis virus or *Aeromonas salmonicida*. Thus, *R. salmoninarum* would need to enter the aquatic environment continuously and/or at high levels for infection to result.

For fish to become infected with *R. salmoninarum*, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. *R. salmoninarum* would be expected to spread between fish under conditions in the Australian aquatic environment.

Infection of non-salmonid fish with *R. salmoninarum* is unlikely to occur, except in exceptional circumstances, for example in fish penned with farmed salmonids affected by an outbreak of BKD. Generally, if infective material entered the aquatic environment and was consumed by fish it would most probably be consumed by non-susceptible species. Therefore, the probability of susceptible salmonids being exposed to and becoming infected with *R. salmoninarum* would be reduced.

It is not expected that there would be sufficient *R. salmoninarum* in eviscerated, apparently healthy salmonids imported for human consumption to induce infection in susceptible host fish.

Repeated high level exposure of susceptible fish to a significant titre of *R. salmoninarum* (for example, from regular discharge of untreated effluent of a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of *R. salmoninarum* into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on salmonids and commercially significant finfish species

The precise magnitude of worldwide economic losses due to BKD is unknown, but it is likely to be considerable (Wiens and Kaattari 1999). The expansion of salmonid culture has helped to spread BKD and, in many areas, it is now recognised as one of the most persistent diseases. In some cases, BKD has caused the loss of up to 80% of Pacific salmon and 40% of Atlantic salmon (Bruno 1986).

Recent advances in treatment and control may reduce the prevalence and clinical effect (including mortality) of BKD. However, effective chemotherapy poses many problems. Some treatments reduce mortality but the benefits are usually transient and mortalities resume once treatment ceases. Erythromycin is currently the drug of choice. It is injected into broodstock before spawning to control vertical transmission, or mixed into the feed of juvenile fish to prevent horizontal transmission. As *R. salmoninarum* can survive and multiply intracellularly, treatment with erythromycin may be used to prevent disease but does not eliminate infection (Fryer and Lannan 1993).

Recent research has focused on developing novel methods to control BKD, including segregation of healthy stock (Stone et al 1997b).

It is expected that all salmonid species in Australia would be susceptible to infection with *R. salmoninarum.* The establishment of BKD in Australia would probably have greatest impact on young farmed freshwater salmonids, which would be most susceptible to infection. Significant losses may also occur in seawater-farmed salmonids. The establishment of *R. salmoninarum* would be expected to reduce hatchery production in the commercial salmonid industry, especially in rainbow trout. Based on the occurrence of clinical BKD in farmed salmonids in Scotland and British Columbia, it is likely that the establishment of *R. salmoninarum* in Australia would also increase costs associated with the production of salmonids for market. It is expected that the establishment of *R. salmoninarum* could cause major losses in production and profitability and would affect the commercial salmonid industry at a national level.

The establishment of BKD would affect farms exporting eyed ova, as they may be required to implement additional testing and certification to preserve their export markets. However, the effects of establishment of BKD would primarily be felt at an individual premises or regional level rather than a whole industry or national level. Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

It is likely that the establishment of *R. salmoninarum* would reduce survival of fish in some wild salmonid populations and would thereby affect the recreational salmonid sector. Based on experience overseas, effects on the recreational salmonid sector may be significant locally or regionally, but not at a national level.

Ecological and environmental effects

Natural infection of non-salmonid fish with *R. salmoninarum* is unusual. If such infection does occur, it is usually in fish penned with, or living in close proximity to, salmonids affected by an outbreak of BKD. *R. salmoninarum* has not been reported to cause disease in wild, non-salmonid finfish under natural conditions overseas. Based on the literature, infection with *R. salmoninarum* is of little pathogenic or economic significance in non-salmonid finfish overseas. There is little evidence to suggest that the establishment of *R. salmoninarum* would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated salmonids of the genus *Oncorhynchus,* and juveniles and sexually mature fish of all salmonid species, the probability of establishment of *R. salmoninarum* would be very low. The consequences of establishment would be of high significance.

From the risk management matrix presented in Section 1.5.3, for *R. salmoninarum*, the risk associated with the unrestricted importation of eviscerated salmonids of the genus *Oncorhynchus*, and juveniles and sexually mature fish of all salmonid species, does not meet Australia's ALOP; and therefore, the implementation of risk management measures is warranted.

For the unrestricted importation of all other eviscerated salmonids, the probability of establishment of *R. salmoninarum* would be lower but still very low. Thus, for *R. salmoninarum*, the risk associated with the unrestricted importation of all other eviscerated salmonids does not meet Australia's ALOP; and and the implementation of risk management measures is warranted. A summary of the risk assessment is shown in Box 4.9. Appropriate measures are discussed in Chapter 5.

Box 4.9
Risk assessment — *Renibacterium salmoninarum* (bacterial kidney disease)

RELEASE ASSESSMENT (R)

The probability of *R. salmoninarum* entering Australia as a consequence of the unrestricted importation of eviscerated salmonids of *Oncorhynchus* spp would be low. Because BKD is primarily expressed in juvenile salmonids and there is a greater probability of a significant bacterial titre in juvenile salmonids and sexually mature salmonids, the probability associated with the unrestricted importation of these lifecycle stages of *Oncorhynchus* spp would be moderate.

For eviscerated salmonids of species other than *Oncorhynchus* spp, the probability would be very low.

For juveniles and sexually mature salmonids of species other than *Oncorhynchus* spp, the probability would be low.

EXPOSURE ASSESSMENT (E)

If *R. salmoninarum* entered Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be very low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of *R. salmoninarum* becoming established in Australia as a consequence of the importation of eviscerated salmonid fish of the genus *Oncorhynchus* and juveniles and sexually mature fish of all salmonid species would be very low (VL).

For the unrestricted importation of all other eviscerated salmonids, the probability of establishment of *R. salmoninarum* would be lower but still very low (VL) (rather than extremely low).

CONSEQUENCE ASSESSMENT

Due primarily to reduced production and profitability in the farmed salmonid industry, the consequences of the establishment of *R. salmoninarum* in Australia would be high (H). Effects on the recreational salmonid sector may be significant locally or regionally, but not at a national level.

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of *R. salmoninarum* would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL
- significance of consequences = H
- importation risk for *R. salmoninarum* = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated salmonids does not meet Australia's ALOP; and
- risk management measures are warranted.

4.2.10 *YERSINIA RUCKERI*, HAGERMAN STRAIN (ENTERIC REDMOUTH DISEASE)

The preferred serotyping scheme for *Yersinia ruckeri* is that devised by Romalde et al (1993) which comprises serotypes O1 to O4. Serotype O1 is subdivided into O1a and O1b. Serotype O1a (formerly Type 1) is the 'Hagerman strain', the most common and virulent of the serotypes (Inglis et al 1993, Austin and Austin 1993).

The IRA addresses the risk associated with serotype O1a, the Hagerman strain of *Y. ruckeri.* This pathogen is on List III of EU Directive 91/67/EEC.

Release assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Enteric redmouth (ERM) is a disease of salmonids caused by pathogenic strains of *Y. ruckeri*, including the Hagerman strain. ERM occurs worldwide in fresh water and saltwater fish.
- The Hagerman strain is the most pathogenic strain of *Y. ruckeri*. It was first isolated in and most commonly affects rainbow trout. Clinical disease most commonly affects farmed juveniles, approximately 7.5 cm in length. The severity of disease peaks at a water temperature of 15–18°C and decreases at temperatures ≤10°C.
- The Hagerman strain of *Y. ruckeri* has been isolated from salmonids other than rainbow trout and several non-salmonid species, including minnows, whitefish, sturgeon, turbot and goldfish.
- Epizootics of ERM in salmonid hatcheries coincide with changes in environmental conditions (water temperature, water quality, overcrowding).
- In salmonids infected in fresh water, disease may persist in the seawater phase of the lifecycle.
- The likelihood of infection with the Hagerman strain would be lower in salmon than in rainbow trout. The prevalence of infection is higher in juvenile fish and is higher in the freshwater than the marine environment. Infection is more common in farmed than wild fish. In British Columbia, 0.31% of wild ripe or spent salmon tested over a 20-year period were positive for *Y. ruckeri*.
- Clinical disease, known as enteric redmouth (ERM), may cause visible abnormalities, including reddening of the mouth and throat, inflammation and erosion of the jaws and palate, darkening of the skin, haemorrhage around the base of the fins, bilateral exophthalmia and sluggish behaviour. Internally, there may be haemorrhage in the muscle, body fat and intestine.
- It is unlikely that clinically infected fish would be harvested and processed for human consumption.
- Disease may be manifested in peracute, acute, subacute and chronic forms. In septicaemic infection, the pathogen may be recovered from most tissues, although it tends to localise in internal organs, particularly the anterior kidney.
- In subclinically and chronically infected fish, *Y. ruckeri* may be recovered from the kidney, lower intestine, spleen and liver in the early stages of infection. Later in the course of infection, the pathogen localises in the lower intestine. Stressed fish shed the pathogen intermittently.
- Chronically infected fish may appear dark and lethargic, with intermittent reversions to an apparently asymptomatic state. However, most chronically infected fish would not be visibly abnormal and would be harvested and processed for human consumption. Evisceration would substantially reduce the titre of pathogen present in such fish.
- In infected market-size fish, *Y. ruckeri* would be present at a low titre (probably not detectable by culture). Most of the pathogens would be in the visceral organs and evisceration would reduce the titre of pathogen in such fish to an extremely low level.

AQIS considered further information, summarised below.

Y. ruckeri has been reported from Australia, Canada, the United States, and most of Europe, as well as Turkey, New Zealand, South Africa, Venezuela and India (Horne

and Barnes 1999, Meier 1986). Most of these reports concern yersiniosis of cold-water fish species, mainly salmonids. The Hagerman strain of *Y. ruckeri* has been reported in Bulgaria, Denmark, France, Italy, Switzerland, West Germany, Canada and the United States (Davies 1991). It has not been reported from Australia or New Zealand.

The Hagerman strain of *Y. ruckeri* has been reported from several non-salmonid species including carp (*Cyprinus carpio*), goldfish (*Carassius auratus*) and sole (*Solea solea*) (reviewed in Horne and Barnes 1999).

ERM is recognised as a disease of farmed rainbow trout, although of more recent concern are the outbreaks of this disease in farmed Atlantic salmon (*Salmo salar*) in fresh and salt water in Norway (Bruno 1990). It is noteworthy that ERM is able to maintain a background level of infection in wild, as well as farmed, populations of fish and appears as a disease only irregularly under stress conditions (A McVicar pers. comm.).

The histopathology of ERM is characterised by a systemic colonisation of the capillaries, in particular those of the gills, kidney, spleen, heart and muscle (Bruno 1990).

Y. ruckeri could be cultured from frozen salmon carcasses after more than six months storage, however the titre of the pathogen was not reported (Anderson et al 1994).

Key findings

Y. ruckeri (Hagerman strain) most commonly affects farmed rainbow trout, particularly juvenile fish. The prevalence of infection is higher in fresh water than in seawater fish and in farmed rather than wild-caught fish. The prevalence of infection in wild-caught salmonids of market size would be extremely low.

Because of the pathological changes associated with this disease, clinically infected salmonids would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. *Y. ruckeri* (Hagerman strain) localises in the viscera, so evisceration would substantially reduce the titre of pathogen present. However, *Y. ruckeri* may remain in tissues such as the gills and the muscle. This proposition is supported by the fact that the recently developed enrichment culture/immunoassay method, which can detect 1–10 cells in a sample, detected 13% carriers in fish from a farm with a history of clinical yersiniosis in Atlantic salmon (Carson 1999).

Inapparently infected salmonids would not be visibly abnormal and would not be detected at inspection. However, in subclinically infected fish, the pathogen occurs at a very low titre and localises in the viscera and, in chronic infections, in the intestine. Thus, evisceration would reduce the titre of pathogen present to an extremely low level.

The pathogen would be expected to survive at freezer temperatures.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- There are many finfish in the Australian freshwater environment that would potentially be susceptible to infection with *Y. ruckeri* (Hagerman strain).
- The most efficient route of infection is unknown; however, *Y. ruckeri* has a direct life cycle and is transmitted horizontally.
- The minimum infective dose is unknown. For many strains of *Y. ruckeri* the LD^{50} appears to be relatively high.
- The agent may survive in the freshwater and brackish environment for considerable periods of time. A study by Fernandez et al (1992) showed that *Y. ruckeri* survived in filtered seawater for 26 days at 20°C and for at least 118 days at 10°C, in the latter case without a marked decline in viable cell counts. However, survival in seawater is generally considered to be limited, relative to that in the freshwater and brackish environment.

AQIS considered further information, summarised below.

Hunter et al (1980) demonstrated the transmission of *Y. ruckeri* from carrier steelhead trout (*Salmo gairdneri* now *Oncorhynchus mykiss*) to healthy fish; the carrier

fish shed pathogens in response to heat stress. The transmission of *Y. ruckeri* from unstressed carrier fish to healthy fish has not been demonstrated (Hunter et al 1980).

Carrier fish may shed large numbers of *Y. ruckeri* into water via faeces. This may cause outbreaks of clinical disease in trout within 3–5 days, particularly in farms with poor husbandry or where the fish are stressed (Bruno 1990).

Busch and Lingg (1975) experimentally challenged rainbow trout by intraperitoneal inoculation of the causative agent of ERM. These authors reported that the pathogen was localised in the lower intestine of 50–75% of fish surviving disease at 60–65 days after infection. Surviving asymptomatic fish had the demonstrable potential for transmission of the disease to other susceptible populations for more than 102 days after infection (Busch and Lingg 1975).

Detection of pathogenic strains of *Y. ruckeri* by conventional culture of rectal swabs from carrier Atlantic salmon, even in groups of fish which have previously suffered an outbreak of clinical disease, was found to be difficult. This suggests that the number of organisms actually excreted in faeces by carriers is relatively low (B Munday pers. comm.).

The spread of *Y. ruckeri* in farmed salmonids is commonly associated with the introduction of inapparently infected salmonids to farms. *Y. ruckeri* has been diagnosed in Canadian salmon, in which inapparent infection commonly occurs (Cornick 1990). Serious epizootics in salmon have been traced to the introduction of carrier fish from infected sources (Cornick 1990).

The movement of carrier trout was implicated as a principal cause for the spread of ERM and seems the likely cause of its introduction to trout farms in Scotland. The causative agent has been found in caged Atlantic salmon parr in Scotland in certain areas. The origin of such infections remains uncertain (Bruno 1990).

Vaccination does not prevent infection of some fish in a population that is exposed to infection. The transfer of vaccinated and apparently healthy carrier fish is a significant route for transmission of ERM to free areas (Bruno 1990).

A study by Flogstad et al (1991) showed that seven different disinfectants could inactivate *Y. ruckeri* in effluent from a salmon slaughterhouse. A three-log reduction in the number of CFU was obtained by heat exposure at 65°C for one minute. Ultraviolet irradiation resulted in poor disinfection, even at very high doses (250m Ws/cm^2). No bacteria could be detected in the water after the addition of ferric salt then chlorine at a rate of 100 mg/L. When used alone, a concentration of 250 mg/L of chlorine was required to inactivate the pathogen. Formic acid at a pH of 2.0 for six minutes provided a three-log reduction in titre of *Y. ruckeri*. Increasing the pH to 12, using sodium hydroxide, resulted in a three-log reduction. The use of a commercial cleaning agent containing chloramine T produced satisfactory results at a dose of 1000 mg/L and exposure for 24 hours.

Long-term survival of *Y. ruckeri* in the aquatic environment at a range of temperatures and salinities has been documented (reviewed in Horne and Barnes 1999). The agent survived more than three months in river, lake and estuarine environments and maintained its virulence in the viable but non-culturable state (Romalde et al 1994). *Y. ruckeri* may be shed in the faeces of wild or farmed fish, aquatic invertebrates and birds; the ability of the organism to persist in an infective state in the aquatic environment is significant (Romalde et al 1994). *Y. ruckeri* may survive for several months in mud sediments, potentially providing a secondary reservoir of infection in pond farms (Bruno 1990).

Romalde et al (1994) reported that during the first 15 days after cells had been added to different environments, the number of culturable cells increased by one log unit in water microcosms and 2–3 log units in sediment systems. The number of culturable bacteria was shown to be greater at 6°C than at 18°C. Each strain studied survived better in river than in estuarine environments.

The expression of ERM is associated with poor husbandry, stress and overcrowding.

Key findings

Y. ruckeri has been isolated from a wide range of finfish hosts. Strains of *Y. ruckeri* other than the Hagerman

strain are routinely isolated from salmonids but not from non-salmonid finfish in Australia. Given the wide host range overseas, it is expected that introduced salmonids, and possibly non-salmonid finfish species, would be susceptible to infection with the Hagerman strain of *Y. ruckeri* should it enter the Australian aquatic environment. Based on the scientific literature, Australian marine salmonids would be more resistant to infection than freshwater salmonids.

ERM may be transferred horizontally; however, transmission of disease has not been shown to occur from unstressed carrier fish to healthy fish. The infective dose may be high in non-stressed fish, relative to fish under stress. It is unlikely that *Y. ruckeri* would be present at a sufficiently high titre in apparently healthy, market-size eviscerated salmonids to induce infection in susceptible hosts.

Waters containing significant populations of Australian salmonids are in the temperature range (15°–18°C) at which clinical disease is expressed. ERM is related to poor husbandry, overcrowding and stress. The relatively high water temperatures in Australia would increase stress on salmonids, however, the pristine nature of the aquatic environment in which salmonids are found and the high standard of husbandry of salmonids in Australia would have a mitigating effect on stress. The endemic strain of *Y. ruckeri* is managed such that infection is largely prevented. It is likely that these strategies would also reduce the probability of infection with the Hagerman strain. The probability of wild salmonids becoming infected would be similarly low.

Y. ruckeri may survive in fresh water and brackish water (particularly in sediment) for a considerable period, but would not persist for as long in the marine environment. In marine waters, *Y. ruckeri* would need to enter the aquatic environment continuously and/or at high levels for infection to result.

For fish to become infected with *Y. ruckeri*, fish of a susceptible species and life-cycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. *Y. ruckeri* would be expected to spread between fish under conditions in the Australian freshwater environment but would be less likely to spread in the marine environment.

Infection of non-salmonid fish with *Y. ruckeri* may occur, but would be more likely to affect freshwater species. It is generally the case that infective material entering the marine environment and being consumed by fish would most probably be consumed by non-susceptible species, reducing the probability of susceptible salmonids being exposed to, and becoming infected with, *Y. ruckeri*.

There would be a greater probability of *Y. ruckeri* becoming established in farmed freshwater salmonids that were overcrowded or otherwise stressed. However, the probability of infected material being discarded into the environment of farmed freshwater fish would be low. Moreover, it is not expected that there would be sufficient *Y. ruckeri* in eviscerated, apparently healthy salmonids imported for human consumption to induce infection in susceptible host fish.

Repeated high-level exposure of susceptible fish to a significant titre of *Y. ruckeri* (for example, from regular discharge of untreated effluent of a salmon processing plant) could result in the establishment of infection. However, sporadic or isolated entries of *Y. ruckeri* into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on salmonids and commercially significant species

In countries where disease due to highly pathogenic strains of *Y. ruckeri* (enteric redmouth — ERM) is endemic in salmonids, mortality rates of 10–15% in affected cohorts are not uncommon (Horne and Barnes 1999). Mortality rates as high as 70% have been recorded in epizootics in fish that were not vaccinated or treated. However, ERM is amenable to prevention, control and treatment through sound husbandry, immunisation, and the use of antimicrobials.

Vaccination is considered to be effective and can achieve the protection of up to 90% of treated fish. Immersion vaccination using commercial preparations is highly effective and has proved cost effective in large-scale commercial use (Bruno 1990). Vaccination has reduced the number of isolations made at the Aberdeen Laboratory from fourteen in 1989 to three in 1998 (D Bruno pers. comm.).

As reported by Rodgers (1991), vaccination against ERM cost farms in England and Wales an average of UK£2495 (UK£399–UK£11,700). If ERM were to become established in Australia, the costs associated with prevention and treatment could be high.

Infection with strains of *Y. ruckeri* that occur in Australia only becomes apparent when parr are moved or otherwise stressed and in smolts after transfer to sea. Losses due to yersiniosis in Australia have not been economically significant, so the development and registration of vaccine has not been warranted to date. In Australia, effective management of stress and reduction in stocking rates prevents disease due to endemic strains of *Y. ruckeri* in farmed salmonids. These strategies would also reduce the probability and the consequences of establishment of the Hagerman strain.

Australian salmonids are susceptible to infection with endemic strains of *Y. ruckeri*, and would be susceptible to infection with the Hagerman strain. However, it is unlikely that infection would become established in wild fish, which are normally subjected to fewer stressors than farmed fish.

Ecological and environmental effects

There are no reports of infection with endemic strains of *Y. ruckeri* in non-salmonid finfish in Australia. Accordingly, it is not expected that the establishment of exotic strains of *Y. ruckeri* in Australia would have significant consequences for non-salmonid finfish or on the environment.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated adult salmonids, the probability of establishment of *Y. ruckeri* (Hagerman strain) would be very low. For juveniles, the probability would be low. The consequences of establishment would be of low to moderate significance.

From the risk management matrix presented in Section 1.5.3, for *Y. ruckeri* (Hagerman strain), the risk associated with the unrestricted importation of eviscerated adult salmonids meets Australia's ALOP and the implementation of risk management measures is not warranted.

The risk associated with the unrestricted importation of eviscerated juvenile salmonids meets Australia's ALOP and the implementation of risk management measures is warranted. A summary of the risk assessment is shown in Box 4.10. Appropriate risk management measures are discussed in Chapter 5.

Box 4.10
Risk assessment —*Yersinia ruckeri*, Hagerman strain (enteric redmouth disease)

RELEASE ASSESSMENT (R)

The probability of *Y. ruckeri* (Hagerman strain) entering Australia as a consequence of the unrestricted importation of eviscerated freshwater salmonids would be very low. The probability of *Y. ruckeri* (Hagerman strain) entering Australia as a consequence of the unrestricted importation of eviscerated marine salmonids (including juveniles) would be lower than that for freshwater salmonids, but still very low.

Because ERM is primarily expressed and there is a greater probability of a significant bacterial titre in juvenile salmonids, the probability associated with the unrestricted importation of juvenile freshwater and marine salmonids would be low.

EXPOSURE ASSESSMENT (E)

If *Y. ruckeri* entered Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of *Y. ruckeri* (Hagerman strain) becoming established in Australia as a consequence of the unrestricted importation of eviscerated freshwater salmonids would be very low (VL).

The probability of *Y. ruckeri* (Hagerman strain) entering Australia as a consequence of the unrestricted importation of eviscerated marine salmonids would be lower than that for freshwater salmonids, but still very low (VL).

Because infection with *Y. ruckeri* (Hagerman strain) is primarily expressed, and there is a greater probability of a significant bacterial titre, in juvenile salmonids, the probability associated with the unrestricted importation of juvenile freshwater and marine salmonids would be low (L).

CONSEQUENCE ASSESSMENT

Due primarily to effects on the farmed and the recreational freshwater salmonid sectors, the consequences of the establishment of *Y. ruckeri* (Hagerman strain) in Australia would be low (L) to moderate (M). Effects on the recreational salmonid sector would not be significant.

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of *Y. ruckeri* (Hagerman strain) would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

Eviscerated adult salmonids

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL
- significance of consequences = L–M
- importation risk for *Y. ruckeri* (Hagerman strain) = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated adult salmonids meets Australia's ALOP; and
- risk management measures are not warranted.

Eviscerated juvenile salmonids

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L
- significance of consequences = L–M
- importation risk for *Y. ruckeri* (Hagerman strain) = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated juvenile salmonids does not meet Australia's ALOP; and
- risk management measures are warranted.

4.2.11 *MICROSPORIDIUM TAKEDAI* (MICROSPORIDIOSIS)

Release assessment

The following key points are based on information in the 1997 report of the New Zealand Government (Stone et al 1997b). This report contains referenced reviews of relevant literature.

- *Microsporidium takedai* infects eight species of freshwater salmonids in Japan.
- In rainbow trout the prevalence may be 100% and in other species the prevalence may be 86–93%.
- Disease outbreaks are restricted to the warmer periods of the year.
- The microspora are obligate intracellular protozoan parasites. Microsporan life cycles are typically direct, with complex development in the host and spore formation. The spore is the infective stage, and natural infestation is by ingestion. Once ingested, the sporoplasm becomes intracellular in the host, typically within macrophages, and the parasite is transported to the target tissues.
- *M. takedai* targets heart and skeletal muscle, producing large (6 mm) spindle-shaped cysts in skeletal muscle and smaller (2 mm) globular cysts in the heart muscle. In acute disease there may be high mortality and up to 130 cysts/g of tissue in the trunk musculature. Heavily infested fish are typically in poor body condition.
- The host mounts an inflammatory response to infestation with phagocytic cells infiltrating the infested tissue. In experimentally infested yearling salmonids, pathological changes started on day 11 post-infestation. Macrophages phagocytise spores and are eventually transported across the epidermis to outside the host. This would reduce the effective spore load within an infested host.

AQIS considered additional information on the distribution and prevalence of *M. takedai*, as follows.

All reports of this parasite are in freshwater salmonids in Japan. Affected species include sockeye salmon, pink salmon, chum salmon, masou salmon, rainbow trout, brown trout and Japanese char (Bruno and Poppe 1996). Mortality associated with *M. takedai* has been observed in wild masou salmon (Urawa 1989).

In chronically infested fish, cysts are generally limited to the heart muscle. There is a strong negative correlation between condition of fish and intensity of infestation (Dykova 1995).

Key findings

M. takedai has only been reported in freshwater salmonids in Japan. The prevalence of infestation with *M. takedai* can be very high, particularly in rainbow trout. Disease outbreaks occur in the warmer months of the year.

In acute infestations, mortality rates may be high and visible spores may be present throughout the skeletal musculature. In chronically infested fish, most spores are found in the heart.

Fish with clinical infestation would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. Chronically infested fish would not be visibly abnormal and would not be detected at inspection. In these fish, most cysts would be in the heart, thus, evisceration would significantly reduce the number of organisms present. There is no evidence to suggest that *M. takedai* would be present in significant numbers in the somatic musculature of chronically infested fish.

Exposure assessment

AQIS considered further information on transmission and agent stability, as summarised below.

M. takedai has a direct lifecycle and disease is transmitted by ingestion of spores in food or by exposure to spores in water (Bruno and Poppe 1996). The mechanism of release of spores from infested fish is unknown; however, it is assumed that acutely infested fish would shed spores.

Data on thermal lability and pH stability are lacking. Based on the characteristics of related microspora, it is likely that *M. takedai* spores would survive freezing (Amigo et al 1996). Some microspora are known to survive in water for up to one year at 4°C (Dykova 1995).

Many chemicals have been tested for efficacy against microsporans. Schmahl and Mehlhorn (1989) recommended immersion of infested fish in 5 or 20 µg toltrazuril/mL of water for 1–4 hours. This treatment should be applied at 2-day intervals for six days, in well aerated water. The chemical kills the vegetative stage but does not affect mature spores.

Key findings

M. takedai causes disease in freshwater salmonids in Japan. The minimum infective dose is unknown.

It is expected that rainbow and brown trout in Australia would be susceptible to infestation. There are no reports of infestation in marine salmonids in Japan, so Australian marine salmonids would not be expected to be susceptible to infestation with *M. takedai*. There are no reports of *M. takedai* infestation in non-salmonid fish in Japan.

The lifecycle of *M. takedai* is direct and disease can be transmitted by ingestion of spores in food or exposure to spores in water. *M. takedai* would be expected to survive in fresh water in Australia for a prolonged period, however, there is no evidence to suggest that it would survive in seawater for a significant period.

For susceptible fish to become infested with *M. takedai*, fish of a susceptible species and life-cycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficient period of time. Infestation would need to be transmitted from the index case of infestation to other susceptible hosts to result in the establishment of disease in the population. *M. takedai* would be expected to spread from one infested fish to another readily under conditions in the Australian freshwater environment.

Repeated high-level exposure of susceptible fish to a significant number of *M. takedai* spores (for example, from regular discharge of untreated effluent from a salmon processing plant) could result in the establishment of infestation. However, sporadic or isolated entries of *M. takedai* into the freshwater environment (for example, via the disposal from pleasure craft of infested food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen.

Consequence assessment

Effects on salmonids and commercially significant finfish species

There is little information on the consequences of *M. takedai* infestation. In outbreaks of acute disease, there may be high mortality in affected salmonids. Fish with visible cysts in the skeletal musculature would not be marketable. Given reports of up to 100% prevalence in rainbow trout in Japan, this may lead to significant short-term effects on individual rainbow trout farms.

No proven treatment is commercially available for microsporan infestation. The most effective means of control seems to be by ensuring that freshwater stocks are not infested before transfer to the sea.

For the farmed salmonid sector, the establishment of *M. takedai* in Australia could cause significant mortality in young rainbow trout farmed in fresh water, which would cause economic losses in the farmed rainbow trout industry. It is expected these effects would be significant locally or regionally, but not at a national level. The establishment of *M. takedai* in Australia is not expected to have a significant effect on the marine-farmed salmonid sector in Australia.

The establishment of *M. takedai* could have some impact on wild populations of rainbow trout and brown trout and, therefore, the recreational salmonid fishery. However, based on information from Japan, there is little evidence to suggest that the effect on wild fish would be significant.

To date, there is no evidence that non-salmonid fish are susceptible to infestation with *M. takedai*. Therefore, the establishment of *M. takedai* in Australia would not be expected to have significant effect on populations of non-salmonid fish.

Ecological and environmental effects

There is no evidence to suggest that the establishment of *M. takedai* would have a significant effect on wild finfish, including native fish in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated freshwater salmonids from Japan the probability of establishment of *M. takedai* would be very low. For the unrestricted importation of marine salmonids from Japan and eviscerated salmonids from other countries, the probability of establishment of *M. takedai* would be negligible. The consequences of establishment would be of low significance.

From the risk management matrix presented in Section 1.5.3, for *M. takedai,* the risk associated with the unrestricted importation of eviscerated salmonids meets Australia's ALOP and the implementation of specific risk management measures is not warranted. A summary of the risk assessment is shown in Box 4.11.

Box 4.11
Risk assessment — *Microsporidium takedai*

RELEASE ASSESSMENT (R)

The probability of *Microsporidium takedai* entering Australia as a consequence of the unrestricted importation of eviscerated freshwater salmonids from Japan would be low.

The probability of *M. takedai* entering Australia as a consequence of the unrestricted importation of eviscerated marine salmonids from Japan and eviscerated salmonids from other countries would be negligible.

EXPOSURE ASSESSMENT (E)

In the event of *M. takedai* entering Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infestation would be very low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of *M. takedai* becoming established in Australia as a consequence of the unrestricted importation of eviscerated freshwater fish from Japan would be very low (VL).

For the unrestricted importation of eviscerated marine salmonids from Japan and eviscerated salmonids from other countries, the probability of *M. takedai* becoming established in Australia would be negligible (N).

CONSEQUENCE ASSESSMENT

Due primarily to the effect on populations of freshwater farmed rainbow trout, the consequences of the establishment of *M. takedai* in Australia would be low (L).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of *M. takedai* would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL (eviscerated freshwater salmonids from Japan) to N (marine salmonids from Japan and eviscerated salmonids from other countries)
- significance of consequences = L
- importation risk for *M. takedai* = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated salmonids meets Australia's ALOP
- risk management measures are not warranted.

4.2.12 *MYXOBOLUS CEREBRALIS* (WHIRLING DISEASE)

Release assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996), which contain referenced reviews of the relevant literature.

- *Myxobolus cerebralis* is present in many salmonid-producing countries of the world including Europe, Africa, and many states of the United States. It is also present in New Zealand at a very low prevalence. It has not been reported in Canada or Alaska.
- The parasite is dependent for completion of its life cycle on the presence of the oligochaete worm *Tubifex tubifex* in which it undergoes an intermediate stage of development to produce triactinomyxon spores, which are then infective for salmonids.
- *M. cerebralis* has only been found in salmonid fish
- Following entry to the body, the parasitic spores invade body cartilage, causing deformity of the body and secondary nervous signs.
- The distribution of spores in clinically infected fish has been found to be approximately 80% in skeletal tissues and 20% in unspecified soft tissues.
- Comparative trials have shown that rainbow trout is the salmonid species that is most susceptible to *M. cerebralis* infection and to the development of clinical disease. Brown trout and coho salmon are more resistant to infection and develop mild or no disease and lake trout are refractory to infection. Sockeye salmon, chinook salmon, Atlantic salmon and brook trout are of intermediate susceptibility to infection and the development of disease.
- Salmonids are most susceptible to infection with *M. cerebralis* at the fry and fingerling stage. Older fish are progressively more resistant to infection and are less likely to develop disease, due to the ossification of cartilage.
- Clinical disease is mostly seen in young freshwater salmonids. The severity of disease is partly dependent on the age of the fish at the time of infection and the magnitude of the infecting dose. Infection of susceptible young fry with *M. cerebralis* can cause up to 100% mortality, yet have little or no effect in fish over six months of age. In hatcheries it is usual to only see mild disease. Disease may not be evident in wild populations.
- Apparently healthy adult fish infected with *M. cerebralis* would not be detected at inspection for human consumption.

AQIS considered other information on *M. cerebralis*, summarised below.

The scientific literature indicates that the manifestation of disease due to this pathogen differ greatly from one salmonid species to another. Rainbow trout are most susceptible to disease and develop the greatest parasitic burden. Coho salmon are relatively resistant to disease; if infected, these fish usually have a lower number of parasitic spores than rainbow trout. Infection due to *M. cerebralis* is rare in anadromous sockeye salmon and Atlantic salmon. Brown trout are much more resistant to disease than rainbow trout.

M. cerebralis has been reliably reported only in salmonids. Some early reports suggested that the parasite was present in tench (*Tinca tinca*), grayling (*Thymallus thymallus*), gudgeon (*Gobio gobio*), perch (*Perca fluviatilis*), pike (*Esox lucius*) and herring (*Clupea harengus*). However, these reports were not confirmed and could be in error due to misidentification of other species of *Myxobolus* (Halliday 1976).

The distribution of the parasite is associated with the distribution of the oligochaete worm *T. tubifex,* which is a normal inhabitant of the freshwater environment and is particularly abundant in organically rich substrates (Markiw 1998). It has been suggested that the prevalence of infection varies in endemic areas according to the topography of the stream habitat: low gradient and slow water flow permitting a build up of silt and tubificid worm populations. Extremes of gradient and river flow have the effect of precluding such a build up (Modin 1998). Variations in environmental conditions may also affect the prevalence and intensity of infection.

There are few data on the prevalence of infection in infected populations. Baldwin et al (1997) reported that

M. cerebralis was present in 7 of 12 species of salmonids in infected rivers, hatcheries and lakes/reservoirs in Montana and that prevalence ranged from 19.4% (brown trout) to 0.5% (cutthroat trout). In contrast, *M. cerebralis* has been detected rarely in rainbow and brown trout in New Zealand which implements an active surveillance program for this pathogen (Boustead 1996).

The distribution of spores in clinically infected fish has been found to be 37% in gill cartilage, 27% in the head cartilage, 16% in spinal column and 20% in unspecified soft tissues. Spores were not detected in the soft tissues of fish with low spore counts in cartilage (Markiw and Wolf 1974). *M. cerebralis* spores have not been found in fish muscle or the parenchymic organs (El-Matbouli 1998 pers. comm.).

The susceptibility of fish to infestation decreases with age as the cartilage ossifies (Halliday 1976). Residual spores may remain in market-size fish. In Scotland, 10 years of examining cartilage from Atlantic salmon heads by the digestion technique failed to detect any evidence of whirling disease (A McVicar pers. comm.).

Key findings

Whirling disease is caused by infestation with *M. cerebralis.* Whirling disease has been reported in many salmonid-producing countries, including New Zealand (where the prevalence is very low). *M. cerebralis* has not been reported in Australia, Alaska or Canada. *M. cerebralis* has only been reported in salmonid fish, the infection being acquired when young salmonid stocks are exposed to a sufficiently high dose of triactinomyxon spores produced in the infected intermediate host, *T. tubifex.* The highest concentration of the intermediate host is found in an organically rich, highly silted, freshwater environment.

The scientific literature indicates that the manifestation of disease due to this pathogen differ greatly from one salmonid species to another. Rainbow trout are most susceptible to disease and develop the greatest parasitic burden. Coho salmon are relatively resistant to disease; if infected, these fish usually have a lower number of parasitic spores than rainbow trout. Disease due to *M. cerebralis* is rare in anadromous sockeye salmon and Atlantic salmon. Brown trout are much more resistant to disease than rainbow trout.

Clinical disease results from exposure of salmonid fry and fingerlings to a sufficiently high dose of triactinomyxon spores. A smaller dose would be required to establish infection in juvenile salmonids than in older fish, and older fish would be unlikely to develop significant disease.

M. cerebralis is mostly confined to cartilage and bone, mainly of the head and gills. The number of spores in infested market-size fish would be significantly lower than in juveniles.

M. cerebralis spores can survive in chilled or frozen product. Infested fish would not be visibly abnormal and would not be detected during inspection.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996), which contain referenced reviews of the relevant literature.

- Salmonid species in Australia would be susceptible to infection; however, brown trout would be relatively resistant to infection.
- *M. cerebralis* has a complex life cycle involving replication in *T. tubifex*. This tubificid worm is the only known intermediate host for *M. cerebralis,* and is present in Australia.
- A dose of 8–350 *M. cerebralis* spores can infect *T. tubifex,* leading to production of triactinomyxon spores after about 110 days. Triactinomyxon spore production can continue for over 12 months, giving triactinomyxon spore yields as high as 5–10 times the infecting dose of *M. cerebralis* spores.
- The infective dose of triactinomyxon spores for fish depends of the viability of the spores and the species and age of the fish. Rainbow trout (the most susceptible species) were resistant to infection when exposed to 1–10 active spores. Infection was established with a dose of ≥100 spores. Adult fish continuously exposed to 100,000 fresh spores per fish for 3.5 months developed asymptomatic infection.

- *M. cerebralis* spores may be present in cartilaginous and bony tissues of eviscerated salmonids. Most spores are found in the cartilage of the head and gill arches. Fewer occur in the vertebral column, ribs and possibly fins.
- *M. cerebralis* spores are relatively stable in chilled or frozen product. They are reported to survive at least 18 days at -18°C and 3 months at -20°C. Hot smoking (eviscerated/brined/air dried at 66°C for 40 minutes) is reported to inactivate all spores. *M. cerebralis* spores can survive in mud at 13°C for at least 5 months and can survive passage through the gastro-intestinal tract of birds.
- Triactinomyxon spores remain viable for a short period of time, becoming non-infective after 4 days at 12.5°C, three days at 20°C and two days at 24°C.
- If it became established in Australia, *M. cerebralis* could be transferred to other sites via the movement of the infected tubificid worm or the infected salmonid host. Such spread would be conditional on the presence of susceptible salmonid hosts and tubificid worms.

AQIS considered additional information on whirling disease, summarised below.

Several *Myxobolus* spp have been detected in Australian fish (Langdon 1990; P Durham pers. comm. 1996, Rothwell et al 1997). However *M. cerebralis* has not been detected in Australian salmonids or other finfish.

T. tubifex has a widespread distribution in Australia, including in regions where there are salmonid populations, although *T. tubifex* is present at a much lower density than other oligochaetes (Pinder pers. comm.). *T. tubifex* is the only oligochaete that is a competent host for *M. cerebralis* and high numbers of the oligochaete are usually associated with bottom substrates that have a high organic content, such as occur in grossly polluted streams or oligotrophic lakes (Pinder and Brinkhurst 1994), where there are few salmonid fish. Such conditions may occur in earthen ponds used for salmonid aquaculture and in sedimentation ponds, which may also contain escaped salmonid fish. While many relatively slow-flowing, organically rich streams in southern Australia contain populations of brown trout, these fish are relatively resistant to infection with *M. cerebralis*. However, rainbow trout may also be found in some of these streams and this species is highly susceptible to disease due to *M. cerebralis*.

Key findings

All Australian salmon species would be susceptible, to some extent, to infection with *M. cerebralis.* Fry and fingerlings, especially of rainbow trout would be most susceptible. Atlantic salmon would be less susceptible and brown trout would be relatively resistant to disease due to *M. cerebralis*.

In eviscerated salmonids, *M. cerebralis* spores may occur in cartilage and bone, especially of the head and gill arches. For infection to occur, infected cartilaginous material would have to be discarded into an aquatic environment containing *T. tubifex*. Salmonid fish would have to be exposed to the relatively short-lived triactinomyxon spores from *T. tubifex,* at a dose sufficient to initiate infection.

T. tubifex has a widespread distribution in Australia, including in regions where there are salmonid populations, although *T. tubifex* is present at a much lower density than other oligochaetes (Pinder pers. comm.). *T. tubifex* is the only oligochaete that is a competent host for *M. cerebralis.* High numbers of *T. tubifex* are usually associated with bottom substrates that have a high organic content, such as occur in grossly polluted streams or oligotrophic lakes (Pinder and Brinkhurst 1994), where there are few salmonid fish. Such conditions may occur in earthen ponds used for salmonid aquaculture and in sedimentation ponds, which may also contain escaped salmonid fish. While many relatively slow-flowing, organically rich streams in southern Australia contain populations of brown trout, these fish are relatively resistant to infection with *M. cerebralis*. However, rainbow trout may also be found in some of these streams and this species is highly susceptible to infestation with *M. cerebralis*.

M. cerebralis spores could survive in mud for many months.

If infected cartilage or bone were to enter salmonid hatchery tanks or waterways containing an accumulation of organically rich sediment containing *T. tubifex, M. cerebralis* could complete its life cycle in juvenile salmonids. However, this would be unlikely to occur.

Consequence assessment

Effects on salmonids and commercially significant finfish species

Whirling disease may cause high losses in affected populations, but this is not always the case. Modin (1998) reported that many endemically infected streams in California supported high quality salmonid populations despite the presence of *M. cerebralis*. Similarly, the presence of *M. cerebralis* (at a very low prevalence) in New Zealand has not caused significant harm to salmonid stocks (Boustead 1996).

However, in Colorado and Montana, whirling disease has been reported as having a significant impact on salmonid populations (Nehring and Walker 1996, Vincent 1996). Nehring and Walker (1996) contrasted the serious impact of whirling disease on wild salmonid populations in the intermountain west of the United States with its minor impact on salmonid populations of the Pacific Coast and Columbia River basin and the eastern and central parts of the United States.

Whirling disease mainly affects young salmonids in fresh water and may cause 100% mortality with no other clinical signs in heavily infected fry in muddy conditions and at high stocking. The only effect of infection on fish over six months of age may be the development of skeletal deformities. The severity of disease depends on the level of challenge, the age and species of fish and the hatchery management system.

Frasca et al (1999) linked mortalities in Atlantic salmon smolts in Ireland with parasitic encephalitis, possibly due to *M. cerebralis* infestation. This syndrome has only been reported from a single farm in Ireland.

Whirling disease can be controlled, or prevented, by avoiding exposure of fish under four months of age to infective spores. This is achieved by adopting hygienic management practices, including the use of clean stream or spring/bore water or water filtration. Young salmonids can be protected from whirling disease by rearing them in clean conditions, on concrete, plastic or metal, with regular removal of silt and debris to prevent exposure to *T. tubifex.* After four months of age, the fish are relatively resistant to infection.

Two outbreaks of whirling disease that occurred in rainbow trout farms in Scotland were successfully managed by rearing young fish in tanks without sediment or mud bottom. The infection was successfully eradicated without significant loss of fish, or decrease in product quality, and there was no recurrence of infection. The success in avoiding the disease by simple farm management methods removed one of the main criteria for the disease being notifiable in the UK, namely the availability of efficacious prevention or treatment methods. Although still on the statute, whirling disease is no longer subject to official control. During subsequent regular inspections of freshwater farms and wild fisheries, there have been no indications of clinical signs of whirling disease (A McVicar pers. comm.).

Other ways to manage whirling disease include health certification of introduced live fish stocks and treatment of infected fish with fumigillin, which has some success in reducing the prevalence of infection and the production of spores (El-Matbouli and Hoffman 1991).

All salmonids in Australia would be susceptible to a lesser or greater extent to infection with *M. cerebralis*. Atlantic salmon are much less susceptible to disease due to *M.cerebralis* than rainbow trout. Under Australian conditions, young Atlantic salmon are normally raised in tanks and concrete raceways until transfer to the sea. Thus, the establishment in Australia of *M. cerebralis* would not be expected to have significant impact on the Australian Atlantic salmon industry.

In Australia, rainbow trout are commonly farmed in earthen ponds with significant populations of oligochaetes. It is expected that the establishment of *M. cerebralis* in Australia would cause significant mortality in young rainbow trout, which would cause economic losses in the farmed rainbow trout industry and may affect the recreational trout-fishing sector. However, because of the environmental conditions found in the areas where Australian salmonids occur, the probability of establishment of whirling disease would be

very low. If the pathogen became established, measures similar to those used overseas could be used to contain its spread. Thus, the impact of whirling disease on the recreational trout-fishing sector could be significant locally or regionally but not at the national level.

Ecological and environmental effects

There is no report of infection with *M. cerebralis* in non-salmonid finfish. Hence, there is no evidence that the establishment of *M. cerebralis* in Australia would have any impact on wild non-salmonid fish or native fish. In New Zealand, galaxid species occur in rivers where *M. cerebralis* is endemic in salmonids and there is no evidence that the galaxids become infected or are susceptible to infection.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated rainbow trout and juvenile salmonids, the probability of establishment of *M. cerebralis* would be low. The consequences of establishment would be of low to moderate significance. From the risk management matrix presented in Section 1.5.3, for *M. cerebralis*, the risk associated with the unrestricted importation of eviscerated rainbow trout and juvenile salmonids does not meet Australia's ALOP and the implementation of risk management measures is warranted.

For the unrestricted importation of eviscerated adult salmonids (other than rainbow trout), the probability of establishment of *M. cerebralis* would be very low. The consequences of establishment would be of low to moderate significance. However, if the syndrome reported in Atlantic salmon at a single farm in Ireland was to occur in Australia as a result of the establishment of *M. cerebralis*, the consequences would be more serious but would still be moderate.

Thus, for *M. cerebralis*, the risk associated with the unrestricted importation of eviscerated adult salmonids (other than rainbow trout) meets Australia's ALOP and the implementation of risk management measures are not warranted. A summary of the risk assessment is shown in Box 4.12. Appropriate measures are discussed in Chapter 5.

Box 4.12
Risk assessment — *Myxobolus cerebralis* (whirling disease)

RELEASE ASSESSMENT (R)

The probability of *M. cerebralis* entering Australia as a consequence of the unrestricted importation of eviscerated salmonids (other than rainbow trout) would be very low. Because *M. cerebralis* is primarily expressed in juvenile salmonids and there is a greater probability of a significant parasitic load in juvenile salmonids, the probability associated with the unrestricted importation of juvenile fish of all salmonid species would be low.

The probability of *M. cerebralis* entering Australia as a consequence of the unrestricted importation of eviscerated rainbow trout would be low. Because *M. cerebralis* is primarily expressed in juvenile rainbow trout and there is a greater probability of a significant parasitic load in juvenile rainbow trout, the probability associated with the unrestricted importation of juvenile rainbow trout would be moderate.

EXPOSURE ASSESSMENT (E)

If *M. cerebralis* entered Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of *M. cerebralis* becoming established in Australia as a consequence of the unrestricted importation of eviscerated adult salmonids (other than rainbow trout) would be very low (VL). For juveniles the probability would be low (L).

For the unrestricted importation of eviscerated rainbow trout, including juveniles, the probability of *M. cerebralis* becoming established in Australia would be low (L).

CONSEQUENCE ASSESSMENT

The consequences of the establishment of *M. cerebralis* in Australia would be low (L) to moderate (M), due primarily to the reduced supply of juvenile rainbow trout and the effect of a reduced population of trout on the recreational sector.

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of *M. cerebralis* would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

For eviscerated rainbow trout and juvenile salmonids

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L
- significance of consequences = L–M
- importation risk for *M. cerebralis* = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated rainbow trout and juvenile salmonids does not meet Australia's ALOP; and
- risk management measures are warranted.

For eviscerated adult salmonids (other than rainbow trout)

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL
- significance of consequences = L–M
- importation risk for *M. cerebralis* = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of eviscerated salmonids (other than rainbow trout and juveniles) meets Australia's ALOP; and
- risk management measures are not warranted.

4.2.13 PROLIFERATIVE KIDNEY DISEASE AGENT (PROLIFERATIVE KIDNEY DISEASE)

Release assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Proliferative kidney disease (PKD) is caused by the myxosporean proliferative kidney disease agent (PKX), probably of the genus *Sphaerospora*.
- PKD has been reported in North America and Europe.
- Natural disease has been seen in America and Europe in rainbow trout, brown trout, steelhead trout, grayling, arctic char, coho salmon, chinook salmon, Atlantic salmon and European pike (*Esox lucius*). The kokanee salmon (*Oncorhynchus nerka*) has been experimentally infected.
- *Oncorhynchus* spp are the salmonid species most susceptible to infestation.
- Clinical disease is most common in 1–2-year-old freshwater salmonids. Disease can also develop after transfer of infested fish to saltwater.
- After infestation of a susceptible host, the organism undergoes a first, extrasporogonic phase of development in the blood vessels and interstitium of the kidney, which causes a severe, long-term inflammatory response in the kidney that is characteristic of PKD. A second, sporogonic phase of development is then thought to occur in the kidney tubules. In clinical disease, extrasporogenic stages of PKX may occur at low titre in the gills, liver, spleen, caeca, pancreas and muscle, provoking an inflammatory response in these tissues.
- The myxosporean spores can be detected from 2–3 weeks after infestation and for several months after recovery from clinical disease. Lesions begin to heal 12 weeks after infestation and may be completely resolved by 20 weeks post-infestation.
- Fish that have been exposed to infestation are immune to subsequent infestation. There is no specific means of detecting carrier or subclinically infected fish.
- It is extremely unlikely that infective stages of these organisms would be present in commercially harvested market-size salmonids.

AQIS considered further information on PKX, summarised below.

Anderson et al (1999) provided molecular data suggesting that bryozoa are hosts of PKX. Further evidence provided by Longshaw et al (1999) suggests that bryozoa are the intermediate or alternative hosts for PKX and that some fish (other than salmonids) are the normal teleost hosts.

Key findings

PKX is present in most salmon-producing countries in Europe and North America. The salmonid species most susceptible to infestation are Pacific salmon (pink, chum, coho, sockeye, and chinook salmon) and rainbow trout. Disease occurs most commonly in 1–2-year-old fish in fresh water, and can occur in fish after transfer to seawater.

The sporogenic phase of PKX does not appear to mature in salmonids. If correct, the probability of salmonids imported into Australia containing PKX organisms infective for intermediate hosts would be negligible. If salmonids are accidental or dead-end hosts for PKX, other finfish species may be the definitive host for PKX.

PKX is mainly found in the kidney, where parasitic spores can be detected from 2–3 weeks after infection and can persist for several months after clinical recovery. In cases of clinical disease, the extrasporogonic stage of PKX (which would not be infective for the intermediate host) can be found in gills, liver, spleen, caeca, pancreas and muscle.

Adult fish are less likely than juvenile fish to have clinical disease.

Because of the pathological changes associated with this disease, salmonids with clinical disease would be visibly abnormal and would be detected and rejected in the course of inspection of fish for human consumption.

Adult fish surviving infestation with PKX may be apparently healthy carriers for several months after clinical recovery. Chronically infected carrier fish would not be visibly abnormal and would not be detected at inspection. In carrier salmonids, the sporogonic stage of PKX that is primarily located in the kidney (mainly in the mesonephros or posterior kidney) may be present for many months. In such fish, evisceration would substantially reduce the titre of parasite; however the pathogen may remain in the carcase at a low titre.

The physical stability of the agent is unknown; however *Sphaerospora* spp are generally not very resistant to physical treatment such as freezing.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- It is thought other finfish species may be the definitive host for PKX and that salmonids may be an accidental or dead-end host, as the sporogenic phase of the pathogen does not appear to mature in salmonids and the myxosporean does not complete its life cycle in these host fish.
- Susceptible hosts in Australia would include *Oncorhynchus* spp (rainbow trout and chinook salmon) and *Salmo* species (Atlantic salmon, brown trout). Natural populations of these species are found in the cooler southern waters of Australia. If non-salmonid finfish are the definitive host for PKX, it is possible that finfish in Australian waters would be susceptible to infestation, however there is little evidence for this at this time.
- PKD cannot be directly transmitted from fish to fish or by keeping susceptible fish in the effluent water of infected fish. Studies have indicated that susceptible fish do not contract infestation when fed on PKX-infected fish.
- Susceptible fish can be infected when held in fresh water containing the pathogen or in fresh water with sediment containing the pathogen. There is evidence that an intermediate invertebrate host is required for completion of the pathogen's life cycle. There is no definitive information on the distribution of suspected intermediate hosts in Australia.
- The minimum infective dose for this agent is unknown.
- The stability of PKX outside the host is not known. However, there is evidence that the infective stage spends part of its lifecycle in the sediment or in the water column. If PKX is a member of the genus *Sphaerospora*, it is unlikely to be highly resistant to physical treatment or chemicals such as chlorine.

Key findings

PKD is primarily a disease of young farmed salmonids in fresh water. To date, there is no evidence that non-salmonid fish (other than European pike) are susceptible to infestation with PKX.

Of the salmonids present in Australia, only chinook salmon and rainbow trout would be expected to be highly susceptible to infestation with PKX, and to develop clinical disease as a result of infestation. Other salmonids in Australia (including Atlantic salmon and brown trout) would be less susceptible to infestation. There is no evidence that non-salmonid fish in Australia would be susceptible to infestation.

PKX is thought to have an indirect lifecycle. Direct transmission from fish to fish is not thought to occur. It has been shown that susceptible fish do not contract infestation with PKX via the ingestion of infected fish. There is no definitive information on the intermediate host(s) of PKX or on the distribution of putative bryozoan or oligochaete intermediate hosts in Australia.

There is limited information on the stability of PKX outside the fish host. The pathogen appears to survive for some time in fresh water or sediment in fresh water.

In order for susceptible fish to become infected, fish of susceptible species and age would need to be exposed to a sufficient dose of the pathogen in combination with the intermediate host for a sufficiently prolonged period. Infestation would need to be transmitted from the index case of infestation to other susceptible hosts to result in the establishment of disease in the population. Lacking definitive information on the intermediate host of PKX,

the ability of the pathogen to spread in Australian waters is unknown.

Repeated high-level exposure of suitable intermediate hosts and susceptible fish to a significant titre of PKX (for example, from regular discharge of untreated effluent of a salmon processing plant) could result in the establishment of infestation. However sporadic or isolated entries of PKX into the aquatic environment (for example, via disposal from pleasure craft of infected food scraps) would be expected to have lesser significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible life stage being exposed to infested intermediate hosts.

Consequence assessment

Effects on salmonids and commercially significant species

PKD is described as one of the most economically significant diseases of farmed rainbow trout in Europe. It causes significant losses in rainbow trout and Pacific salmon populations of western North America. In Europe, the disease has been estimated to cost fish farmers US$2.5 million per annum due to reduction of food conversion efficiency and fish quality and costs associated with the control of disease. Losses of up to 95% of chinook salmon, 13% of coho salmon and 18% of steelhead trout have been recorded. In rainbow trout, mortality rates up to 20% have been recorded. However, the presence of intercurrent disease complicates attempts to gauge the contribution of PKD to mortality.

Atlantic salmon are susceptible to infestation with PKX and may develop clinical disease, the severity of which may vary with the strain of fish. Atlantic salmon infected with PKX generally show less serious pathological effects and lower rates of mortality than reported in brown trout and rainbow trout. It has been shown that the use of diluted seawater reduces the pathogenic effects of PKD in Atlantic salmon parr (O'Hara, cited by A McVicar pers. comm.).

The mortality associated with PKD probably reflects the contribution of other stressors in addition to infestation by PKX. Good husbandry practices, such as low stocking density, are effective means of control. Treatment with malachite green (banned in many countries) or fumagillin may also be effective.

Of the salmonids present in Australia, only chinook salmon and rainbow trout would be expected to be highly susceptible to infestation with PKX, and to develop clinical disease as a result of infestation. Other salmonids in Australia (including Atlantic salmon, brown trout and brook trout) would be less susceptible to infestation.

For the farmed salmonid sector, the establishment of PKX in Australia could cause significant mortality in young rainbow trout, which would cause economic losses in the farmed rainbow trout industry. Based on overseas experience, it is expected that the consequences could be significant for the Australian Atlantic salmon industry locally or regionally, but not at a national level.

It is likely that the establishment of PKX would have some impact on wild populations of rainbow trout and chinook salmon and, therefore, the recreational salmonid fishery. It is expected that PKD would reduce wild salmonid populations through its effect on salmonid survival. The establishment of PKX would be expected to have a significant effect on the recreational fishing industry locally or regionally, but not at a national level.

To date, there is no evidence that non-salmonid fish in Australia would be susceptible to infestation with PKX.

Ecological and environmental effects

PKX has not been reported to infest non-salmonid finfish, other than European pike (*Esox lucius*), under natural conditions overseas. *E. lucius* and other members of the Family Esocidae occur only in the Northern hemisphere, thus the probability of PKX infesting non-salmonid finfish including native fish in Australia would be low.

Based on the literature, infection with PKX is of little pathogenic or economic significance in non-salmonid finfish overseas. There is little evidence to suggest that the establishment of PKX would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of salmonids

For the unrestricted importation of eviscerated salmonids, including *Oncorhynchus* spp, the probability of establishment of PKX would be very low. The consequences of establishment would be of low to moderate significance.

From the risk management matrix presented in Section 1.5.3, for PKX, the risk associated with the unrestricted importation of eviscerated salmonids meets Australia's ALOP and the implementation of risk management measures is not warranted. A summary of the risk assessment is shown in Box 4.13.

Box 4.13
Risk assessment — proliferative kidney disease agent

RELEASE ASSESSMENT (R)

The probability of proliferative kidney disease (PKX) entering Australia as a consequence of the unrestricted importation of eviscerated *Oncorhynchus* spp would be low.

The probability of PKX entering Australia as a consequence of the unrestricted importation of salmonids other than *Oncorhynchus* spp would be very low.

EXPOSURE ASSESSMENT (E)

In the event of PKX entering Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infestation would be very low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability that PKX would become established in Australia as a consequence of the unrestricted importation of eviscerated salmonids, including *Oncorhynchus* spp, would be very low (VL).

CONSEQUENCE ASSESSMENT

Due primarily to the effect on populations of farmed and wild *Oncorhynchus* spp, the consequences of establishment of PKX in Australia would be low (L) to moderate (M).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of PKX would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL
- significance of consequences = L–M
- importation risk for PKX = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of eviscerated salmonids, including juveniles and sexually mature fish, meets Australia's ALOP; and
- risk management measures are not warranted.

4.2.14 *GYRODACTYLUS SALARIS* (GYRODACTYLOSIS)

This parasite was not considered in the previous AQIS reports (DPIE 1995, 1996), or in the 1997 report of the New Zealand Government (Stone et al 1997b).

Release assessment

Geographical distribution

Gyrodactylus salaris occurs in Europe, including Spain, Germany, Russia, Finland, Sweden, Norway, Denmark, Portugal, Ukraine, Georgia and France (Soleng and Bakke 1997). There are now serious doubts about the actual identity of the parasite recorded as *G. salaris* from Bosnia-Herzogovina (former Yugoslavia). *G. salaris* has not been reported in the UK after active surveillance for the parasite and protective measures have been introduced to restrict trade within the EU as a consequence. It is probable that the widespread distribution of *G. salaris* through several countries in continental Europe is associated with the movement of live rainbow trout for farming and restocking purposes (A McVicar pers. comm.).

G. salaris is listed by the OIE as an 'other significant' disease.

Host range and prevalence

Self-sustaining populations of *G. salaris* occur on salmonids in fresh water and brackish water. Populations of viable *G. salaris* have only been recorded from Atlantic salmon, Arctic char and rainbow trout (A McVicar pers. comm.). Although *G. salaris* will transiently parasitise various non-salmonid finfish, the parasite does not reproduce on non-salmonid fish (Bakke et al 1996).

On infested fish, including Atlantic salmon in the Baltic, the number of *G. salaris* is normally limited by the host's immune response. However, on some strains of Norwegian Atlantic salmon the number of parasites increases until the host dies. It is probable that all strains of Norwegian salmon and western Swedish salmon are vulnerable to serious disease from *G. salaris* (ie salmon feeding in the North Atlantic/Norwegian Sea as opposed to the Baltic race of Atlantic salmon which do not leave the Baltic Sea). This has been shown under natural conditions in Norway and western Sweden and experimentally with Scottish and Norwegian Atlantic salmon (A McVicar pers. comm.).

Disease due to infestation with *G. salaris* is rare in other salmonid species, probably due to innate resistance and the effectiveness of the host response. In parasitised rainbow trout and brook trout, the host response 8–20 days after infection limited the numbers of the parasite and on brown trout, whitefish and lake trout, the number of parasites was not significant (Bakke et al 1992). The susceptibility of individual fish in a population may be affected by genetically determined factors.

G. salaris cannot sustain a viable population on brown trout and the parasite appears to die very rapidly; that is, there is preliminary evidence for an active process of elimination of *G. salaris* from parasitised *Salmo trutta* (A McVicar, pers. comm.).

High numbers of *G. salaris* can cause serious disease in juvenile salmonids and in some instances cause significant losses of Atlantic salmon parr. Brown trout in the same locations did not show significant losses (Johnsen and Jensen 1991). There is preliminary evidence that *G. salaris* dies more rapidly when in contact with brown trout than in the absence of a fish host (from information supplied by TA Mo to A McVicar). Adult Atlantic salmon may be infested with *G. salaris* in the absence of clinical disease.

The prevalence of *G. salaris* varies widely and may depend on factors such as water temperature and host species. Prevalence ranged from 71% to 88% in smolts in a Norwegian river system. In adult salmon returned to spawn the prevalence was 0–100% (Soleng et al 1998). In Finland, the recorded prevalence of *G. salaris* on farmed fingerlings, yearlings and smolt was <1%–17.7% (Rintamaki-Kinnunen and Tellervo Valtonen 1996, Rintamaki and Valtonen 1994), and in northern Finland, *G. salaris* was reported in 39% of salmonid farms (Koski and Malmberg 1995). Most of these Finnish figures refer to Baltic race Atlantic salmon and to rainbow trout. In Northern Ireland, *G. salaris* was not reported in a survey

of 831 fish on 17 farms and 163 fish from 7 rivers (Platten et al 1994).

G. salaris may be present at a very low number on fish without any signs of clinical disease. This occurs occasionally on Atlantic salmon, but is more common with other salmonid species, especially rainbow trout. Infestation with a single parasite is common (OIE Aquatic Manual).

Detection and organs affected

G. salaris is quite large and can usually be seen with the naked eye. However, the OIE recommends the use of a binocular dissecting microscope to detect the parasite. Identification of *Gyrodactylus* spp is based on morphology and morphometry of marginal hooks, anchors (hamuli) and bars in the opisthaptor (the attachment organ) (OIE Aquatic Manual).

G. salaris most commonly attaches to the fins, especially the dorsal and pectoral fins, but it may occur anywhere on the skin of the host, including the gills and the oral cavity (OIE Aquatic Manual).

There is little published information on the survival of *G. salaris* on fish removed from the aquatic environment; however, it is considered that *G. salaris* would not survive on fish passed for human consumption because of the handling conditions and delay from slaughter. This is the basis for the absence of controls for *G. salaris* on non-viable susceptible fish from areas not shown to be free of *G. salaris* to United Kingdom markets. Moreover, *G. salaris* would not survive freezing or cooking (A McVicar pers. comm.). Gyrodactylus dies rapidly if not covered with water and the parasite often leaves the host soon after its death (OIE Aquatic Manual).

Key findings

G. salaris only occurs in the countries of continental Europe. It has been reported on many salmonid species but pathological effects are usually only significant in Atlantic salmon. Self-sustaining populations of *G. salaris* are restricted to fish in fresh water and brackish water; however, the parasite has been shown to survive but not reproduce in water at higher salinity. The prevalence of infestation in salmonids varies widely, but is generally highest in parr and smolts.

Juvenile salmonid fish (which are the lifecycle stage most likely to have clinical disease) are not usually harvested for human consumption. Fish with clinical infection would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. Adult fish are less likely than juvenile fish to have a significant level of infestation or clinical disease due to *G. salaris*. Such fish would appear normal and would not be detected at inspection. The detection of scars resulting from infestation with *G. salaris* may result in the direction of fish to further processing but would not be expected to result in their being rejected for human consumption.

Evisceration would have no effect on the number of *G. salaris* if present on salmonids for human consumption, however, some parasites may be removed in the course of processing and inspection.

Viable *G. salaris* would not be expected to survive on salmonids harvested and processed for human consumption, particularly in frozen or cooked product (A McVicar pers. comm.). There would be a negligible probability of salmonid product imported to Australia from continental Europe containing viable *G. salaris*.

Unrestricted risk estimate for importation of salmonids

Taking into account the release assessment documented above, the probability of infective *G. salaris* entering Australia as a consequence of the unrestricted importation of eviscerated salmonids would be negligible. Therefore, the probability of establishment of disease would also be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of *G. salaris* in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted. A summary of the risk assessment is shown in Box 4.14.

Box 4.14
Risk assessment — *Gyrodactylus salaris*

RELEASE ASSESSMENT (R)

The probability of *G. salaris* entering Australia as a consequence of the unrestricted importation of eviscerated salmonids, including juvenile fish, would be negligible (N).

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because there is negligible probability of establishment
- importation risk for *G. salaris* = acceptable ('yes' in Figure 1.1).

That is:

- regardless of the consequences of establishment of *G. salaris* in Australia, the risk associated with the unrestricted importation of eviscerated salmonids meets Australia's ALOP; and
- risk management measures are not warranted.

4.2.15 *LEPEOPHTHEIRUS SALMONIS* (SEA LICE DISEASE)

Release assessment

This parasite was not considered in the previous AQIS reports (DPIE 1995, 1996), or in the 1997 report of the New Zealand Government (Stone et al 1997b).

Geographic distribution

Lepeophtheirus salmonis has a circumpolar distribution in the northern hemisphere. Sea lice infestations cause significant problems mainly in Norway, Canada and the United Kingdom (MacKinnon 1997). They also cause problems in Ireland and the Faroe Islands (A McVicar pers. comm.).

Host range and prevalence

L. salmonis is a parasite of salmonids, primarily infesting fish in the genera *Salmo*, *Salvelinus* and *Oncorhynchus*. Other hosts may occasionally harbour specimens but do not provide adequate conditions for development and reproduction (MacKinnon 1997).

L. salmonis occurs commonly on wild salmonids but is generally present in low numbers and responsible for only minor damage to the host tissues, such as dermal abrasion, dark colouration and haemorrhage in the perianal region (Johnson et al 1996). Prevalence depends on many factors, including the host age and species, water temperature and salinity (Cusack 1995). Prevalence varies from 0–100% in salmonid populations in which *L. salmonis* is endemic (Johnson et al 1996, Birkeland 1996, Birkeland and Grimmes 1993, Birkeland et al 1997). Epidemic infestations of *L. salmonis* are most common in farmed salmonids (Bjordal 1994). However, the parasite can also occur in significant numbers on wild-caught salmonids (Jakobsen et al 1992). In wild-caught Atlantic salmon in Norway, the number of adults and preadult *L. salmonis* was low, while the number of chalimus larvae was higher (Jakobsen et al 1992).

Detection and organs affected

L. salmonis is up to 1.8 cm long (not including the eggstrings that may be up to 2 cm long) and adult parasites can be seen with the naked eye. The infective

copepodid stage is smaller and may not be easily seen with the naked eye.

Adult male and female *L. salmonis* can move freely over the surface of salmon although they tend to aggregate on the back of the head, around the anus and behind the dorsal fin. Eggs from mature females are shed into the water and hatch as planktonic nauplii, which develop into infective copepods. Once this stage finds a suitable host it will attach and remain attached until it develops into an adult. Adult copepods can move about or leave the host and may occur as free swimming copepods. Salmonids seem necessary for attachment and development of copepodid stages (B Jones pers. comm.).

The rate of successful development from egg to adult parasite is normally very low (A McVicar pers. comm.). Egg production is usually very high, with short generation times dependant on temperature (B Jones pers. comm.).

These parasites are susceptible to desiccation when out of water and would not be expected to survive more than one or two days on fish for human consumption. *L. salmonis* would not survive freezing or heat treatment and would be unlikely to survive chilling for an extended period (A McVicar pers. comm.)

Key findings

There is a high likelihood that marine salmonids from the northern hemisphere would be infected with *L. salmonis.* Infection may occur in fish of all ages. Clinical disease may develop in fish that are not treated.

Given that *L. salmonis* is visible to the naked eye and infestation causes visible abnormality, including, epidermal damage and haemorrhage, it is expected that infested fish would be detected in the course of inspection for human consumption. Fish with visible lesions may be directed to further processing.

The entry of lifecycle stages other than mature, gravid females of *L. salmonis* to Australia would have minimal quarantine significance. In the case of fish infested with the early stage (ie the copepodid and chalimus) of *L. salmonis*, the parasite could not attach to another host. The entry of male parasites or immature females would be unlikely to lead to the establishment of *L. salmonis* in Australia.

It is extremely unlikely that *L. salmonis* would survive beyond approximately 48 hours on fish harvested and processed for human consumption.

Unrestricted risk estimate for importation of salmonids

Taking into account the release assessment documented above, the probability of infective *L. salmonis* entering Australia as a consequence of the unrestricted importation of eviscerated salmonids would be negligible. Therefore, the probability of establishment of disease would also be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of *L. salmonis* in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted. A summary of the risk assessment is shown in Box 4.15.

Box 4.15
Risk assessment — *Lepeophtheirus salmonis* (sea lice disease)

RELEASE ASSESSMENT (R)

The probability of *L. salmonis* entering Australia as a consequence of the unrestricted importation of eviscerated salmonids would be negligible (N).

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF EVISCERATED SALMONIDS

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because there is a negligible probability of establishment
- importation risk for *L. salmonis* = acceptable ('yes' in Figure 1.1).

That is:

- regardless of the consequences of establishment of *L. salmonis* in Australia, the risk associated with the unrestricted importation of eviscerated salmonids meets Australia's ALOP; and
- risk management measures are not warranted.

4.3 Summary of import risk assessment for salmonids

A summary of the import risk assessment for salmonids is shown in Table 4.2.

Table 4.2

Summary of import risk assessment for salmonids

DISEASE AGENT	ESTABLISHMENT[a]	CONSEQUENCES[b]	RISK MANAGEMENT[c]
Infectious haematopoietic necrosis virus	VL	M–H	Yes
Infectious pancreatic necrosis virus	Juveniles—L All others—EL	M–H M–H	Yes No
Infectious salmon anaemia virus	Atlantic salmon—L All others—N	H H	Yes No
Oncorhynchus masou virus	Oncorhynchus spp from Japan—VL All others—N	M M	No No
Salmon pancreas disease virus	Atlantic salmon, brown trout and rainbow trout—VL All others—N	M M	No No
Viral haemorrhagic septicaemia virus	VL	M—Freshwater European strains L—All others	No
Aeromonas salmonicida (typical)	Wild ocean-caught Pacific salmon—EL All others—L	M–H	No Yes
Aeromonas salmonicida (atypical)	Wild ocean-caught Pacific salmon—EL All others—L	M	No Yes
Piscirickettsia salmonis	Farmed marine salmonids and all juveniles—VL Farmed freshwater adult salmonids—EL All others—N	M—Chilean strain L—All others M—Chilean strain L—All others M—Chilean strain L—All others	No No No
Renibacterium salmoninarum	VL	H	Yes
Yersinia ruckeri (Hagerman strain)	Juveniles—L All others—VL	L–M L–M	Yes No
Microsporidium takedai	Freshwater salmonids from Japan—VL All others—N	L L	No No
Myxobolus cerebralis	All rainbow trout and all juveniles—L All others—VL	L–M L–M	Yes No
Proliferative kidney disease agent	VL	L–M	No
Gyrodactylus salaris	N		No
Lepeophtheirus salmonis	N		No

a Level of probability: H=high, M=moderate, L=low, VL=very low, EL=extremely low, N=negligible.
b Level of significance: C=catastrophic, H=high, M=moderate, L=low, N=negligible.
c Risk categorisation (see Figure 1.1).
Yes = risk management is required.
No = the risk is acceptable and importation can be permitted without further risk management.

Chapter 5
Risk management: salmonids

5.1 General principles

THIS CHAPTER CONSIDERS THE RISK management measures that will be required to address the quarantine risks associated with disease agents of salmonids. The risk assessment for the unrestricted importation of eviscerated salmonids (see Chapter 4), showed that the risk associated with the establishment of some disease agents would not meet Australia's appropriate level of protection (ALOP). The next step was to consider how risk management measures could be implemented to reduce the unrestricted risk to a level that would meet the ALOP.

If the risk from the proposed importation of a commodity is determined to be greater than Australia's ALOP, that is, the risk associated with the unrestricted importation is unacceptable, implementation of risk management measures must be considered, consistent with Section 70 of Quarantine Proclamation (QP) 1998:

> In deciding whether to grant a permit to import a thing into Australia, a Director of Quarantine… must consider whether, if the permit were granted, the imposition of conditions on it would be necessary, to limit the quarantine risk to a level that would be acceptably low.

Such consideration of measures is consistent with Australia's international obligations under the World Trade Organization (WTO) Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement).

The risk management measures chosen must be the least trade restrictive necessary to meet Australia's ALOP. In developing measures, Australia must consider matters such as practicability and ease of implementation, cost of compliance, cost-effectiveness of the measures and impact on trade, subject to the over-riding requirement that measures reliably achieve the ALOP. Additionally, under Article 4 of the SPS Agreement, if an exporting country can objectively demonstrate that measures other than those initially proposed by Australia would deliver the level of protection we require, the alternative measures should be acceptable.

Quarantine measures must be specified and applied in a way that does not discriminate between the commodities of different exporting countries, taking into account differences in assessed risk associated with commodities from each source. Similarly, measures applied to limit risk from imported commodities must not be more restrictive than measures applied to address similar risks from domestic commodities. Furthermore, quarantine measures imposed by Australia must not make arbitrary or unjustified distinctions in the acceptable level of quarantine risk from imported commodities (considering both the likelihood and consequences of establishment) if such distinctions restrict trade; that is, quarantine risk must be managed consistently.

Consistent with the SPS Agreement, Australia's policy is to adopt international standards if their use will meet our ALOP. As noted in Chapter 1, relevant international standards, including for several diseases considered in this risk analysis, have been determined by the Office International des Epizooties (OIE, or World Organistion for Animal Health). For importing countries that are free of specified diseases and are sourcing fish from countries or regions that are not free, the OIE recommends, as a minimum risk-management measure, the evisceration of fish imported for human consumption.

Section 5.2 describes the general measures available for managing quarantine risks. Section 5.3 describes the risk management measures proposed for the diseases identified in Section 4.1.2 as requiring assessment with high priority (group 1). Section 5.4 shows the overall measures required for import of eviscerated salmonids to manage the risks associated with the group 1 diseases.

Finally, the diseases identified in Section 4.1.2 as requiring assessment with lower priority (group 2) are described in Section 5.5. This is to assess if the risk management measures identified in Section 5.4 would also meet Australia's ALOP against group 2 pathogens if eviscerated salmonids are imported.

5.2 Available quarantine measures

Quarantine measures aim to reduce the likelihood that the importation of products would lead to exotic disease agents being introduced into and becoming established in Australia. There are two principal methods of achieving this outcome:

- reducing the likelihood of disease agents entering Australia in imported product by imposing conditions relevant to the source population from which the product is derived, and/or by treating the product to reduce the number of disease agents (if any) present; and
- reducing the likelihood that susceptible host species in Australia would be exposed to imported product or derived waste likely to transmit disease.

Measures can be applied in the country of origin before export and/or in Australia after import to modify the level of risk. Factors relevant to the identification of appropriate risk management measures are discussed in Sections 1.6 and 1.7.

5.2.1 PRE-EXPORT REQUIREMENTS FOR COUNTRY OF ORIGIN

Pre-export requirements aim to reduce the likelihood that fish containing pathogens are exported to Australia and/or to reduce the titre of disease agents likely to occur in such fish. General factors affecting the prevalence of disease agents in imported product are discussed in Section 1.6. There are various measures that would reduce the likelihood of disease agents entering Australia in imported salmonids. These include inspection and grading and processing practices, such as washing, evisceration, removal of the head and gills, removal of the tail, fins and skin, filleting and processing of product to a consumer-ready state. In this risk analysis, products such as fillets (without skin) of any size, skin-on fillets or cutlets if less than 450g and headless fish of 'pan-size' (ie less than 450g) are considered to be consumer-ready.

Exporting countries may provide statements in official certification to confirm the application of these procedures and any other conditions that the importing country may impose on the importation of the commodity.

Export certification

Official certification may be used to provide assurances for those measures whose implementation cannot be readily confirmed on the basis of post-arrival examination. Certification may also be used as an alternative to more costly or trade restrictive methods, such as inspection and testing of product on arrival.

Certifying authorities must have systems in place to support the issuance of accurate, valid certification. The key elements of such systems include:

- legislation providing for the notification and control of animal diseases;
- official programs for disease surveillance and monitoring;
- animal health services supported by competent diagnostic laboratories;
- systems for the inspection of animals and product, including for approval and control of premises processing product for human consumption; and
- legislation concerning the issuance of certification, with appropriate sanctions to discourage the issuance of false statements.

Government certification is the basis of international trade in many commodities. Countries involved in international trade normally accept that government certificates are accurate and are supported by systems to ensure their accuracy. Importing countries have the right to take appropriate steps to verify that certificates and certification systems are reliable.

Before approving the importation of products for which certification is an important part of the risk management arrangements, an appropriate evaluation of the certifying authority should be made. The Australian Quarantine and Inspection Service (AQIS) would normally approve countries with a history of exporting to Australia animals/products certified as meeting Australia's quarantine requirements. This includes countries that regularly export to Australia commercial consignments of goods that are subject to quarantine control, such as live animals, genetic material and animal products.

Canada, the United States, New Zealand and some countries of the European Union have an established history of exporting a large range of animals (including fish) and animal products to Australia. Appendix 2 provides an overview of the inspection and certification systems of some of these countries.

AQIS may conduct a specific evaluation of the competent authority(ies) of countries that do not have an established history of exporting to Australia animals/products certified as meeting Australia's quarantine requirements. Animal Quarantine Policy Memorandum 1999/41[1] provides draft guidelines for the approval of countries to export animals (including fish) and their products to Australia.

An exporting country may provide certification on matters relevant to quarantine risk, such as:

- the nature and source of exported fish/product;
- the health status of populations from which the fish/product was derived;
- results of health surveillance and monitoring;
- the processing of the product; and
- the system of inspection and grading to which the product was subjected.

The nature and source of an imported product will affect the prevalence of disease agents, if present, in the product. An importing country may require official certification as to the source of a product, including the species, geographical location where it was caught or harvested and production system, for example, whether the fish was farmed or wild caught.

1 Australian Quarantine and Inspection Service, Animal Quarantine Policy Memorandum 1999/41. Guidelines for the approval of countries to export animals (including fish) and their products to Australia.

Health status of the population from which the fish were derived

Certification can be used to provide assurances that countries or regions are free of specified pathogens or diseases. For diseases listed by the OIE, countries provide regular annual and, as required, emergency reports of their disease status. The exporting country is normally in the best position to have current and accurate information on fish health, based on the scientific and technical resources of government, industry, research organisations and academia.

Surveillance and monitoring

Surveillance and monitoring underpins the provision of health certification. A documented fish health surveillance and monitoring program will provide up-to-date information on the health of the population from which imported fish were derived. Such information can be used by an importing country to confirm that it has identified and addressed all diseases in the exporting country that it considers to present a significant hazard. These programs provide information needed for confidence about disease status and related matters, including rapid detection of disease and early recognition of the emergence of new pathogens. Surveillance and monitoring programs must be designed and implemented as appropriate to the target population and pathogens of interest.

The emergence of a new pathogen or disease syndrome may necessitate the adoption of additional or alternative risk management measures to maintain consistency with Australia's ALOP. An effective surveillance and monitoring program provides the necessary underpinning for countries to detect the emergence of significant new pathogens. Conditions for the importation of animals and their products would normally include requirements for exporting countries to report relevant changes in animal health status (as relevant to the exported fish or product) to AQIS in a timely manner. Moreover, AQIS reserves the right to modify, suspend or revoke import conditions. This would apply if there were changes in the health status of an exporting country, and such changes are judged to substantially affect the quarantine risks presented by imported animals or products.

Regionalisation

AQIS would normally accept information provided by an authority that AQIS recognises as being a competent authority on the presence or absence of pathogens in susceptible populations of fish. However, to assist evaluation of claims for the absence of specified pathogen(s) in a country or part of a country, AQIS may require the competent authority to present a scientific submission supporting its claims. The submission should include information obtained from ongoing surveillance and monitoring for the pathogen in question and details of controls to exclude the disease agent from the country or free region. AQIS would formally evaluate the submissions of exporting countries having regard to the epidemiology of the disease agents and effectiveness of surveillance, monitoring and control measures.

Disease control measures

Many of the diseases considered in this import risk assessment (IRA) are significant pathogens. Some are OIE listed and/or are listed in legislation of national governments. Others are the subject of official controls, including compulsory slaughter of disease populations. The slaughter of farmed fish populations under official direction to control an outbreak of disease could present a particular risk factor, as a significant proportion of apparently healthy fish may have a high titre of pathogen in their body tissues. Under these circumstances, the risk of disease becoming established in Australia may be higher than the estimate provided in Chapter 4.

If the increase in risk is such that the new risk would not be expected to meet Australia's ALOP, AQIS could exclude fish slaughtered under official direction to control an outbreak of disease or impose additional controls over such fish. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia were not derived from a population slaughtered as an official disease control measure.

Commercial operators may also decide to slaughter farmed fish affected by disease or other conditions that decrease efficiency of production. Taking into account the risk management measures discussed in this

section, operator-initiated slaughter from time to time of farmed fish affected by disease would not significantly increase the risk associated with the importation of eviscerated salmonids overall.

Equivalence and national treatment

In considering the effectiveness of an exporting country's surveillance and monitoring program, an importing country should have regard to the principles, in the SPS Agreement, of equivalence and national treatment. In considering minimum requirements for disease surveillance by exporting countries, there are limits to what Australia can demand of exporting countries. Thus, it cannot ask them to conduct significantly more intensive national surveillance to demonstrate the absence of specified diseases than that deemed sufficient to support Australia's claims to freedom from the same diseases (all other technical issues being equal).

Age of fish

In Chapter 4 it is concluded that for several of the disease agents, the probability of the pathogen being present would be higher for juvenile fish or sexually mature fish than for commercially harvested, market-size salmonids. These agents include: infectious haematopoietic necrosis virus (IHNV), *Renibacterium salmoninarum* and viral haemorrhagic septicaemia virus (VHSV) for juvenile fish or sexually mature fish; and infectious pancreatic necrosis virus (IPNV), *Myxobolus cerebralis* and *Yersinia ruckeri* for juvenile fish only.

For the purposes of this IRA, juvenile salmonid fish are fish that, in headless, eviscerated presentation, weigh less than 200 g. Sexually mature salmonid fish are fish in milt or in spawn; that is, with developed gonads. These lifecycle stages are not traded under normal commercial conditions, due to size and quality considerations.

Some pathogens are clinically expressed in and/or would be present at a higher prevalence or titre in these lifecycle stages than in commercially harvested, market-size salmonids. If the increased risk is such that it would not meet Australia's ALOP, AQIS could exclude fish in these lifecycle stages or impose additional controls. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia are not juvenile salmonids or spawners.

Processing of the product

Procedures conducted in the course of normal processing for human consumption may also have the effect of reducing the level of risk. Public health authorities require that premises processing fish for human consumption operate in a sanitary manner to ensure that the product is free from contamination and fit for human consumption. While this risk analysis is not primarily concerned with pathogens of public health significance, inspection controls for public health purposes may simultaneously serve quarantine objectives. For example, appropriate sanitary controls would reduce the likelihood of biofilms and cross contamination in processing plants, thus reducing the probability of pathogens contaminating salmonids exported to Australia. Having regard to the principles in the SPS Agreement, AQIS could require plants to operate in accordance with sanitary standards that would address identified quarantine risks.

Plants exporting salmonids to Australia could be approved by a competent authority of the exporting country and subject to inspection and control by that competent authority to ensure the maintenance of appropriate standards. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia were processed in a plant approved and controlled by the competent authority and subjected to regular inspection to confirm the exported product meets Australia's import requirements.

AQIS would conduct reviews of national systems including audits of plants to confirm that acceptable sanitary standards were being maintained.

System of inspection and grading

Fish for human consumption are inspected in many countries under a program approved and supervised by a competent authority. Such an authority can provide certification attesting to the fitness of the product and that it meets specified conditions, including those of an importing country. Such inspection systems aim to ensure that product is safe for human consumption and meets other specified requirements (including those of

importing countries). Such systems do not have the primary objective of detecting the presence of fish disease agents.

Commercial inspection and grading programs are commonly used to ensure that the product is wholesome and meets commercial specifications, which normally include correct processing and presentation, size/weight of carcase, absence of blemishes (including lesions associated with infectious disease) and freedom from signs of sexual maturity.

Fish with visible lesions would normally be downgraded and/or diverted for further processing. Fish with generalised lesions or evidence of septicaemia would normally be rejected from human consumption. Apparently healthy fish (which may include fish with chronic infection, inapparent lesions and fish incubating disease) would normally pass inspection. While inspection would not detect all infected fish, it would detect most visibly abnormal fish, which are often associated with higher titres of disease agents. Thus, inspection and grading of fish for human consumption could contribute to the reduction of quarantine risk.

The Australian retailer or consumer would be likely to discard visibly abnormal fish because it would be unacceptable for consumption. If discarded into the domestic sewerage or solid waste disposal system, such fish would present a negligible likelihood of disease establishment. However, disposal of such fish into water containing significant populations of susceptible fish could present a higher likelihood of disease establishment. While this possibility cannot be discounted, in most cases product discarded by retailers and consumers would be more likely to enter the domestic sewerage or solid waste disposal system than to be discarded directly into the aquatic environment.

In Chapter 4, AQIS concluded that for many of the disease agents, inspection and grading for human consumption would increase the likelihood that diseased fish would be detected and would reduce the likelihood of disease agents entering Australia with imported salmonids.

Inspection and grading would detect fish that were visibly affected by disease from pathogens including IHNV, IPNV, infectious salmon anaemia virus (ISAV), oncorhychus masou virus (OMV), salmon pancreas disease virus (SPDV), VHSV, *Aeromonas salmonicida*, *Piscirickettsia salmonis*, *Renibacterium salmoninarum*, *Yersinia ruckeri*, *Hexamita salmonis*, *Microsporidium takedai*, *Myxobolus cerebralis*, proliferative kidney disease agent (PKX), *Gyrodactylus salaris* and *Lepeophtherius salmonis*. The efficiency of detection would vary from plant to plant. Fish with generalised infection and moribund fish would be reliably detected, while some fish with low-grade pathological lesions (which could contain a significant titre of pathogens) may pass inspection. Inspection and grading would reliably detect juvenile salmonids, sexually mature fish and fish that were not processed to meet Australian entry requirements. Thus, inspection and grading would contribute to a reduction in disease risk overall.

Inspection and grading is a routine part of the processing of salmonids for human consumption under normal commercial conditions. Accordingly, AQIS could introduce a requirement for inspection and grading of salmonids exported to Australia for human consumption and this would not present a significant impediment to trade. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia had been inspected and graded and that they meet relevant conditions of importation.

Deheading

The fish's head is usually not eaten and, in the case of fish larger than 'pan-size', is normally removed before the product is cooked. The head represents about 10% of the body mass of the fish. Certain pathogens may localise in tissues of the head, such as the gills, brain or retrobulbar blood sinuses. Thus, deheading would be an appropriate risk-reduction requirement for certain disease agents. Importation of head-on salmonids into Australia may present a significant exposure pathway, as fish heads are used by some industries as fishing bait.

AQIS could require the removal of the head and gills from salmonids before importation into Australia. An appropriate measure would be for the competent authority of the exporting country to certify that only fish that have had their head and gills removed were exported to Australia.

5.2.2 POST-IMPORT MEASURES IN AUSTRALIA

This strategy aims to reduce the probability of imported product or derived waste entering the aquatic environment and susceptible hosts being exposed to a dose of pathogen sufficient to cause infection.

As discussed above, when product imported for human consumption is consumed by humans and waste product (cooked or uncooked) is discarded into the domestic sewerage or solid waste disposal systems, there is a negligible probability of disease establishment. If the product or waste is handled in a manner that increases the likelihood of it entering the aquatic environment in untreated form (eg the use of trimmings as bait or berley) or when untreated waste products bypass the domestic waste disposal or sewerage systems, there may be a high probability of pathogens entering the aquatic environment. If there are significant populations of susceptible hosts in waters containing pathogens at high concentration, disease could become established in these fish.

Measures that may be applied to reduce risk potentially associated with imported fish include:

- restricting the type/presentation of product, to increase the probability of it being used in a low-risk manner;
- restricting the type/presentation of product to reduce the amount of waste generated after arrival in Australia;
- processing the product to reduce the likelihood of it containing aquatic pathogens in an infective form; and
- restricting the distribution or end use of imported product.

These measures may be applied singly or in combination (when they would be expected to have a cumulative effect on the reduction of quarantine risk).

Restrictions on product type

The attractiveness of a product for use other than for human consumption may be reduced by controls on the type or presentation of product. Most fish are prepared for human consumption by processes such as evisceration, deheading, filleting and skinning. Some, but not all, fish are cooked. It is increasingly the case that consumers purchase product that is ready to cook/eat without further preparation or trimming. Consumer-ready products, individually packaged to protect their quality and improve presentation, have a higher unit value. Accordingly, such products are much less likely to be used for fish feed or bait than a whole, round or eviscerated fish. For pathogens that are present at higher titre in tissues such as the head or skin, consumer-ready product would present an extremely low quarantine risk because there would be minimal waste potentially containing pathogens.

There may be instances where imported consumer-ready product gets contaminated or spoiled and is no longer fit for human consumption. The possibility of such product being discarded in a 'higher risk' manner (eg used as fish bait) cannot be discounted. However, importers would be more likely to dispose of spoiled fish via the domestic, solid-waste disposal systems. While there is a potential risk associated with inappropriate disposal of contaminated or spoiled imported fish, this would not significantly increase the risk associated with the importation of eviscerated fish in total; thus the imposition of additional specific measures would not be warranted.

Although AQIS's ability to control the further processing of imported salmonid products in Australia is limited (see section on waste treatment below), it is not appropriate to simply ban importation on these grounds. Rather, AQIS must consider the application of alternative risk management measures. For example, it may be appropriate to require that product is consumer-ready before its release from quarantine. In this risk analysis, products such as fillets (without skin) of any size, skin-on fillets or cutlets of less than 450g weight and headless fish of 'pan-size' (ie less than 450g weight) are considered to be consumer-ready.

Restrictions on end use

For many disease agents, the Australian fish species known to be susceptible to infection have a limited distribution. Accordingly, domestic controls could be imposed on the use and distribution of product as a means of reducing risk. For example, economically

significant populations of salmonids mainly occur in the waters of Tasmania and parts of Victoria and NSW. Controls that reduce the supply of certain types of imported product to these areas would reduce the likelihood of salmonids being exposed to pathogens, if present, in imported product.

To be effective, such measures would be based on internal quarantine of fish products in relation to specified water catchments. This would require the introduction of new controls over products that are currently free of movement restrictions. Under the Quarantine Act AQIS can restrict the use and distribution of goods that are subject to quarantine but has limited authority over the movement of goods once they are released from quarantine. Accordingly, AQIS could restrict the location of quarantine approved fish processing premises, but not the wholesale or retail distribution of product released from quarantine. Regional controls over distribution or labelling of this nature may be most appropriately based on State or Territory government legislation. To meet Australia's SPS obligations, such controls must be consistent with current interstate quarantine regimes and have regard to the memorandum of understanding (MOU) between the State/Territory governments and the Commonwealth Government on SPS issues.

Waste treatment

Exposure pathways

The disposal of waste may provide a pathway for the establishment of pathogens in Australia as discussed in Section 1.7. The disposal of domestic and HRI (hotel, restaurant and institution) waste via domestic sewerage or solid waste disposal systems would generally present a negligible likelihood of pathogens becoming established.

Several of the pathogens considered in the IRA could survive in imported salmonid product. Fish waste is attractive to scavengers and product containing pathogens may be transported by birds or mammals and may eventually enter the aquatic environment. Commercial and trade premises are generally required to keep putrescible wastes covered and dispose of them quickly via the domestic waste management system, to preserve environmental quality and protect public health. Wastes from fish processing plants are putrescible and their disposal may be difficult or expensive. Commercial operators may be inclined to dispose of large volumes of waste in a manner that increases the probability of the waste entering the aquatic environment.

The commercial processing of imported salmonids in Australia could generate a significant volume of solid or liquid waste at the premises' point of discharge. For historical reasons, many fish processing plants are located near or on waterways. Large-scale discharge (deliberate or accidental) into the aquatic environment of untreated waste from imported salmonids would increase the risk of establishment of pathogens, if present in imported product. Continuous long-term release of untreated waste at the premises' point of discharge could result in infective material building up to a biologically significant level in the aquatic environment. Accordingly, it may be appropriate to introduce controls over the disposal of waste from the commercial processing of imported salmonids.

Some types of waste, particularly fish heads, may be attractive for use as fish feed or bait. Based on current industry practices, salmonid heads would be more likely to be used for fish feed or bait than other wastes produced in the course of commercial processing.

Commercial processing

In this IRA, 'commercial' processing is defined as the activities undertaken at a commercial premises that produce product for consumption at another premises or location, and incidentally generate 'waste' — that is, product that will not be used for human consumption. This definition does not include premises where the product is consumed on site or premises that only supply the consumer or end-user.

To ensure that the quarantine risks associated with the commercial processing of imported products in Australia are the subject of appropriate risk management measures, AQIS could require that imported salmonids were under quarantine control until further processed. AQIS would only release consumer-ready product, which would be unattractive for further commercial processing (which may generate a significant volume of waste).

AQIS would not place restrictions on the movement or end-use of consumer-ready product — whether processed overseas or in Australia.

In most cases, commercial processing plants in Australia would import salmonids with the intention of processing them. Applicants for a permit to import salmonid products would be required to advise AQIS of the presentation/form of the product. In the case of product that is not in 'consumer ready' form (ie suitable for further commercial processing), AQIS could order the goods into quarantine at a premises approved under Section 46A, or subject to a compliance agreement under Section 66B, of the Quarantine Act. In considering whether to approve a plant for the purpose of processing imported salmonid products, AQIS would take into account the following factors:

- the location and physical security of the premises;
- the nature of imported product, the intended processing and the volume and type of waste that would be produced;
- the control of scavengers and pests in and around the plant;
- the competency of the management to meet quarantine requirements;
- the availability of systems for maintenance of appropriate records of the processing of imported product and waste disposal;
- the availability of competent personnel to supervise quarantine-approved processes (such personnel would be expected to have a thorough knowledge of quarantine requirements);
- methods for the disposal of waste material, including arrangements for transport, storage, treatment and disposal, and the effectiveness of procedures in preventing the entry of imported product and derived waste into the aquatic environment; and
- the proximity of the plant to economically significant populations of salmonids.

Criteria for approval of premises to process imported salmonids

AQIS would address applications for approval of premises on a case-by-case basis. Key considerations in deciding whether to approve an application would be as follows.

AQIS would consider the location of commercial processing plants proposed for approval relative to economically significant populations of salmonids such as occur in Tasmania, Victoria (around Lake Eildon-Delatite Shire and Murrindindi Shire) and in the alpine areas of southern NSW. Commercial processing would not be permitted in regions where there are economically significant populations of salmonid fish. This would reduce the probability that susceptible fish would be exposed to imported fish or derived waste.

In considering methods for the treatment of liquid waste, AQIS would accept discharge into a municipal sewerage system providing that processing and dilution was judged to be sufficient to reduce risk to an acceptable level. AQIS would also accept treatment on site (eg by heating, disinfection or an equivalent process) that was judged to be sufficient to reduce risk to an acceptable level. AQIS would require that solid waste was covered and access of scavengers prevented until final disposal by an AQIS-approved method, such as deep burial at an approved facility or heating.

AQIS would also require that premises approved for the further processing of imported salmonids were located to allow quarantine inspectors and auditors ready access and to facilitate regular announced and unannounced inspection. It is likely that most, if not all, approved processing plants would be located in metropolitan centres of mainland Australia.

5.2.3 CONCLUSIONS

Measures may be used singly or in combination to manage risk. Exporting countries seeking to modify any of these measures could provide a submission, supported by relevant scientific information, for consideration by AQIS. Australia may accept equivalent approaches to risk management generally or specifically, on the basis of a case-by-case assessment.

Other risk management measures, such as freezing, chilling, special packaging, heating or chemical treatment of product, could contribute to a reduced probability of a pathogen becoming established in Australia. Exporting countries seeking to adopt alternative measures should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supportive scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

5.3 Risk management for specific disease agents (group 1 diseases)

This section considers the risk management measures that could be applied to address the quarantine risks associated with individual high priority (group 1) disease agents. On the basis of the risk assessment in Chapter 4, it was shown that for the unrestricted importation of eviscerated salmonids, the risk associated with the establishment of some of the group 1 disease agents do not meet Australia's ALOP. The disease agents for which the importation of eviscerated salmonids does not meet the ALOP were identified as:

- infectious haematopoietic necrosis virus;
- infectious pancreatic necrosis virus — for juveniles only;
- infectious salmon anaemia virus — for Atlantic salmon only;
- *Aeromonas salmonicida* (typical and atypical strains) — all salmonids except for wild ocean-caught Pacific salmon;
- *Renibacterium salmoninarum*;
- *Yersinia ruckeri* (Hagerman strain) — for juveniles only; and
- *Myxobolus cerebralis* — for rainbow trout and for juveniles of all salmonid species.

The next step was to consider for each disease how risk management measures could be implemented to reduce the unrestricted risk to a level that would meet the ALOP.

5.3.1 INFECTIOUS HAEMATOPOIETIC NECROSIS VIRUS (INFECTIOUS HAEMATOPOIETIC NECROSIS)

Risk assessment conclusions

In Chapter 4, AQIS concluded that for the unrestricted importation of eviscerated salmonids, including juveniles and sexually mature fish, the probability of the establishment of infectious haematopoietic necrosis virus (IHNV) would be very low. The consequences of establishment would be of moderate to high significance.

Thus, for IHNV, the risk associated with the unrestricted importation of eviscerated salmonids, including juveniles and sexually mature fish, does not meet Australia's ALOP and the implementation of risk management measures is warranted (see Box 4.1).

Key risk factors

1. IHN is a serious disease. If detected in the course of official surveillance and monitoring of the health of salmonid populations, IHNV may the subject of official controls, including compulsory slaughter of diseased populations.
2. The risk associated with juvenile fish and sexually mature fish would be higher than that associated with commercially harvested, market-size salmonids?
3. Clinically affected fish would have a high titre of IHNV in their body tissues.
4. IHNV may be present in covertly infected fish, including in the brain and viscera.
5. IHNV could survive in tissues and in the aquatic environment for a significant period.
6. IHNV could accumulate in the aquatic environment as a result of the uncontrolled disposal of waste from commercial processing of imported salmonids.

Risk management measures

The following risk management measures would reduce the risk associated with the establishment of IHNV via the importation of eviscerated salmonids into Australia.

Health status

- requirement that the fish are derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority; and
- requirement that the fish are not derived from a population slaughtered as an official disease control measure.

Age of fish

- requirement that the fish are not juvenile salmonids or sexually mature fish (spawners).

Inspection and grading

- to remove clinically diseased fish.

Processing

- removal of the head and gills;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- requirement that the fish are processed in a premises under the control of a competent authority.

Export certification

- requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Waste disposal

- control over the processing of imported salmonids in Australia; and
- control over the form and presentation of imported salmonid product released from quarantine to reduce the volume of waste generated in Australia.

Health status of the population from which the imported fish were derived

IHNV is listed by the OIE as a notifiable disease and there are official control programs for IHNV in many regions. Countries or regions that are free from IHNV would maintain surveillance for this pathogen. Moreover, the presence of IHNV would readily be detected in the course of surveillance for other OIE-listed viral diseases of salmonids, such as VHSV. However, countries in which IHNV is endemic and not the subject of control may not maintain active surveillance for this pathogen.

The slaughter of farmed fish under official direction, to control an outbreak of IHN, presents a particular risk factor, as a significant proportion of apparently healthy fish may be expected to have IHNV in their body tissues. Under these circumstances, the risk of IHNV becoming established in Australia would be higher than the estimate provided in Chapter 4. In these circumstances, the risk would not be expected to meet Australia's ALOP, necessitating the imposition of additional controls. AQIS could require that the competent authority of the exporting country provide certification confirming that the fish were not derived from a population slaughtered under official direction to control an outbreak of IHN. This would substantially address risk factor 1.

Age of fish

As discussed in Section 5.2.1, for IHNV the risk associated with the importation of juvenile salmonids and sexually mature fish (spawners) would be higher than that associated with commercially harvested, market-size salmonids. AQIS could require the exporting country to provide certification confirming that fish exported to Australia were not juvenile salmonids or spawners. This would address risk factor 2.

Inspection and grading

Inspection and grading would provide for the detection of fish with clinical disease due to IHNV, addressing the third risk factor identified above. Inspection and grading

would also provide for the identification of juvenile and sexually mature fish and fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factors 2 and 3.

Processing of the product

Inspection and grading would not detect covertly infected fish. IHNV could be present in the tissues of such fish, particularly in the brain and viscera. Commonly used commercial processes (removal of the head and thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with these factors.

Removal of the head and gills before importation into Australia would significantly reduce risk, as the head is not normally consumed and is (except for pan-size salmonids) usually removed before the fish is cooked. Disposal of the head by inappropriate means (such as by use as fishing bait) could present a high risk.

However, such processing would not totally eliminate risk; for example, washing would not remove all remnants of the anterior kidney on the skeleton.

AQIS could require the processing of imported salmonids to a specified standard, that is, removal of the head and gills and thorough cleaning and washing of internal surfaces. This would substantially address risk factor 4.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority. An appropriate measure would be for the competent authority of the exporting country to certify that the fish exported to Australia were inspected, graded and processed in accordance with Australia's conditions.

Waste disposal

The commercial processing of imported salmonids in Australia could generate a significant volume of solid or liquid waste at the premises' point of discharge. Continuous long-term release of untreated waste could result in the build-up of IHNV to a biologically significant level in the aquatic environment. For IHNV, waste tissues of concern would be the head and gills and the parts of the skeleton with attached remnants of the anterior kidney. As discussed in Section 5.2.2, AQIS could implement controls over commercial plants processing imported salmonid products with regard to location, waste disposal and related matters that would substantially address risk factors 5 and 6.

To ensure that imported salmonids were not commercially processed in non-approved premises, AQIS could permit release from quarantine of consumer-ready product only. Processing of such product for human consumption would normally generate minimal waste and, but this would not be expected to increase the overall risk of IHNV establishing.

Conclusions

To mitigate risks associated with the importation of eviscerated salmonids in relation to the establishment in Australia of IHNV, AQIS will permit the importation of eviscerated salmonids subject to the conditions shown in Box 5.1.

For IHNV, the implementation of these measures singly would reduce risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 5.1 would meet Australia's ALOP; importation of eviscerated salmonids will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supportive scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 5.1
Risk management measures for infectious haematopoietic necrosis virus

PRE-EXPORT REQUIREMENTS

- The fish must be derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority.
- The fish should not be derived from a population slaughtered as an official disease control measure.
- The fish must not be juvenile salmonids or sexually mature fish.
- The head and gills must be removed and internal surfaces thoroughly washed.
- The fish must be inspected and graded under the supervision of a competent authority.
- The product for export must be free from visible lesions associated with infectious disease and fit for human consumption.
- The fish must be processed in a premises approved by and under the control of a competent authority.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

POST-IMPORT MEASURES

- Only premises approved by AQIS will be permitted to commercially process imported salmonids in Australia.
- Only consumer-ready product will be released from quarantine.

5.3.2 INFECTIOUS PANCREATIC NECROSIS VIRUS (INFECTIOUS PANCREATIC NECROSIS)

Risk assessment conclusions

In Chapter 4, AQIS concluded that, for the unrestricted importation of eviscerated salmonids, the probability of the establishment of infectious pancreatic necrosis virus (IPNV) would be extremely low. For juvenile fish the probability would be low. The consequences of establishment would be of moderate to high significance.

Thus, for IPNV, the risk associated with the unrestricted importation of eviscerated adult salmonids meets Australia's ALOP and the implementation of risk management measures is not warranted.

For juvenile salmonids the risk does not meet Australia's ALOP and the implementation of risk management measures is warranted (see Box 4.2).

Key risk factors

- IPN is a serious disease which is normally clinically expressed in juvenile salmonids. IPN is reported in major salmonid-producing countries but not in Australia and New Zealand. In affected countries it is not normally the subject of official controls and it is unlikely that infected fish would be slaughtered under official direction.
- The risk associated with juvenile fish would be higher than that associated with commercially harvested, market-size salmonids because infection is usually clinically expressed in juvenile salmonids so there is a greater probability of a significant viral titre in these fish.
- IPNV may be present in covertly infected fish, particularly in the viscera.
- IPNV could survive in tissues and in the aquatic environment for a significant period.
- IPNV could accumulate in the aquatic environment as a result of the uncontrolled disposal of waste from commercial processing of imported salmonids.

Risk management measures

The following risk management measures would reduce the risk associated with the establishment of IPNV via the importation of eviscerated salmonids into Australia.

Age of fish

- requirement that the fish are not juvenile salmonids.
- *Inspection and grading*
- to remove clinically diseased fish.

Processing

- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- requirement that the fish are processed in a premises under the control of a competent authority.

Export certification

- requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Health status of the population from which the imported fish were derived

The implementation of specific risk management to address risk factor 1 is not warranted because the other risk management measures identified would substantially address risks associated with the entry and establishment of IPNV in Australia.

Age of fish

Disease would be unlikely to occur in adult fish, thus for IPNV the risk associated with the importation of juvenile salmonids would be higher than that associated with commercially harvested, market-size salmonids. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia were not juvenile salmonids. This would address risk factor 2.

Inspection and grading

Inspection and grading would provide for the identification of juvenile salmonids and fish that were not processed in accordance with Australia's import conditions. The risk assessment for IPNV concluded that fish clinically affected by IPNV would be visibly abnormal. Such fish would be detected and removed in the course of inspection for human consumption. This would substantially address risk factor 2.

Processing of the product

Inspection and grading would not detect covertly infected fish. IPNV could be present in the tissues of such fish, particularly in the viscera. Commonly used commercial processes (evisceration, followed by thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with these factors.

However, such processing would not totally eliminate risk; for example, washing would not remove all remnants of the anterior kidney on the skeleton.

AQIS could require the processing of imported salmonids to a specified standard, that is, evisceration and thorough cleaning and washing of internal surfaces. This would substantially address risk factor 3.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority. An appropriate measure would be for the competent authority of the exporting country to certify that the fish exported to Australia were inspected, graded and processed in accordance with Australia's conditions.

Waste disposal

The implementation of specific risk management to address risk factors 4 and 5 is not warranted because the other risk management measures identified would effectively prevent the entry of IPNV into the aquatic environment.

Conclusions

To mitigate risks associated with the importation of eviscerated salmonids in relation to the establishment in

Australia of IPNV, AQIS will permit the importation of eviscerated salmonids subject to the conditions shown in Box 5.2.

For IPNV, implementation of the measures singly would reduce risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 5.2 would meet Australia's ALOP; importation of eviscerated salmonids will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supportive scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 5.2
Risk management measures for infectious pancreatic necrosis virus

PRE-EXPORT REQUIREMENTS

- The fish must not be juvenile salmonids.
- The internal surfaces must be thoroughly cleaned and washed.
- The fish must be inspected and graded under the supervision of a competent authority.
- The product for export must be free from visible lesions associated with infectious disease and fit for human consumption.
- The fish must be processed in a premises approved by and under the control of a competent authority.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

5.3.3 INFECTIOUS SALMON ANAEMIA VIRUS (INFECTIOUS SALMON ANAEMIA)

Risk assessment conclusions

In Chapter 4, AQIS concluded that, for the unrestricted importation of eviscerated Atlantic salmon from areas infected with infectious salmon anaemia virus (ISAV) or affected by HKS, the probability of establishment of ISAV would be low. The consequences of establishment would be of high significance.

Thus, for ISAV, the risk associated with the unrestricted importation of eviscerated Atlantic salmon from ISAV-infected and HKS-affected areas does not meet Australia's ALOP and the implementation of risk management measures is warranted.

For salmonids other than Atlantic salmon (and for non-salmonid fish), from areas infected with ISAV or affected by HKS the probability would be negligible. For salmonids (and non-salmonid fish) from areas that have not reported the presence of ISAV or HKS the probability would be negligible. No risk management is warranted in these cases (see Box 4.3).

Key risk factors

(**Note:** these key risk factors only apply to Atlantic salmon from ISAV-infected and/or HKS-affected countries.) As of July 1999, ISA has been reported from Scotland, Norway and Canada.

1. ISA is a serious disease. If detected in the course of official surveillance and monitoring of the health of salmonid populations, ISAV may the subject of official controls, including compulsory slaughter of diseased populations.
2. Clinically affected Atlantic salmon would have a high titre of ISAV in their body tissues.
3. Preclinically infected Atlantic salmon may also have a significant titre of ISAV in their body. In such fish and in covertly infected salmonids if such infections occur, ISAV may be present in many tissues, including skin mucus, gills, blood and viscera.
4. ISAV could survive in waste tissues and the aquatic environment for a significant period.

5. ISAV could accumulate in the aquatic environment as a result of the uncontrolled disposal of waste from commercial processing of imported salmonids.

An official surveillance and monitoring program would be required to confirm the status of a country exporting Atlantic salmon to Australia with respect to ISAV/HKS. An appropriate measure would be for the competent authority of the exporting country to certify that the fish for export to Australia were derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority.

Risk management measures

The following risk management measures would reduce the risk associated with the establishment of ISAV via the importation of eviscerated salmonids into Australia.

Health status

- requirement that the fish are derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority; and
- requirement that Atlantic salmon do not come from a farm known or officially suspected of being affected by an outbreak of ISA.

Inspection and grading

- to remove clinically diseased fish.

Processing

- removal of the head and gills;
- thorough cleaning and washing of external surfaces to remove as much skin mucus as practicable;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- requirement that the fish were processed in a premises under the control of a competent authority.

Export certification

- requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Waste disposal

- control over the processing of imported salmonids in Australia; and
- control over the form and presentation of imported salmonid product released from quarantine to reduce the volume of waste generated in Australia.

Health status of the population from which the imported fish were derived

ISAV is listed by the OIE as an 'other significant disease' and there are official control programs for ISAV in many regions. Countries or regions that are free from ISAV would maintain surveillance for this pathogen in Atlantic salmon. Confidence regarding the presence or absence of ISAV would depend on the level of surveillance and monitoring of susceptible populations. However, the presence of ISAV would be readily detectable because the disease causes high morbidity and mortality.

The slaughter of farmed Atlantic salmon under official direction, to control an outbreak of ISAV, presents a particular risk factor, as a significant proportion of apparently healthy fish may be expected to have ISAV in their body tissues. Under these circumstances, the risk of ISAV becoming established in Australia would be higher than the estimate provided in Chapter 4. In these circumstances, the risk would not be expected to meet Australia's ALOP, necessitating the imposition of additional controls. AQIS could require that the competent authority of the exporting country provide certification confirming that Atlantic salmon did not come from a farm known or officially suspected of being affected by an outbreak of ISA. This would substantially address risk factor 1.

Inspection and grading

Inspection and grading would provide for the detection of fish with clinical disease due to ISAV, addressing risk factor 2. Inspection and grading would also provide for the identification of fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factor 3.

Processing of the product

Inspection and grading would not detect covertly infected Atlantic salmon. ISAV could be present in the tissues of such fish, particularly the skin mucus, gills, blood and viscera. Commonly used commercial processes (removal of the head and thorough cleaning and washing of internal and external surfaces to remove visceral remnants and skin mucus respectively) would substantially reduce risks associated with these factors.

Removal of the head and gills before importation into Australia would significantly reduce risk, as the head is not normally consumed and is (except for pan-size salmonids) usually removed before the fish is cooked. Disposal of the head by inappropriate means (such as by use as fishing bait) could present a high risk.

However, such processing would not totally eliminate risk; for example, washing would not remove all of the skin mucus or remnants of the anterior kidney on the skeleton.

AQIS could require the processing of imported salmonids to a specified standard, that is, removal of the head and gills and thorough cleaning and washing of internal and external surfaces. This would substantially address risk factor 3.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority. An appropriate measure would be for the competent authority of the exporting country to certify that the fish exported to Australia were inspected, graded and processed in accordance with Australia's conditions.

Waste disposal

The commercial processing of imported salmonids in Australia could generate a significant volume of solid or liquid waste at the premises' point of discharge. Continuous long-term release of untreated waste could result in the build-up of ISAV to a biologically significant level in the aquatic environment. For ISAV, waste tissues of concern would include the head and gills, skin and associated skin mucus, effluent contaminated by blood and the parts of the skeleton with attached remnants of the anterior kidney. As discussed in Section 5.2.2, AQIS could implement controls over commercial plants processing imported salmonid products with regard to location, waste disposal and related matters that would substantially address risk factors 4 and 5.

To ensure that imported salmonids were not commercially processed in non-approved premises, AQIS could permit release from quarantine of consumer-ready product only. Processing of such product for human consumption would normally generate minimal waste and this would not be expected to increase the overall risk of ISAV establishing.

Conclusions

To mitigate risks associated with the importation of eviscerated salmonids in relation to the establishment in Australia of ISAV, AQIS will permit the importation of eviscerated Atlantic salmon subject to the conditions shown in Box 5.3. The importation of eviscerated commercially harvested market-size salmonids other than Atlantic salmon does not warrant specific risk management for ISAV.

For ISAV, implementation of the measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 5.3 would meet Australia's ALOP; importation of eviscerated Atlantic salmon will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supportive scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 5.3
Risk management measures for infectious salmon anaemia virus for Atlantic salmon

PRE-EXPORT REQUIREMENTS

- The fish must be derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority.
- The Atlantic salmon must not come from a farm known or officially suspected of being affected by an outbreak of ISA.
- The head and gills must be removed and internal and external surfaces thoroughly washed.
- The fish must be inspected and graded under the supervision of a competent authority.
- The product for export must be free from visible lesions associated with infectious disease and fit for human consumption.
- The fish must be processed in a premises approved by and under the control of a competent authority.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

POST-IMPORT MEASURES

- Only premises approved by AQIS will be permitted to commercially process imported salmonids in Australia.
- Only consumer-ready product will be released from quarantine.

5.3.4 *AEROMONAS SALMONICIDA* (TYPICAL AND ATYPICAL)

Risk assessment conclusions

In Chapter 4, AQIS concluded that for the unrestricted importation of eviscerated salmonids (other than wild ocean-caught Pacific salmon, for which the probability would be extremely low) the probability of establishment of typical *A. salmonicida* would be low. The consequences of establishment would be of moderate to high significance. Thus, for typical *A. salmonicida*, the risk associated with the unrestricted importation of eviscerated salmonids (other than wild ocean-caught Pacific salmon) does not meet Australia's ALOP and the implementation of risk management measures is warranted.

For the unrestricted importation of eviscerated salmonids (other than wild ocean-caught Pacific salmon), the probability of establishment of additional strains of atypical *A. salmonicida* would be low. The consequences of establishment would be of moderate significance. Thus, for atypical *A. salmonicida*, the risk associated with the unrestricted importation of eviscerated salmonids does not meet Australia's ALOP and the implementation of risk management measures is warranted (see Box 4.7).

Key risk factors

(**Note:** these risk factors do not apply to wild, ocean-caught, Pacific salmon.)

1. Furunculosis is a serious disease. If detected in the course of official surveillance and monitoring of the health of salmonid populations, *A. salmonicida* may the subject of official controls, including compulsory slaughter of diseased populations.
2. The risk associated with juvenile fish and sexually mature fish (spawners) would be higher than that associated with commercially harvested, market-size salmonids.

3. Clinically infected fish would have a high titre of *A. salmonicida* in their body tissues.
4. *A. salmonicida* may be present in covertly infected fish, including in the gills, skin mucus and viscera.
5. *A. salmonicida* could survive in tissues and in the aquatic environment for a significant period.
6. *A. salmonicida* could accumulate in the aquatic environment as a result of the uncontrolled disposal of waste from commercial processing of imported salmonids.

Risk management measures

The following risk management measures would reduce the risk associated with the establishment of *A. salmonicida* via the importation of eviscerated salmonids into Australia.

Health status

- requirement that the fish are derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority; and
- requirement that the fish are not derived from a population slaughtered as an official disease control measure.

Age of fish

- requirement that the fish are not juvenile salmonids or sexually mature fish (spawners).

Inspection and grading

- to remove clinically diseased fish.

Processing

- removal of the head and gills;
- thorough cleaning and washing of external surfaces to remove as much skin mucus as practicable;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- requirement that the fish were processed in a premises under the control of a competent authority.

Export certification

- a requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Waste disposal

- control over the processing of imported salmonids in Australia; and
- control over the form and presentation of imported salmonid product released from quarantine to reduce the volume of waste generated in Australia.

Health status of the population from which the imported fish were derived

A. salmonicida is no longer listed by the OIE. Countries or regions that are free from *A. salmonicida* would maintain surveillance for this pathogen. The presence of *A. salmonicida* would readily become apparent because of the significance of the disease caused by the bacterium. The prevalence of furunculosis is higher in farmed than in wild salmonids. The prevalence is extremely low to negligible in wild, ocean-caught Pacific salmon. The prevalence and economic significance of furunculosis in farmed fish is decreasing with the use of improved vaccines and the practice of emergency slaughter to minimise losses is now uncommon.

The slaughter of farmed fish under official direction, to control an outbreak of furunculosis, presents a particular risk factor, as a significant proportion of apparently healthy fish may be expected to have *A. salmonicida* in their body tissues. Under these circumstances, the risk of *A. salmonicida* becoming established in Australia would be higher than the estimate provided in Chapter 4. In these circumstances, the risk would not be expected to meet Australia's ALOP, necessitating the imposition of additional controls. AQIS could require that the competent authority of the exporting country provide certification confirming that the fish were not derived from a population slaughtered as an official disease control measure due to an outbreak of furunculosis. This would substantially address risk factor 1.

Age of fish

As discussed in Section 5.2.1, for *A. salmonicida* the risk associated with the importation of juvenile salmonids and sexually mature fish (spawners) would be higher than that associated with commercially harvested, market-size salmonids. AQIS could require the exporting country to provide certification confirming that fish exported to Australia were not juvenile salmonids or spawners. This would address risk factor 2.

Inspection and grading

Inspection and grading would provide for the detection of fish with clinical disease due to *A. salmonicida*, addressing the third risk factor identified above. Inspection and grading would also provide for the identification of juvenile and sexually mature fish and fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factors 2 and 3.

Processing of the product

Inspection and grading would not detect covertly infected fish. *A. salmonicida* could be present in the tissues of such fish, particularly in the gills, skin mucus and viscera. Commonly used commercial processes (removal of the head and thorough cleaning and washing of internal and external surfaces to remove visceral remnants and skin mucus respectively) would substantially reduce risks associated with these factors.

Removal of the head and gills before importation into Australia would significantly reduce risk, as the head is not normally consumed and is (except for pan-size salmonids) usually removed before the fish is cooked. Disposal of the head by inappropriate means (such as by use as fishing bait) could present a high risk.

However, such processing would not totally eliminate risk; for example, washing would not remove all of the skin mucus or remnants of the anterior kidney on the skeleton.

AQIS could require the processing of imported salmonids to a specified standard, that is, removal of the head and gills and thorough cleaning and washing of internal and external surfaces. This would substantially address risk factor 4.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority. An appropriate measure would be for the competent authority of the exporting country to certify that the fish exported to Australia were inspected, graded and processed in accordance with Australia's conditions.

Waste disposal

The commercial processing of imported salmonids in Australia could generate a significant volume of solid or liquid waste at the premises' point of discharge. Continuous long-term release of untreated waste could result in the build-up of *A. salmonicida* to a biologically significant level in the aquatic environment. For *A. salmonicida*, waste tissues of concern would be the head and gills, skin and associated skin mucus and the parts of the skeleton with attached remnants of the anterior kidney. As discussed in Section 5.2.2, AQIS could implement controls over commercial plants processing imported salmonid products with regard to location, waste disposal and related matters that would substantially address risk factors 5 and 6.

To ensure that imported salmonids were not commercially processed in non-approved premises, AQIS could permit release from quarantine of consumer-ready product only. Processing of such product for human consumption would normally generate minimal waste and this would not be expected to increase the risk of establishment of *A. salmonica* overall.

Conclusions

To mitigate risks associated with the importation of eviscerated salmonids, other than wild, ocean-caught Pacific salmon, in relation to the establishment in Australia of *A. salmonicida*, AQIS will permit the importation of eviscerated salmonids subject to the conditions shown in Box 5.4.

For *A. salmonicida*, the implementation of these measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 5.4 would meet

Australia's ALOP; importation of eviscerated salmonids will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supportive scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 5.4
Risk management measures for *A. salmonicida*

Note: These risk management measures do not apply to wild, ocean-caught Pacific salmon.

PRE-EXPORT REQUIREMENTS

- The fish must be derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority.
- The fish must not be derived from a population slaughtered as an official disease control measure.
- The fish must not be juvenile salmonids or spawners.
- The head and gills must be removed and internal and external surfaces thoroughly washed.
- The fish must be inspected and graded under the supervision of a competent authority.
- The product for export must be free from visible lesions associated with infectious disease and fit for human consumption.
- The fish must be processed in a premises approved by and under the control of a competent authority.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

POST-IMPORT MEASURES

- Only premises approved by AQIS will be permitted to commercially process imported salmonids in Australia.
- Only consumer-ready product will be released from quarantine.

5.3.5 *RENIBACTERIUM SALMONINARUM* (BACTERIAL KIDNEY DISEASE)

Risk assessment conclusions

In Chapter 4, AQIS concluded that for the unrestricted importation of eviscerated salmonids of the genus *Oncorhynchus* and juveniles and sexually mature fish of all salmonid species, the probability of establishment of *R. salmoninarum* would be very low. The consequences of establishment would be of high significance.

Thus, for *R. salmoninarum*, the risk associated with the unrestricted importation of eviscerated salmonids of the genus *Oncorhynchus*, and juveniles and sexually mature fish of all salmonid species, does not meet Australia's ALOP. Therefore, the implementation of risk management measures is warranted.

For the unrestricted importation of all other eviscerated salmonids, the probability of establishment of *R. salmoninarum* would be lower but still very low. Thus, for *R. salmoninarum*, the risk associated with the unrestricted importation of all other eviscerated salmonids does not meet Australia's ALOP and the implementation of risk management measures is warranted (see Box 4.9).

Key risk factors

1. Bacterial kidney disease is a serious disease. If detected in the course of official surveillance and monitoring of the health of salmonid populations, *R. salmoninarum* may be the subject of official controls, including compulsory slaughter of diseased populations.
2. The risk associated with juvenile fish and sexually mature fish (spawners) would be higher than that associated with commercially harvested, market-size salmonids.
3. Clinically affected fish would have a high titre of *R. salmoninarum* in their body tissues.
4. *R. salmoninarum* may be present in covertly infected fish, including in the brain and viscera, particularly the anterior kidney.
5. *R. salmoninarum* could survive in tissues and in the aquatic environment for a significant period.
6. *R. salmoninarum* could accumulate in the aquatic environment as a result of the uncontrolled disposal of waste from commercial processing of imported salmonids.

Risk management measures

The following risk management measures would reduce the risk associated with the establishment of *R. salmoninarum* via the importation of eviscerated salmonids into Australia.

Health status

- requirement that the fish are derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority; and
- requirement that the fish are not derived from a population slaughtered as an official disease control measure.

Age of fish

- requirement that the fish are not juvenile salmonids or sexually mature fish (spawners).

Inspection and grading

- to remove clinically diseased fish.

Processsing

- removal of the head and gills;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- requirement that the fish were processed in a premises under the control of a competent authority.

Export certification

- requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Waste disposal

- control over the processing of imported salmonids in Australia; and
- control over the form and presentation of imported salmonid product released from quarantine to reduce the volume of waste generated in Australia.

Health status of the population from which the imported fish were derived

R. salmoninarum is listed by the OIE as an 'other significant' disease and there may be official control programs for *R. salmoninarum* in some regions. Countries or regions that are free from *R. salmoninarum* would maintain surveillance for this pathogen. Confidence regarding the presence or absence of *R. salmoninarum* will depend on the level of surveillance and monitoring of susceptible populations. However, the presence of *R. salmoninarum* would be readily detectable because of the high morbidity and significant pathological changes associated with bacterial kidney disease.

The slaughter of farmed fish under official direction, to control an outbreak of bacterial kidney disease, presents a particular risk factor, as a significant proportion of apparently healthy fish may be expected to have *R. salmoninarum* in their body tissues. Under these circumstances, the risk of *R. salmoninarum* becoming established in Australia would be higher than the estimate provided in Chapter 4. In these circumstances, the risk would not be expected to meet Australia's ALOP, necessitating the imposition of additional controls. AQIS could require that the competent authority of the exporting country provide certification confirming that the fish were not derived from a population slaughtered as an official disease control measure due to an outbreak of bacterial kidney disease. This would substantially address risk factor 1.

Age of fish

As discussed in Sefction 5.2.1, for *R. salmoninarum* the risk associated with the importation of juvenile salmonids and sexually mature fish (spawners) would be higher than that associated with commercially harvested, market-size salmonids. AQIS could require the exporting country to provide certification confirming that fish exported to Australia were not juvenile salmonids or spawners. This would address risk factor 2.

Inspection and grading

Inspection and grading would provide for the detection of fish with clinical disease due to *R. salmoninarum*, addressing the third risk factor identified above. Inspection and grading would also provide for the identification of juvenile and sexually mature fish and fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factors 2 and 3.

Processing of the product

Inspection and grading would not detect covertly infected fish. *R. salmoninarum* could be present in the tissues of such fish, particularly in the brain and viscera, particularly the anterior kidney. Commonly used commercial processes (removal of the head and thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with these factors.

Removal of the head and gills before importation into Australia would significantly reduce risk, as the head is not normally consumed and is (except for pan-size salmonids) usually removed before the fish is cooked. Disposal of the head by inappropriate means (such as by use as fishing bait) could present a high risk.

However, such processing would not totally eliminate risk; for example, washing would not remove all remnants of the anterior kidney on the skeleton.

AQIS could require the processing of imported salmonids to a specified standard, that is, removal of the head and gills and thorough cleaning and washing of internal surfaces. This would substantially address risk factor 4.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority. An appropriate measure would be for the competent authority of the exporting country to certify that the fish exported to Australia were inspected, graded and processed in accordance with Australia's conditions.

Waste disposal

The commercial processing of imported salmonids in Australia could generate a significant volume of solid or liquid waste at the premises' point of discharge. Continuous long-term release of untreated waste could result in the build-up of *R. salmoninarum* to a biologically significant level in the aquatic environment. For *R. salmoninarum*, waste tissues of concern would be the head and gills and the parts of the skeleton with attached remnants of the anterior kidney. As discussed in Section 5.2.2, AQIS could implement controls over commercial plants processing imported salmonid products with regard to location, waste disposal and related matters that would substantially address risk factors 5 and 6.

To ensure that imported salmonids were not commercially processed in non-approved premises, AQIS could permit release from quarantine of consumer-ready product only. Processing of such product for human consumption would normally generate minimal waste and this would not be expected to increase the risk of establishment of *R. salmoninarum* overall.

Conclusions

To mitigate risks associated with the importation of eviscerated salmonids in relation to the establishment in Australia of *R. salmoninarum*, AQIS will permit the importation of eviscerated salmonids subject to the conditions shown in Box 5.5.

For *R. salmoninarum*, the implementation of these measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 5.5 would meet Australia's ALOP; importation of eviscerated salmonids will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supportive scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 5.5
Risk management measures for *R. salmoninarum*

PRE-EXPORT REQUIREMENTS

- The fish must be derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority.
- The fish must not be derived from a population slaughtered as an official disease control measure.
- The fish must not be juvenile salmonids or spawners.
- The head and gills must be removed and internal surfaces thoroughly washed.
- The fish must be inspected and graded under the supervision of a competent authority.
- The product for export must be free from visible lesions associated with infectious disease and fit for human consumption.
- The fish must be processed in a premises approved by and under the control of a competent authority.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

POST-IMPORT MEASURES

- Only premises approved by AQIS will be permitted to commercially process imported salmonids in Australia.
- Only consumer-ready product will be released from quarantine.

5.3.6 *YERSINIA RUCKERI* (HAGERMAN STRAIN) (ENTERIC REDMOUTH DISEASE)

Risk assessment conclusions

In Chapter 4, AQIS concluded that for the unrestricted importation of eviscerated adult salmonids, the probability of establishment of *Y. ruckeri* (Hagerman strain) would be very low. For juveniles, the probability would be low. The consequences of establishment would be of low to moderate significance.

Thus, for *Y. ruckeri* (Hagerman strain), the risk associated with the unrestricted importation of eviscerated adult salmonids, meets Australia's ALOP and the implementation of risk management measures is not warranted. The risk associated with the unrestricted importation of eviscerated juvenile salmonids, would not meet Australia's ALOP and the implementation of risk management measures is warranted (see Box 4.10).

Key risk factors

1. The risk associated with juvenile fish would be higher than that associated with commercially harvested, market-size salmonids because infection is usually clinically expressed in juvenile salmonids; thus there is a greater probability of a significant bacterial titre in these fish.
2. *Y. ruckeri* may be present in covertly infected fish, particularly in the viscera.
3. *Y. ruckeri* could survive in tissues and in the aquatic environment for a significant period.
4. *Y. ruckeri* could accumulate in the aquatic environment as a result of the uncontrolled disposal of waste from commercial processing of imported salmonids.

Risk management measures

The following risk management measures would reduce the risk associated with the establishment of *Y. ruckeri* (Hagerman strain) via the importation of eviscerated salmonids into Australia.

Age of fish

- requirement that the fish are not juvenile salmonids.

Inspection and grading

- to remove clinically diseased fish.

Processsing

- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- requirement that the fish were processed in a premises under the control of a competent authority.

Export certification

- requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Age of fish

Infection and disease would be unlikely to occur in adult fish, thus for *Y. ruckeri* the risk associated with the importation of juvenile salmonids would be higher than that associated with commercially harvested, market-size salmonids. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia were not juvenile salmonids. This would address risk factor 1.

Inspection and grading

Inspection and grading would provide for the identification of juvenile salmonids and fish that were not processed in accordance with Australia's import conditions. The risk assessment for *Y. ruckeri* concluded that fish clinically affected by *Y. ruckeri* would be visibly abnormal. Such fish would be detected and removed in the course of inspection for human consumption. This would substantially address risk factors 1 and 2.

Processing of the product

Inspection and grading would not detect covertly infected fish. *Y. ruckeri* (Hagerman strain) could be present in the tissues of such fish, particularly in the viscera.

Commonly used commercial processes (evisceration and thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with these factors.

However, such processing would not totally eliminate risk; for example, washing would not remove all remnants of the anterior kidney on the skeleton.

AQIS could require the processing of imported salmonids to a specified standard, that is, thorough cleaning and washing of internal surfaces. This would substantially address risk factor 2.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority. An appropriate measure would be for the competent authority of the exporting country to certify that the fish exported to Australia were inspected, graded and processed in accordance with Australia's conditions.

Waste disposal

The implementation of specific risk management to address risk factors 4 and 5 is not warranted because the other risk management measures identified would effectively prevent the entry of *Y. ruckeri* into the aquatic environment.

Conclusions

To mitigate risks associated with the importation of eviscerated salmonids in relation to the establishment in Australia of *Y. ruckeri*, AQIS will permit the importation of eviscerated salmonids subject to the conditions shown in Box 5.6.

For *Y. ruckeri*, implementation of the measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 5.6 would meet Australia's ALOP; importation of eviscerated salmonids will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supportive scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 5.6
Risk management measures for *Y. ruckeri*

PRE-EXPORT REQUIREMENTS

- The fish must not be juvenile salmonids.
- The internal surfaces must be thoroughly cleaned and washed of to remove remnants of the viscera as far as practicable.
- The fish must be inspected and graded under the supervision of a competent authority.
- The product for export must be free from visible lesions associated with infectious disease and fit for human consumption.
- The fish must be processed in a premises approved by and under the control of a competent authority.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

5.3.7 *MYXOBOLUS CEREBRALIS* (WHIRLING DISEASE)

Risk assessment conclusions

In Chapter 4, AQIS concluded that for the unrestricted importation of eviscerated rainbow trout and juvenile salmonids, the probability of establishment of *M. cerebralis* would be low. The consequences of establishment would be of low to moderate significance. Thus, for *M. cerebralis*, the risk associated with the unrestricted importation of eviscerated rainbow trout and juvenile salmonids does not meet Australia's ALOP and the implementation of risk management measures is warranted.

For the unrestricted importation of eviscerated adult salmonids (other than rainbow trout), the probability of establishment of *M. cerebralis* would be very low. The consequences of establishment would be of low to moderate significance. Thus, for *M. cerebralis*, the risk associated with the unrestricted importation of eviscerated adult salmonids (other than rainbow trout) meets Australia's ALOP and the implementation of risk management measures is not warranted (see Box 4.12).

Key risk factors for rainbow trout and juvenile salmonids

1. Whirling disease is a significant disease of rainbow trout. If detected in the course of official surveillance and monitoring, *M. cerebralis* may be the subject of official controls.
2. The risk associated with juvenile fish would be higher than that associated with commercially harvested, market-size salmonids.
3. Clinically affected fish would have a high titre of *M. cerebralis* in cartilaginous and bony tissue of the body.
4. *M. cerebralis* may be present in covertly infested fish in cartilage and bone, particularly in the head and gills.
5. *M. cerebralis* could survive in tissues and in the aquatic environment for a significant period.
6. *M. cerebralis* spores could accumulate in the aquatic environment as a result of the uncontrolled disposal of waste from commercial processing of imported salmonids.
7. *T. tubifex* has a widespread distribution in Australia including in regions were there are salmonid populations, although *T. tubifex* is thought to be present at much lower density than other oligochaetes. *T. tubifex* is the only oligochaete that is known to be a competent host for *M. cerebralis*.

Risk management measures

The following risk management measures would reduce the risk associated with the establishment of *M. cerebralis* via the importation of eviscerated rainbow trout (and juvenile salmonids of all species) into Australia:

Health status

- requirement that the fish are derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority; and
- requirement that the fish are not derived from a population slaughtered as an official disease control measure.

Age of fish

- requirement that the fish are not juvenile salmonids.

Inspection and grading

- to remove clinically diseased fish.

Processing

- removal of the head and gills.

Export certification

- requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Waste disposal

- control over the processing of imported salmonids in Australia; and
- control over the form and presentation of imported salmonid product released from quarantine to reduce the volume of waste generated in Australia.

Health status of the population from which the imported fish were derived

M. cerebralis is no longer listed by the OIE. Countries or regions that are free from *M. cerebralis* would maintain surveillance for this pathogen. The presence of *M. cerebralis* would readily become apparent in susceptible rainbow trout because of the nature of the clinical signs of the disease caused by this parasite. In other salmonids, infestation with the parasite would be less apparent and its presence could be undetected for some time. By the time an outbreak of whirling disease was detected, the intermediate host (*T. tubifex*) would have already been infected and official slaughter may not be warranted, but other controls may be implemented. There would be little cartilage in commercially harvested, market-size rainbow trout and the number of spores in infested fish would be much lower than that in juvenile fish.

The slaughter of farmed rainbow trout under official direction, to control an outbreak of whirling disease presents a particular risk factor, as a significant proportion of apparently healthy fish may be expected to have *M. cerebralis* in their cartilage/bone. Given the epidemiology of whirling disease, it is questionable that such practice would significantly increase the risk of *M. cerebralis* becoming established in Australia above that estimated in Chapter 4. However, to address any additional element of risk, AQIS could require that the competent authority of the exporting country provide certification confirming that the rainbow trout were not derived from a population slaughtered as an official disease control measure due to an outbreak of whirling disease. This would substantially address risk factor 1.

Age of fish

As discussed in Section 5.2.1, for *M. cerebralis* the risk associated with the importation of juvenile salmonids would be higher than that associated with commercially harvested, market-size salmonids. AQIS could require the exporting country to provide certification confirming that fish exported to Australia were not juvenile salmonids. This would address risk factor 2.

Inspection and grading

Inspection and grading would provide for the detection of rainbow trout with clinical disease due to *M. cerebralis* and for the identification of juvenile fish and fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factors 2 and 3.

Processing of the product

Inspection and grading would not detect covertly infested rainbow trout. *M. cerebralis* could be present in the cartilage and bone of such fish, particularly in the head and gills. Commonly used commercial processes (removal of the head and gills) would substantially reduce risks associated with these factors.

Removal of the head and gills before importation into Australia would significantly reduce risk, as the head is not normally consumed and is (except for pan-size salmonids) usually removed before the fish is cooked. Disposal of the head by inappropriate means (such as by use as fishing bait) could present a high risk.

However, such processing would not totally eliminate risk; for example, spores could still be present in the vertebral column and elsewhere in the body.

AQIS could require the processing of imported rainbow trout to a specified standard, that is, removal of the head and gills. This would substantially address risk factor 4.

Export certification

To support the provision of certification, AQIS could also require that the rainbow trout were processed in a premises approved by and under the control of a competent authority. An appropriate measure would be for the competent authority of the exporting country to certify that the fish exported to Australia were inspected, graded and processed in accordance with Australia's conditions.

Waste disposal

The commercial processing of imported rainbow trout in Australia could generate a significant volume of solid or liquid waste at the premises' point of discharge. Continuous long-term release of untreated waste could result in the build-up of *M. cerebralis* spores in the

aquatic environment. For example, the Lake Eildon and Goulburn River regions of Victoria, which include Australia's major commercial trout raising areas, have fish processing plants, including smokehouses, on the banks of waterways. The discharge or accidental spillage of untreated wastes from these plants into the waterway could be a significant pathway for the establishment of *M. cerebralis*. Tubificid oligochaetes are very common in this region, although *T. tubifex* is thought to be present at a much lower density than other oligochaetes.

For *M. cerebralis*, waste tissues of concern would be the head and gills, and cartilage and bone elsewhere in the body. As discussed in Section 5.2.2, AQIS could implement controls over commercial plants processing imported salmonid products with regard to location, waste disposal and related matters that would substantially address risk factors 5, 6 and 7.

In order to ensure that imported rainbow trout were not commercially processed in non-approved premises, AQIS could permit release from quarantine of consumer-ready product only. Processing of such product for human consumption would normally generate minimal waste and this would not be expected to increase the risk of establishment of *M. cerebralis* overall.

Conclusions

To mitigate risks associated with the importation of eviscerated salmonids in relation to the establishment in Australia of *M. cerebralis*, AQIS will permit the importation of eviscerated rainbow trout subject to the conditions shown in Box 5.7. The importation of eviscerated commercially harvested market-size salmonids other than rainbow trout does not warrant specific risk management for *M. cerebralis*.

For *M. cerebralis*, the implementation of these measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 5.7 would meet Australia's ALOP; importation of eviscerated rainbow trout will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supportive scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 5.7
Risk management measures for *M. cerebralis*

Note: These risk management measures only apply to rainbow trout and juvenile salmonids.

PRE-EXPORT REQUIREMENTS

- The fish must be derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority.
- The fish must not be derived from a population slaughtered as an official disease control measure.
- The fish must not be juvenile salmonids.
- The head and gills must be removed.
- The fish must be inspected and graded under the supervision of a competent authority.
- The product for export must be free from visible lesions associated with infectious disease and fit for human consumption.
- The fish must be processed in a premises approved by and under the control of a competent authority.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

POST-IMPORT MEASURES:

- Only premises approved by AQIS will be permitted to commercially process imported salmonids in Australia.
- Only consumer-ready product will be released from quarantine.

5.4 Overall risk management for eviscerated salmonids

In Section 5.3 AQIS concluded that, as warranted by the risk analysis, the importation of eviscerated salmonids would be permitted, subject to a series of measures that would have the effect of mitigating risks associated with specified diseases. For eviscerated, commercially-harvested, market-size salmonids,[2] the disease agents that require specific risk management are:

- infectious haematopoietic necrosis virus (IHNV);
- infectious salmon anaemia virus (ISAV) (for Atlantic salmon);
- *Aeromonas salmonicida* (not for wild, ocean-caught Pacific salmon);
- *Renibacterium salmoninarum*; and
- *Myxobolus cerebralis* (for rainbow trout).

As these diseases are either not reported in New Zealand or (for *M. cerebralis*) occur at extremely low prevalence in New Zealand Pacific salmon, these measures would not apply to imports of Pacific salmon from New Zealand.

- the fish are derived from a population for which there is a documented system of health surveillance and monitoring administered by a competent authority;
- the fish are not derived from a population slaughtered as an official disease control measure;
- the fish are not juvenile salmonids or spawners;
- the head and gills were removed and the internal and external surfaces were thoroughly washed;
- the fish were inspected and graded under the supervision of a competent authority;
- the product is free from visible lesions associated with infectious disease;
- the fish were processed in a premises approved by and under the control of a competent authority;
- consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full;
- only premises approved by AQIS are permitted to commercially process imported salmonids in Australia; and
- only product that is consumer-ready will be released from quarantine.

In addition, for countries in which infectious salmon anaemia (ISA) occurs[3] there is a requirement that Atlantic salmon do not come from a farm known or officially suspected of being affected by an outbreak of ISA.

In this risk analysis, the following products are considered to be 'consumer-ready':

- cutlets — including central bone and external skin but excluding fins — of less than 450g in weight;
- skinless fillets — excluding the belly flap and all bone except the pin bones, of any weight;
- skin-on fillets — excluding the belly flap and all bone except the pin bones — of less than 450g in weight;
- eviscerated, headless 'pan-size' fish of less than 450g in weight; and
- product that is processed further than the stage described above.

5.5 Risk management for lower priority diseases (group 2)

The next step was to consider whether the application of the general risk management strategies outlined above would address the risk associated with the importation of eviscerated salmonids in relation to the establishment in Australia of the pathogens in group 2 (see Section 4.1.1). The following disease agents were identified to be of lower priority in the import risk analysis on salmonids (group 2):

2 AQIS will not generally permit the importation of juvenile salmonids and sexually mature adult salmonids (spawners) as this would present an unacceptably high quarantine risk for certain disease agents (specified in Chapter 5).

3 As at July 1999, ISA has been reported from Scotland, Norway and Canada.

- erythrocytic necrosis virus (viral erythrocytic necrosis);
- new Japan virus;
- salmon anaemia virus (erythrocytic inclusion body syndrome);
- salmon leukaemia virus (plasmacytoid leukaemia);
- *Vibrio salmonicida* (hitra disease);
- *Ceratomyxa shasta* (ceratomyxosis);
- *Enterocytozoon salmonis* (or *Nucleospora salmonis*);
- *Henneguya salminicola* (henneguyosis);
- *Hexamita salmonis* (hexamitosis);
- *Loma salmonae*;
- nervous mortality syndrome; and
- rosette agent.

Sections 5.5.1 to 5.5.12 consider the expected effect of the general risk management strategies on the risk of establishment of these diseases as a result of the importation of eviscerated salmonids.

5.5.1 ERYTHROCYTIC NECROSIS VIRUS (VIRAL ERYTHROCYTIC NECROSIS)

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Viral erythrocytic necrosis (VEN) caused by erythrocytic necrosis virus (ENV)[4] has been reported in Europe, the United States, Canada, and Greenland. ENV is not OIE listed.
- Erythrocytic abnormalities associated with ENV infection have been recorded in at least 17 families of marine and anadromous fish including Atlantic cod, Atlantic and Pacific herring, Atlantic salmon and Pacific salmon.
- VEN does not cause high morbidity or mortality; rather, it impairs fish health and production. Outbreaks of clinical disease are often associated with intercurrent infection with other pathogens.
- The clinical signs of VEN include pallor of the gills and internal organs.
- ENV infects erythrocytes and occurs at a significant titre tissues containing concentrations of haematopoietic cells (eg kidney, spleen, liver and intestinal submucosa).
- ENV is known to survive freezing at –70°C but is inactivated at 60°C for 15 minutes.

Key considerations

Fish affected by clinical disease would be visibly abnormal and would be detected and rejected in the course of inspection and grading for human consumption. Because ENV infects erythrocytes, it may occur in the somatic musculature in clinically infected fish. However, most virus would be in blood-rich organs, thus evisceration and other processing would substantially reduce the titre of ENV present.

There is no information on the propensity of fish with chronic infection to become inapparent carriers of ENV. Covertly infected fish would not be detected at inspection. Because this virus infects erythrocytes, it may occur in the somatic musculature of such fish. However, there is no evidence to suggest that ENV would occur at a significant titre in the muscle of commercially harvested, market-size fish.

Because other common pathogens which require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries which report ENV, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on ENV, the implementation of these measures would reduce the risk of establishment of ENV to a level similar to that for other pathogens considered in the risk analysis.

4 In this chapter of the IRA, ENV is defined as the iridovirus that causes VEN. Other viruses that can cause erythrocytic necrosis in salmonids, such as the togavirus which causes erythrocytic inclusion body syndrome (EIBS), are considered in appropriate sections of this chapter.

The consequences of establishment of ENV in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For ENV, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.2 NEW JAPAN VIRUS

The following points are based on information in the 1997 report of the New Zealand Government (Stone et al 1997b). This report contains referenced reviews of the relevant literature.

- New Japan virus has only been reported in Japan and is yet to be characterised.
- The virus has been isolated from coho salmon, rainbow trout, iwana char and ayu and may be transmitted to masou salmon and ito under experimental conditions.
- Diseased fish exhibit abnormal swimming behaviour and lethargy.
- Viral antigen can be detected in kidney, brain, and blood cells of infected fish.

AQIS considered further information, summarised below.

Oh et al (1995) found that New Japan virus was not inactivated by ether or chloroform. It was partially inactivated at pH 1; however, treatment in a range of pH 2–9 reduced viral infectivity. The isolates were also stable at a high temperature, remaining infective at 60°C for 30 minutes.

Key considerations

There are few data on this disease agent. Fish affected by clinical disease may not be visibly abnormal and may not be detected in the course of inspection and grading for human consumption. Because New Japan virus infects erythrocytes, it may occur in the somatic musculature of clinically infected fish. However, most virus would be in blood-rich organs, thus evisceration and other processing would substantially reduce the titre of virus present.

There is no information on the propensity of fish with chronic infection to become inapparent carriers of New Japan virus. Covertly infected fish would not be detected at inspection. Because this virus infects erythrocytes, it may occur in the somatic musculature of such fish. However, there is no evidence to suggest that New Japan virus would occur at a significant titre in the muscle of commercially harvested, market-size fish.

Other common pathogens that require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium*, occur in countries that report New Japan virus. Therefore, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on New Japan virus, the implementation of these measures would reduce the risk of establishment of New Japan virus to a level similar to that for other pathogens considered in the risk analysis.

The consequences of establishment of New Japan virus in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For New Japan virus, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.3 PACIFIC SALMON ANAEMIA VIRUS (ERYTHROCYTIC INCLUSION BODY SYNDROME)

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Erythocytic inclusion body syndrome (EIBS) has been reported in the United States, Norway, Ireland, Scotland and Japan. It is not OIE listed.
- EIBS has been recorded in chinook salmon, coho salmon, Atlantic salmon, rainbow trout and cutthroat trout. It has not been reported in non-salmonid finfish.

- It is thought that the causative virus (salmon anaemia virus) is a togavirus but it has not been isolated.
- EIBS is a disease of salmonids characterised by severe anaemia. Its main effect is to compromise overall fish health. The outcome of infection (mortality or recovery) depends on the presence of intercurrent infection.
- The signs of clinical disease include anaemia, lethargy, pallor of the liver and haemorrhage in kidney and, sometimes, in skeletal muscle.
- Juveniles and adult salmonids may be infected under natural conditions; however, the highest prevalence of infection is in juvenile fish.
- There is no information available on the stability of the causative virus.

Key considerations

Juveniles are more likely to be infected with EIBS than adult salmonids.

Fish affected by clinical disease would be visibly abnormal and would be detected and rejected in the course of inspection and grading for human consumption. Because EIBS infects erythrocytes, it may occur in the somatic musculature of clinically infected fish. However, most virus would be in blood-rich organs, thus evisceration and other processing would substantially reduce the titre of EIBS present.

There is no information on the propensity of fish with chronic infection to become inapparent carriers of EIBS. Covertly infected fish would not be detected at inspection. Because this virus infects erythrocytes, it may occur in the somatic musculature of such fish. However, there is no evidence to suggest that EIBS would occur at a significant titre in the muscle of commercially harvested, market-size fish.

Because other common pathogens which require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries which report EIBS, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on EIBS, the implementation of these measures would reduce the risk of establishment of EIBS to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of EIBS in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For EIBS, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.4 SALMON LEUKAEMIA VIRUS (PLASMACYTOID LEUKAEMIA)

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Plasmacytoid leukaemia (PL) has only been reported from Canada; however, pathological changes similar to those seen with PL have been observed in salmonids in the United States. PL is not listed by the OIE.
- PL has only been recorded in chinook salmon under natural conditions. Coho salmon, sockeye salmon and Atlantic salmon may be infected by injection with infected material. PL has not been reported from non-salmonid finfish.
- The agent that causes PL is yet to be fully characterised, but it is thought to be a retrovirus (see Section 5.5.7).
- PL is principally a disease of marine chinook salmon. It has been reported in wild and farmed fish.
- Signs in clinically diseased fish include anaemia, exophthalmia, enlargement of the spleen and kidney, petechial haemorrhage and ascites.
- There are limited data on the tissues that harbour the virus. Infection has been transmitted by injection of kidney and spleen extracts from naturally infected chinook salmon. Infected plasmablasts, which are associated with virus-like particles, may be found in

many tissues. Based on this information, it is possible that all tissues would contain infective material; however, the titre would be higher in blood-rich tissues.

Key considerations

Fish affected by clinical disease would be visibly abnormal and would be detected and rejected in the course of inspection and grading for human consumption. Because PL infects erythrocytes, it may occur in the somatic musculature in clinically infected fish. However, most virus would be in blood-rich organs, thus evisceration would substantially reduce the titre of PL present.

There is no information on the propensity of fish with chronic infection to become inapparent carriers of PL. Covertly infected fish would not be detected at inspection. Because this virus infects erythrocytes, it may occur in the somatic musculature of such fish. However, there is no evidence to suggest that PL would occur at a significant titre in the muscle of commercially harvested, market-size fish.

Other common pathogens which require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries that report PL. Therefore, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on PL, the implementation of these measures would reduce the risk of establishment of PL to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of PL in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For PL, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.5 *VIBRIO SALMONICIDA* (HITRA DISEASE)

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- *V. salmonicida* is reported from many countries including North America, Norway, Scotland, Iceland and the Faroe Islands.
- Natural infections have been reported in Atlantic salmon, rainbow trout, Atlantic cod (*Gadus morhua*) and coal fish (*Gadus virens*). There is some evidence that non-salmonid fish are more resistant to disease than salmonids.
- Disease caused by *V. salmonicida* is characterised by severe haemorrhage and necrosis of the internal organs. Clinically diseased fish have deep-seated necrotic lesions in muscle and other tissues. In fish with chronic infections, muscle lesions may be replaced by scar tissue.
- Outbreaks of clinical disease are only reported in salmonids in seawater or brackish water.
- In clinically diseased fish, *V. salmonicida* may occur throughout the vascular system and may be found in the heart, intestine, blood, liver, kidney, spleen, muscle and faeces.
- It is thought that important sources of infection include carrier fish and/or sediment. There is no information on the distribution of the pathogen in the tissues of carrier fish.
- *V. salmonicida* has been shown to survive in the marine environment for more than 14 months. It does not grow at temperatures >22°C.
- Mortality rates as high as 95% have been recorded in disease epizootics. However, the introduction of an effective vaccine (administered by immersion, in food or by injection) and the use of antibiotics has significantly reduced the incidence of disease and associated mortality rates.

Key considerations

In clinically diseased salmonids*, V. salmonicida* is predominantly found in visceral organs but may also be found in muscle tissues; a factor distinguishing this agent from many other significant pathogens that are almost exclusively located in the visceral organs. In covertly infected fish, the pathogen may be found in kidney tissues, gills and other blood-rich organs, generally at low titre. The titre may be higher in inapparently infected fish sourced from a population experiencing an acute epizootic or just before an outbreak of disease due to *V. salmonicida*.

For both clinically and covertly infected fish, evisceration would substantially reduce the titre of V*. salmonicida* present. However, the pathogen may remain in other parts of the body, including the somatic musculature. The titre of *V. salmonicida* in muscle of covertly infected fish would normally be very low. The titre may be higher in muscle of salmonids affected by clinical disease.

Clinically infected salmonids are likely to be detected and rejected in the course of inspection for human consumption. Adult carrier fish would not be visibly abnormal and would not be detected at inspection. However, the bacterial titre in eviscerated adult carrier fish would be extremely low, unless these fish had been derived from a population affected by an acute disease epizootic.

Other common pathogens that require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries which report *V. salmonicida.* Therefore, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on *V. salmonicida*, the implementation of these measures would reduce the risk of establishment of *V. salmonicida* to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of *V. salmonicida* in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For *V. salmonicida*, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.6 *CERATOMYXA SHASTA* (CERATOMYXOSIS)

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- *Ceratomyxa shasta* has only been reported from the Pacific coast of Canada and the United States. *C. shasta* appears to be limited to a few rivers within these areas and has not shown a propensity to spread beyond this limited geographic range.
- It has been reported in rainbow trout, cutthroat trout, pink salmon, chinook salmon, coho salmon and chum salmon. There are no reports of *C. shasta* infesting non-salmonid fish.
- Infestations have been reported in fresh water and marine salmonids. Clinical disease is only seen in juvenile salmonids and sexually mature Pacific salmonids in fresh water.
- Clinically infected fish have lesions in the kidneys, pyloric caeca and intestine and external swellings. Parasitic stages of the protozoan may be found in the alimentary tract, liver, gall bladder, spleen, gonads, kidney, heart, gills, skin and musculature of the trunk.
- Infestation is initially limited to the alimentary tract, particularly the intestines. In subclinically infected fish most of the parasites may be limited to the alimentary tract.
- The source of infection is not known. Mature spores released from fish are not infective for susceptible fish species. The infective stage is suspected to be an actinosporan that develops in an oligochaete intermediate host, but this is yet to be confirmed.
- Developmental and sporogonic stages of *C. shasta* remain infective after freezing in dimethylsufoxide

(DMSO) at –80°C. If the parasite requires an intermediate host to complete its lifecycle it would be expected that spores would survive in the aquatic environment for a significant period.

AQIS considered further information from Bartholomew et al (1997), who provided a definitive description of the lifecycle of *C. shasta*. The intermediate host is the freshwater polychaete *Manayunkia speciosa.*

Key considerations

Clinical disease has only been reported in juvenile wild Pacific salmon and sexually mature adult wild Pacific salmon returned to fresh water. *C. shasta* appears to be limited to a few rivers within these areas and has not shown a propensity to spread beyond this limited geographic range.

In clinically diseased salmonids, *C. shasta* is predominantly found in visceral organs but may also be found in other tissues. Clinically infected salmonids are likely to be detected and rejected in the course of inspection for human consumption.

In covertly infected fish, most of the parasites would generally be localised in the alimentary tract. Evisceration would substantially reduce the numbers of *C. shasta* present. However, the pathogen may remain in other parts of the body.

Other common pathogens that require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries that report *C. shasta.* Therefore, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on *C. shasta*, the implementation of these measures would reduce the risk of establishment of *C. shasta* to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of *C. shasta* in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For *C. shasta*, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.7 *ENTEROCYTOZOON SALMONIS* (OR *NUCLEOSPORA SALMONIS*)

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- *E. salmonis* has been reported in Canada, United States, France and Chile.
- Natural infestations have been reported in chinook salmon, Atlantic salmon and rainbow trout. Sockeye salmon, coho salmon, brook trout and golden trout may be experimentally infested. *E. salmonis* has not been recorded from non-salmonid fish.
- Clinical disease is most common in young salmonids in fresh water.
- Signs associated with clinical infestations include anaemia, lethargy, exophthalmia, lymphoproliferation, enlarged kidneys and spleen and intestinal swelling.
- The organism is generally found in the haematopoietic tissues such as the kidney and spleen, and also in smaller numbers in the skeletal muscle, brain, intestine, eye and gills.
- *E. salmonis* is susceptible to freezing at –20°C. There is little further information on the susceptibility of *E. salmonis* to other treatments.

Key considerations

Clinical infestation is most common in juvenile salmonids.

In clinically diseased salmonids, *E. salmonis* is predominantly found in visceral organs but may also be found in muscle tissues. In covertly infested fish, the parasite would be expected to occur mainly in the kidney and spleen.

For both clinically and covertly infested fish, evisceration would substantially reduce the numbers of *E. salmonis* present. However, the pathogen may remain in other parts of the body, including the somatic musculature.

Clinically infested salmonids are likely to be detected and rejected in the course of inspection for human consumption. Covertly infested fish would not be detected at inspection. This protozoan has been reported in the somatic musculature of diseased fish. However, there is no evidence to suggest that *E. salmonis* would occur at a significant titre in the muscle of commercially harvested, market-size fish. There is no information on the propensity of fish with chronic infestation to become inapparent carriers of *E. salmonis.*

Other common pathogens that require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries that report *E. salmonis.* Therefore, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on *E. salmonis*, the implementation of these measures would reduce the risk of establishment of *E. salmonis* to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of *E. salmonis* in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For *E. salmonis*, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.8 *HENNEGUYA SALMINICOLA* (HENNEGUYOSIS)

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- The myxozoan parasite, *H. salminicola* has only been recorded in Canada and the United States.
- Natural infestation has been reported in coho salmon, sockeye salmon, chinook salmon, chum salmon, pink salmon and rainbow trout.
- Infestation with *H. salminicola* does not cause clinical disease in fish, but cystic lesions associated with postmortem myoliquifaction in affected fish reduces the value of the carcass.
- The number of cysts increases as the fish host ages. *H. salminicola* spores are found in cysts only after smolts have been in seawater for a year or longer.
- In infested fish, cysts may be found in the musculature in the region of the dorsal fin to caudal peduncle. They are also found, less frequently, on the lower jaw, spine, in the retrobulbar tissues and in the kidneys.
- As with other freshwater myxosporeans, the lifecycle of *H. salminicola* is not direct and is thought to include an oligochaete or other unknown host.
- The prevalence of infection may be high in certain parts of Canada and the United States, but the distribution of the parasite is limited, probably due to the limited distribution of the intermediate host.

Key considerations

In salmonids infested with *H. salminicola* cystic lesions are predominantly found in muscle tissues; a factor distinguishing this agent from many other pathogens that are almost exclusively located in the visceral organs. In covertly infested fish, the pathogen may also occur in muscles.

Infested salmonids are likely to be detected and rejected in the course of inspection for human consumption. There is no information on the propensity of fish with chronic infestation to become inapparent carriers of *H. salminicola*. Covertly infested fish would not be detected at inspection. Because cystic lesions are highly visible and spores are only found in these lesions after a considerable period, it is unlikely that fish with significant numbers of infective organisms would pass inspection and grading for human consumption.

Because other common pathogens which require the implementation of risk management, such as IPNV, IHNV,

Aeromonas salmonicida and *Renibacterium salmoninarum*, occur in countries which report *H. salminicola*, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on *H. salminicola*, the implementation of these measures would reduce the risk of establishment of *H. salminicola* to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of *H. salminicola* in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For *H. salminicola*, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.9 *HEXAMITA SALMONIS* (HEXAMITOSIS)

The following points are based on information in the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- *H. salmonis* is reported in Europe, North America and Asia.
- *H. salmonis* infests many species of freshwater and marine fishes.
- Of the salmonids, rainbow trout and Atlantic salmon are the species most commonly infested and fingerlings, yearlings and smolts are the lifecycle stage most commonly infested. Clinical disease is occasionally reported in larger fish. High mortality rates have been recorded in chinook salmon in Canada.
- Infestation occurs sporadically in farmed fish and rarely in wild fish.
- Clinical signs of infestation include anorexia, emaciation, pallor of the gills, abdominal distension, ascites, darkening and exophthalmia. In some cases petechial haemorrhage may occur throughout the skeletal musculature.
- Severe systemic infestation associated with high mortality has been reported infrequently. Systemically infested Atlantic salmon may be in good condition but significantly smaller than non-infected fish. It is thought that more invasive strains of *H. salmonis* may cause systemic infestation.
- The organism normally occurs only in the intestine of diseased fish. However, in systemic infestations, the pathogen may occur throughout the body.

Key considerations

In clinically diseased salmonids, *H. salmonis* is predominantly found in the intestine but may also be found in muscle tissues. In covertly infected fish the organism would be likely to be limited to the intestine.

For both clinically and covertly infected fish, evisceration would substantially reduce the numbers of *H. salmonis* present. However, the pathogen may remain in other parts of the body, including the somatic musculature. Clinically infected salmonids are likely to be detected and rejected in the course of inspection for human consumption. Covertly infected fish would not be detected at inspection. However, the numbers of *H. salmonis* in muscle of covertly infected fish (if any were present) would be very low.

There is no information on the propensity of fish with chronic infection to become inapparent carriers of *H. salmonis*.

Other common pathogens that require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries that report *H. salmonis.* Therefore, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on *H. salmonis*, the implementation of these measures would reduce the risk of establishment of *H. salmonis* to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of *H. salmonis* in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For *H. salmonis*, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.10 *LOMA SALMONAE*

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- *L. salmonae* occurs in North America, Japan and France.
- Infestation has been reported in rainbow trout, sockeye salmon, chinook salmon, coho salmon, and masou salmon.
- Infestation may be widespread in wild and hatchery-reared salmonids.
- Infection usually does not cause significant disease problems; however, outbreaks of clinical disease with mortality rates up to 10–12% have been reported (usually after transfer of smolts to seawater).
- The pathological changes associated with clinical disease include severe inflammatory gill lesions associated with the formation of xenomas, exophthalmia, ascites, haemorrhagic pyloric caeca and fins, petechial haemorrhage on opercula and skin, and darkened tails.
- The primary site of infection is the gills, in which xenomas occur. Parasitic spores may also be found in the heart, spleen, kidney, head and skeletal muscle.

Key considerations

Clinical disease is most common in juvenile salmonids, after transfer to seawater.

In clinically diseased salmonids, *L. salmonae* is predominantly found in the gills but may also be found in visceral tissues and muscle.

Clinically infected salmonids would be detected and rejected in the course of inspection for human consumption. Covertly infected fish would not be detected at inspection. However, there is no evidence to suggest that *L. salmonae* would occur at a significant titre in the muscle of commercially harvested, market-size fish. A higher number of organisms would be expected to occur in the gills.

There is no information on the propensity of fish with chronic infection to become inapparent carriers of *L. salmonae*.

Other common pathogens that require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries that report *L. salmonae.* Therefore, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on *L. salmonae*, the implementation of these measures would reduce the risk of establishment of *L. salmonae* to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of *L. salmonae* in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

Accordingly, for *L. salmonae*, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.11 NERVOUS MORTALITY SYNDROME

The following points are based on information in the 1997 report of the New Zealand Government (Stone et al 1997b). This report contains referenced reviews of the relevant literature.

- This syndrome has only been reported in Atlantic salmon at a single location in Ireland.
- Clinical disease is most common in post-smolts 6–8 weeks after transfer to seawater and may be associated with mortality rates of up to 90% in these fish.

- Fish that survive infection grow normally and are not affected by further outbreaks of the syndrome.
- Clinical signs of disease include lethargy, abnormal swimming behaviour, loss of balance and unconsciousness.
- Parasites associated with nervous mortality syndrome appear to be similar to the extrasporogenic stages of a myxosporean. They may be found in the brain and spinal cord of infected fish.
- AQIS considered further information from Frasca et al (1999), who described the parasite associated with nervous mortality syndrome and demonstrated in molecular studies that it probably is *Myxobolus cerebralis.*

Key considerations

There are limited data on this syndrome.

Juveniles are more likely to be infected with nervous mortality syndrome than adult salmonids. There is no evidence to suggest that spores would be present in tissues other than the central nervous system of clinically or subclinically infected fish. Furthermore, as the spores found in infected fish are presporogonic they would not be infectious for the intermediate or final host.

Because other common pathogens which require the implementation of risk management, such as *Aeromonas salmonicida*, occur in Ireland, the only country that reports nervous mortality syndrome, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above. Based on current scientific information on nervous mortality syndrome, the implementation of these measures would reduce the risk of establishment of nervous mortality syndrome to a similar extent as for the risk associated with other pathogens considered in the risk analysis. The consequences of establishment of nervous mortality syndrome in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

Accordingly, for nervous mortality syndrome, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.5.12 ROSETTE AGENT

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- The rosette agent has not yet been defined; however, it has some similarities to a *Dermocystidium* sp.
- Rosette agent has been reported in Canada and United States.
- Infections with rosette agent have been reported in chinook salmon, Atlantic salmon, brown trout and rainbow trout.
- In some cases infection with rosette agent has resulted in mortality rates of more than 95% of infected fish.
- Infection with rosette agent results in a chronic inflammatory disease characterised by the formation of granulomas in the kidney, spleen, liver, and gonads.
- In clinically infected fish, organisms may be found in the peripheral blood and vascular spaces of kidney, spleen, liver, gonad, heart, brain and intestinal mucosa.
- The organism appears to infect and replicate in fixed macrophages of the spleen and kidney.
- There are conflicting data on this pathogen's lability to freezing. It can remain infective for cell culture after being held in phosphate buffered saline for 44 days at 5°C and in tissue culture media for five months.

Key considerations

There are limited data on this pathogen.

Fish affected by clinical disease would be visibly abnormal and would be detected and rejected in the course of inspection and grading for human consumption. If rosette agent infects macrophages, it may occur in the somatic musculature in clinically infected fish. However, most virus would be in blood-rich organs, thus evisceration would substantially reduce the titre of rosette agent present.

There is no information on the propensity of fish with chronic infection to become inapparent carriers of rosette agent. Covertly infected fish would not be detected at inspection. Because this virus infects erythrocytes, it may occur in the somatic musculature of such fish. However, there is no evidence to suggest that rosette agent would occur at a significant titre in the muscle of commercially harvested, market-size fish.

Other common pathogens that require the implementation of risk management, such as IPNV, IHNV, *Aeromonas salmonicida* and *Renibacterium salmoninarum*, occur in countries that report rosette agent. Therefore, the importation into Australia of eviscerated salmonids from these countries would be subject to risk management measures outlined above.

Based on current scientific information on rosette agent, the implementation of these measures would reduce the risk of establishment of rosette agent to a level similar to that for other pathogens considered in the risk analysis. The consequences of establishment of rosette agent in Australia would not exceed those associated with the establishment of any other pathogen considered in the risk analysis.

Conclusion

For rosette agent, the risk associated with the importation of eviscerated salmonids in accordance with the general conditions specified above meets Australia's ALOP.

5.6 Summary of risk management measures required for importation of salmonids

A summary of risk management measures appropriate to the importation of eviscerated salmonids into Australia is shown in Table 5.1.

Table 5.1

Summary of risk management measures appropriate to the importation of salmonids

DISEASE AGENT	AGE RESTRICTIONS	MONITORED POPULATION	OFFICIAL SLAUGHTER	INSPECTION & GRADING	DEHEADING	WASH	CERTIFICATION & PREMISE	PROCESSING CONTROLS	CONSUMER READY
Infectious haematopoietic necrosis virus	✓(J & S)	✓	✓	✓	✓	✓	✓	✓	✓
Infectious pancreatic necrosis virus (for juvenile salmonids only)	✓(J)	×	×	✓	×	✓	✓	×	×
Infectious salmon anaemia virus (for Atlantic salmon from ISAV infected and/or HKS-affected countries only)	×	✓	✓	✓	✓	✓	✓	✓	✓
Aeromonas salmonicida (not for wild ocean-caught Pacific salmon)	✓(J & S)	✓	✓	✓	✓	✓	✓	✓	✓
Renibacterium salmoninarum	✓(J & S)	✓	✓	✓	✓	✓	✓	✓	✓
Yersinia ruckeri (for juvenile salmonids only)	✓(J)	×	×	✓	×	✓	✓	×	×
Myxobolus cerebralis (for rainbow trout and all juvenile salmonids)	✓(J)	✓a	✓a	✓a	✓a	X	✓a	✓a	✓a

✓= risk management measure applies;

J = juveniles; S = sexually mature fish/spawners.

a risk management applies for rainbow trout only.

Part 3
Non-salmonid marine finfish

Chapter 6
Hazard identification: non-salmonid marine finfish

6.1 Method

MANY DISEASE AGENTS HAVE BEEN REPORTED in association with non-salmonid marine finfish. The Australian Quarantine and Inspection Service (AQIS) has identified the disease agents that may be present in non-viable marine finfish product on the basis of the agent being reported in a host found in marine waters and/or being reported from a wide range of marine hosts, indicating a low host-specificity.

In preparing this chapter, AQIS reviewed the scientific literature and other relevant information, including the *International Aquatic Animal Health Code* (OIE 1997a), known as the Aquatic Code, the Western Australian Fishing Industry Council report on the importation of frozen fish as bait (Jones and Gibson 1997), and the review by Humphrey (1995). This chapter includes the disease agents considered important by Humphrey (ie those agents given a 'quarantine importance' of ≥15). For this reason, Chapter 6 includes several pathogens that are not in Chapter 3. However, the information in this chapter has been cross-checked with Chapter 3 to ensure that agents included in this part of the import risk analysis (IRA) were also evaluated for quarantine significance to salmonids.

In identifying hazards that may be associated with products derived from non-salmonid marine finfish, AQIS is aware that the availability of scientific data reflects the research effort committed to the investigation of disease in relevant species. For example, most publications on the diseases of marine finfish relate to the North Sea, the Baltic and the north-west Atlantic. Thus, there would be a greater likelihood of unrecognised diseases occurring in fish from other areas (A McVicar pers. comm.).

Moreover, there is more information on disease in aquacultured species than in wild fish. Keeping fish in the artificial environment of a farm or aquarium makes it easier to detect disease, because these fish can be more closely observed and because suboptimal environmental conditions or husbandry may result in the clinical expression of otherwise unapparent infections.

This section classifies disease agents for further consideration in the IRA. The classification criteria are shown in Section 1.5 (Box 1.2) and also in Section 3.1. Table 6.1 shows the classification of disease agents of non-salmonid marine finfish.

Table 6.1
Classification of disease agents of non-salmonid marine finfish

DISEASE AGENT/PEST	1 DISEASE AGENT IS INFECTIOUS	2A AGENT OR STRAIN EXOTIC TO AUSTRALIA	2B CONTROL PROGRAM IN AUSTRALIA	3A OIE-LISTED	3B SIGNIFICANT DISEASE	FURTHER CONSIDERATION OF DISEASE AGENT IS REQUIRED
Viruses						
Aquabirnaviruses	Y	Y[a]	Y[a]	Y	Y	Y
Erythrocytic necrosis virus	Y	Y	N	N	Y	Y
Infectious haematopoietic necrosis virus	Y	Y	N	Y	Y	Y
Iridovirus of red sea bream	Y	Y	N	N	Y	Y
Lymphocystis	Y	N	N	N	Y	N
Pilchard herpes virus	Y	N	N	N	Y	N
Viral encephalopathy and retinopathy virus	Y	Y[b]	N[c]	Y	Y	Y
Viral haemorrhagic septicaemia virus	Y	Y	N	Y	Y	Y
Bacteria						
Aeromonas hydrophila	Y	N	N	N	N	N
Aeromonas salmonicida — atypical	Y	Y[b]	N[c]	N	Y	Y
Aeromonas salmonicida — typical	Y	Y	N	N	Y	Y
Citrobacter freundii	Y	N	N	N	N	N
Edwardsiella tarda	Y	N	N	N	Y	N
Epitheliocystis spp	Y	N	N	N	Y	N
Flexibacter maritimus	Y	N	N	N	Y	N
Nocardia spp	Y	N	N	N	N	N
Photobacterium damsela piscicida	Y	Y	N	N	Y	Y
Pseudomonas anguilliseptica	Y	Y	N	N	Y	Y
Renibacterium salmoninarum	Y	Y	N	Y	Y	Y
Streptococcus iniae	Y	N	N	N	Y	N
Vibrio anguillarum	Y	Y[b]	N	N	Y	N[d]
Vibrio ordalii	Y	N	N	N	Y	N
Vibrio salmonicida	Y	Y	N	N	Y	Y
Yersinia ruckeri (Hagerman strain)	Y	Y	N	N	Y	N[d]
Fungi						
Aphanomyces invadans	*Y*	N	N	N	Y	N
Exophiala spp	Y	N	N	N	N	N
Protozoa[g]						
Brooklynella hostilis	Y	Y	N	N	Y	Y
Cryptocaryon irritans	Y	N	N	N	N	N
Eimeria sardinae[e]	Y	Y	N	N	N	N
Glugea stephani[e]	Y	Y	N	N	Y	Y
Goussia gadi[e]	Y	Y	N	N	Y	Y
Henneguya spp (excluding *H. salminicola*)[e]	Y	Y	N	N	N	N
Ichthyophonus hoferi	Y	N	N	N	Y	N
Kudoa spp	Y	N[f]	N	N	N	N
Microsporidium seriolae[e]	Y	Y	N	N	Y	Y
Parvicapsula spp	Y	N[f]	N	N	N	N
Pleistophora spp	Y	N[f]	N	N	N	N
Sphaerospora spp	Y	N[f]	N	N	N	N
Trichodina spp	Y	N[f]	N	N	N	N
Trichodinella spp	Y	N[f]	N	N	N	N
Trypanoplasma spp	Y	N[f]	N	N	N	N
Trypanosoma spp	Y	N[f]	N	N	N	N
Metazoa[fg]	Y	N[f]	N	N	N	N

N = no; Y = yes

a There are no restrictions on the movement of non-viable wild salmonids from the infected area. However, non-viable farmed salmonids must be gilled and gutted.

b Some strains occur.

c No movement controls apply to non-viable fish/fish products.

d *V. anguillarum* and *Yersinia ruckeri* (Hagerman strain) were rated 'Y' for further consideration in the draft report. The rationale for changing this rating is set out in the text.

e This species has not been reported but other members of the genus have been reported in Australia.

f Numerous species have been reported but few identified at species level.

g The myxosporeans are now classified as metazoans rather than as protozoans. However, *Henneguya* spp, *Kudoa* spp, *Parvicapsula* spp and *Sphaerospora* spp are listed with the protozoa in this chapter.

6.1.1 DISEASES DUE TO INFECTION WITH VIRUSES OR BACTERIA

In a personal communication to AQIS, McVicar stated that there is increasing evidence that several 'species' of virus include substantially different organisms under the same name, and that the inadequacy of the diagnostic methods currently available prevents their distinction. Thus, infectious pancreatic necrosis (IPN) and viral haemorrhagic septicaemia (VHS) are known to include quite diverse infective agents, as indicated by their host ranges and pathogenicity. Similarly, the range of diseases associated with nodaviruses is assuming much greater significance in fish farmed in seawater. McVicar further commented that fish farmed in both warm water and cold water appear to be susceptible to infection with nodaviruses, and there may be several virus species involved.

Although scientific knowledge about many strains of viruses and other pathogens is not sufficiently well developed to provide a basis for legislative controls, AQIS acknowledges that there is evidence that different pathogens are present in foreign countries, and takes this into account in the IRA.

AQIS has carefully considered the evidence for the presence overseas of exotic strains of pathogens that occur in Australia. In the case of agents that have been reported sporadically or exceptionally in Australia and for which there are few data, it may be difficult to determine if strains reported overseas are more pathogenic and should be considered in the IRA. This is particularly the case for agents that have not been identified to species level. Many pathogenic bacteria (eg *Mycobacterium* spp, *Nocardia* spp *Edwardsiella tarda* and *Vibrio ordalii*) have been excluded from further consideration in the IRA on the basis that strains of similar or greater virulence to those that occur overseas are found in Australia. However, where there is evidence for the existence of significantly more pathogenic strains overseas, these agents have been included in the IRA for further consideration (eg *Photobacterium damsela piscicida, Aeromonas salmonicida* (atypical forms), and aquatic birnaviruses, known as aquabirnaviruses).

6.1.2 DISEASES DUE TO PROTOZOAN AND METAZOAN PATHOGENS

In a personal communication to AQIS, A McVicar stated that, in general, diseases due to parasitic infestations are not considered to present the same level of risk of being introduced into and becoming established in a new area as those caused by bacteria and viruses. Indeed, many parasites (protozoan and metazoan) have a sufficiently discontinuous distribution to be used as natural indicators of host stock history (ie there is a sufficiently strong pattern of distribution for a scientific discipline to be established around the phenomenon).

Further complicating attempts to categorise parasitic pathogens for the risk analysis, data on the different strains of parasites that occur around the world are notably deficient. For example, the relatively well-studied protistan, *Ichthyophonus hoferi*, is known to cause serious disease epizootics in wild fish on both sides of the north Atlantic, but there is still insufficient information on whether there is one species or more in different parts of the world.

For diseases due to protozoan parasites, many agents have been shown to cause significant pathology in individual fish, but there are few data on the effects on wild fish populations. Protozoan infestations can cause serious diseases and the species listed in this section for further consideration are recognised as among the most significant pathogens in this group. For most protozoans, it is unlikely that free-living stages would survive for any significant period in a dead fish (A McVicar pers. comm.).

AQIS has considered the parasitic metazoans associated with marine finfish. This is a very large group of organisms and for many species/genera there is little information on the distribution, host range and significance of infestation. There are very few records of serious disease epizootics due to metazoan infestations in wild fish. With certain exceptions (see Section 3.2.5), AQIS will not give metazoans further consideration in this IRA, for the following reasons.

Generally speaking, infestation with metazoan organisms, in the absence of additional stressors such as overcrowding, insanitary environmental conditions or intercurrent disease, is of minor significance to the vertebrate host. Some exceptions to this are cited in Section 3.2.5.

Most of the metazoa are obligatory parasites that display varying degrees of host-specificity. Many (but not all) have lifecycles that involve several host animals. Although some species have free-living stages, in general parasites would not survive beyond about 48 hours in a dead host that has been removed from the aquatic environment. Moreover, freezing the product would rapidly kill any metazoan parasites that may be present (this is an important step in treating fish for consumption in raw form that may contain metazoan parasites of public health concern).

Many metazoan parasites are sufficiently large to be seen on the fish and removed during inspection of the product. Most of the metazoans that infest the internal organs and the gastrointestinal tract would be removed from the product at the time of evisceration.

In a personal communication to AQIS, B Jones (1999) stated that many genera of metazoan parasites have been recorded in fish in Australian waters, and in most cases these species have not been defined. There is a growing literature on the taxonomic relationships of the Australian aquatic parasite fauna with the parasite fauna of neighbouring regions. These studies show that the relationships are complex and often reflect the faunal groupings of the host animals and historical migration and movement patterns. There are no mandatory controls in Australia to address endemic diseases due to metazoan parasites.

6.1.3 USE OF CONSERVATIVE JUDGMENT

Where definitive data relevant to this process of classification are lacking, AQIS makes conservative judgments based on current scientific information and the advice of experts in relevant fields.

6.2 Diseases / disease agents of non-salmonid marine finfish

6.2.1 VIRUSES

Aquabirnaviruses

Aquabirnaviruses include those members of the Family Birnaviridae that have been isolated from aquatic hosts. Aquabirnaviruses are ubiquitous in aquatic species and are commonly isolated from healthy fish. The aquabirnaviruses essentially have a global distribution, although serogroupings have been identified that are generally restricted in geographic distribution to either North America or Europe, Asia and Japan (review by Reno 1999).

Some aquabirnaviruses cause infectious pancreatic necrosis (IPN), an acute disease of juvenile salmonids; these viruses are categorised as infectious pancreatic necrosis virus (IPNV). Based on transmission studies conducted in juvenile brook trout, IPNV has been recovered from the following non-salmonid marine fish: striped bass (*Morone saxatilis*), southern flounder (*Paralichthys lethostigma*), Atlantic menhaden (*Brevoortia tyrannus*) and Japanese eel (*Anguilla japonica*) (McAllister and Owens 1995). IPNV in salmonids is widespread in Europe (including the United Kingdom), North America and Asia, and continues to be the main viral problem in both farmed Atlantic salmon smolts following transfer to seawater and in many freshwater salmon hatcheries in Norway. It has also been reported from Chile after being undetected for over 10 years (OIE 1999). Classical IPN of salmonids is not reported in Australia or New Zealand.

Classical IPN of salmonids is an acute, highly contagious disease that causes mortality in salmonid fry and fingerlings, but rarely affects yearling or older fish. Clinical disease is characterised by behavioural changes and gross external and internal histopathological lesions, but no specific pathognomonic signs exist (review by Reno 1999). Microscopically, there is focal coagulative necrosis of the acinar and islet cells of the pancreas and of the haematopoietic cells of the kidney (Wolf 1988), although

classic pancreatic necrosis may not be observed in clinically affected non-salmonid species (McAllister and Stoskopf 1993). A range of isolates has been recovered, with highly virulent strains resulting in mortalities approaching 100%. Survivors of IPNV infection develop immunity but may become covert carriers of infection for the rest of their lives (Wolf 1988).

Yellowtail ascites virus (YAV) and viral deformity virus (VDV) (of yellowtail) are significant birnavirus disease agents in cultured marine fish in Japan (Nakajima et al 1998). Eel virus European (EVE) is a pathogenic aquabirnavirus affecting eel species (review by Reno 1999). Another pathogenic aquabirnavirus has caused significant mortality in farmed juvenile halibut (*Hippoglossus hippoglossus*) in Norway (review by Biering 1997) and the United Kingdom, where it caused mortality rates greater than 70% (Rodger and Frerichs 1997). This virus has been described as IPNV, based on serological and genotypic relatedness to the Sp reference strain of IPNV (Biering et al 1997). In the absence of data confirming the virulence of the virus (isolated from halibut) for salmonids, it will be referred to as 'halibut birnavirus' (HBV) in this IRA.

Other non-salmonid marine fish species in which aquabirnaviruses have been associated with disease include turbot (*Scophthalmus maximus*), Japanese flounder (*Paralichthys olivaceus*), European sea bass (*Dicentrarchus labrax*), Senegalese sole (*Solea senegalensis*) and Atlantic cod (*Gadus morhua*) (review by Biering 1997). As the role of these viruses in causing disease is not clear, they are not evaluated individually in this IRA. It is likely that similar conclusions would apply to them as apply to the 'proven' pathogenic aquabirnaviruses further considered in this IRA.

In a personal communication, Dr M Crane advised AQIS that in 1998 an aquabirnavirus was isolated in Australia from farmed Atlantic salmon (in apparently healthy fish and in 'pinheads'), and from rainbow trout, wild flounder, cod, spiked dogfish and ling, all on the west coast of Tasmania. This virus is currently being characterised and its precise relationship to other aquabirnaviruses is not yet known. Polymerase chain reaction analysis of viral nucleic acid indicates that the virus appears to be more closely related to IPNV fr21 and N1 isolates than other birnavirus isolates available for comparison. The Australian isolate is neutralised by an antiserum raised against IPNV Ab strain and by a commercial IPNV monoclonal antibody. Further analysis is required to confirm this relationship. Experimental transmission of this virus to young salmonid species indicated that the virus is of low pathogenicity to brook trout and Atlantic salmon, and hence should not be described as IPNV (M Crane pers. comm.).

As a result of the discovery of the aquabirnavirus in Macquarie Harbour on the west coast of Tasmania, this area has been proclaimed a disease control zone. Restrictions on movement of live farmed (but not wild) salmonids from the zone and protocols for treatment of nets and processed fish have been developed. Farmed fish harvested from the infected area must be gilled and eviscerated, and gills and viscera must be buried. There are no restrictions on the movement of wild-caught non-viable fish or eviscerated non-viable farmed salmonids from the infected area.

A marine aquabirnavirus with some characteristics in common with the IPN group of viruses has been detected a number of times in healthy sea-run quinnat salmon in New Zealand. Clinical disease due to aquabirnavirus infection has never been observed in New Zealand and the virus has had no impact on salmon farming (Anderson 1996).

The World Organisation for Animal Health (Office International des Epizooties, OIE) lists IPNV as an 'other significant' disease. Disease due to IPNV or other pathogenic aquabirnaviruses has not been reported in Australia. Accordingly, IPNV, EVE, HBV, VDV and YAV will be further considered in this IRA.

Erythrocytic necrosis virus or viral erythrocytic necrosis

In the draft report (Chapter 6, posted 12 May 1999), AQIS made reference to four groups of viruses that have been associated with erythrocytic abnormalities, including necrosis. Pacific salmon anaemia virus (thought to be caused by a togavirus) is considered in Section 5.5.3. There are few data on the other virus groups and their causal association with significant disease has not been proven. Accordingly, only erythrocytic necrosis virus (ENV) is further considered in this chapter.

ENV causes viral erythrocytic necrosis (VEN), characterised by the development of erythrocytic abnormalities, in at least 17 families of marine and anadromous fish, including Atlantic cod, Atlantic and Pacific herring, and Pacific salmonids (review by Humphrey 1995).

Infection is not usually associated with high mortality (Nicholson and Reno 1981), although disease epizootics have been recorded in Pacific herring (Meyers et al 1986). In tropical marine species, VEN has been reported in wrasse and comb-toothed blennies, and is likely to affect other reef species (McAllister and Stoskopf 1993).

The disease has been reported from the United Kingdom, United States, Canada, Taiwan, Greenland and Chile, and is suspected to be present off the coast of Portugal (review by Humphrey 1995, review by Dannevig and Thorud 1999).

Infection with ENV normally causes chronic disease with no external physical signs. Histopathological findings include damage to the nuclei of infected erythrocytes, which contain DNA-positive cytoplasmic inclusions (review by Humphrey 1995). Postmortem findings ascribed to VEN include pallor of the gills and internal organs, and hyperplasia of haematopoietic tissue (Bernoth and Crane 1995, review by Dannevig and Thorud 1999).

Disease associated with ENV has not been reported in Australia. Accordingly, ENV is further considered in this IRA.

Infectious haematopoietic necrosis virus

Infection with infectious haematopoietic necrosis virus (IHNV) causes disease in wild and cultured salmonids. IHNV has also been isolated from Pacific herring (*Clupea pallasi*), shiner perch (*Cymatogaster aggregata*) and tubesnout (*Aulorhynchus flavidus*) (Kent et al 1998). White sturgeon (*Acipencer transmonatus*) are susceptible to infection under experimental conditions (LaPatra et al 1995, OIE 1997b).

IHNV has been reported from the Pacific rim of North America, continental Europe, China, Taiwan, Korea and Japan (review by Botland and Leong 1999).

In salmonid fry and fingerlings, mortality rates may be as high as 100%. Pathological effects include extensive necrosis of kidney, haematopoietic tissues, pancreas, gastrointestinal tract and the adrenal cortex. Fish that survive IHNV infection develop a protective immunity (Wolf 1988, OIE 1997b).

IHNV is listed by the OIE as a notifiable disease. It has not been reported from Australia; accordingly, IHNV is further considered in this IRA.

Iridovirus of red sea bream

Iridoviruses recorded in fish in the Asia–Pacific region cause serious disease in a number of cultured fish species. Disease associated with iridovirus infection has been recorded in Japan, Hong Kong, Taiwan, Singapore and Thailand. Red sea bream iridovirus (RSIV) is the most important fish iridovirus in western Japan, producing significant mortality (up to 70%) of juvenile red sea bream (review by Nakajima et al 1998). The characterisation and relatedness of the recorded iridoviral disease agents are under review, but preliminary evidence indicates that an iridovirus with a single origin is widespread (Miyata et al 1997). RSIV is serologically cross-reactive with other pathogenic iridoviruses, such as epizootic haematopoietic necrosis virus, sheatfish iridovirus and grouper iridovirus, but is antigenically distinct.

Disease due to RSIV (or closely related iridoviruses) has been reported in the following species of cultured marine fish: red sea bream (*Pagrus major*), crimson sea bream (*Evynnis japonica*), spotted parrotfish (*Oplegnathus punctatus*), Japanese parrotfish (*O. fasciatus*), Japanese sea bass (*Lateolabrax* spp), yellowtail (*Seriola quinqueradiata*), amberjack (*S. dumerili*), gold-striped amberjack (*S. aureovittata*), Japanese flounder (*Paralichthys olivaceus*), striped jack (*Pseudocaranx dentex*), red spotted grouper (*Epinephelus akaara*), brown spotted grouper (*E. tauvina*, *E. malabaricus*), albacore (*Thunnus thynnus)* and tiger puffer (*Takifugu rubripes*) (Miyata et al 1997, Nakajima et al 1998).

The highest prevalence of RSIV is in red sea bream. Molecular diagnostic studies conducted in Japan suggested that RSIV causes significant disease in numerous farmed marine finfish species, including

Japanese parrotfish, striped jack, sea bass, yellowtail, amberjack and albacore. The specificity of the nucleic acid diagnostic technique for RSIV is yet to be confirmed (Kurita et al 1998).

Signs of clinical infection with RSIV are usually limited to petechial haemorrhage of the gills (Nakajima et al 1998). Gross pathological changes include anaemia and enlargement of the spleen. Histologically, large, deeply-stained cells may be observed in Giemsa-stained tissue sections of spleen, heart, kidney, liver and gill.

RSIV causes significant disease in marine fish and has not been reported from Australia. Accordingly, RSIV is further considered in this IRA.

Lymphocystis

Lymphocystis is an iridoviral infection that results in massively hypertrophied cells, forming nodular, pale, wart-like masses or nodules in the epidermis of infected fish. Infection is usually considered benign.

Lymphocystis has a wide host range in both freshwater and marine species, but has not been detected in salmonids (review by Humphrey 1995). The virus has a global distribution and occurs in Australia. There are no movement controls associated with lymphocystis in Australia; accordingly, it is not further considered in this IRA.

Pilchard herpes virus

Pilchard herpes virus is a recently identified virus that has been associated with extensive mortality in pilchard (*Sardinops sagax*) populations in Australia and New Zealand (Fletcher et al 1997). The virus appears to be highly host-specific. It appears to cause gill lesions that may result in the death of some affected fish (M Crane pers. comm.). There is no evidence for the presence of different strains of pilchard herpes virus overseas.

The virus has been reported only in Australia and New Zealand. In the absence of a disease outbreak, Australia applies no mandatory controls in relation to pilchard herpes virus. Accordingly, this agent is not further considered in this IRA.

Viral encephalopathy and retinopathy virus

Infection with viral encephalopathy and retinopathy virus (VERV) causes epizootic disease characterised by high mortality rates in larvae and juvenile fish of several marine species. This disease is known as viral encephalopathy and retinopathy (VER) or viral nervous necrosis (VNN).

Fish found in fresh and marine waters may be affected, and include turbot (*Scophthalmus maximus*), striped jack (*Pseudocaranx dentex*), redspotted grouper (*Epinephelus akaara*), halibut (*Hippoglossus hippoglossus*), European sea bass (*Dicentrarchus labrax*) and barramundi (*Lates calcarifer*). Although there is no nodavirus conclusively associated with salmonids so far, it is suspected that cardiac myopathy syndrome in Norwegian salmon is linked with this group of viruses (A McVicar pers.comm). VERV has been detected in Australia in association with mass mortality in hatchery-raised larval and juvenile barramundi (Munday et al 1992). The virus has been reported from Europe (OIE 1997b). Molecular studies of isolates recovered from a range of non-salmonid marine hosts, including an Australian isolate from barramundi, indicate that strains occurring overseas can be differentiated from the virus present in Australia. Comparative data on pathogenicity of the various strains are lacking, although the host range appears to differ across strains (Nishizawa et al 1997).

There are some interstate movement restrictions on live barramundi (and other species) with respect to VERV, but there are no restrictions on the movement of non-viable barramundi or other species for this pathogen.

The OIE lists VER as an 'other significant' disease (OIE 1997a). In the draft report (Chapter 6, posted on the Internet on 12 May 1999), AQIS proposed that VERV not be further considered in the IRA. However, in light of the fact that exotic strains of VERV reported overseas could affect a different host range from or could be more pathogenic than the strain of virus found in Australia, AQIS decided that VERV would be further considered in this IRA.

Viral haemorrhagic septicaemia virus

Viral haemorrhagic septicaemia (VHS) is caused by infection with a rhabdovirus, viral haemorrhagic septicaemia virus (VHSV), which causes acute to chronic systemic disease, characterised by haemorrhage and necrosis of the viscera.

Rainbow trout (*Oncorhynchus mykiss*) are the salmonid species most susceptible to disease and may be infected at any stage in the life cycle (Meier et al 1994). VHSV has also been isolated from non-salmonid marine finfish, including Atlantic herring (*Clupea harengus harengus*) (Dixon et al 1997), Atlantic cod (*Gadus morhua*) (Jensen et al 1979), turbot (*Scophthalmus maximus*) (Ross et al 1994), Pacific herring (*C. harengus pallasi*) (Meyers et al 1994), Pacific cod (*Gadus macrocephalus*) (Meyers et al 1992) and pilchards (*Sardinops sagax*) (OIE 1999). Clinical disease due to VHSV infection in non-salmonid marine fish has been reported in farmed turbot in Scotland and Germany (Ross et al 1994, Schlotfeldt et al 1991), in wild-caught Pacific herring in the United States (Marty et al 1998) and in Atlantic cod and haddock in the North Sea (Smail 1999). Mortality rates as high as 100% have been reported in Pacific herring under experimental conditions (Kocan et al 1997). A major outbreak of VHS that killed several thousand metric tonnes of pilchards and some other marine fish species was recently reported in Canada (OIE 1999).

A number of biotypes of VHSV have been identified by genomic analysis, which has shown that isolates from Europe and North America are genotypically heterogeneous. Isolates from non-salmonid finfish have been identified as related to, but distinct from, those found in salmonids (Oshima et al 1993).

The OIE lists VHS as a notifiable disease.

VHSV has not been recorded in Australia (review by Humphrey 1995), and causes serious disease overseas. Accordingly, this agent is further considered in this IRA.

6.2.2 BACTERIA

Aeromonas hydrophila

Aeromonas hydrophila occurs in fresh waters throughout the world, including in Australia (review by Humphrey 1995). This agent may be associated with disease, but *A. hydrophila* is generally considered to be a secondary pathogen (Austin and Austin 1993).

This agent has not been shown to be causally associated with serious disease, except when fish are stressed or environmental conditions are suboptimal. *A. hydrophila* is present in Australia; accordingly, it is not further considered in this IRA.

Aeromonas salmonicida — *'atypical' and 'typical' strains*

Infection with *Aeromonas salmonicida* causes a number of acute to chronic disease syndromes of fish, including furunculosis, goldfish ulcer disease, carp erythrodermatitis and ulcer disease of flounder. There are currently four recognised subspecies of *Aeromonas salmonicida: A. salmonicida salmonicida*, *A. salmonicida masoucida*, *A. salmonicida achromogenes* and *A. salmonicida smithia* (Whittington et al 1995). The subspecies *salmonicida* includes isolates also described as 'typical'. Other subspecies are described as 'atypical'. The terms 'typical' and 'atypical' relate to growth and biochemical characteristics of isolates in culture.

Infection with *A. salmonicida* may develop into a septicaemic condition (more commonly associated with typical isolates) or may be restricted to cutaneous ulcerative lesions (commonly associated with atypical isolates).

Infection with *A. salmonicida salmonicida* has been recorded from both salmonids and non-salmonids (Hammel 1995), not always in association with clinical disease (Bricknell et al 1996). Non-salmonid species in which infection has been detected include wrasse (*Labridae* spp) (Treasurer and Laidler 1994), turbot (*Scophthalmus maximus* (L.)) (Nougayrede et al 1990), Atlantic cod (*Gadus morhua* (L.)) and coalfish (*Pollachius viriens* (L.)) (Willumsen 1990). Infection with this bacterium causes disease epizootics and major losses in wild and cultured salmonids. It is considered one of the most serious diseases of salmonids cultured in Canada, Norway and Scotland (Husevag and Lunestad 1995).

The typical and many atypical forms of *A. salmonicida* do not occur in Australia.

The number of published reports of disease outbreaks associated with atypical strains has increased significantly during the last decade, and these strains have been isolated from an increasing number of fish species and geographical areas (Wahli et al 1992). Atypical strains have been isolated from non-salmonid species belonging to a range of marine taxa, including flatfish (order Pleuronectiformes), codfish (order Gadiformes), turbot (*S. maximus*), haddock (*Melanogrammus aeglefinus*), Pacific herring (*Clupea harengus pallasi*) and catadromous eels (family Anguillidae) (Wiklund and Dalsgaard 1998, review by Hiney and Olivier 1999).

Atypical strains of *A. salmonicida* have been associated with high cumulative mortality in sea trout in Sweden (Wichardt 1983) and Atlantic salmon in Canada (Paterson et al 1980). These strains have caused losses of 15–25% of total production in Iceland (G¸omunds¸ttir et al 1995), and in Japan atypical infection has also led to mortality in salmonids (Bruno and Poppe 1996). Atypical *A. salmonicida* is a major economic constraint to salmonid culture in Newfoundland, with mortality rates of up to 29% (Groman et al 1992).

The atypical strain of *A. salmonicida* that causes goldfish ulcer disease is usually reported from non-salmonid species, but may cause disease in salmonids under experimental conditions (Carson and Handlinger 1988; Whittington and Cullis 1988). This pathogen has been isolated from goldfish, koi carp and silver perch and it has been visualised in roach. It is endemic to some regions of Australia. Some Australian States have adopted internal quarantine measures for live fish to prevent the spread of this disease; however, there are no restrictions on the movement of non-viable fish products (Carson and Handlinger 1988). Atypical infection with a marine strain distinct to the goldfish ulcer disease isolate has also been reported in flounder in Australia (Whittington et al 1995).

Exotic atypical strains and typical *A. salmonicida* may cause significant disease. As they are not present in Australia, they are further considered in this IRA.

Citrobacter freundii

Infection with *Citrobacter freundii*, a member of the Enterobacteriaceae, has been associated with disease in rainbow trout and Atlantic salmon in Spain, the United States and Scotland. Infection has not been reported in mariculture or wild-caught non-salmonid marine species. *C. freundii* has been reported in sunfish (*Mola mola*), in aquaria (Austin and Austin 1993) and in goldfish (*Carassius auratus*) in Australia (L Owens pers. comm.).

C. freundii occurs in Australia and is not the subject of statutory controls; accordingly, it is not further considered in this IRA.

Edwardsiella tarda

Infection with *Edwardsiella tarda* may cause septicaemia and abscessation of muscle, skin, gills and internal organs. Affected marine species include catfish, eels and salmonids. Infection has also been reported in whales, waterfowl and reptiles (J Carson pers. comm.), ornamental fish imported into Australia (Humphrey et al 1986), Australian eels (Ketterer et al 1990) and diseased rainbow trout (Reddacliff et al 1996).

E. tarda occurs widely, including in the United States, Asia and Africa.

E. tarda is present in Australia and is not the subject of statutory controls; accordingly, it is not further considered in this IRA.

Epitheliocystis

The disease epitheliocystis is considered to be due to infection with a chlamydia-like organism. It is generally a benign or chronic proliferative disease, characterised by the formation of cysts in the branchial epithelia of the host. Infection may cause death, especially in young, hatchery-reared fish (review by Humphrey 1995).

Epitheliocystis has been reported in most countries of the world, including Australia (review by Humphrey 1995). Australia imposes no statutory controls in relation to this disease. Accordingly, it is not further considered in this IRA.

Flexibacter maritimus

Infection with *Flexibacter maritimus* causes saltwater columnaris disease in marine finfish. The disease causes erosion of the epithelial tissues of the mouth, fins and tail (Inglis et al 1993).

The agent has been found in marine waters throughout the world, including Australia. Australia imposes no statutory controls in relation to this disease. Accordingly, this agent is not further considered in this IRA.

Nocardia *spp* (N. asteroides, N. kampachi syn. seriolae)

The histopathological similarities between infections caused by *Nocardia* and mycobacteria give rise to some uncertainty when reviewing the scientific literature, as many reports are based on histological studies (Austin and Austin 1993, Inglis et al 1993). In marine finfish, nocardiosis typically presents clinical signs of scale loss, exophthalmia, opacity of the eyeball, skin ulceration, listlessness, and anorexia. Granulomas may be seen in the dermis, gills, muscles and internal organs.

N. asteroides has been isolated from freshwater ornamental species, salmonids and cultured yellowtail (*Seriola quinqueradiata)* (Stoskopf 1993). *N. kampachi* is an important pathogen of yellowtail in Japan but does not appear to have been recorded from other species of fish (Inglis et al 1993, Austin and Austin 1993) or other regions.

N. asteroides is reported to have a worldwide distribution and has been detected in livestock in Australia (Buddle 1985). Nocardiosis (due to *Nocardia* spp) has also been recorded in salmonids in Australia (Munday 1996).

Nocardia spp occur in Australia and there are no statutory control measures to address disease associated with these bacteria. Accordingly, *Nocardia* spp are not further considered in this IRA.

Photobacterium damsela piscicida

Infection with *Photobacterium damsela piscicida* (formerly known as *Pasteurella piscicida*) typically causes the formation of granulomatous pseudo-tubercles in the kidney and spleen, and generalised necrosis in infected hosts.

Infection has caused serious disease in cultured marine finfish, particularly in Japan, where infection of yellowtail (*Seriola quinqueradiata*) may result in losses of up to 50% on individual farms (Inglis et al 1993; review by Daly 1999). Mass mortalities in wild fish populations in the United States have been associated with this agent, and infections have also been recorded in a wide range of marine species including sea bream (*Sparus aurata*), turbot (*Scophthalmus maximus*) and sea bass (*Dicentrarchus labrax*) (review by Daly 1999).

P. damsela piscicida is a cause of significant disease. This agent has not been reported in Australia (review by Humphrey 1995). Accordingly, *P. damsela piscicida* is further considered in this IRA.

Pseudomonas anguilliseptica

Infection with *Pseudomonas anguilliseptica* may cause serious disease in pond-cultured eels (*Anguilla japonica*) in Japan (Austin and Austin 1993). This pathogen has also been isolated from other non-salmonid finfish, including Baltic herring (*Clupea harengus membras*) (L^nnstr^m et al 1994), sea bream (*Pagrus aurata*) and turbot (*Scophthalmus maximus*). The disease causes similar pathological changes in all susceptible species, predominantly petechial haemorrhage of the skin, peritoneum and liver. Liquefactive necrosis of the kidney has also been recorded (review by Daly 1999).

Disease due to infection with *P. anguilliseptica* has been reported from Japan, Scotland, Taiwan, France and Finland (review by Daly 1999).

P. anguilliseptica may cause serious disease. This agent has not been reported in Australia (review by Humphrey 1995). Accordingly, *P. anguilliseptica* is further considered in this IRA.

Renibacterium salmoninarum

Renibacterium salmoninarum is the causative agent of bacterial kidney disease (BKD). BKD is primarily a disease of salmonids, infecting both farmed and wild stocks. Fish are most commonly infected with BKD in the freshwater stage of their life cycle, with disease being carried through to the marine phase. Serological evidence of infection with *R. salmoninarum* has been

detected in Japan in greenling (*Hexagrammos otakii*) and flathead (*Platycephalus indicus*) (Sakai and Kobayashi 1992), and in Canada in healthy Pacific herring (*Clupea harengus pallasi*) and moribund Pacific hake (*Merluccius productus*) (Kent et al 1998). In each report, the infected fish had been caught close to salmonid net pens.

Clinical signs of BKD have only been reported in salmonids, where the signs may not be evident until disease is well established. External signs typically include exophthalmos and skin lesions, which may take the form of unruptured cysts containing blood cells, necrotic tissue and large numbers of *R. salmoninarum.* In advanced cases, lesions may take the form of large shallow ulcers (Bullock and Herman 1988). Internal lesions include necrosis of the kidney and haemorrhages in the body wall and hind-gut.

BKD impairs the adaptation of juvenile fish to seawater and causes death (Bullock and Herman 1988). BKD may be transmitted vertically. It is not readily prevented or treated.

The OIE classifies BKD as an 'other significant' disease. It has not been identified in Australia and is further considered in this IRA.

Streptococcus *spp* (Streptococcus iniae)

Infection with *Streptococcus* spp may cause disease in freshwater and marine fish species. *Streptococcus iniae* is the most significant pathogen and has been associated with disease in flounder (*Paralichthys olivaceous*), sardines (*Sardinops melanostictus*), menhaden (*Brevoortia patronus*) and striped bass (*Morone saxatilis*) (Austin and Austin 1993, Eldar et al 1995).

Infection with *S. iniae* can cause septicaemic disease characterised by exophthalmia, petechial haemmorhage within the opercula, and congestion of the mouth and pectoral and caudal fins (review by Kusuda and Salati 1999).

S. iniae has been reported in barramundi (*Lates calcarifer*) in Australia (L Owens pers. comm.) and no statutory controls are imposed for this disease. Accordingly, *S. iniae* is not further considered in this IRA.

Vibrio anguillarum, V. ordalii and V. salmonicida

The term 'vibriosis' is used to describe disease caused by infection with bacteria belonging to the genus *Vibrio*. Members of the genus are ubiquitous in marine environments and include significant pathogens such as *Vibrio anguillarum, V. ordalii* and *V. salmonicida*.

V. anguillarum is the most common and widespread of the pathogenic *Vibrio* species affecting fish (Egidius 1987). It is associated with systemic infection and localised infection of skin, resulting in ulceration. Mortality rates of up to 100% have been recorded in infected salmonids (Ransom et al 1984). Sixteen different serotypes of *V. anguillarum* have been reported. Most disease outbreaks have been ascribed to serotypes 1 and 2 (Grisez and Ollevier 1995). Disease due to infection with *V. anguillarum* occurs in all major fish-rearing countries in the northern hemisphere, including the United States, Japan, Canada, Norway, Denmark and Scotland. It is associated with systemic infection and skin ulceration in a wide range of non-salmonid marine species including turbot (*S. maximus*), red sea bream (*Pagrus major*), cod (*Gadus morhua*) and winter flounder (*Pseudopleuronectes americanus*) (Austin and Austin 1993, review by Actis et al 1999).

V. anguillarum serotype 1 occurs in Australia (Carson 1990), where disease is controlled by immersion vaccination of juvenile salmonids before stocking to sea pens. There are no statutory controls for this disease in Australia; accordingly, *V. anguillarum* is not further considered in the IRA.

V. anguillarum serotype 2 has not been reported in Australia. Because strains of the agent are present in Australia and the disease is under effective management, it is expected that, if any new strain of *V. anguillarum* became established, it could be controlled with similar methods. Accordingly, *V. anguillarum* is not further considered in this IRA.

Infection with *V. ordalii* may cause a haemorrhagic septicaemia in salmonids similar to, but less severe than, the disease caused by infection with *V. anguillarum* (Austin and Austin 1993). Although this pathogen is

usually isolated from salmonids, it has also caused disease in the fingerlings of cultured black rockfish (*Sebastes schlegeli*) in Japan (Wards et al 1991, Muroga et al 1986).

V. ordalii has been isolated from the water column and sediment in Tasmania (Cameron et al 1988) and it is not uncommonly isolated from diseased fish in Western Australia (B Jones pers. comm.). No statutory controls are imposed for this disease; accordingly, *V. ordalii* is not further considered in the IRA.

Infection with *V. salmonicida* may cause coldwater vibriosis or 'Hitra disease'. *V. salmonicida* is widespread in North America, Norway and Scotland (Actis et al 1999) and in Iceland and the Faroes (DPIE 1996). Infections have been recorded in salmonids and gadoids. Disease due to *V. salmonicida* is characterised by severe haemorrhage and necrosis of the internal organs (Neilsen and Dalsgaard 1991, Jorgensen et al 1989).

Infection with *V. salmonicida* may cause serious disease. This disease has not been recorded in Australia. Accordingly, *V. salmonicida* is further considered in the IRA.

Yersinia ruckeri *(Hagerman strain)*

Infection with *Yersinia ruckeri* may cause a systemic disease known as enteric redmouth in salmonids, the severity of which varies with the biotype of pathogen and the age and species of salmonid host. At least five serotypes of *Y. ruckeri* have been identified. The three most virulent serotypes may be grouped into type 1 (Inglis et al 1993, Austin and Austin 1993), also referred to as the 'Hagerman strain'. The other serotypes are considered to be relatively avirulent.

Although enteric redmouth occurs most commonly in salmonid species under intensive culture, it has also been isolated from a number of non-salmonid marine species including turbot (*Scophthalmus maximus*), European eel (*Anguilla anguilla*) and sole (*Solea solea*) (review by Horne and Barnes 1999).

Two clonal types of *Y. ruckeri* occur in Australia, one of which shares characteristics with isolates from Europe and the United States, and one which appears to be unique to Australia. Neither of the Australian isolates is classified as type 1 *Y. ruckeri*.

Infection with type 1 *Y. ruckeri* has not been reported in non-salmonid marine fish (B Munday pers. comm.). Accordingly, this disease agent is not further considered in this part of the IRA.

6.2.3 FUNGI

Aphanomyces invadans

Infection with *Aphanomyces invadans* is currently considered to cause the disease known as epizootic ulcerative syndrome, a serious disease of wild and farmed fish. Over 100 freshwater and several brackish water species are susceptible to infection (OIE 1997b). Epizootic ulcerative syndrome is considered to be the same disease as red-spot disease, which affects various fish species in Australia (Lilley et al 1997, Callinan et al 1995).

Epizootic ulcerative syndrome is listed by the OIE as an 'other significant' disease (OIE 1997a). *A. invadans* is present in Australia and no statutory control measures are in place. Accordingly, this agent is not further considered in this IRA.

Exophiala *spp*

Infection with *Exophiala* spp has been associated with granulomatous inflammation (similar to that caused by mycobacteria) in marine species in aquaria, including trigger fish, (*Xanthichthys ringens)* and Atlantic cod (*Gadus morhua)* (review by Humphrey 1995). *Exophiala* spp are not considered to cause significant disease in marine finfish (Sindermann 1990).

E. pisciphila has been recorded as the cause of cranial mycosis in Atlantic salmon in Australia (review by Humphrey 1995). It has also been isolated from whiting in South Australia (R Reuter pers comm). Australia imposes no statutory controls for *Exophiala* spp. Accordingly, this agent is not further considered in this IRA.

6.2.4 PROTOZOA

Brooklynella hostilis

Brooklynella hostilis is a protozoan parasite that infests the gills of marine finfish. It primarily affects cultured fish

and causes disease characterised by epithelial necrosis, haemorrhage, respiratory dysfunction and death. *B. hostilis* was reported as a cause of disease in cultured red sea bream (*Sparus aurata*) (Diamant 1998).

A member of the genus *Brooklynella* has been reported in Australia (review by Humphrey 1995). This agent has not been defined to the species level, but as this isolation was from a healthy fish it may be assumed that more pathogenic strains of *Brooklynella* spp found overseas are not present in Australia. Accordingly, *B. hostilis* is further considered in this IRA.

Cryptocaryon irritans

Infestation with *Cryptocaryon irritans* causes marine white-spot, which may lead to serious disease epizootics in aquarium and farmed fish (review by Humphrey 1995). This agent is the marine counterpart to *Ichthyophthirius multifiliis*. Disease is characterised by darkening of the body, opacity of the eye, excessive mucus production, respiratory distress, and lethargy and death (Beck et al 1996).

In Australia, *C. irritans* has been recorded from more than 13 species of fish (review by Humphrey 1995, Diggles and Lester 1996) and there are no statutory movement controls in place. Accordingly, *C. irritans* is not further considered in this IRA.

Eimeria sardinae

Eimeria sardinae is a coccidian parasite that infests the reproductive organs of clupeid fish, including Atlantic herring (*Clupea harengus*), European sardine (*Sardina pilchardus*), round sardine (*Sardinella aurita*) and Madeiran sardine (*Sardinella maderensis*).

Infestation reportedly occurs at a high prevalence in the seminiferous tubules of clupeids, fluctuating from a peak in March to a low in June to September (Lom and Dykova 1992).

Heavy infestations of the testes are associated with proliferation of epithelial and connective tissue and the replacement of the testicular tissue, leading to reduced production of sperm (Sindermann 1990, Lom and Dykova 1992). The infection appears to be ubiquitous in clupeid populations in the northern hemisphere, with a reported prevalence of 11–100% (Draoui et al 1995).

Infestation may impair reproductive function but does not appear to be associated with significant mortality or disease (Lom and Dykova 1992). Accordingly, although *E. sardinae* does not occur in Australia, it is not further considered in this IRA.

Glugea stephani

Glugea species may cause significant disease in cultured and wild marine finfish stocks (Noga 1996, Sindermann 1990). Infection with *G. stephani* has been reported in 11 species of flatfish in Europe and the North Atlantic (Sindermann 1990, Lom and Dykova 1992).

G. stephani produces tissue cysts (xenomas) in the intestine and other organs of flatfish, and mortalities have been associated with impairment of intestinal function. This disease has also been associated with immunosuppression (Lom and Dykova 1992).

G. atherinae has been reported in marine fish (atherinids) in Tasmania (X Su pers. comm.) and several *Glugea*-like species have been reported as incidental findings in pilchards and galaxids in Australia (Langdon 1992). However, *G. stephani* has not been reported in Australia (review by Humphrey 1995, X Su pers. comm.).

G. stephani may cause significant disease. This agent has not been reported in Australia; accordingly, *G. stephani* is further considered in this IRA.

Goussia gadi

Goussia gadi infests the swim-bladder of gadoid fish (eg haddock). Heavy infections may result in death of the host due to dysfunction of the swim-bladder (Sindermann 1990, Lom and Dykova 1992).

Infection has been reported in gadoid fish from the North and Baltic seas and the North Atlantic Ocean.

More than 69 species of *Goussia* have been described, of which six have been reported in Australia (review by Humphrey 1995). *G. auxidis* has a wide distribution and has been identified in liver, spleen and kidney tissue of pelagic fish in the South Pacific Ocean (Jones 1990). It is not considered to cause serious disease. Although not reported, *G. auxidis* is considered likely to be present in Australian waters (Jones and Gibson 1997).

G. gadi may cause serious disease in gadoid fish. This agent has not been reported in Australia. Accordingly *G. gadi* is further considered in this IRA.

Henneguya

A number of species in the genus *Henneguya* are reported as pathogens of non-salmonid marine fish. Infection by *H. ocellata* has been reported in red drum (*Sciaenops ocellata*) in Florida (Sindermann 1990), and *H. lagodon* has been reported in the pinfish (*Lagodon rhomboides*) by Hall and Iversen (1967). Infestation with *Henneguya* spp such as *H. vitiensis, H. otolithi* and *H. sebasta* may cause pericardial adhesions and formation of abscesses in marine finfish (Sindermann 1990).

Infestation with *H. salminicola* may cause serious disease in Pacific salmon (see Section 3.2.3).

Henneguya spp (unspeciated) have been detected in numerous Australian fish, including barramundi (*Lates calcarifer*) and yellowfin bream (*Acanthopagrus australis*) (Reddacliff 1985, Roubal 1994, review by Humphrey 1995).

As *H. salminicola* has not been reported in Australia, it is further considered in this IRA (see Section 5.5.8).

Several unspeciated *Henneguya* spp have been reported in Australia and there are no statutory control measures in place for these disease agents. Accordingly, *Henneguya* spp of non-salmonid marine fish are not further considered in this IRA.

Ichthyophonus hoferi

Most of the literature on diseases of marine fish refers to *Ichthyophonus hoferi* as a fungal disease agent, although it has recently been reclassified as a protozoan (A McVicar pers. comm.).

Infection with *I. hoferi* is typically associated with the formation of lesions in the highly vascularised organs such as the heart, liver and spleen (Noga 1996). This pathogen may infect several species of marine finfish. Major epizootics of disease have been reported in herring (Mellergaard and Spangaard 1997).

I. hoferi has been isolated from fish in Australia (review by Humphrey 1995), where there are no statutory controls for this disease. Accordingly, this disease agent is not further considered in this IRA.

Kudoa *spp*

The genus *Kudoa* contains numerous species of pathogenic and economic importance. Infection with *K. thyrsites* does not usually cause mortality, but reduces the market value of infected fish due to postmortem myoliquefaction (Cheung 1993). *K. clupeidae* infections in clupeids may cause significant mortalities in young fish (Lom and Dykova 1992). *K. paniformis* causes serious pathological changes in the musculature of whiting (*Merluccius productus*) from the Pacific coast of North America, while *K. cerebralis* parasitises the connective tissue surrounding the central nervous system of adult striped bass (*Morone saxatilis*) in the Chesapeake Bay region of the United States (Sindermann 1990). *K. amamiensis* has been reported as a serious pathogen of cultured yellowtail (*Seriola quinqueradiata*) in Japan. Infection is thought to arise from fish being fed on reef fish of the genera *Abudefduf*, *Chromis* and *Chrysiptera* (Egusa and Nakajima 1978).

Numerous species of *Kudoa* have been identified in Australia including *K. thyrsites*, *K. nova* and *K. clupeidae*. *Kudoa* spp have been reported in cerebral infections of barramundi fry (*Lates calcarifer*), kingfish (*Seriola grandis*) and yellowtail kingfish (*Seriola lalandi*), showing pathological changes in muscle tissues (review by Humphrey 1995). Infestation of southern bluefin tuna (*Thunnus maccoyii*) has also been reported (Munday 1996).

Kudoa spp occur in several finfish species in Australia and there are no statutory controls for these agents. Accordingly, *Kudoa* spp are not further considered in this IRA.

Microsporidium *spp*

The genus *Microsporidium* includes about 15 species and was formed to contain identifiable species of unknown genus. Infestation with *Microsporidium* spp causes formation of tissue cysts (xenomas) and necrosis of the musculature. *M. cotti* causes the formation of xenomas in the testes of long-spined bullhead (*Taurulus bubalis)* in Europe (Lom and Dykova 1992).

M. seriolae causes myoliquefaction in cultured yellowtail in Japan, referred to as 'Beko' disease (Sindermann 1990, review by Dykong 1995). 'Beko' has also been described in aquacultured red sea bream (*Pagrus major)* (Egusa et al 1988).

The status of *Microsporidium* spp in Australia is uncertain (review by Humphrey 1995). *M. hepaticum sp nov* has been reported in healthy flounder in Tasmania (X Su pers. comm.).

M. seriolae is the cause of serious disease in cultured non-salmonid marine fish in Japan and has not been reported in Australia. *Microsporidium* spp are present in Australia and no statutory control measures are in place for these agents. On this basis, AQIS had proposed that *Microsporidium* spp would not be further considered in this IRA. However, there is considerable interest in Australia in the mariculture of *Seriola* spp and, since the establishment of *M. seriolae* in Australia would be an impediment to the development of such an industry, *M. seriolae* is further considered in this IRA.

Parvicapsula

Infestation with a *Parvicapsula* sp found in the kidney of pen-reared marine coho salmon and other salmonid species was reported to cause severe disease in the early 1980s on the northern Pacific coast of the United States (Hoffman 1984). However, the pathogenic significance of this parasite was unclear, as concurrent infection with *R. salmoninarum* and *Vibrio* spp was frequently reported. Johnstone (1984) also reported infection with *Parvicapsula* spp in chinook, Atlantic and masou salmon and cutthroat trout.

More recently, Kent et al (1992) described *P. minibicornis* from the kidney of wild sockeye salmon in British Columbia. No lesions were associated with this infection. It is difficult to ascertain whether *P. minibicornis* is the same species that was recorded in the studies by Hoffman (1984) and Johnstone (1984), because descriptions of the latter were taken from preserved material. Nevertheless, these descriptions suggest differences between the two *Parvicapsula* species. Furthermore, there are no records of disease caused by *Parvicapsula* spp beyond those in 1984. Kent et al (1994) do not consider *Parvicapsula* spp to be an important pathogen in salmonids in British Columbia. *Parvicapsula* spp described from non-salmonid marine species include *P. renalis* from the kidney of red drum (*Sciaenops ocellatus)* (Landsberg 1993) and *P. hoffmani* from the intestinal musculature of large-scaled mullet (*Liza macrolepis)* (Padma and Kalavati 1993).

Infestation with *Parvicapsula* spp has been reported from two species of marine finfish in Australia. Like the *Parvicapsula* spp discussed above, the Australian species were not speciated.

There is little evidence for a causal association of *Parvicapsula* spp with significant clinical disease. *Parvicapsula* spp occur in Australia and there are no statutory controls for these agents. Accordingly, they are not further considered in this IRA.

Pleistophora (Plistophora) *spp*

Infestation with *Pleistophora* spp has been associated with disease epizootics and high mortality rates. Pleistophorans are not host-specific. Three *Pleistophora* spp are considered to cause serious pathological changes in the muscle tissue of infested marine finfish (Sindermann 1990).

Infestation of ocean pout (*Macrozoarces americanus*) by a pleistophoran resulted in the curtailment of a developing fishery for that species in North America (Sindermann 1990). *P. hippoglossoideos* forms parasitic cysts in the musculature of common flatfish (*Drepanopsetta hippoglossoides, Solea solea* and *Hippoglossoides platessoides*), while infection with *P. ehrenbaumi* is common in wolf-fish (*Anarhichas lupus*) and spotted wolf-fish (*A. minor*) in the North Sea. *P. gadi* infestation has been associated with the formation of tumours in the body musculature of infested gadoids (Sindermann 1990).

Pleistophora spp are reported commonly in Australia (Jones and Gibson 1997) but the pathogenic significance of these agents is unclear. *P. sciaena* has been associated with the formation of ovarian cysts in Brisbane River perch (*Sciaena australis*) (review by Humphrey 1995). Several unidentified microsporidian parasites have been reported in muscle cysts in clupeids (Langdon et al 1992).

Pleistophora spp occur in Australia and there are no statutory controls for these agents. Accordingly, they are not further considered in this IRA.

Sphaerospora *spp*

Many species of *Sphaerospora* have been reported to infest fish, generally occurring in the urinary tract of freshwater and marine species, or infesting the gills and skin (Fioravanti et al 1994).

S. epinephali causes epithelial hyperplasia and severe renal disease in grouper (*Epinephalus malabaricus)* (Supamattaya et al 1991, 1993).

Proliferative kidney disease (PKD) is an economically significant disease of salmonids, thought to be caused by an unidentified species of *Sphaerospora*. Kokanee salmon, chinook salmon and other salmonids from British Columbia and the United States have been observed with natural infections of PKD associated with *Sphaerospora oncorhynchi*-like spores (Kent et al 1993, 1995). *Sphaerospora* parasites have been recorded in sticklebacks (*Gasterosteus aculeatus*) in direct contact with salmonids with PKD (Hedrick et al 1988).
S. testicularis has been reported to infect the seminiferous tubules of cultured sea bass (*Dicentrarchus labrax*), greatly reducing male reproductive performance (review by Lom and Dykova 1995).

S. aldrichettae, *S. mayi* and several unspeciated *Sphaerospora* have been recorded in Australia (review by Humphrey 1995) and there are no statutory controls for these agents. There is no evidence that non-salmonid marine fish are associated with the spread of PKD of salmonids. Accordingly, *Sphaerospora* spp other than as discussed in Section 4.2.13 (PKX) will not be further considered in this IRA.

Trichodina *spp*

Most *Trichodina* spp are opportunistic pathogens that are capable of rapid proliferation and infestation of the gills of fish exposed to poor water quality or variations in water temperature (Langdon 1990). *T. jadranica* has been detected on gills of red mullet (*Mullus barbatus*) and other marine fish and causes disease in cultured eels. *T. murmanica* is commonly reported on the skin of cod (*Gadus morhua*), coastal gadoid and perciform fish in the North Atlantic (review by Lom 1999).

There is little information on the pathogenic significance of marine *Trichodina* spp in mariculture. Few marine trichodinids have been speciated, in comparison with those described from freshwater fish, in part because identification techniques are more difficult to apply in marine specimens (review by Lom 1999).

It is considered that the pathogenic potential of *Trichodina* spp that may be exotic to Australia is not sufficient to warrant further assessment of these agents in this IRA.

Trichodinella *spp*

Trichodinella spp are common parasites of freshwater and marine fish and are usually considered to be opportunistic pathogens. There are approximately 10 known species, of which *T. epizootica* is considered the most pathogenic. In his review, Humphrey (1995) considered that several of the species reported in Australia are probably synonymous with *T. epizootica*.

It is considered that the pathogenic potential of *Trichodinella* spp that may be exotic to Australia is not sufficient to warrant further assessment of these agents in this IRA.

Trypanoplasma bullocki

Of the *Trypanoplasma* spp, *T. bullocki* is the most important pathogen of marine finfish. *T. bullocki* is a haematozoic parasite widely distributed in at least 13 finfish species on the Atlantic coast of North America and in the Gulf of Mexico. Infection is most common in flatfish. An ectoparasitic phase has been demonstrated in the mucus on the surface of the host (review by Woo and Poynton 1995). *T. bullocki* infestation has been associated with mortality in yearling flatfish. Pathological changes include anaemia, splenomegaly and severe ascites (Sindermann 1990). The host normally develops an acquired immunity so that the level of parasitaemia is markedly reduced in older fish (Lom and Dykova 1992).

Trypanoplasma species found in Australia include *T. parmae* in New South Wales, an unidentified *Trypanoplasma* associated with anaemia in goldfish

(review by Humphrey 1995) and *Trypanoplasma* spp detected in unapparent infections of eels (Munday 1996).

It is considered that the pathogenic potential of *Trypanoplasma* spp that may be exotic to Australia is not sufficient to warrant further assessment of these agents in this IRA.

Trypanosoma *spp*

Piscine trypanosomes are transmitted by blood-sucking vectors. In most cases, the host recovers and is refractory to further infection (Lom and Dykova 1992). In the marine environment, infection with *T. murmanensis* is common in the Atlantic cod (*Gadus morhua*) and may cause anaemia, emaciation and lethargy; natural infections have also been recorded in Pleuronectiformes and Perciformes (review by Woo and Poynton 1995). *T. murmanensis* has caused mortality in experimentally infected juvenile cod and flounder, while pathogenicity was reduced in older fish (Lom and Dykova 1992). Most species of *Trypanosoma* spp are not known to cause significant disease, although anaemia may occur.

Eight species of piscine trypanosomes and one undefined trypanosome have been recorded in Australia, associated with subclinical infections in eels and other native Australian fish (review by Humphrey 1995).

It is considered that the pathogenic potential of *Trypanosoma* spp that may be exotic to Australia is not sufficient to warrant further assessment of these agents in this IRA.

Chapter 7
Risk assessment: non-salmonid marine finfish

7.1 Methods

IN CHAPTER 6, THE AUSTRALIAN QUARANTINE AND Inspection Service (AQIS) identified the disease agents that would be subject to further consideration in the risk analysis, based on defined criteria. The criteria include the absence of the agent from Australia and features of the disease agent, including its ability to cause serious disease and its status according to the Office International des Epizooties (OIE, or World Organisation for Animal Health).

7.1.1 PRIORITY RANKING OF DISEASES

As a next step, AQIS identified the disease agents to be considered with higher priority, based on the probability of the disease becoming established in Australia, the consequences that would arise from such establishment and the assessment of disease agents in the Humphrey review (1995) (see Section 1.5.2). Disease agents for consideration with high priority were placed in group 1 and those for consideration with lower priority were placed in group 2. The priority rating of each pathogen is set out in Table 7.1.

This chapter covers all disease agents in group 1. Chapter 8 contains an assessment of disease agents in group 2 (see Section 8.5).

7.1.2 RISK ASSESSMENT

The risk assessment covers:

- *Release assessment* — the probability that the agent will enter Australia as a consequence of the importation of whole, round, non-salmonid marine finfish.[1]
- *Exposure assessment* — if the disease agent entered Australia in whole, round, non-salmonid marine finfish, the probability of susceptible fish being exposed to a dose sufficient to cause infection.
- *Probability of disease establishment* — combining the release and exposure assessment.

1 Most product of non-salmonid marine finfish imported into Australia is highly processed (eg consumer ready). However, a significant demand exists for the importation of whole, round product (Food Factotum 1999). To ensure consistency in the risk assessment process, non-salmonid marine finfish are assessed from the starting point of whole, round product.

- *Consequence assessment* — the consequences of the disease agent becoming established in Australia.

Each of the above assessments is defined and described in qualitative terms in Section 1.5.3.

Table 7.1
Non-salmonid marine finfish disease agents — priority in IRA

DISEASE AGENT	PROBABILITY OF ESTAB.	IMPACT OF ESTAB.	HUMPHREY SCORE[a]	PRIORITY	COMMENT
Viruses					
Aquabirnaviruses (IPNV, VDV, YAV, EVE, HBV)[b]	++/+++	++/+++	IPNV 26 EVE 20 YAV 19 VDV NA	1	
Erythrocytic necrosis virus	+	+	24	2	Reason for score 24 is not clear. ENV does not characteristically cause high morbidity/mortality overseas. It occurs in many countries, but there is no evidence of active international spread.
Infectious haematopoietic necrosis virus	+/++	++/+++	27	1	
Viral encephalopathy and retinopathy virus	+	+/++	15	2	Strain(s) of this virus occur in Australia. The probability and impact of the establishment of new strains would be expected to be low.
Viral haemorrhagic septicaemia virus	+++	+++	26	1	
Iridovirus of red sea bream	++	+	NA	1	Causes economic loss and significant pathology in snapper and other cultured marine fish in Japan.
Bacteria[c]					
Aeromonas salmonicida — atypical	++	++	28	1	
Aeromonas salmonicida — typical	+	+++	28	1	
Photobacterium damsela piscicida	+/++	++	20	1	Disease may have serious impact if it became established.
Pseudomonas anguilliseptica	++	+/++	18	2	Humphrey score <21
Renibacterium salmoninarum	+	++/+++	29	1	
Vibrio salmonicida	+	+/++	19	2	Probability of establishment and impact of disease would be expected to be low.
Protozoa[d]					
Brooklynella hostilis	++	+	22	1	
Glugea stephani	+/++	+	20	2	Members of this genus occur in Australia. The probability and impact of the establishment of new species would be expected to be low.
Goussia gadi	+/++	+	21	2	Members of this genus occur in Australia. The probability and impact of the establishment of new species would be expected to be low.
Microsporidium seriolae[e]	+/++	++	21 (*Microsporidium* spp)	1	

a Disease score given by Humphrey (1995); NA = not scored.

b Infectious pancreatic necrosis virus (IPNV); viral deformity virus (VDV); yellowhead ascites virus (YAV); eel virus European (EVE); and halibut birnavirus (HBV).

c *Vibrio anguillarum* was eliminated at the hazard identification stage, because strains of the agent are present in Australia and the disease is under effective management. If any new strain of *V. anguillarum* were to become established, it is expected that it could be controlled with similar methods. For *Yersinia ruckeri*, the Hagerman strain is the only strain being considered. As this strain does not infect marine finfish it has been eliminated at the hazard identification stage.

d *Sphaerospora* spp/PKX/PKD was eliminated at the hazard identification stage because there is not strong evidence to link PKX/PKD to *Sphaerospora* spp that occur in non-salmonid marine finfish. *Sphaerospora* spp that occur in non-salmonid marine finfish are not considered to cause significant disease.

e In the hazard identification, this agent was identified as *Microsporidium* spp.

7.1.3 UNRESTRICTED RISK ESTIMATE

The combined probability and consequences of disease establishment represent the unrestricted risk assessment (ie the risk if no management measures are applied). As presented in the risk evaluation matrix in Section 1.5.3, the unrestricted risk estimate either exceeds or meets the 'appropriate level of protection (ALOP). Risk management measures would be required (in the former case) or would not be justified (in the latter case).

The conclusions are summarised at the end of the assessment for each disease agent.

7.2 Risk assessments for high priority diseases

7.2.1 AQUABIRNAVIRUSES

Aquabirnaviruses are those members of the Family Birnaviridae that have been isolated from aquatic hosts. Aquabirnaviruses are ubiquitous in aquatic species and are commonly isolated from healthy fish. Some aquabirnaviruses cause an acute disease of juvenile salmonids known as infectious pancreatic necrosis (IPN); these viruses are categorised as infectious pancreatic necrosis virus (IPNV). IPNV is recognised as the most significant pathogen in the aquabirnavirus group. In this analysis, the quarantine risk associated with IPNV has been assessed separately (see Section 7.2.2) from that associated with other pathogenic aquabirnaviruses and the term 'pathogenic aquabirnavirus' does not include IPNV.

Yellowtail ascites virus (YAV) and viral deformity virus (of yellowtail) (VDV) are significant aquabirnavirus disease agents in cultured marine fish in Japan (review by Nakajima et al 1998). Eel virus European (EVE) is a pathogenic aquabirnavirus affecting eel species (review by Reno 1999). Another pathogenic aquabirnavirus has caused significant mortality in farmed juvenile halibut (*Hippoglossus hippoglossus*) in Norway (review by Biering 1997) and the United Kingdom (mortality >90%) (Rodger and Frerichs 1997). This virus has been described as IPNV based on serological and genotypic relatedness to the Sp reference strain of IPNV (Biering et al 1997).

In the absence of confirmatory data on virulence of the virus isolated from halibut for salmonids, it will be referred to as halibut birnavirus (HBV) in this IRA.

Non-salmonid marine fish species in which pathogenic aquabirnaviruses have been associated with disease are identified in Table 7.2. As seen in that table, apart from YAV, VDV, EVE and HBV, aquabirnaviruses have also been associated with disease in farmed turbot (*Scophthalmus maximus*) and Japanese flounder (*Paralichthys olivaceus*) (review by Biering 1997). Furthermore, there have been reports on aquabirnaviruses in association with disease in European sea bass (*Dicentrarchus labrax*), Senegalese sole (*Solea senegalensis*) and Atlantic cod (*Gadus morhua*) (review by Biering 1997). The role of these viruses in the causation of disease is not clear. These viruses will not be evaluated individually in this IRA; however, it is likely that similar conclusions would apply to them as apply to the 'proven' pathogenic aquabirnaviruses considered in this IRA.

In a personal communication, Dr M Crane advised AQIS that in 1998 an aquabirnavirus was isolated in Australia from farmed Atlantic salmon (apparently healthy fish and 'pinheads'), rainbow trout, wild flounder, cod, spiked dogfish and ling on the west-coast of Tasmania. This virus is currently being characterised and its precise relationship to other aquabirnaviruses is not yet known. PCR analysis of viral nucleic acid indicates that the virus appears to be more closely related to IPNV fr21 and N1 isolates than other birnavirus isolates available for comparison. The Australian isolate is neutralised by an antiserum raised against IPNV Ab strain and by a commercial IPNV monoclonal antibody. Further analysis is required to confirm this relationship. Experimental transmission of this virus to young salmonid species indicated that the virus is of low pathogenicity to brook trout and Atlantic salmon and hence should not be described as IPNV (M Crane pers. comm.).

There is little information on many of the other aquabirnaviruses reported overseas. In this IRA, the most significant representatives of the aquabirnavirus group are considered, and it is recognised that similar principles would apply to other members of the group.

Table 7.2

Disease effects caused by, or associated with, aquabirnavirus infections in non-salmonid marine fish (modified from Nakajima et al 1998 and Reno 1999)

SPECIES	SYNDROME	VIRUS ACRONYM	MORTALITY RATE	GROSS PATHOLOGY / SIGNS
Turbot *Scophthalmus maximus*	haemorrhagic syndrome	na[a]	natural 6–25% experimental >50%	muscle haemorrhage, anaemic gills and liver.
Yellowtail *Seriola quinqueradiata*	yellowtail ascites	YAV	80–90%	ascites, catarrh, haemorrhage of liver, stomach and pyloric caeca; pale spleen, kidney, gills.
Yellowtail *S. quinqueradiata*	viral deformity	VDV	high (unspecified)	congestion of liver and brain, oedema, anaemia of the kidney and spleen.
Eel *Anguilla anguilla*, *A. japonica*	eel nephritis (eel virus European)	EVE	50–75%	reddening of anal fin, abdomen and gills; ascites, kidney enlargement.
Japanese flounder *Paralichthys olivaceus*	Japanese flounder ascites	JFAV[a]	5–60%	ascites, cranial haemorrhage.
Halibut *Hippoglossus hippoglossus*		HBV[b]	natural high (unspecified) experimental up to 100%	necrosis of pancreas, intestine, liver and kidney.

a The role of these viruses in the causation of disease is not clear. These viruses will not be evaluated individually in this IRA; however, it is likely that similar conclusions would apply to them as apply to the 'proven' pathogenic aquabirnaviruses considered in this IRA.

b Halibut birnavirus (HBV) is the term adopted in this IRA to describe this virus.

Release assessment

Geographic distribution

YAV and VDV have been reported only from Japan. Molecular analysis of YAV isolates indicates that these viruses belong to a new genogroup within the aquabirnaviruses; a number of strains of each virus have been identified (Hosono et al 1996). EVE has been recorded in diseased eels (*Anguilla anguilla*, *A. japonica*) in Japan and Taiwan; clinically inapparent infection with a closely related virus has been recorded in eels in the United Kingdom (review by Humphrey 1995; Reno 1999). HBV has been reported in Norway and the United Kingdom (review by Biering 1997; Rodger and Frerichs 1997).

Host range and prevalence

The following table summarises the known host range and geographical distribution of EVE, YAV, VDV and HBV.

Table 7.3
Host range and geographic distribution of EVE, HBV[a], YAV and VDV (after Nakajima and Sorimachi 1994; Biering 1997; Nakajima et al 1998; Reno 1999)

DISEASE AGENT	HOST SPECIES	REGION
EVE	European eel (*Anguilla anguilla*) Japanese eel (*Anguilla japonica*)	Japan, Taiwan
YAV	three-line grunt (*Parapristipoma trilineatum*) yellowtail (*Seriola quinqueradiata*) gold-striped amberjack (*S. aureovittata*)[b] greater amberjack (*S. dumerilii*) file fish (*Stephanolepis cirrhifer*)	Japan
VDV[c]	yellowtail (*Seriola quinqueradiata*)	Japan
HBV[a]	halibut (*Hippoglossus hippoglossus*)	Norway, United Kingdom

a Halibut birnavirus (HBV) is the term adopted in this IRA to describe this virus.

b *syn. S. lalandi.*

c VDV is closely related to YAV and could be cross-infective to hosts of YAV.

YAV has been isolated from yellowtail (*Seriola quinqueradiata*), gold-striped amberjack (*S. aureovittata*), greater amberjack (*S. dumerilii*) and threeline grunt (*Parapristipoma trilineatum*) (Nakajima and Sorimachi 1994). It has also been isolated from wild-caught file fish (*Stephanolepis cirrhifer*) (Nakajima et al 1998). YAV was detected in 15% of wild yellowtail fingerlings; nearly half of the infected fingerlings developed disease when they were transferred to the laboratory for culture (Reno 1999).

Infection with VDV has only been reported in yellowtail but, based on its relatedness to YAV, VDV is likely to be infective in other species.

EVE infects the European eel (*Anguilla anguilla*) and the Japanese eel (*A. japonica*). EVE is closely related to IPNV (Ab serotype) but has been shown to be non-pathogenic for rainbow trout (Wolf 1988; Sano et al 1992). Clinical disease mainly occurs in winter, when water temperatures are low (review by Reno 1999).

Infection with HBV has been reported only in larvae and fry of halibut.

Clinical disease due to EVE, HBV, VDV and YAV has been reported only in cultured fish stocks although YAV has been recovered from wild-caught fish (review by Reno 1999). Data on the prevalence of clinical disease and

the carrier status in farmed fish were not found in the literature.

Detection and organs affected

The pathogenic aquabirnaviruses are virulent primarily for juvenile fish. Gross pathology in juvenile yellowtail infected with YAV includes pallor of the gills, liver haemorrhage and severe ascites. Histopathology is characterised by extensive necrosis of the pancreatic acinar cells and hepatic parenchyma. Pathology observed in juvenile yellowtail infected with VDV includes pallor of the kidneys and spleen with congestion of the liver and some brain tissues (review by Nakajima et al 1998).

Juvenile halibut naturally infected with HBV exhibited darkening of the skin and pallor of the gills and liver. Histopathology revealed multifocal necrosis of pancreatic acinar tissue, necrosis of the epithelium of the gastrointestinal tract and focal necrosis of the haematopoietic tissue of the kidney and spleen (Rodger and Frerichs 1997). Necrosis of the pancreatic tissues did not occur in halibut experimentally infected with HBV (Biering 1997).

Congestion of the anal fin and abdominal skin occurs in some eels clinically infected with EVE. Internally, renal hypertrophy and ascites are present. Histopathology is characterised by glomerulonephritis; focal necrosis of the liver and extensive necrosis of the spleen may occur in some affected fish (Wolf 1988).

The visceral organs (particularly the kidney) and gonadal products are typically the main source of virus in birnavirus-infected fish (Wolf 1988). YAV has frequently been detected in the eggs and ovarian fluid of infected yellowtail broodstock after gonadotropic hormone treatment (Nakajima et al 1998).

An asymptomatic carrier state has been identified in Japanese eel (EVE) and yellowtail (YAV) and may occur in other fish species (review by Reno 1999).

Detection of virus in host tissues requires isolation and identification in cell culture. Direct nucleic acid probe techniques have been used to identify EVE and YAV but cannot differentiate between infective and non-infective virus (Hedrick et al 1983, Hosono et al 1996, review by Reno 1999). Genotypic differences between YAV and VDV are being investigated (review by Nakajima et al 1998).

Key findings

Aquabirnaviruses are found in a wide range of non-salmonid marine fish species, however, the presence of pathogenic non-IPNV aquabirnaviruses (eg EVE, HBV, VDV and YAV) has been confirmed in a limited number of species only.

Clinical infections with pathogenic birnaviruses are usually restricted to juvenile, farmed fish. The importation of farmed finfish would present a greater risk than that associated with wild-caught fish.

Pathological changes in diseased fish are most prominent in internal organs. Externally detectable pathology (eg congestion of fins/skin, pale gills) is generally present in clinically affected fish. Such fish would be detected and rejected in the course of inspection for human consumption.

Farmed yellowtail, eel and halibut are higher quality fish and are normally imported for human consumption as inspected, eviscerated carcases or as further processed product. Evisceration would reduce the viral titre in infected fish. It is unlikely that the species recorded as being susceptible to pathogenic aquabirnaviruses would be imported for use as bait or fish feed.

Covertly infected fish would not be visibly abnormal and would not be detected at inspection. In such fish, most pathogens would be in the visceral organs.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature. There is little information on the epizootiology of aquabirnaviruses other than IPNV.

Transmission

- IPNV has a direct life cycle. It may be transmitted horizontally via ingestion and across the gills and vertically, via sperm. There is no reason to assume that non-IPNV aquabirnaviruses would differ in lifecycle and means of transmission.

- Carrier fish may shed IPNV intermittently for a prolonged period, providing an enhanced opportunity for the spread of infection.
- IPNV is a relatively robust virus and would be expected to survive for considerable periods in the environment. IPNV can survive for several years at –70°C and for several months at 4°C. It is highly resistant to low pH and can survive for 22 hours at 50°C. In municipal tap water, IPNV survived for 7 months at 10°C. At chlorine concentrations of 0.2 mg/mL, IPNV was inactivated in 10 minutes in soft water; in hard water at a concentration of 0.7 mg/mL inactivation occurred in two minutes. 90 mg/L ozone inactivated IPNV in 10 minutes in hard water and 30 seconds in soft water.

AQIS considered further information, summarised below.

Transmission studies demonstrated that EVE is not cross-infective for rainbow trout (review by Wolf 1988).

For IPNV, titres in five asymptomatically infected, adult, brook trout ranged from $10^{6.7}$ $TCID_{50}$ (median tissue culture infective dose) per gram of kidney to $10^{0.3}$ $TCID_{50}$/g of muscle. This information indicates that viral titres in muscle tissue are likely to be several orders of magnitude (possibly 10^3 to 10^6) lower than in kidney tissue. IPNV has also been detected in the leucocytic fraction of blood and in the ovarian fluid (mainly in association with cells) of carrier brook trout. IPNV may be isolated from viscera of asymptomatic carriers (Reno 1999).

Vertical transmission is thought to be important in the spread of YAV because virus is readily isolated from gonadal products of mature fish. Horizontal transmission is also suspected but is yet to be proven experimentally (Nakajima et al 1998). Information is lacking on the mode of transmission of EVE, HBV and VDV. It is expected that transmission may occur horizontally and vertically, as with IPNV in salmonids.

Challenge studies in Norway demonstrated that the halibut birnavirus was highly pathogenic (100% mortality) for yolk-sac larvae exposed to a high waterborne viral titre (10^7 $TCID_{50}$/mL) but no significant effect was observed when larvae were exposed to a low (10^3 $TCID_{50}$/mL) or moderate (10^5 $TCID_{50}$/mL) viral titre (Biering and Berg 1996).

Data on the minimum infective dose for other pathogenic birnaviruses are not reported in the literature.

The isolation of a 'non-IPNV' aquabirnavirus in Tasmania indicates that natural pathways exist in Australia for the transfer and establishment of pathogens in the aquabirnavirus family.

Yellowtail kingfish (also known as gold-striped amberjack) (*Seriola lalandi syn. aureovittata*), samson fish (*S. hippos*) and greater amberjack (*S. dumerili*) in Australia would be expected to be susceptible to infection with YAV and VDV. Definitive information on the susceptibility of eel species in Australia to EVE is lacking but the long-finned eel (*Anguilla reinhardti*) and the short-finned eel (*A. australis*) would be expected to be susceptible. Atlantic halibut (*H. hippoglossus*) is a species and genus not present in Australia. The infectivity of HBV for flounder species present in Australia (eg greenback flounder and longsnout flounder) in the same family as halibut (Family Pleuronectidae) is unknown.

Agent stability

As for IPNV, it is expected that carrier fish may shed non-IPNV aquabirnaviruses intermittently for a prolonged period and these viruses would be expected to survive well in the environment, providing an enhanced opportunity for the spread of infection.

YAV and VDV have a physicochemical stability similar to IPNV, that is, they are stable at a pH range of 3–11, resistant to ether and chloroform, and stable at 56°C for 30 minutes (Nakajima et al 1998). It can be assumed that EVE and HBV would have similar stability characteristics. Based on IPNV stability parameters, the pathogenic birnaviruses would be expected to be resistant to freezing, chilling and heating and to persist well in marine, brackish and freshwater environments.

Key findings

In infected fish, visceral tissues and gonadal products would be the main source of virus and aquabirnaviruses would be expected to persist in infected tissue under environmental conditions in Australia.

Vertical transmission is likely to be the primary mode of transmission of aquabirnaviruses but, based on information on IPNV (in salmonids and non-salmonids), horizontal transmission may also occur.

There are likely to be susceptible hosts in Australia for YAV, VDV and EVE. It is less likely that there are susceptible hosts for HBV in Australia. EVE is not cross-infective for rainbow trout. Data on the cross-infectivity of HBV, YAV and VDV for salmonids are lacking.

For susceptible fish to become infected with pathogenic aquabirnaviruses, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficient period of time. Infection would need to be transmitted from the index case of infection to other susceptible hosts for the disease to establish in the population. Given the information on IPNV, it would be expected that pathogenic aquabirnaviruses would spread between fish readily under conditions in the Australian aquatic environment.

Repeated high level exposure of susceptible fish to a significant number of pathogenic aquabirnaviruses (for example, from regular discharge of untreated effluent from a fish processing plant) could result in the establishment of infection. However, sporadic or isolated entries of pathogenic aquabirnaviruses into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen.

Consequence assessment

Effects on commercially significant finfish species

Aquabirnaviruses can have significant impact on the health of cultured fish, however IPNV has by far the most serious effects of the pathogenic aquabirnaviruses (Nakajima and Sorimachi 1994, Reno 1999).

Based on available information, the establishment of a pathogenic aquabirnavirus would primarily affect juvenile fish of susceptible species maintained at high population density (ie farmed fish). There is little information on how these diseases might affect wild fish but such impact is not likely to be significant.

If a pathogenic aquabirnavirus were to become established in wild marine finfish it would not be amenable to control/eradication. Wild fish would provide a reservoir of infection for farmed finfish of susceptible species.

Yellowtail kingfish (also known as gold-striped amberjack), samson fish and greater amberjack in Australia would be expected to be susceptible to infection with YAV and VDV. They are economically significant in commercial and recreational fisheries in Australia. Considerable interest exists in the development of mariculture of yellowtail kingfish in Australia, for which the presence of YAV and VDV would be an impediment. Mortality rates of 80–90% of cultured juvenile yellowtail due to YAV have occurred in some farms in Japan.

The long-finned eel and the short-finned eel would be expected to be susceptible to EVE. The extent of intensive aquaculture of eels in Australia is insignificant. Most eel production (annual value of $A4–6 million) in Australia is based on wild-caught stocks that are grown out to marketable size. The establishment of EVE would not be expected to have a significant impact on wild eel stocks, but may become apparent once fish are confined.

The significance of establishment of HBV is uncertain since there are no members of the genus *Hippoglossus* in Australia. Members of the Family Pleuronectidae (to which the genus belongs) are, however, present in Australia and there is considerable interest in the culture of some species (eg greenback flounder).

Taking into account these factors, AQIS considers the establishment of pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV) may have a significant effect locally or regionally, but not at a national level.

Ecological and environmental effects

Based on the scientific literature, infections with pathogenic aquabirnaviruses are of little pathogenic or economic significance in wild finfish overseas. There is little evidence to suggest that the establishment of pathogenic aquabirnaviruses would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of non-salmonid marine finfish

For the unrestricted importation for human consumption of whole, round, non-salmonid, marine fish of susceptible species,[2] the probability of the establishment of pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV) would be low. For juvenile fish of susceptible species, the probability would also be low. For whole, round, non-salmonid marine fish of susceptible species imported for use as bait or as fish feed the probability would be moderate. The consequences of establishment would be of low to moderate significance.

Thus, for pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV), the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish of susceptible species does not meet Australia's ALOP and the implementation of risk management measures is warranted.

For the unrestricted importation of whole, round, non-salmonid marine fish of other species, the probability of establishment of pathogenic aquabirnaviruses would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of pathogenic aquabirnaviruses establishing in Australia, the risk meets Australia's ALOP. Therefore, the implementation of specific risk management measures is not warranted.

A summary of the risk assessment is shown in Box 7.1. Appropriate risk management measures are discussed in Chapter 8.

Box 7.1
Risk assessment — aquabirnaviruses

RELEASE ASSESSMENT (R)

The probability of the pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV) entering Australia as a consequence of the unrestricted importation for human consumption, bait or fish feed of whole, round, non-salmonid marine fish of susceptible species (*Anguilla* spp for EVE; *Hippoglossus* spp for HBV; *Seriola* spp, *Stephanolepis* spp and *Parapristipoma* spp for VDV and YAV) would be low.

Because the pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV) are primarily clinically expressed in juveniles, there is a greater probability of a significant viral titre in juvenile fish of the susceptible species. The probability associated with the unrestricted importation of juvenile fish of susceptible species would be moderate.

The probability of pathogenic aquabirnaviruses entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine finfish of other species would be negligible.

EXPOSURE ASSESSMENT (E)

If pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV) entered Australia in whole, round, non-salmonid marine fish for human consumption, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low.

If pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV) entered Australia in whole, round, non-salmonid marine fish for use as bait or fish feed, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be moderate.

2 These conclusions apply to: *Anguilla* spp for EVE; *Hippoglossus* spp for HBV; *Seriola* spp, *Stephanolepis* spp and *Parapristipoma* spp for VDV and YAV.

Box 7.1 (continued)
Risk assessment — aquabirnaviruses

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV) becoming established in Australia as a consequence of the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of susceptible species would be low (L). For juvenile fish, the probability would be higher but still low (L).

For whole, round, non-salmonid marine fish of susceptible species imported for use as bait or as fish feed the probability would be moderate (M).

The probability of pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV) becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of other species would be negligible (N).

CONSEQUENCE ASSESSMENT

The consequences of the establishment of pathogenic aquabirnavirus (eg EVE, HBV, VDV or YAV) in Australia would be low (L) to moderate (M).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of pathogenic aquabirnavirus (eg EVE, HBV, VDV or YAV) would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID MARINE FINFISH

For susceptible species (*Anguilla *spp,* Seriola *spp,* Stephanolepis *spp,* Parapristipoma *spp and* Hippoglossus *spp), including juveniles

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L (for fish imported for human consumption) to M (for fish imported for use as bait or fish feed)
- significance of consequences = L–M
- Importation risk for pathogenic aquabirnaviruses = unacceptable ('no' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish of susceptible species does not meet Australia's ALOP; and
- risk management measures are warranted.

For other species

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because the probability of disease establishment is negligible
- importation risk for pathogenic aquabirnaviruses = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish of other species meets Australia's ALOP; and
- risk management measures are not warranted.

7.2.2 INFECTIOUS PANCREATIC NECROSIS VIRUS (INFECTIOUS PANCREATIC NECROSIS)

In this IRA, infectious pancreatic necrosis (IPN) describes the acute disease of juvenile salmonids caused by infection with an aquabirnavirus. The various strains of virus that cause IPN — referred to as infectious pancreatic necrosis virus (IPNV) — differ in virulence and serological characteristics. IPN is listed by the OIE as an 'other significant disease', and is included in List III of the European Union Directive 93/54/EEC.

The OIE Code (1997a) provides the following international recommendation for countries with an official control policy for IPN:

'When importing live fish of a susceptible species or their gametes or eggs or dead uneviscerated fish, the Competent Authority of the importing country with an official control policy for infectious pancreatic necrosis may wish to require the presentation of an international aquatic animal health certificate issued by the Competent Authority in the exporting country, attesting that the aquaculture establishment, zone or country of origin has been regularly subjected to appropriate tests for infectious pancreatic necrosis with negative results.'

Hill and Way (1995) reviewed the serological classification of aquabirnaviruses, many of which are serologically related to reference strains (Ab, Sp and VR299) of IPNV. Some of these viruses were isolated from non-salmonid fish and can be called IPNV as they produce IPN in salmonid fry. There is no evidence that many of the aquabirnaviruses that are serologically related to IPNV are pathogenic in salmonids; they should not therefore be described as IPNV (Hill and Way 1995).

In reviewing the scientific literature on aquabirnaviruses, Reno (1999) noted that it was difficult to evaluate the virulence of non-salmonid isolates for salmonid fish as many different experimental protocols had been used. Water-borne infectivity trials demonstrated that IPN occurred in brook trout downstream from striped bass (*M. saxatilis*) infected with an aquabirnavirus (McAllister and McAllister 1988). Immersion challenge of juvenile brook trout with aquabirnaviruses isolated from various aquatic hosts gave clear evidence of the presence of IPNV in non-salmonid fish and other aquatic hosts (McAllister and Owens 1995).

Release assessment

Geographic distribution

IPNV has been identified in non-salmonid marine fish in the United States and in Taiwan (McAllister and Owens 1995). IPNV is known to occur in salmonid fish in continental Europe, Scandinavia, the United Kingdom, North America, South America and North Asia.

Host range and prevalence

There are few data on the prevalence of IPNV infection in non-salmonid marine hosts.

IPNV has been identified in Atlantic menhaden (*Brevoortia tyrannus*), striped bass (*Morone saxatilis*) and southern flounder (*Paralichthys lethostigma*) in the United States and Japanese eel (*Anguilla japonica*) in Taiwan. There is little information on the clinical effect of infection with IPNV in these hosts. While IPNV has been associated with disease epizootics in southern flounder (McAllister et al 1983), striped bass fry (Schutz et al 1984) and juvenile menhaden (Stephens et al 1980), its causative role has not been confirmed (Wechsler et al 1987b, Wolf 1988). The IPNV isolated from Japanese eel was apparently avirulent for that species (McAllister and Owens 1995). Virus from each species reacted with IPNV-specific polyvalent antiserum and produced IPN with acute mortality in brook trout fry following immersion challenge (five hours in water containing 10^5 plaque-forming units [PFU] per mL) (McAllister and Owens 1995).

IPNV-specific neutralising activity was demonstrated in the serum of 15 of 143 (9.6%) wild-caught, 1–3-year-old striped bass; however, virus was not isolated from tissues (Wechsler et al 1987a).

In a personal communication, Dr M Crane advised AQIS that in 1998 an aquabirnavirus was isolated in Australia from farmed Atlantic salmon (apparently healthy fish and 'pinheads'), rainbow trout, wild flounder, cod, spiked dogfish and ling on the west-coast of Tasmania. This virus is currently being characterised and its precise relationship to other aquabirnaviruses is not yet known. Polymerase chain reaction (PCR) analysis of viral nucleic acid indicates that the virus appears to be more closely related to IPNV fr21 and N1 isolates than other birnavirus isolates available for comparison. The

Australian isolate is neutralised by an antiserum raised against IPNV Ab strain and by a commercial IPNV monoclonal antibody. Further analysis is required to confirm this relationship. Experimental transmission of this virus to young salmonid species indicated that the virus is of low pathogenicity to brook trout and Atlantic salmon and hence should not be described as IPNV (M Crane pers. comm.).

Detection and organs affected

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- Clinical infection of salmonids usually occurs in fish younger than four months.
- While older salmonids (eg six months) may become infected with IPNV, they are less susceptible (or refractory) to clinical disease when compared with juvenile salmonids. Clinical disease would be rare in adult fish of market size for human consumption.
- Salmonids that survive infection with IPNV may become chronic carriers and shed virus via faeces and reproductive fluid for the rest of their lives.
- In clinically diseased salmonids, IPNV may be found in many organs, with the highest viral titres being reported in the kidney.
- Carrier salmonids of market-size may contain viral titres as high as $10^{6.7}$ $TCID_{50}$/g in the viscera, especially the kidney. Virus may also be present in the muscle tissue at a lower titre ($10^{0.3}$ $TCID_{50}$/g).
- Virus isolation in cell culture is a sensitive diagnostic method as IPNV readily grows in a number of standard cell lines. Direct methods for the detection of viral nucleic acid are also available but cannot be used to distinguish viable from non-viable virus.

AQIS considered further information on IPNV in non-salmonid marine fish, summarised below.

Wechsler et al (1987a) reported that IPNV could not be isolated from tissues of 1–3-year-old, wild-caught striped bass that were seropositive for IPNV. Experimental studies described below suggest that the titre of IPNV in the viscera of inapparently infected striped bass may range from undetectable to moderate. The establishment of the carrier status appears to depend upon exposure to IPNV via ingestion of infected fish or other species (Wechsler et al 1987b).

There is little information on the titre of IPNV in muscle tissue of infected striped bass. Based on findings in salmonids, the titre of IPNV would be expected to be several orders of magnitude lower in muscle than in visceral tissues.

Based on information on IPNV in salmonids and the preceding information for striped bass, it is likely that IPNV infection of other non-salmonid marine hosts (eel, menhaden and southern flounder) would be inapparent, particularly in fish of market size. In carrier fish, virus would mainly be present in visceral organs. Virus has been detected in the brain of infected menhaden (review by Reno 1999).

Limited laboratory testing conducted at CSIRO-AAHL in 1996 of imported pilchards for salmonid pathogens, including the OIE-listed agents, infectious haematopoietic necrosis virus, infectious pancreatic necrosis virus, epizootic haematopoietic necrosis virus, *Oncorhynchus masou* virus and viral haemorrhagic septicaemia virus, did not reveal any evidence of those viruses.

Key findings

IPNV occurs in non-salmonid marine hosts but is rarely associated with clinical disease or not causally related to disease. There are no data on the prevalence of inapparent IPNV infection of non-salmonid finfish. It has been reported that apparently healthy striped bass may be infected with IPNV, and this may also apply to Japanese eel, southern flounder or menhaden.[3]

3 Japanese eel, southern flounder and striped bass may be imported into Australia for human consumption; menhaden is commonly used for bait in North America.

Carrier fish would not be visibly abnormal and would not be detected when being inspected for human consumption.

In infected fish, most virus would be located in the visceral organs. Virus may also be present in the brain of infected menhaden.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

Transmission

- All salmonid species present in Australia would be susceptible to infection with IPNV. *Brevoortia* spp, *Paralichthys* spp and *Morone* spp do not occur in Australia; however *Anguilla* spp are present. Other non-salmonid marine finfish may be susceptible to infection.
- IPNV has a direct life cycle. It may be transmitted horizontally via ingestion and across the gills, and vertically via sperm.
- The minimum infective dose of IPNV is unknown. Infection has been initiated by feeding brook trout fry a dose of 10^3 $TCID_{50}$ virus/mL per 100 fish in a two-day period. This resulted in greater than 70% mortality; hence, the infective dose was somewhat less than this.
- The isolation of 'non-IPNV' aquabirnavirus in Tasmania indicates that natural pathways exist in Australia for the transfer and establishment of pathogens in the aquabirnavirus family.
- Carrier fish may shed IPNV intermittently for a prolonged period, providing an enhanced opportunity for the spread of infection.
- Virus can be spread mechanically via vectors (eg piscivorous birds).

AQIS considered further information on IPNV in non-salmonids, summarised below.

Wechsler et al (1987a) reported that IPNV could not be isolated from tissues of 1–3-year-old, wild-caught striped bass that were seropositive for IPNV; however, low viral titres were detected in striped bass up to 14 months after intraperitoneal inoculation. Viral titres in visceral tissues of striped bass fed IPNV-infected brook trout (that contained between 10^2 and 10^5 PFU/fish) were 10^3 PFU/g at two weeks post-exposure. Titres subsequently declined to 10^2 PFU/g at four weeks post-exposure and 10^1 PFU/g at 33 weeks post-exposure (Wechsler et al 1987b). Striped bass fry were transiently infected after waterborne infection with IPNV. Fish challenged per os or by intraperitoneal inoculation were inapparently infected (Wechsler et al 1987b). These findings suggest that the titre of IPNV in the viscera of inapparently infected striped bass may range from undetectable to moderate. The establishment of the carrier status appears to depend upon exposure to IPNV via ingestion of infected fish or other species.

IPNV was not detected in the gonadal products of adult striped bass, including fertilised eggs and fry, four months post-inoculation (Wechsler et al 1987b). Vertical transmission does not appear to be a significant route for transmission of IPNV infection in striped bass.

Infected striped bass can shed IPNV and can transmit the virus to brook trout (McAllister and McAllister 1988).

In addition to the preceding points, chronic, low-level exposure to IPNV in stream water is not considered to pose a significant risk of spread of infection to resident salmonid and non-salmonid fish (McAllister and Bebak 1997). Exposure to lower levels (about 10^2 PFU/L) of virus compared to higher levels (about 10^4 PFU/L) in hatchery effluent did not result in infection in downstream adult salmonid and non-salmonid fish, although one out of nine salmonid fingerlings was virus positive. These authors commented that laboratory immersion challenges generally use high levels of virus (about 10^5 PFU/mL with short exposure times (about five hours) to assure consistent levels of mortality. The virus levels found in stream water were about 10^7-fold lower than the levels used in immersion challenge. Therefore, even though stream fish were exposed continuously to IPNV, infection might not have occurred because virus concentration in the water was too low or because natural defence

mechanisms of the fish effectively controlled low-level virus exposure (McAllister and Bebak 1997).

Agent stability

IPNV is a relatively robust virus and would be expected to survive for considerable periods in the environment. IPNV would be expected to be resistant to freezing, chilling and heating. It is highly resistant to low pH and can survive for 22 hours at 50°C. In municipal tap water, IPNV survived for seven months at 10°C. At chlorine concentrations of 0.2 mg/mL IPNV was inactivated in 10 minutes in soft water; in hard water at a concentration of 0.7 mg/mL inactivation occurred in two minutes. IPNV was inactivated by 90mg/L ozone in 10 minutes in hard water and 30 seconds in soft water.

Key findings

Freshwater salmonids, in particular juvenile fish, are susceptible to infection with IPNV, whereas salmonids older than six months and salmonid and non-salmonid marine fish are relatively resistant to infection. Infection may be transmitted horizontally via exposure to a relatively high titre of virus in the aquatic environment. An even higher titre of virus would be required to initiate infection in adult fish or in the marine environment. Exposure to lower titres of virus would need to be maintained for a prolonged period to initiate an index case of infection.

Carrier fish of susceptible non-salmonid marine fish species would not be detected at inspection. The viscera of carrier fish would be expected to provide a significant source of virus, however, the titre of virus (if present) in eviscerated carrier fish would be expected to be extremely low. Non-salmonid marine fish of susceptible species may be imported as whole, round, product for use as bait (eg menhaden). The viscera and brain of infected menhaden may provide a significant source of virus in carrier fish.

IPNV is relatively resistant to inactivation by environmental factors (eg temperature) or chemical treatments (eg chlorine). If IPNV entered the aquatic environment, it would be expected to survive in infective form for a prolonged period.

For susceptible fish to become infected with IPNV, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts for the disease to establish in the population. IPNV would be expected to spread readily between fish, under conditions in the Australian aquatic environment.

Repeated high-level exposure of susceptible fish to a significant titre of IPNV (for example, from regular discharge of untreated effluent of a fish processing plant or via frequent and extensive use of bait or fish feed) could result in the establishment of infection. However, sporadic or isolated entries of IPNV into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen.

Consequence assessment

Effects on commercially significant finfish species

Isolates of IPNV from striped bass, menhaden and Japanese eel were highly virulent for challenged brook trout, and caused mortality rates of 87%–95% of infected fish. IPNV from southern flounder demonstrated low virulence (mean mortality rate of 23%) for brook trout after immersion challenge (McAllister and Owens 1995).

Disease due to IPNV causes substantial loss of young salmonids in northern Europe and North America, especially under conditions of stress or high temperature. In countries where infection with IPNV is endemic, mortality rates of up to 70% have been reported among fry and fingerlings up to 20 weeks of age. Under experimental conditions, highly virulent strains of IPNV have been reported to cause mortality rates higher than 90%.

IPNV has also been linked with serious pathology, morbidity and mortality problems in the immediate post-smolt period and as a consequence, IPN is considered to be one of the most economically significant diseases in salmon farming in Norway (A McVicar pers. comm.).

Failed smolt syndrome may cause substantial loss of Atlantic salmon post-smolts.

There are no effective chemotherapeutic agents or proven vaccines available for the treatment or control of IPN. Moreover, there is no evidence that maternally transferred immunity or heritable resistance protects against disease. Overseas, the disease is controlled by maintaining strict hatchery hygiene, screening broodstock and minimising stress.

It is expected that the establishment of IPNV in Australia would cause significant mortality in young rainbow trout, which would cause economic losses in the farmed rainbow trout industry and may affect the recreational trout-fishing sector. The occurrence of 'failed-smolt syndrome' could cause significant mortality in individual batches of Atlantic salmon smolts but would not be expected to cause major losses in production or profitability in the Atlantic salmon industry nationally.

Internationally, IPNV has a significant economic effect on fish farming industries. It is a notifiable disease in several countries and requires certification that broodstock are free of infection before use of eggs within countries and for export. Costs are associated with testing and the inability to use gonadal products from certain populations or individual fish (A McVicar pers. comm.).

The establishment of IPNV would affect farms exporting eyed ova, as the level of testing required to maintain access to export markets would probably increase. However, the effects of establishment of IPNV would primarily be felt at an individual premises or regional level, rather than a whole industry or national level. Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

IPNV has occasionally been recovered from non-salmonid hosts (eg menhaden, striped bass and southern flounder) during disease epizootics, but its role in causing disease in these hosts has not been established. Experimental transmission studies, including intraperitoneal inoculation with a high titre of IPNV, failed to produce disease in juvenile striped bass (*Morone saxatilis*) (Wechsler et al 1987b). IPNV isolated from Japanese eel (*Anguilla japonica*) appeared to be avirulent for that species (McAllister and Owens 1995). Generally the detection of IPNV in non-salmonid marine finfish is an incidental finding that is not associated with disease.

Ecological and environmental effects

Based on the scientific literature, infection with IPNV is of little pathogenic or economic significance in wild salmonids or non-salmonid finfish overseas. There is little evidence to suggest that the establishment of IPNV would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of non-salmonid marine finfish

For the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of susceptible species,[4] the probability of establishment of IPNV would be low to moderate. For whole, round, non-salmonid marine fish of susceptible species imported for use as bait or fish feed, the probability would be moderate. The consequences of establishment of IPNV would be of moderate to high significance.

Thus, for IPNV, the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish of susceptible species does not meet Australia's ALOP and the implementation of specific risk management measures is warranted.

For the unrestricted importation of whole, round, non-salmonid marine fish of other species, the probability of establishment of IPNV would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of IPNV in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

A summary of the risk assessment is shown in Box 7.2. Appropriate measures are discussed in Chapter 8.

4 These conclusions apply to *Anguilla* spp, *Paralichthys* spp, *Morone* spp and *Brevoortia* spp.

Box 7.2
Risk assessment — infectious pancreatic necrosis virus

RELEASE ASSESSMENT (R)

The probability of infectious pancreatic necrosis virus (IPNV) entering Australia as a consequence of the unrestricted importation for human consumption, bait or fish feed of whole, round, non-salmonid marine fish of susceptible species (*Anguilla* spp, *Paralichthys* spp, *Morone* spp and *Brevoortia* spp) would be low to moderate. The probability for whole, round, finfish of non-salmonid marine fish of other species would be negligible.

EXPOSURE ASSESSMENT (E)

If IPNV entered Australia in whole, round, non-salmonid marine fish for human consumption, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low to moderate.

If IPNV entered Australia in whole, round, non-salmonid marine fish for use as bait or fish feed, the probability would be moderate.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

For whole, round, non-salmonid marine fish of susceptible species imported for human consumption, the probability would be low (L) to moderate (M).

For whole, round, non-salmonid marine fish of susceptible species imported for use as bait or as fish feed the probability would be moderate (M).

The probability of IPNV becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of other species would be negligible (N).

CONSEQUENCE ASSESSMENT

Due primarily to effects on the farmed and recreational freshwater salmonid sectors, the consequences of the establishment of IPNV in Australia would be moderate (M) to high (H).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of IPNV would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID MARINE FINFISH

For susceptible species (*Anguilla *spp,* Paralichthys *spp,* Morone *spp and* Brevoortia *spp)

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L–M (for fish imported for human consumption) to M (for fish imported for use as bait or fish feed)
- significance of consequences = M–H
- importation risk for IPNV = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of susceptible species of whole, round, non-salmonid marine fish does not meet Australia's ALOP; and
- risk management measures are warranted.

For other species

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because the probability of disease establishment is negligible
- importation risk for IPNV = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish of other species meets Australia's ALOP; and

risk management measures are not warranted.

7.2.3 INFECTIOUS HAEMATOPOIETIC NECROSIS VIRUS (INFECTIOUS HAEMATOPOIETIC NECROSIS)

Release assessment

Infectious haematopoietic necrosis (IHN) is listed under 'diseases notifiable to the OIE' in the OIE International Aquatic Animal Health Code (1997a) and is included in List II of the European Union Directive 93/54/EEC.

The OIE Code (1997a) provides the following international standard for countries officially declared free of IHN:

> 'The Competent Authorities in countries officially declared to be IHN-free should demand that dead fish for importation from countries not free from IHN be eviscerated before transit.'

Geographic distribution

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Infectious haematopoietic necrosis virus (IHNV) was confined to the Pacific coast of North America (from California to Alaska) until the early 1970s. The disease subsequently spread[5] to Japan, Taiwan, Korea, France, Belgium, Germany, Austria and Italy. The virus also spread to eastern North America, but has since been eradicated from this region.

Host range and prevalence

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Challenge experiments on marine fish commonly found in and around net pens in British Columbia showed that tubesnout (*Aulorhynchus flavidus*), shiner perch (*Cymatogaster aggrregata*) and Pacific herring (*Clupea harengus pallasi*) are all susceptible to intraperitoneal inoculation with IHNV, with losses exceeding 50%. Herring were the species most susceptible to immersion challenge, with losses of 25% reported.

AQIS considered further information on IHNV in non-salmonid marine fish, summarised below.

Low-prevalence natural infection with IHNV has recently been reported in non-salmonid marine hosts in coastal waters of British Columbia, Canada. Infection was detected in Pacific herring, shiner perch and tubesnout at prevalences of 0.03% (1/289), 0.03% (1/318) and 2.7% (2/72), respectively. Tubesnout and shiner perch were collected from an Atlantic salmon farm experiencing an outbreak of IHN (Kent et al 1998). The infected herring was collected approximately 20km from Atlantic salmon net pens. Six weeks after the removal of salmonids from the farm affected by IHN, virus could not be detected in shiner perch or tubesnout from that locality (Kent et al 1998). IHNV has not been detected in any other non-salmonid marine species, including 66 finfish species, for which surveillance data are available (Kent et al 1998).

Other studies in Pacific and Atlantic herring have reported the finding of the related rhabdovirus, viral haemorrhagic septicaemia virus (Meyers et al 1994, Dixon et al 1997) but not IHNV.

Detection and organs affected

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

5 In a personal communication to AQIS, Dr B Hill noted that there is no firm evidence for the spread of IHNV from North America to other countries in the world. Rather, it has been assumed by some people that the first time occurrence of this disease in a country, particularly in a different continent, must have been due to importation of salmonid eyed-ova from North America. Dr Hill stated that this is an assumption that is not supported by hard scientific evidence. It is quite possible that the virus had been naturally present for a long time in some affected countries but only recently detected by chance, or due to increasing skills and facilities for fish disease investigation.

- In acutely ill fish, virus can be isolated from all major organs, though it is accepted that the virus is most abundant in the kidney, spleen, encephalon and digestive tract and virus is shed via the faeces, urine, sexual fluids and external mucus.
- Salmonids may survive infection to become chronic carriers. The location of virus in carrier fish is unknown, and virus can only be isolated immediately before, during or after spawning. In pre-spawning female salmon, viral titres were highest in the gills (10^2–10^5 PFU/g) and lower (10^0–10^4 PFU/g) in kidney, spleen, pyloric caecae, brain and eggs. In spawning fish, high titres of virus (10^6–10^9 PFU/g tissue), can be detected in most major organs including gill, kidney, spleen and pyloric caeca. In spawning females, titres as high as 10^8 PFU/mL and 10^6 PFU/mL have been reported in ovarian fluids and in mucus respectively.
- IHNV has been isolated from wild marine salmonid fish but this is an unusual event.

AQIS considered additional information on IHNV in non-salmonid marine fish, summarised below.

Where IHNV has been isolated from non-salmonid marine fish, it has, in all cases, been from apparently healthy fish (Kent et al 1998). Titres of IHNV in infected tubesnout were 2 x 10^3 PFU/g and 8.8 x 10^3 PFU/g, with a titre of 5.8 x 10^2 PFU/g in shiner perch. Virus titration was not performed in the infected Pacific herring.

Waterborne challenge of 20 five-month-old, laboratory-reared herring with $10^{6.4}$ PFU/mL IHNV caused the death of one fish, whereas a similar challenge of trout or salmon at this age would be expected to cause nearly 100% mortality. Many of the herring in this experiment were infected but had low viral titres suggesting that herring are highly resistant to natural challenge (Kocan et al 1997).

Limited laboratory testing conducted at CSIRO-AAHL in 1996 of imported pilchards for salmonid pathogens, including the OIE-listed agents, infectious haematopoietic necrosis virus, infectious pancreatic necrosis virus, epizootic haematopoietic necrosis virus, *Oncorhynchus masou* virus and viral haemorrhagic septicaemia virus, did not reveal any evidence of those viruses.

Key findings

Surveillance data indicate that the prevalence of IHNV infection in non-salmonid marine fish is rare. Infection has, in almost all cases, been reported in fish in, or associated with, salmonid net pens. Notwithstanding the extensive surveillance for IHNV in Pacific herring, there is only one report of infection (in a single fish) in this species. Moreover, the results of studies on infection via water suggest that herring are refractory to infection with IHNV.

Based on the information in the literature, AQIS considers that there would be a negligible likelihood that non-salmonid marine finfish would be infected with IHNV.

Unrestricted risk estimate for importation of non-salmonid marine finfish

The probability of IHNV entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine finfish would be negligible. Therefore, the probability of establishment of disease would also be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of IHNV in Australia, the risk meets Australia's ALOP and specific risk management measures are not warranted. A summary of the risk assessment is shown in Box 7.3.

Box 7.3
Risk assessment — infectious haematopoietic necrosis virus

RELEASE ASSESSMENT (R)

The probability of infectious haematopoietic necrosis virus (IHNV) entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid, marine finfish would be negligible.

EXPOSURE ASSESSMENT (E)

Because there is a negligible probability fo IHNV entering Australia as a result of the unrestricted importation of whole, round, non-salmonid marine finfish, the probability of susceptible fish being exposed to a dose sufficient to cause infection would also be negligible.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

Because there is a negligible probability of IHNV entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid, marine finfish, the probability of disease establishment would also be negligible (N).

CONSEQUENCE ASSESSMENT

Because there is a negligible probability of IHNV entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine finfish, the consequences of establishment were not considered further.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID, MARINE FINFISH

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because the probability of disease establishment is negligible
- importation risk for IHNV = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine finfish meets Australia's ALOP; and
- risk management measures are not warranted.

7.2.4 RED SEA BREAM IRIDOVIRUS

The OIE lists Red sea bream iridovirus disease as an 'other significant' disease. Iridoviruses recorded from fish in the Asia-Pacific region cause serious disease in a number of cultured fish species. Disease associated with iridovirus infection has been recorded in Japan, Hong Kong, Taiwan, Singapore and Thailand. Red sea bream iridovirus (RSIV) is the most important fish iridovirus in western Japan, producing significant mortality (of up to 70%) of juvenile red sea bream (review by Nakajima et al 1998). The characterisation and relatedness of the recorded iridoviral disease agents is under review but preliminary evidence indicates that an iridovirus with a single origin is widespread (Miyata et al 1997). RSIV is serologically cross-reactive with other pathogenic iridoviruses, such as epizootic haematopoietic necrosis virus (EHNV), sheatfish iridovirus (SFIV) and grouper iridovirus (GIV), but is antigenically distinct.

There is limited information on many iridoviruses reported in the scientific literature. On the basis of current information RSIV is the most significant of the iridoviruses. AQIS considers that the analysis for RSIV would apply to other iridoviruses.

Release assessment

Geographic distribution

RSIV has been reported in Japan. Closely related iridoviruses have been isolated from diseased grouper in Thailand, Hong Kong, Taiwan and Singapore (Chua et al 1993, Chou et al 1998).

Host range and prevalence

Disease due to RSIV (or closely related iridoviruses) has been reported in the following species of cultured marine fish: red sea bream (*Pagrus major*), crimson sea bream (*Evynnis japonica*), spotted parrotfish (*Oplegnathus punctatus*), Japanese parrotfish (*O. fasciatus*), Japanese sea bass (*Lateolabrax* spp), yellowtail (*Seriola quinqueradiata*), amberjack (*S. dumerili*), goldstriped amberjack (*S. aureovittata*), Japanese flounder (*Paralichthys olivaceus*), striped jack (*Pseudocaranx dentex*), red spotted grouper (*Epinephelus akaara*), brown spotted grouper (*E. tauvina*, *E. malabaricus*), albacore (*Thunnus thynnus*) and tiger puffer (*Takifugu rubripes*) (Miyata et al 1997, Nakajima et al 1998).

The highest prevalence of RSIV is in red sea bream. Molecular diagnostic studies conducted in Japan suggested that RSIV causes significant disease in numerous farmed marine finfish species including Japanese parrotfish, striped jack, sea bass, yellowtail, amberjack and albacore. The specificity of the nucleic acid diagnostic technique for RSIV is yet to be confirmed (Kurita et al 1998).

Iridoviruses that are closely related to RSIV (based on serology and molecular profiles) appear to cause 'iridoviral disease' in various other marine fish species cultured in Japan and are cross-infective for red sea bream (Nakajima et al 1995, Nakajima and Maeno 1998).

Mortality primarily occurs in juvenile red sea bream although market-size fish have also been infected (Nakajima et al 1998).

Iridoviral disease has only been reported in cultured marine finfish. Disease primarily occurs in summer months and has not been reported in winter months (Nakajima et al 1998).

Detection and organs affected

Signs of clinical infection with RSIV are usually limited to petechial haemorrhage of the gills (review by Nakajima et al 1998). Gross pathological changes include anaemia and enlargement of the spleen. Histologically, large, deeply stained cells may be observed in Giemsa-stained tissue sections of spleen, heart, kidney, liver and gill. An immunofluorescent antibody technique (IFAT) using a monoclonal antibody is commonly used for rapid field diagnosis of RSIV infection. RSIV may be grown in culture on a number of standard fish cell lines. Molecular diagnostic techniques using polymerase chain reaction (PCR) have also been applied (Miyata et al 1997, Kurita et al 1998).

There is little information on the epizootiology of RSIV (eg establishment of carrier status in fish that survive infection) under natural conditions. Virus could not be detected by PCR in the spleen of experimentally infected red sea bream three months after intraperitoneal challenge (Kurita et al 1998).

Key findings

RSIV has been reported only in cultured marine fish in Japan, although closely related iridoviruses have been reported in cultured marine fish throughout the Asia-Pacific region.

Clinical infections with iridoviruses are most common in juvenile farmed fish, although market-size fish can also be infected. Infection has not been reported in wild fish. The importation of farmed finfish would present a greater risk than that associated with wild-caught fish.

In diseased fish, pathological changes are most prominent in internal organs and include anaemia. Externally detectable pathological changes (eg petechial haemorrhage of the gills) are generally present in fish with clinical infection. Visibly abnormal fish would be detected and rejected in the course of inspection for human consumption.

Experimental evidence indicates that fish surviving infection do not become long-term (>3 months) carriers. Covertly infected fish would not be visibly abnormal and would not be detected at inspection. In such fish, the viscera would be the main source of virus.

Species susceptible to RSIV and related iridoviruses are mainly higher-quality fish that are normally imported for human consumption as inspected, eviscerated carcasses or as further processed product[6]. Evisceration would reduce the viral titre in infected fish.

Exposure assessment

Transmission

In red sea bream that had died from RSIV infection, viral particles were widely distributed throughout the body tissues. Large numbers of virus-containing cells were observed in the intestine, kidney, liver, spleen, heart and gills (Jung et al 1997).

A mortality rate of 90% occurred in red sea bream after immersion challenge (virus titre of $10^{2.7}$ $TCID_{50}$/g for 1 hour) (Jung et al 1997), demonstrating that RSIV can be spread horizontally. Vertical transmission is unlikely as RSIV infections have not been reported in hatcheries (Nakajima et al 1998).

Numerous fish species known to be susceptible to infection with RSIV are present in Australia. Some of the more important species include snapper (also known as gilthead sea bream) (*Sparus aurata*), yellowtail kingfish (*Seriola lalandi*) (also known as gold-striped amberjack) and the greater amberjack (*S. dumerili*). *Thunnus* spp present in Australia (eg southern bluefin tuna, yellowfin tuna) may also be susceptible.

Agent stability

RSIV is sensitive to acid pH (99% reduction at pH 3 for 4 hours at 4°C) but stable at pH 11. The virus is heat-sensitive (greater than 99.9% reduction at 56°C for 30 minutes) but stable under repeated freeze-thaw cycles (Nakajima and Sorimachi 1994). Iridoviruses are sensitive to organic solvents (ether and chloroform), indicating the presence of a viral envelope (Chou et al 1998).

Data on persistence in the environment are lacking. A related iridovirus, epizootic haematopoietic necrosis virus (EHNV) is present in Australia and is reported to persist well in the aquatic environment (Munday 1990).

Key findings

Disease has only been reported in cultured marine fish. Several commercially significant species (such as snapper, yellowtail kingfish, amberjack and tuna species) present in Australia would be susceptible to infection with RSIV (or closely related iridoviruses). Snapper and southern bluefin tuna are susceptible species that are cultured in Australia.

RSIV can be transmitted horizontally.

Water temperature appears to be associated with the expression and transmission of disease; disease due to infection with RSIV has only been reported in cultured marine fish in Japan in summer. The temperature of coastal Australian waters would generally be suitable for transmission of disease (except in the winter months in southern Australia), while other waters may be suitable for disease transmission throughout the year.

RSIV will survive freezing and thawing and would be expected to persist in the aquatic environment.

For susceptible fish to become infected with RSIV, or closely related iridoviruses, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficient period of time. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. RSIV or closely related iridoviruses would be expected to spread readily between fish under conditions in the Australian aquatic environment, except in water at a low temperature.

Repeated high-level exposure of susceptible fish to a significant titre of RSIV or closely related iridoviruses

6 Striped jack (silver trevally) are also imported for use as lobster bait in Australia.

(for example, from regular discharge of untreated effluent from a fish processing plant) could result in the establishment of infection. However, sporadic or isolated entries of RSIV or closely related iridoviruses into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen.

Consequence assessment

Effects on commercially significant finfish species

Iridoviruses can cause significant pathological effects on cultured fish. From available information, the establishment of pathogenic iridoviruses would primarily affect juvenile fish of susceptible species maintained at high population density (ie farmed fish). There is little information on how these pathogens affect wild fish and no evidence to suggest that they would have a significant effect on the health of wild populations.

If a pathogenic iridovirus were to become established in wild marine finfish in Australia, it would not be amenable to control/eradication. Preliminary field studies have demonstrated that an inactivated vaccine is reasonably effective in prevention of disease in farmed red sea bream but its efficacy in other susceptible species is yet to be examined (Nakajima et al 1998).

The most economically significant mariculture industries in Australia are based on Atlantic salmon, ocean trout and tuna. The establishment of iridoviruses in Australia could have an effect on farmed tuna, due to reduced stocks of young fish; however, the effect on adult fish would not be significant. RSIV or closely related iridoviruses have not been reported as a cause of disease in salmonids.

Other marine farming industries in Australia are at a relatively early stage of development. Some species (eg snapper, barramundi) that are being considered or trialled for potential use in mariculture are in the same taxa as species reported to be susceptible to iridoviruses. The establishment of iridoviruses in Australia could impede the development of mariculture of snapper or other susceptible species. Given the current stage of development of the mariculture industries based on susceptible species, the consequences of establishment would not be expected to cause significant losses at a national level; however, it could limit the prospects of developing industries.

Many of the species that could be susceptible to infection with iridoviruses are also economically significant in commercial and recreational fisheries in Australia.

Taking account of these factors, AQIS considers the establishment of pathogenic iridoviruses could have a significant effect locally or regionally, but not at a national level.

Ecological and environmental effects

No information was found in the literature on the impact of RSIV (or closely related iridoviruses) in wild marine finfish overseas. Disease has only been reported in cultured, non-salmonid marine fish and the impact of disease on wild native (eg barramundi, snapper, yellowtail, tuna) fish stocks in Australia is unknown. EHNV is a related iridovirus present in Australia that causes seasonal (in summer months) outbreaks of disease in fresh water in redfin perch, and occasionally in rainbow trout in the summer months. EHNV has not had a significant impact on wild finfish, including native species.

There is little evidence to suggest that the establishment of RSIV (or closely related iridoviruses) would have a significant effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of non-salmonid marine finfish

For the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of susceptible species[7], the probability of establishment of RSIV or closely related iridoviruses would be low, and importation

7 conclusions apply to *Epinephelus* spp (eg grouper); *Evynnis* spp (eg crimson sea bream), *Lateolabrax* spp (eg Japanese sea bass); *Oplegnathus* spp (eg Japanese parrotfish); *Pagrus* spp (eg red sea bream), *Paralichthys* spp (eg Japanese flounder); *Pseudocaranx* spp (eg striped jack); *Seriola* spp(eg yellowtail); *Takifugu* spp (eg tiger pufferfish) and *Thunnus* spp (eg albacore).

of such fish for use as bait or fish feed would also present a low probability. The consequences of establishment would be of low to moderate significance.

Thus, for RSIV or closely related iridoviruses, the risk associated with the unrestricted importation of whole, round, non-salmonid, marine fish of susceptible species for human consumption or for use as bait or fish feed does not meet Australia's ALOP and the implementation of specific risk management measures is warranted.

For the unrestricted importation of whole, round, non-salmonid marine fish of other species the probability of establishment of RSIV or closely related iridoviruses would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of RSIV or closely related iridoviruses in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

A summary of the risk assessment is shown in Box 7.4. Appropriate measures are discussed in Chapter 8.

Box 7.4
Risk assessment — red sea bream iridovirus

RELEASE ASSESSMENT (R)

The probability of red sea bream iridovirus (RSIV) or closely related iridoviruses entering Australia as a consequence of the unrestricted importation for human consumption, bait or fish feed of whole, round, non-salmonid marine fish of susceptible species (*Epinephelus* spp, eg grouper; *Evynnis* spp, eg crimson sea bream; *Lateolabrax* spp, eg Japanese sea bass; *Oplegnathus* spp, eg Japanese parrotfish; *Pagrus* spp, eg red sea bream; *Paralichthys* spp, eg Japanese flounder; *Pseudocaranx* spp, eg striped jack; *Seriola* spp, eg yellowtail; *Takifugu* spp, eg tiger pufferfish; and *Thunnus* spp, eg albacore) would be low (for wild fish) to moderate (for farmed fish).

The probability for whole, round finfish of other species would be negligible.

EXPOSURE ASSESSMENT (E)

If RSIV or closely related iridoviruses entered Australia in whole, round, non-salmonid marine fish for human consumption, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low (L).

In the case of importation for use as bait or fish feed, the probability would be moderate (M).

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of RSIV or closely related iridoviruses becoming established in Australia as a consequence of the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of susceptible species would be low (L).

For whole, round, non-salmonid marine fish of susceptible species imported for use as bait or fish feed the probability would be low (L) for wild fish to moderate (M) for farmed fish.

The probability of RSIV or closely related iridoviruses becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of other species would be negligible (N).

CONSEQUENCE ASSESSMENT

The consequences of the establishment of RSIV or closely related iridoviruses in Australia would be low (L) to moderate (M).

Box 7.4 (continued)
Risk assessment — red sea bream iridovirus

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID, MARINE FINFISH

For susceptible species (see above)

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L (for human consumption) to M (for use as bait or fish feed)
- significance of consequences = M
- importation risk for RSIV or closely related iridoviruses = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish of susceptible species does not meet Australia's ALOP; and
- risk management measures are warranted.

For other species

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because the probabality of disease establishment is negligible
- importation risk for RSIV or closely related iridoviruses = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish of other species meets Australia's ALOP; and
- risk management measures are not warranted.

7.2.5 VIRAL HAEMORRHAGIC SEPTICAEMIA VIRUS (VIRAL HAEMORRHAGIC SEPTICAEMIA)

Release assessment

The OIE lists viral haemorrhagic septicaemia (VHS) as a notifiable disease and it is included in List II of the European Union Directive 93/54/EEC.

The Aquatic Code (1997a) provides the following international standard for countries officially declared free of VHS:

> 'The Competent Authorities in countries officially declared to be VHS-free should demand that dead fish for importation from countries not free from VHS be eviscerated before transit.'

Geographic distribution

Viral haemorrhagic septicaemia virus (VHSV) has been reported in salmonid and non-salmonid fish in Europe and North America. Isolates from Europe and North America are genetically distinct (Oshima et al 1993). European strains derived from marine hosts are genetically distinct from freshwater isolates, although an isolate from diseased turbot on the Baltic Coast was closely related to virus associated with disease in rainbow trout (Oshima et al 1993, Batts et al 1993, Stone et al 1997a). It has been hypothesised that the virus reported in trout evolved from that in marine fish in European waters (Dixon 1999). VHSV has not been reported from Asia, South America or Oceania (review by Smail 1999).

Host range and prevalence

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- In Europe, disease epizootics occur primarily in farmed rainbow and brown trout in fresh water. In the United States, infection with VHSV occurs

naturally in chinook salmon, coho salmon and steelhead trout; however disease has not been reported. Clinical disease has not been recorded in wild salmonids infected with VHSV in North America.

- In salmonids, VHSV is more common in farmed fish. Based on limited testing of wild salmonid populations the prevalence of VHSV infection in wild salmonids is considered to be extremely low.
- In Europe, rainbow trout is the salmonid species most susceptible to infection and clinical infection is most common in rainbow trout reared in fresh water.
- VHSV can infect fish of all ages; however, clinical infection is more severe and the mortality rate is higher in young fish. Where infection occurs in wild Pacific salmon it is most often in sexually mature fish in fresh water.

AQIS considered further information on VHSV in non-salmonids, summarised below.

According to OIE (1997b) non-salmonid marine fish susceptible to infection with VHSV are turbot (*Scophthalmus maximus*), Pacific cod (*Gadus macrocephalus*), Pacific herring (*Clupea pallasi*), Atlantic cod (*G. morhua*), haddock (*G. aeglefinus*), rockling (*Onos mustela*) and sprat and herring (*Clupea* spp) in the Atlantic Ocean and Baltic Sea.

Natural infection has also been reported in shiner perch (*Cymatogaster aggregata*), pollock (*Pollachius virens*) and hake (*Merluccius* spp) (T Meyers pers. comm.), stickleback (*Gasterosteus aculeatus*), tubesnout (*Aulorhynchus flavidus*) (Kent et al 1998), Norway pout (*Trisopterus esmarkii*) (A McVicar pers. comm.) and pilchard (also known as Pacific sardine) (*Sardinops sagax*) (OIE 1999).

Surveillance of herring on the Pacific coast of the United States and Canada indicates that the prevalence of infection with VHSV varies widely and may be very high (eg 80%) in populations affected by a disease epizootic (Meyers and Winton 1995). In Alaska, Marty et al (1998) isolated virus from 4.7% (11/233) of Pacific herring (*C. harengus pallasi*) sampled, and found that infection was associated with myocardial mineralisation, hepatocellular necrosis, submucosal gastritis and meningoencephalitis. Further studies suggested that 10–15% of Pacific herring in Alaska were subclinically infected with VHSV and that clinical disease occurred when fish were stressed (Marty et al 1998). Recent surveillance from British Columbia (Canada) found VHSV in 17% (50/289) of Pacific herring (Kent et al 1998). Surveillance data from Alaska and Canada indicated that VHSV infection in Pacific cod was rare (Meyers and Winton 1995, Kent et al 1998). VHSV infection was reported in Atlantic and Baltic herring and Atlantic cod although prevalence was not stated (Dixon et al 1997, A. McVicar pers.comm.). There is no evidence that infection rates fluctuate seasonally. It is expected that viral titres would be higher in spawning fish (Meyers and Winton 1995).

A. Munro (cited by the Tasmanian Salmon Growers Association) reported that VHSV had been isolated in 12 marine species from more than 24 species tested from the Baltic Sea and North Sea and Atlantic Ocean, west of Scotland. The highest prevalence was in herring and sprats in the Baltic Sea off the coast of Denmark.

VHSV infection may occur exceptionally in pilchards (*Sardinops sagax*) (OIE 1999). However there is only one record of disease and this appears to have been an unusual event, related to particular environmental circumstances. There is no evidence that VHSV is endemic in pilchard populations of North America (G Traxler pers. comm.).

Limited laboratory testing conducted at CSIRO–AAHL in 1996 of imported pilchards for salmonid pathogens, including the OIE-listed agents, infectious haematopoietic necrosis virus, infectious pancreatic necrosis virus, epizootic haematopoietic necrosis virus, *Oncorhychus masou* virus and viral haemorrhagic septicaemia virus, did not reveal any evidence of those viruses.

Detection and organs affected

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- In clinically infected rainbow trout, the highest titre of virus is in the kidney and spleen. Virus is also found in milt, ovarian fluid, liver, heart and muscle. A neurological form of VHS is associated with a high titre of virus in the brain, and possibly the spinal cord.

- In salmonids, signs of clinical infection may include lethargy, darkening of the skin, exophthalmia, anaemia, haemorrhage in the eyes, skin, gills and at the base of the fins.
- Clinically infected fish would be visibly abnormal and it would be expected that such fish would be detected and rejected in the course of inspection for human consumption. Carrier fish would not be visibly abnormal and would not be detected at inspection.
- Salmonids that survive infection may become apparently healthy carriers of VHSV. The carrier state is less prevalent in fish in water at a higher temperature. Virus can be isolated from the kidney, spleen, brain and ovarian fluid of carrier fish. While the titre of virus in carrier fish is not known, it is expected that, as for other viruses, the titre would be lower and the tissue distribution would be relatively limited as compared with fish affected by clinical disease.

AQIS considered further information on VHSV in non-salmonids, summarised below.

Clinical signs of VHSV infection in Pacific herring include ulceration of the skin and localised subdermal haemorrhage of the skin and fins. Skin ulceration has been reported in Pacific and Atlantic cod infected with VHSV (Meyers et al 1994). Clinically affected turbot showed marked exophthalmus, abdominal distension and diffuse haemorrhage of the ventral surface (Ross et al 1994). Haemorrhage may be less evident in chronically infected fish (Wolf 1988) and more typical in fish concurrently infected with other pathogens (Kocan et al 1997).

Virus may be detected in host tissues by isolation in cell culture and identification by serological methods. Direct nucleic acid detection methods can be used to detect VHSV but cannot be used to distinguish between viable and non-viable virus (Smail 1999).

Virus is readily isolated from the visceral organs (kidney, spleen) of infected herring (Meyers and Winton 1995) and turbot (Ross et al 1994). High viral titres have also been found in skin lesions of clinically infected herring and cod. Virus has not been isolated from the viscera of infected Pacific cod and VHSV is considered to have a limited tissue distribution (eg in skin lesions) in that species (Meyers and Winton 1995, Kent et al 1998).

The neurological syndrome associated with high titres of VHSV in the nervous tissues of infected salmonids has not been reported in non-salmonid species.

The titre of virus in clinically infected herring may be high ($>10^6$ PFU/g), while titres in subclinically infected fish may range from undetectable to high (Kocan et al 1997).

Key findings

From available data it can be concluded that populations of Pacific herring are endemically infected with VHSV, with an expected prevalence of infection ranging from 5–20%. VHSV has been isolated from Atlantic and Baltic herring. While prevalence data are not available for these species (A. McVicar pers. comm), the prevalence of infection is likely to be similar to that in Pacific herring. VHSV infection occurs sporadically in other species (eg Pacific cod, Atlantic cod, Norway pout).

In non-salmonid marine fish, herring and sprats (*Clupea* spp) appear to be the natural reservoir hosts of VHSV. The prevalence of VHSV infection in herring varies from low to high (in populations affected by epizootic disease). The probability of entry of VHSV through the importation of *Clupea* spp is likely to be higher than that for other non-salmonid marine species in which infection with VHSV occurs sporadically.

Pilchards (*Sardinops sagax*) may be infected under exceptional circumstances but are not a normal host for VHSV.

Clinically infected non-salmonid marine fish would be visibly abnormal (Meyers and Winton 1995) and would be detected in the course of inspection/grading for human consumption. In clinically infected whole round fish, the highest titre of virus would occur in the visceral organs and skin lesions. Diseased fish (eg herring) that are harvested and processed for use as bait (ie not inspected) would not be removed from a consignment.

Herring that survive infection with VHSV may become carriers, with viral titres ranging from undetectable to high. Subclinically infected fish would not be visibly abnormal and would not be detected in the course of

inspection for human consumption. The viscera would be the main source of virus in subclinically infected fish.

Exposure assessment

Transmission

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- Rainbow and brown trout are the salmonid species present in Australia that would be most susceptible to infection with virulent strains of VHSV. Brook trout, Atlantic salmon and chinook salmon appear to be refractory to infection.
- The likelihood of VHSV mutating and becoming pathogenic for additional species is unknown, but this has been considered possible.
- It appears that water temperature is associated with the transmission and expression of disease. Disease transmission is known to occur at water temperatures of 1–12°C and is not recorded at temperatures greater than 15°C. Most Australian coastal marine waters are warmer than 15°C for a large part of the year, while the temperature of some inland waters of southern Australia is regularly less than 15°C.
- Most virus would be located in the visceral organs of infected salmonids and in the brain in neurological disease.
- Infection may be transmitted horizontally.

AQIS considered further information on VHSV in non-salmonid marine fish, summarised below.

Bath challenge of laboratory-reared (specific pathogen free) Pacific herring (age 5–13 months) with a North American strain of VHSV resulted in mortality rates ranging from 65–100% in groups of fish exposed to medium or high levels of virus ($10^{3.5}$–$10^{6.5}$ PFU/mL) for one hour. Infection was established in 6/9 groups of fish exposed to lower viral concentrations ($10^{1.5}$–$10^{2.5}$PFU/mL). A minimum infective dose in the range of $10^{1.5}$–$10^{2.0}$ PFU/mL for one hour was proposed for waterborne infection of juvenile herring (Kocan et al 1997). In this experiment, infected fish shed virus at a rate sufficient to induce infection and mortality in healthy herring, suggesting that disease could be maintained in a population under field conditions. Most visceral tissues from fish that died during the study contained more than 10^{6} PFU/g while tissues of fish that survived infection contained low/undetectable levels of virus (< $10^{2.6}$ PFU/g) at 21 days post-exposure.

In a study on the pathogenicity of marine VHSV isolates from the North and Baltic seas and Atlantic west coast of Scotland, all marine isolates were of negligible virulence for salmon and rainbow trout but several were virulent for turbot. The freshwater strains of VHS that were virulent for rainbow trout were not virulent for Atlantic salmon (via bath exposure) (A Munro, cited by TSGA).

Non-salmonid marine finfish species known to be susceptible to VHSV infection overseas, including *Clupea harengus* and members of the Family Gadidae, do not occur in Australian waters. Pilchards (*Sardinops sagax*) are susceptible to infection with VHSV only in exceptional circumstances.

Agent stability

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- Based on in vitro studies with bovine serum, VHSV can survive for several weeks in infected tissue in the aquatic environment (half-life 3–10 days at water temperature 17–22°C).
- VHSV would remain viable in frozen and chilled product although the freeze-thaw cycle would be expected to reduce the titre of virus (North American strain) by an order of magnitude. The virus is stable at pH 5–10 and labile at pH <3.

AQIS considered further information on stability of VHSV, summarised below.

In vitro studies with North American isolates of VHSV demonstrated a 200-fold reduction in titre after one hour in fresh water and a 10-fold reduction in titre in saltwater, indicating they are significantly less stable in the freshwater aquatic environment (Winton et al 1991).

Key findings

Herring and sprats (*Clupea* spp) appear to be the main marine reservoir hosts for VHSV overseas. Southern sprat (*C. bassensis*) is the only *Clupea* sp present in Australian waters. Pilchards (*Sardinops sagax*) may be infected under exceptional circumstances but are not a normal host for VHSV.

Freshwater salmonids (and possibly some non-salmonid marine finfish eg southern sprat, greenback flounder) in Australia would be susceptible to infection.

Infection with VHSV normally occurs at water temperatures below 15°C. The higher temperature of coastal waters of Australia (significantly warmer than those of the north Pacific region of North America) would be expected to reduce the probability of transmission and establishment of VHSV in marine waters.

VHSV has not been reported in Australia, despite ongoing importation of herring for use in marine waters as lobster bait and pilchards as feed for caged tuna. For example, approximately 16,354 tonnes of North Sea herring (*C. harengus*) were imported into Western Australia in the period 1989–97 (Western Australian Fishing Industry Council), and there is no evidence that this practice has resulted in any adverse disease developments. Imports of herring have primarily been from Holland. Herring exported from Holland is required to be graded fit for human consumption (B Jones pers. comm., citing Dr P van Banning, pers. comm.). Fish showing evidence of disease (ie clinically infected) would not be exported, thus reducing Australia's exposure to the virus. It may be concluded that there are factors mitigating against the introduction and establishment of the pathogen by this route. One such factor may be the relatively high inshore water temperatures (>12°C) around areas where herring and pilchards are used in large quantities (Jones and Gibson 1997, Fletcher et al 1997), given that VHSV is not normally transmitted at water temperatures greater than 15°C.

Transmission of VHSV could occur at the temperatures recorded in some coastal waters and in southern inland waters in the winter months.

VHSV isolated from marine fish could survive in fish tissues in the aquatic environment, but would not persist as well in fresh water as in seawater.

Consequence assessment

Effects on commercially significant finfish species

Based on overseas experience, the effect of the establishment of VHSV would depend on the strain and its characteristics, particularly its pathogenicity and host specificity. The most significant consequences would be expected to arise if a freshwater strain of VHSV virulent for salmonids were to become established in Australia.

The establishment in Australia of European strains of VHSV isolated from non-salmonid marine finfish, which have been shown to be of low virulence, would have little consequence for salmonids or other finfish in Australia.

The establishment of North American strains of VHSV would also be of low significance for salmonids, as these strains appear to be of low virulence for these species, although the potential for the virus to mutate and become more virulent cannot be dismissed (Meyers and Winton 1995). Inapparent natural infection with VHSV has been recorded in coho and chinook salmon in North America. Mortality rates of 0–7% were recorded in eight species of salmonids challenged by immersion with four North American isolates (Stone et al 1997b citing Winton, pers. comm.).

The North American strain of VHSV has been isolated from farmed Atlantic salmon, but not associated with any clinical disease or significant losses (G Traxler pers. comm.).

The establishment of North American strains of VHSV in Australia would be expected to have little consequence for salmonids or other finfish in Australia.

In Europe, infection with the freshwater salmonid strains of VHSV causes mortality of up to 80–100% of rainbow trout fry. Fingerlings and growers are also susceptible to VHSV and virulent strains produce mortality rates of 10–50%. Significant commercial losses (US$40 million per year) were associated with VHS in freshwater salmonids (cited in Humphrey 1995).

In the freshwater environment, husbandry measures such as de-stocking and disinfection of hatcheries, followed by re-stocking from pathogen-free sources, can be used to prevent and control VHSV infection. Surviving fish are resistant to reinfection (Wolf 1988).

Immunisation with a DNA-based vaccine has been shown to confer protective immunity to rainbow trout (Lorenzen et al 1998).

The establishment of freshwater European strains of VHSV in Australia would be expected to cause significant mortality in young rainbow and brown trout. This would cause economic losses in the farmed rainbow trout industry and may affect the recreational trout-fishing sector. Based on the low virulence of freshwater European strains of VHSV for Atlantic salmon, the establishment in Australia of these strains of VHSV would be of very low significance for the Atlantic salmon industry.

The establishment of any strain of VHSV would affect farms exporting eyed ova, as they would be required to implement testing and certification to preserve their export markets. However, the effects of establishment of VHSV would primarily be felt at an individual premises or regional level rather than a whole industry or national level.

Based on current OIE requirements, any effect on trade in product for human consumption would be limited to uneviscerated fish, which is not a significant export for the Australian salmonid industry.

There is limited information on the effect of VHSV on wild salmonid populations. The establishment of VHSV would be expected to cause some reduction in wild populations of rainbow and brown trout and to have significant effect on the recreational fishing industry locally or regionally rather than at a national level.

Ecological and environmental effects

There is no evidence to suggest that the establishment of strains of VHSV virulent to salmonids would lead to disease or mortality in native or other fish species in Australia.

Overseas, the main hosts for marine strains of VHSV are herring (*Clupea harengus*) and members of the Family Gadidae, neither of which occurs in Australia. The potential for marine finfish in Australian waters, including other members of the Family Clupeidae such as southern sprat (*Sprattus novaehollandiae*), bony bream (*Nematolosa come*), southern herring (*Harengula abbreviata*) and pilchards (*Sardinops sagax*), to become infected and provide a reservoir for VHSV is uncertain. As VHSV has a wide host range and has shown the potential to adapt to new hosts under overseas conditions, it is expected that some marine finfish in Australia would be susceptible to infection. Given that marine strains of VHSV appear to be avirulent for salmonids, and there have been no records of these strains causing disease in other freshwater species, it appears unlikely that there would be significant effects on freshwater finfish species, including native fish, in Australia.

Unrestricted risk estimate for importation of non-salmonid marine finfish

For the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of susceptible species[8] the probability of establishment of VHSV would be low, while importation of such fish for use as bait or fish feed would present a low to moderate probability. For herring and sprat (*Clupea* spp), the probability would be higher, but still moderate. The consequences of establishment would be of low to moderate significance.

Thus, for VHSV, the risk associated with the unrestricted importation of whole, round, marine fish of susceptible species for human consumption or for use as bait or fish feed does not meet Australia's ALOP and the implementation of specific risk management measures is warranted.

For the unrestricted importation of whole, round, non-salmonid marine fish of other species, the probability of establishment of VHSV would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of VHSV in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

A summary of the risk assessment is shown in Box 7.5. Appropriate risk management measurements are discussed in Chapter 8.

8 These conclusions apply to species in the Families Gadidae (eg Atlantic cod, haddock, blue whiting, pollock), Scophthalmidae (eg turbot), Gasterosteidae (eg tubesnout, three-spined stickleback), Embiotocidae (eg shiner perch), Lotidae (eg rockling), Pleuronectidae (dab, plaice), *Clupea* spp (eg herring, sprat) and *Merluccius* spp (hake).

Box 7.5
Risk assessment — viral haemorrhagic septicaemia virus

RELEASE ASSESSMENT (R)

The probability of viral haemorrhagic septicaemia virus (VHSV) entering Australia as a consequence of the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of susceptible species — that is, species in the families Gadidae (eg Atlantic cod, haddock, blue whiting, pollock); Scophthalmidae (eg turbot); Gasterosteidae (eg tubesnout, three-spined stickleback); Embiotocidae (eg shiner perch); Lotidae (eg rockling); Pleuronectidae (dab, plaice); *Clupea* spp (eg herring, sprat) and *Merluccius* spp (hake)— would be moderate. Importation for use as bait or fish feed would present a moderate to high probability. For herring and sprat, the probability would be high.

The probability of VHSV entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of other species would be negligible.

EXPOSURE ASSESSMENT (E)

If VHSV entered Australia in whole, round, non-salmonid marine fish of susceptible species for human consumption, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low.

In the case of importation for use as bait or fish feed the probability would be low to moderate.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of VHSV becoming established as a consequence of the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of susceptible species would be low (L).

Importation of such fish for use as bait or fish feed would present a low (L) to moderate (M) probability.

For herring and sprat (*Clupea* spp) the probability would be higher, but still moderate (M).

The probability of VHSV becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of other species would be negligible (N).

CONSEQUENCE ASSESSMENT

The consequences of the establishment of freshwater European strains of VHSV in Australia would be moderate (M), due primarily to effects on commercial and recreational trout stocks in Australia. The effect on the Atlantic salmon industry would not be significant. The effect on the recreational salmonid sector would be limited to the regional level.

The consequences of the establishment of marine European strains and North American strains of VHSV would be low (L), due primarily to the limited impact that these strains of VHSV would have on salmonids and other finfish species in Australia.

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of VHSV would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE ROUND NON-SALMONID MARINE FINFISH

For susceptible species (see above)

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L (fish for human consumption) to L–M (fish for use as bait or fish feed)
- significance of consequences = L–M
- importation risk for VHSV = unacceptable ('no' in Figure 1.1)

Box 7.5 (continued)
Risk assessment — viral haemorrhagic septicaemia virus

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish for human consumption or for use as bait of susceptible species does not meet Australia's ALOP; and
- risk management measures are warranted.

For other species

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because the probability of disease establishment is negligible
- importation risk for VHSV = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish of other species meets Australia's ALOP; and
- risk management measures are not warranted.

7.2.6 *AEROMONAS SALMONICIDA*, TYPICAL (FURUNCULOSIS) AND ATYPICAL STRAINS

In view of the significance of this disease, AQIS has undertaken a review of the literature (see Appendix 7) as a basis for this section of the risk analysis which also draws upon information in salmonids from previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b).

Release assessment

Geographic distribution

(a) Typical A. salmonicida

Typical *A. salmonicida* has been reported in non-salmonid marine fish in Norway (Willumsen 1990), France (Nougayrede et al 1990), Scotland (Treasurer and Laidler 1994), Denmark (Pedersen and Larsen 1996), and Spain (Real et al 1994).

(b) Atypical A. salmonicida

Atypical strains of *A. salmonicida* have been reported in non-salmonid marine fish in Denmark, Japan, Canada, UK, Iceland, Norway, France, Finland, Sweden and Australia (after Wiklund and Dalsgaard 1998).

Host range and prevalence

(a) Typical A. salmonicida

Infection with typical *A. salmonicida* has been reported in numerous non-salmonid marine fish but only in farmed fish or fish in close proximity to infected salmonids. Infection of non-salmonid marine fish with typical *A. salmonicida* is not necessarily associated with disease (Willumsen 1990). Infection has been identified in turbot (*Scophthalmus maximus*) (Nougayrede et al 1990, Toranzo and Barja 1992), goldsinny wrasse (*Ctenolabrus rupestris*), cuckoo wrasse (*Labrus bimaculatus*), rock cook (*Centrolabrus exoletus*) (Treasurer and Cox 1991), Atlantic cod (*Gadus morhua*), coalfish (*Pollachius virens*) (Willumsen 1990), gilthead sea bream (*Sparus aurata*) (Real et al 1994) and halibut (*Hippoglossus hippoglossus*) (Bergh et al 1997).

In a study of 519 wild fish (comprising 40 non-salmonid marine species, including Pacific herring) captured

around salmonid net pens and from open waters, typical *A. salmonicida* was not detected in any species (Kent et al 1998).

Clinical disease in non-salmonid marine fish due to infection with *A. salmonicida salmonicida* has only been reported in farmed fish or in species that are known to cohabit with intensively reared salmonids. Without exception, reports have been from countries where furunculosis occurs in cultured salmonids. There are no records of ongoing disease problems in non-salmonid marine fish; rather, reports are of isolated instances of disease and mortality.

In a study of wrasse (of various species) sourced from salmonid farms, Treasurer and Cox (1991) reported that 7.8% (16/204) of fish were infected with typical *A. salmonicida*. The pathogen was not detected in wild-caught wrasse (0/139) examined in this study.

Other studies have not reported prevalence, but rather the effects of infection. In a population of 1200 farmed turbot maintained in close proximity to infected salmonids, a 15% cumulative mortality occurred over one month due to infection with typical *A. salmonicida* (Toranza and Barja 1992). Another outbreak of disease in farmed turbot due to typical *A. salmonicida* resulted in mortality of approximately 25% of fish in affected tanks. Salmonids had previously been cultured at the affected locality (Pedersen and Larsen 1996). High mortality was also recorded in turbot in France, with 2.5–3% of infected fish dying daily until treatment commenced. Treatment significantly reduced the mortality rate although deaths continued to occur. Furunculosis had occurred some months earlier at the affected locality in recently introduced coho salmon (Nougayrede et al 1990).

In Spain, infection with typical *A. salmonicida* caused the death of 6–7% of cultured juvenile gilthead sea bream (*Sparus aurata*) in the first three days of a disease outbreak (Real et al 1994).

(b) Atypical A. salmonicida

Atypical strains of *A. salmonicida* have been reported in approximately 19 species of non-salmonid marine fish, primarily in cultured fish. Species in which infection has been reported include American eel (*Anguilla rostrata*), European eel (*A. anguilla*), Japanese eel (*A. japonica*), Atlantic cod (*Gadus morhua*), flounder (*Platichthys flesus*), greenback flounder (*Rhombosolea tapirina*), Pacific herring (*Clupea harengus pallasi*), plaice (*Pleuronectes platessa*), American plaice (*Hippoglossoides platessoides*), four-bearded rockling (*Enchelyopus cimbrius*), haddock (*Melanogrammus aeglefinus*), wolffish (*Anarhichas lupus*), turbot (*Scophthalmus maximus*), Schlegel's black rockfish (*Sebastes schlegeli*), Japanese flounder (*Paralichthys olivaceus*), sand-eels (*Ammodytes lancea*; *Hyperoplus lanceolatus*), shotted halibut (*Eopsetta grigorjewi*) (Nakatsugawa 1994) and goldsinny wrasse (*Ctenolabrus rupestris*) (after Wiklund and Dalsgaard 1998).

There is little information on the prevalence of infection with atypical *A. salmonicida* in non-salmonid marine fish. Disease associated with atypical strains of *A. salmonicida* has been reported as an occasional finding in cultured non-salmonid marine fish (Wiklund and Dalsgaard 1998).

In a survey of 40 wild-caught non-salmonid marine species (Kent et al 1998), the only isolation of atypical *A. salmonicida* was from the kidney of an apparently healthy lingcod (*Ophiodon elongatus*). Wiklund and Bylund (1993) studied 6890 flounder caught off the coast of Finland and reported epidermal ulceration in 5.9% of fish. Atypical *A. salmonicida* was isolated from 54% of ulcers examined (162 fish specimens). The bacterium was isolated from the visceral organs of 1.9% (3/162) of these specimens.

There are few disease conditions associated with atypical *A. salmonicida* infections in non-salmonid fish. Examples are ulcer disease of flounder (UDF), carp erythrodermatitis and goldfish ulcer disease. However in some cases, *A. salmonicida* has not been detected and other (opportunistic) pathogens have been isolated (Wiklund and Dalsgaard 1998). Such observations are not unexpected as atypical strains of *A. salmonicida* are fastidious and often difficult to isolate. Immunological and/or molecular studies may help clarify such situations.

Detection and organs affected

(a) Typical A. salmonicida

Subdermal haemorrhage at the base of the pectoral fins and multiple skin ulcers have been reported as prominent external pathology in farmed turbot clinically infected with typical *A. salmonicida* (Toranzo and Barja 1992, Pedersen and Larsen 1996). Internally, livers of affected turbot were pale with petechial haemorrhages (Toranzo and Barja 1992). In both disease outbreaks, typical *A. salmonicida* was isolated from skin ulcers and kidney tissues of affected fish. Toranzo and Barja (1992) also isolated the bacteria from spleen and liver specimens.

There are few data on the titres of *A. salmonicida* (typical) in infected non-salmonid marine fish. Given that most non-salmonid marine fish species are more resistant than salmonids to infection with typical strains of *A. salmonicida*, it is expected that bacterial titres would be lower in non-salmonid hosts than in salmonids.

(b) Atypical A. salmonicida

Fish clinically infected with atypical strains of *A. salmonicida* normally show gross epidermal lesions from which bacteria can be recovered. Data on bacterial titres in clinically infected non-salmonid marine fish are lacking. Covertly infected fish would yield lower bacterial titres than fish with clinical disease (epidermal ulceration). The bacterium has only occasionally been isolated from the viscera of clinically infected, non-salmonid marine fish; internal pathological changes are not a feature of disease due to infection with atypical strains of *A. salmonicida* (Wiklund and Dalsgaard 1998).

Key findings

In comparison with other subspecies, typical *A. salmonicida* is an unusual cause of infection or disease in non-salmonid marine fish (Real et al 1994). With the exception of turbot, non-salmonid marine fish appear to be far more resistant than salmonids to infection with typical *A. salmonicida*. Cases of disease in non-salmonid marine hosts are usually reported from fish that cohabit with infected salmonids or that are farmed in the vicinity of salmonid farms. Most reports of infection with typical *A. salmonicida* in non-salmonid marine fish are isolated cases, not associated with epizootic disease in the non-salmonid host.

While infection with atypical strains of *A. salmonicida* in non-salmonid marine fish is more frequently reported than infection with typical *A. salmonicida*, the overall prevalence appears low. In recent years, the number of reports of atypical infection in non-salmonid species has increased; some scientists consider that infection with atypical strains may become a limiting factor in aquaculture of both non-salmonids and salmonids (Wiklund and Dalsgaard 1998).

From data presented above it can be seen that the prevalence of infection with *A. salmonicida* (typical and atypical strains) is very low in farmed non-salmonid marine fish and extremely low in wild-caught non-salmonid marine fish.

Because of the pathological changes associated with infection (both typical and atypical), clinically infected, non-salmonid marine fish would be visibly abnormal and would be detected in the course of inspection of fish for human consumption.

The titre of *A. salmonicida* likely to be present in non-salmonid marine fish is not known, but would depend on several factors including the subspecies of *A. salmonicida* and the species of the host fish. Non-salmonid marine fish generally appear to be less susceptible than salmonids to infection with *A. salmonicida.* Therefore bacterial titres would be expected to be lower in non-salmonids than in salmonids.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996), the 1997 report of the New Zealand Government (Stone et al 1997b) and information in Appendix 7. These reports contain referenced reviews of relevant literature.

Transmission

- *A. salmonicida* has a direct lifecycle.
- *A. salmonicida* is transmitted horizontally, via water, contaminated equipment, food or direct contact between fish. Vertical transmission may be possible but is not thought to be epidemiologically significant.
- The minimum infective dose of *A. salmonicida* (typical) for Atlantic salmon in seawater by short

duration (1–3 days) bath exposure has been measured at 10^4 colony-forming units/mL, and for long duration immersion (three weeks) at 10^2 CFU/mL. Immersion in concentrations of 10^2 CFU/mL for periods up to one week failed to cause infection. Intragastric intubation required doses of > 10^5 CFU/fish to establish infection in Atlantic salmon.

- Environmental conditions in some parts of Australia would be suitable for transmission of infection.
- There is epidemiological evidence that infected fish may 'shed' bacteria for a substantial period of time.
- Fish with damaged skin and mucus are more susceptible to infection. In Australia, frequent treatment for amoebic gill disease increases the prevalence of skin abrasions in cultured Atlantic salmon.

Agent stability

- *A. salmonicida* (typical) is adversely affected by freezing. Freezing the flesh of infected salmon for 5–7 days at –20°C reduces the bacterial titre by 99% (data are lacking for atypical strains).
- *A. salmonicida* (typical) is reported to be stable for 28 days in kidney tissue and for 32 days in muscle tissues at 4°C. *A. salmonicida* (typical) is resistant to pH 4 at 22°C.
- *A. salmonicida* (typical) has been reported to survive in fresh water for 17 days, in brackish water for 24 days and in seawater for eight days at temperatures of 11–13°C. In sediment it may survive for up to 29 days.

AQIS considered further information on *A. salmonicida* in non-salmonid marine fish.

Cross-infection of strains between different wild or farmed host species has rarely been reported (Wiklund and Dalsgaard 1998). However, in Australia the goldfish ulcer disease organism has been shown to infect goldfish, koi carp, silver perch and probably roach and the greenback flounder. *A. salmonicida* has been isolated from Atlantic salmon and striped trumpeter in contact with clinically diseased flounder (B Munday pers. comm.).

Studies by Wiklund (1995), using an atypical strain of *A. salmonicida* isolated from flounder with skin ulcerations, showed that the dose required to cause significant mortality in fresh water fish varied considerably depending on the species of fish challenged.

For typical strains of *A. salmonicida*, an intraperitoneal dose of 2 x10^4 CFU/fish induced 50% mortality in 30g turbot. The minimum lethal dose by bath for the same fish was 10^5 CFU/mL after exposure for 12 hours. These fish could be infected with a lower dose than that required to infect rainbow trout (Perez et al 1996).

Treasurer and Laidler (1994) reported that three species of wrasse were less susceptible than Atlantic salmon to infection with a typical strain of *A. salmonicida*. Goldsinny wrasse (*Centolabrus rupestris*), cuckoo wrasse (*Labrus bimaculatus*) and rock cook (*Centolabrus exoletus*) cohabited with salmon post-smolts, in a bath containing 1 x10^5 cells/mL for 24 hours. At 9–10 days after challenge, the Atlantic salmon post-smolts began to die and *A. salmonicida* could be isolated from these fish. None of the wrasse died, and bacteriological tests for *A. salmonicida* were negative. These results were confirmed by Bricknell et al (1996), in a study with Atlantic salmon, all of which died after the administration of 10^4 cells/fish intraperitoneally. Doses ≥10^7 cells/fish were required to cause mortality in goldsinny wrasse. A dose of 10^5 CFU/mL to eggs caused a cumulative mortality of approximately 20% (compared to 10% of controls) of larval turbot. A dose of 10^6 CFU/mL to eggs caused mortality of approximately 60% (compared to 30% of controls) of larval halibut. *A. salmonicida* was not reisolated from turbot in these studies.

With the exception of typical *A. salmonicida* in turbot the minimum infective dose of *A. salmonicida* is likely to be significantly higher in non-salmonid marine fish than in salmonids.

Experimental studies indicate that atypical strains may survive in sediment in brackish waters in excess of 60 days. Survival is significantly reduced (<14 days) in the absence of sediment (Wiklund 1995).

Atypical strains have a slow growth habit and may readily be overgrown in lesions by opportunistic pathogens (Wiklund and Dalsgaard 1998).

Key findings

All salmonids farmed in Australia would be susceptible to infection with typical and some atypical strains of *A. salmonicida*. While non-salmonid freshwater and marine finfish may be susceptible to infection with typical and some atypical strains of *A. salmonicida*, the available evidence indicates that non-salmonid species would be more resistant to infection than trout. The one exception reported is turbot, a non-salmonid species that has been shown experimentally to be more susceptible (lower infectious doses) than rainbow trout for infection with typical *A. salmonicida*. Non-salmonid finfish in Australia would be more likely to become infected with atypical strains than with typical *A. salmonicida*, should these pathogens enter Australia.

Infection may be transmitted horizontally, via exposure to a significant titre of the pathogen in the aquatic environment. A higher titre of typical *A. salmonicida* would generally be required to initiate infection in non-salmonid fish than in salmonid fish. Exposure to a low titre of the pathogen would need to be maintained for a prolonged period for infection to result.

Were typical *A. salmonicida* to enter a freshwater or brackish aquatic environment, it would be expected to survive for a prolonged period in organic material and sediment; however, it would not be expected to survive in the marine environment for a significant period. While there is little definite evidence that atypical strains of *A. salmonicida* would persist to the same extent as the typical strain, this possibility cannot be discounted.

For susceptible fish to become infected with typical or atypical *A. salmonicida*, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. Typical or atypical *A. salmonicida* would be expected to spread readily between fish under conditions in the Australian aquatic environment.

Repeated high-level exposure of susceptible fish to a significant titre of typical or atypical *A. salmonicida* (for example, from regular discharge of untreated contaminated effluent from a fish processing plant) could result in the establishment of infection. However, sporadic or isolated entries of *A. salmonicida* into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have lesser significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen by this route.

Consequence assessment

Effects on commercially significant finfish species

Infection with typical *A. salmonicida* may cause serious disease in farmed salmonids but is of little pathogenic or economic significance in other finfish. The significance of this disease in salmonids has decreased greatly in recent years with the adoption of effective management strategies; however, furunculosis due to typical *A. salmonicida* is still one of the economically significant diseases of farmed salmonids in northern Europe and North America.

Experience in Europe shows that management and veterinary strategies can be used to prevent clinical disease but infection will still occur. Vaccines are available commercially in Europe and Canada. Oil adjuvant vaccines appear to be effective in controlling outbreaks of disease, however they also have adverse effects, including the development of lesions in the carcase, increased cost of production and reduced growth rate (Lillehaug et al 1996, Midtlyng 1996). The use of vaccines may also mask the presence of infection. It is possible to vaccinate hatchery fish to be used for the replenishment of wild stocks. However, vaccination provides a limited period of protection, hence there is current research interest in the development of an oral vaccine that could be used to boost immunity (A. McVicar, pers. comm.).

If disease due to *A. salmonicida* became established in Australia, control measures similar to those used overseas could be implemented but may be less effective than in overseas countries, depending on local conditions. This would necessitate the use of antibiotics that would have a direct cost and could also harm the product image of Australian salmon. The establishment of antibiotic-resistant strains of *A. salmonicida* would add

to costs and limit the effectiveness of control measures. The introduction of practices, such as 'all in-all out' management, would add to the cost of production, especially for Atlantic salmon farms using out-of-season smolts. Attempts could be made to eradicate disease if it was detected in an isolated locality; however, it is unlikely that disease in wild fish or at multiple sites could be eradicated.

ABARE (1994) reported that reduced fish survival, loss of product and increased costs could threaten the viability of the Australian salmonid industry if furunculosis became established. Effective strategies for the management and prevention of furunculosis have been adopted in countries affected by this disease since ABARE conducted this study. Dr A McVicar (pers. comm.) advised 'because of the success of control, furunculosis has now dropped well down the ranking in importance of diseasesÖcurrently affecting the Scottish salmon farming industry'. If disease due to typical *A. salmonicida* was to become established in Australia, it is likely that similar management measures would be adopted. The impact of establishment may be lower than that predicted by ABARE, but it is likely that establishment would result in increased costs and reduced profitability for the salmonid farming industry. Australia's 'disease and chemical residue free' image could also be harmed, reducing the price premium that Australian salmon attracts.

The establishment of disease due to typical *A. salmonicida* in wild freshwater salmonids would be expected to affect the recreational fishery (primarily trout angling) at a local/regional level due to disease-associated mortality in young and adult fish in naive populations. Although it is not likely that the disease could be eradicated from wild salmonids, experience in the UK suggests that the initial high impact would eventually be reduced as salmonids developed resistance to the pathogen (A McVicar pers. comm.). The adoption of management strategies to prevent the spread of disease to additional freshwater catchments would be expected to prevent the disease having a significant impact on the recreational sector at the national level.

Infection with atypical strains of *A. salmonicida* has caused significant disease in farmed Atlantic salmon in some cases, but has been of little economic significance in other finfish to date.

Based on experience overseas, the establishment of typical or atypical *A. salmonicida* in non-salmonid fish would not be expected to have significant consequences at a regional or national level. Perhaps the most significant aspect of the establishment of infection in non-salmonids would be the potential for these fish to serve as a reservoir of the pathogen for freshwater salmonids.

Taking into account the expected effects on the farmed and the recreational salmonid sectors, AQIS concludes that the establishment of disease due to typical *A. salmonicida* in Australia would have moderate to high consequences. Taking into account the capability of some atypical strains of *A. salmonicida* to cause disease and mortality in farmed salmonids overseas, the consequences of the establishment of additional atypical strains of *A. salmonicida* in Australia would be moderate.

Ecological and environmental effects

Based on the literature, infection with typical *A. salmonicida* is of little pathogenic or economic significance in non-salmonid finfish, including native fish, overseas. Non-salmonid fish in fresh water would be more likely to be infected with atypical strains than with the typical strain of *A. salmonicida* (A McVicar, pers. comm.).

It has been suggested that the establishment of *A. salmonicida* (typical or atypical strains) would threaten the survival of native freshwater species in Australia. For non-salmonid freshwater species, the most common hosts of *A. salmonicida* infection overseas are members of the Family Cyprinidae. There is little evidence that Australian native fish, none of which are closely related to the Family Cyprinidae, would be particularly susceptible to infection with typical or atypical strains of *A. salmonicida*. While Australian experience of infection with *A. salmonicida* is limited, atypical strains occur, including the GUD biovar and *A. salmonicida* in greenback flounder. An atypical strain of *A. salmonicida* was detected by an indirect fluorescent antibody test (IFAT) but not isolated in roach with ulcerative dermatitis in a Victorian lake (cited by Whittington et al 1995). A single case of disease due to the GUD biovar of

A. salmonicida was reported in native fish (silver perch) at a farm where goldfish had been infected.

The following conclusions can be drawn from the behaviour of these pathogens under Australian conditions. The presence of the GUD variant of *A. salmonicida* in Australia has had little consequence other than for the specific premises affected. It has had no discernible effect on wild fish or the environment and has had no significance in terms of the status of vulnerable or endangered native fish. Similarly, the presence of other atypical strains of *A. salmonicida* in Tasmania and in Victoria has not been associated with disease under natural conditions and has had little consequence for farmed or wild salmonids or native finfish.

AQIS has considered how the entry and establishment of typical *A. salmonicida* or more virulent atypical strains might affect the environment and native fish. The finfish species listed by Environment Australia as vulnerable and/or endangered under the *Endangered Species Protection Act 1992* belong to 13 genera, as listed in Appendix 5. Several factors have led to the current status of these species. The more important contributing factors include predation (including by introduced salmonid species such as brown trout) and degradation of habitat. Equally, it is important to prevent the establishment of exotic diseases that could affect the survival of native species. On the other hand, it could be argued that the establishment of a pathogen that had its main pathogenic effects on introduced salmonid species (such as brown trout) could have positive consequences for vulnerable species such as the galaxids, through a reduction in the population of key predators.

Overseas experience shows that the presence of *A. salmonicida* has had no significant effect on populations of wild non-salmonid fish. Therefore, while the effect of establishment of additional, more virulent strains of *A. salmonicida* cannot be discounted, there is no reason to expect that this would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

Unrestricted risk estimate for importation of non-salmonid marine finfish

Typical A. salmonicida

For the unrestricted importation of whole, round, non-salmonid marine fish for human consumption, the probability of establishment of *A. salmonicida* (typical) would be extremely low (for *wild-caught* fish) to very low (for *farmed* fish). The probability would be extremely low for whole, round non-salmonid marine fish imported for use as bait or as fish feed (ie *wild caught*). The consequences of establishment of typical *A. salmonicida* would be of moderate to high significance.

Thus, for typical *A. salmonicida*, the risk associated with the unrestricted importation of whole, round, *wild-caught* non-salmonid marine fish for human consumption, bait or fish feed meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

For the unrestricted importation of whole, round, *farmed*, non-salmonid marine fish for human consumption the risk does not meet Australia's ALOP and the implementation of specific risk management measures is warranted.

Atypical A. salmonicida

For the unrestricted importation of whole, round, non-salmonid marine fish for human consumption, the probability of establishment of *A. salmonicida* (atypical) would be very low (for *wild-caught* fish) to low (for *farmed* fish). The probability would be very low for whole, round, non-salmonid marine fish imported for use as bait or as fish feed (ie *wild caught*). The consequences of establishment of atypical *A. salmonicida* would be of moderate significance.

Thus for atypical *A. salmonicida*, the risk associated with the unrestricted importation of whole, round, *wild-caught* non-salmonid marine fish for human consumption, bait or fish feed meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

For the unrestricted importation of whole, round, *farmed* non-salmonid marine fish for human consumption the risk does not meet Australia's ALOP and the implementation of risk management measures is warranted.

A summary of the risk assessment is shown in Box 7.6. Appropriate measures are discussed in Chapter 8.

Box 7.6
Risk assessment — *Aeromonas salmonicida* (typical and atypical)

RELEASE ASSESSMENT (R)

The probability of *A. salmonicida* (typical) entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish for human consumption would be extremely low (for *wild-caught* fish) to very low (for *farmed* fish). For whole, round, non-salmonid marine fish imported for use as bait or as fish feed (ie *wild caught*) the probability would be extremely low.

The probability of *A. salmonicida* (atypical) entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish for human consumption would be very low (for *wild-caught* fish) to low (for *farmed* fish). For whole, round, wild-caught non-salmonid marine fish imported for use as bait or as fish feed (ie *wild caught*) the probability would be very low.

EXPOSURE ASSESSMENT (E)

If *A. salmonicida* (typical or atypical) entered Australia as a result of the unrestricted importation of whole, round, non-salmonid fish (wild-caught or farmed) for human consumption, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low.

The probability would be low to moderate, were *A. salmonicida* to enter via whole, round, wild caught non-salmonid marine fish imported for use as bait or as fish feed (ie *wild caught*).

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

The probability of *A. salmonicida* (typical) becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish for human consumption would be extremely low (EL) (for *wild-caught* fish) to very low (VL) (for *farmed* fish). The probability would be extremely low (EL) for the importation of whole, round non-salmonid marine fish for use as bait or as fish feed (ie *wild caught*).

The probability of *A. salmonicida* (atypical) becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish for human consumption would be very low (VL) (for wild-caught fish) to low (L) (for farmed fish). The probability would be very low (VL) for the importation of whole, round, wild-caught non-salmonid marine fish for use as bait or as fish feed (ie wild caught).

CONSEQUENCE ASSESSMENT

The consequences of the establishment of typical *A. salmonicida* in Australia would be moderate (M) to high (H), due primarily to effects on the farmed and the recreational salmonid sectors. Taking into account the capability of some atypical strains of *A. salmonicida* to cause disease and mortality in farmed salmonids overseas, the consequences of the establishment of additional atypical strains of *A. salmonicida* in Australia would be moderate (M).

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID MARINE FINFISH

Typical A. salmonicida

Wild-caught fish

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = EL
- significance of consequences = M–H
- importation risk for typical *A. salmonicida* = acceptable ('yes' in Figure 1.1)

Box 7.6 (continued)
Risk assessment — *Aeromonas salmonicida* (typical and atypical)

That is:

- the risk associated with the unrestricted importation of wild-caught whole, round, non-salmonid marine fish for human consumption, bait or fish feed meets Australia's ALOP; and
- risk management measures are not warranted.

Farmed fish

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL (fish for human consumption)
- significance of consequences = M–H
- importation risk for typical *A. salmonicida* = unacceptable ('no' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, farmed non-salmonid marine fish does not meet Australia's ALOP; and
- risk management measures are warranted.

Atypical A. salmonicida

Wild-caught fish

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL
- significance of consequences = M
- importation risk for *A. salmonicida* = acceptable ('yes' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of whole, round, wild-caught non-salmonid marine fish for human consumption, bait or fish feed meets Australia's ALOP; and
- risk management measures are not warranted.

Farmed fish

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L (fish for human consumption) to VL (for fish for bait or as fish food).
- significance of consequences = M
- importation risk for *A. salmonicida* = unacceptable ('no' in Figure 1.1).

That is:

- the risk associated with the unrestricted importation of whole, round, farmed non-salmonid marine fish does not meet Australia's ALOP; and
- risk management measures are warranted.

7.2.7 *PHOTOBACTERIUM DAMSELA PISCICIDA* (PASTEURELLOSIS)

Photobacterium damsela piscicida causes a disease in fish known as 'pasteurellosis' or pseudotuberculosis. Pasteurellosis is a serious disease in many non-salmonid marine finfish in Japan and Europe. Historically, infection with *P. damsela piscicida* has caused severe mortality in wild marine finfish populations in the United States; however there are no recent reports of disease in wild fish populations in the literature.

Release assessment

Geographic distribution

P. damsela piscicida has been isolated from diseased fish in the United States, Japan, Taiwan, Spain, France, Greece, Italy, Portugal and Norway. Isolates from several European countries, Japan and the United States are biochemically and antigenically similar (review by Daly 1999).

Host range and prevalence

P. damsela piscicida has been isolated from at least 15 non-salmonid marine finfish species. Historically, the bacterium has been associated with significant disease epizootics (and mortality) in wild fish (striped bass, white perch) in the United States (review by Daly 1999). More recently, infection with *P. damsela piscicida* caused high losses in cultured marine fish in Japan (eg up to 50% mortality in yellowtail on individual farms) and Europe, particularly the Mediterranean (eg in cultured European sea bass and gilthead sea bream) (review by Daly 1999). Disease occurs more commonly in warm water (20–25°C) and may affect and cause mortality in juveniles and older fish (Le Breton 1999).

Non-salmonid marine species from which *P. damsela piscicida* has been isolated include: white perch (*Morone americana*), striped bass (*Morone saxatilis*), Atlantic menhaden (*Brevoortia tyrannus*), striped mullet (*Mugil cephalus*), yellowtail (*Seriola quinqueradiata*), black sea bream (*Mylio macrocephalus*), red sea bream (*Pagrus major*), oval file fish (*Navodan modestus*), red grouper (*Epinephelus okaara*), gilthead sea bream (*Sparus aurata*), turbot (*Scophthalmus maximus*), sole (*Solea solea*), mullet (*Mugil cephalus*), sea bass (*Dicentrarchus labrax*), and sea bream (*Pagrus pagrus*). The bacterium has also been isolated from Atlantic salmon (review by Daly 1999).

Detection and organs affected

Disease may be manifest in acute or chronic form. Infected fish usually show no external signs. Chronically infected fish may have grossly visible granulomatous inflammatory lesions, throughout the internal viscera, particularly in the kidney and spleen (review by Daly 1999, Kusuda and Kawai 1998). *P. damsela piscicida* has not been isolated from tissues other than the viscera. There is no information on the propensity of infected fish to become inapparent carriers of infection. Covertly infected fish would have no signs of infection but could contain the pathogen in their tissues (especially the viscera).

Diagnosis is based on culture of the pathogen and biochemical identification. *P. damsela piscicida* can be cultured on most bacteriological media provided sodium chloride is added to a final concentration of 0.5%.

Key findings

P. damsela piscicida has a wide host range.

Disease is more commonly reported in farmed than in wild marine fish. Disease occurs more commonly in fish in warm water than in cold water. *P. damsela piscicida* can cause disease in juvenile and older fish.

In diseased fish pathological changes primarily affect the viscera. Except for non-specific signs of generalised septicaemia (ie in moribund fish) there are usually no external signs of disease. Cultured marine warm-water fish are higher quality fish and are normally imported for human consumption as inspected, eviscerated carcases or as further processed product. In this case clinically infected fish would be detected and rejected in the course of inspection for human consumption.

Covertly infected fish would not be visibly abnormal and would not be detected at inspection. The pathogen has not been isolated from tissues other than the viscera, thus, evisceration would substantially reduce the titre of pathogen in covertly infected fish.

Exposure assessment

Transmission

Horizontal transmission by direct fish-to-fish contact is the likely mode of spread. Oral infection is considered the most important route of entry. Perbranchial and percutaneous routes of infection may also be possible. The bacterium has been reported to enter a 'viable but non-culturable state' in which virulence is retained. However, the epidemiological significance of such organisms and their capability to cause disease has not been established (review by Daly 1999).

A range of non-salmonid marine finfish species farmed in warm water (20–25°C) in Australia (eg snapper, barramundi) would be susceptible to infection. *P. damsela piscicidia* has been isolated from Atlantic salmon overseas. The temperature of coastal waters of southern Australia may be suitable for the occurrence of disease in that species in summer months.

Agent stability

The bacterium does not appear to survive in seawater for periods of greater than 3–5 days (review by Daly 1999). There are no published data on the pathogen's stability to thermal treatment or pH.

Key findings

Clinical disease mainly affects farmed marine fish, although clinical disease has been reported in some wild fish species.

Infection may be transmitted horizontally. The highest titre of the pathogen would be found in the viscera.

A range of non-salmonid marine finfish species farmed in warm-water (20–25°C) in Australia (eg snapper, barramundi) would be susceptible to infection.

Water temperatures conducive to infection (eg 20–25°C) occur in most coastal waters of Australia, including southern waters in summer.

For susceptible fish to become infected with *P. damsela piscicida*, fish of a susceptible species and lifecycle stage would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. *P. damsela piscicida* would be expected to spread readily between fish under conditions in the Australian aquatic environment, except at low water temperatures.

Repeated high-level exposure of susceptible fish to a significant titre of *P. damsela piscicida* (for example, from regular discharge of untreated effluent of a fish processing plant or via frequent and extensive use of bait or fish feed) could result in the establishment of infection. However, sporadic or isolated entries of *P. damsela piscicida* into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen.

Consequence assessment

Effects on commercially significant finfish species

P. damsela piscicida could potentially infect several economically significant commercial and recreational marine fish species in Australia. Based on overseas experience, the species most likely to be affected would be marine species farmed in warm water 20–25°C (eg snapper and barramundi).

Antibiotic resistant strains of *P. damsela piscicida* have been isolated from infected fish. A number of vaccines (inactivated and live attenuated) have been trialed by various routes of exposure, but reliable, reproducible results have not yet been obtained (Kusuda and Kawai 1998, Le Breton 1999). If *P. damsela piscicida* were to become established in the Australian marine environment it would not be amenable to eradication.

While Atlantic salmon are susceptible to infection, there are no reports of significant consequences in Atlantic salmon resulting from infection with *P. damsela piscicida*. The temperature of coastal waters of southern Australia may be suitable for the occurrence of disease in that species in summer months; however, the impact of *P. damsela piscicida* on farmed Atlantic salmon would not be expected to be significant at a national level.

The most economically significant mariculture industries in Australia are based on Atlantic salmon, ocean trout and tuna. Other marine farming industries in Australia are at a relatively early stage of development. Some species being considered or trialled for potential use in mariculture are in the same taxa as species reported to be susceptible to the pathogen. The establishment of *P. damsela piscicida* in Australia could impede the development of mariculture of snapper or other susceptible species. Given the current stage of development of the mariculture industries based on susceptible species, the consequences of establishment would not be expected to cause significant losses at a national level; however, it could limit the prospects of developing industries.

Ecological and environmental effects

Historically, infection with *P. damsela piscicida* has been associated with significant mortality in wild-caught marine fish (white perch, striped bass) in the United States. Recent reports of mortality due to pasteurellosis have been confined to marine species farmed in warm water. *P. damsela piscicida* could infect wild, non-salmonid marine finfish species in temperate and subtropical coastal waters of Australia.

There is little evidence to suggest that the establishment of this pathogen would have a significant long-term effect on wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of non-salmonid marine finfish

For the unrestricted importation for human consumption of whole, round, non-salmonid marine fish (farmed or wild caught) of susceptible species[9] the probability of establishment would be very low. For whole, round, wild-caught non-salmonid marine fish imported for use as bait or as fish feed the probability would be low. The consequences of establishment would be of moderate significance.

Thus, for *P. damsela piscicida*, the risk associated with the unrestricted importation for human consumption of whole, round, non-salmonid marine fish meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

The risk associated with the unrestricted importation for bait and fish feed of whole, round, wild-caught non-salmonid, marine fish of specified species does not meet Australia's ALOP and the implementation of specific risk management measures is warranted.

For the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of other species the probability of the establishment of *P. damsela piscicida* would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of *P. damsela piscicida* in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

A summary of the risk assessment is shown in Box 7.7. Appropriate risk management measures are discussed in Chapter 8.

9 These conclusions apply to: *Morone* spp, *Brevoortia* spp, *Mugil* spp, *Seriola* spp, *Mylio* spp, *Pagrus* spp, *Navodan* spp, *Epinephalus* spp, *Sparus* spp, *Scophthalmus* spp, *Solea* spp and *Dicentrarchus* spp.

Box 7.7
Risk assessment — *Photobacterium damsela piscicida* (pasteurellosis)

RELEASE ASSESSMENT (R)

The probability of *P. damsela piscicida* entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of susceptible species (*Morone* spp, *Brevoortia* spp, *Mugil* spp, *Seriola* spp, *Mylio* spp, *Pagrus* spp, *Navodan* spp, *Epinephelus* spp, *Sparus* spp, *Scophthalmus* spp, *Solea* spp and *Dicentrarchus* spp) for human consumption would be very low (for wild-caught fish) to low (for farmed fish).

For whole, round, wild-caught non-salmonid marine fish imported for use as bait or as fish feed (ie wild caught) the probability would be low.

The probability of *P. damsela piscicida* entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of other species would be negligible.

EXPOSURE ASSESSMENT (E)

If *P. damsela piscicida* entered Australia in whole, round, non-salmonid marine fish (farmed or wild-caught) for human consumption, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be very low. If *P. damsela piscicida* entered Australia in whole, round, non-salmonid, marine fish for use as bait or fish feed (ie wild caught), the probability would be low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

For whole, round, non-salmonid marine fish of susceptible species (*Morone* spp, *Brevoortia* spp, *Mugil* spp, *Seriola* spp, *Mylio* spp, *Pagrus* spp, *Navodan* spp, *Epinephelus* spp, *Sparus* spp, *Scophthalmus* spp, *Solea* spp, *Mugil* spp and *Dicentrarchus* spp) imported for human consumption, the probability would be very low (VL).

For whole, round, non-salmonid marine fish imported for use as bait or as fish feed (ie wild caught) the probability would be low (L).

The probability of *P. damsela piscicida* becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of other species would be negligible (N).

CONSEQUENCE ASSESSMENT

The consequences of the establishment of *P. damsela piscicida* in Australia would be moderate (M).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of *P. damsela piscicida* would affect the survival of any vulnerable or endangered species in Australia or have any significant long-term effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID, MARINE FINFISH

For susceptible species (see above)

For fish for human consumption
From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL
- significance of consequences = M
- importation risk for *P. damsela piscida* = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish for human consumption meets Australia's ALOP; and
- risk management measures are not warranted.

Box 7.7 (continued)
Risk assessment — *Photobacterium damsela piscicida* (pasteurellosis)

For fish for bait or as fish feed (ie wild caught)
From Figure 1.1 (risk evaluation matrix):

- probability of establishment = L
- significance of consequences = M
- importation risk for *P. damsela piscida* = unacceptable ('no' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, non-salmonid marine fish for bait and fish feed does not meet Australia's ALOP; and
- risk management measures are warranted.

For other species
From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because the probability of disease establishment is negligible
- importation risk for *P. damsela piscida* = acceptable ('yes' in Figure 1.1)

That is:

- regardless of the consequences of establishment of *P. damsela piscida* in Australia, the risk associated with the unrestricted importation meets Australia's ALOP; and
- risk management measures are not warranted.

7.2.8 *RENIBACTERIUM SALMONINARUM* (BACTERIAL KIDNEY DISEASE)

Release assessment

BKD is listed by the OIE as an 'other significant disease', and is included in List III of the European Union Directive 93/54/EEC.

The OIE Code (1997a) provides the following international recommendation for countries with an official control policy for bacterial kidney disease:

> 'When importing live fish of a susceptible species or their gametes or eggs or dead uneviscerated fish, the Competent Authority of the importing country with an official control policy for bacterial kidney disease may wish to require the presentation of an international aquatic animal health certificate issued by the Competent Authority in the exporting country, attesting that the aquaculture establishment, zone or country of origin has been regularly subjected to appropriate tests for bacterial kidney disease with negative results.'

Geographic distribution

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.

- BKD caused by *R. salmoninarum* is recognised internationally as one of the most prevalent diseases of cultured salmonids. It has a wide geographical distribution that includes most salmon producing countries.

AQIS considered further information on *R. salmoninarum* in non-salmonid marine fish, summarised below.

R. salmoninarum has been detected serologically in non-salmonid marine fish collected from salmonid farms in Japan (Sakai and Kobayashi 1992) and Canada (Kent et al 1998).

Host range and prevalence

- The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of relevant literature.
- Natural outbreaks of BKD are restricted to members of the family Salmonidae. The greatest losses are recorded in fish of the genus *Oncorhynchus*.
- Clinical disease is more common in farmed salmonids but may also be observed in wild salmonids.
- Outbreaks of clinical disease usually occur when smolts are transferred to sea; infection hinders adaptation to seawater and death commonly follows.

AQIS considered further information on *R. salmoninarum* in non-salmonid marine finfish.

Clinical disease due to natural infection with *R. salmoninarum* has not been reported in non-salmonid marine finfish.

In Japan the bacterium was detected by indirect dot blot assay and IFAT in the kidney tissue of apparently normal greenling (*Hexagrammos otakii*) (1/5) and flathead (*Platycephalus indicus*) (6/22) (Sakai and Kobayashi 1992). Bacterial culture yielded negative results. In Canada *R. salmoninarum* antigen was detected in the kidney tissue of Pacific herring (3/19) and moribund Pacific hake (2/8) using ELISA and direct FAT (Kent et al 1998). There was no evidence of clinical disease and bacterial culture was not attempted.

The findings of these studies indicate that non-salmonid fish may become infected with *R. salmoninarum* when closely associated with infected salmonids. This is an occasional finding and is not associated with pathological changes; thus, it appears that *R. salmoninarum* does not establish infection in non-salmonid finfish as a consequence of natural exposure. The positive results obtained in these studies may result from the detection of antigen associated with non-viable bacteria that have been inactivated by the host's immune response. Antigen of *R. salmoninarum* has been reported to persist in uninfected fish for up to three months (Kent et al 1998).

Under experimental conditions, disease was induced in juvenile Pacific herring by intraperitoneal challenge (Traxler and Bell 1988). Infection with *R. salmoninarum* has also been established under experimental conditions in sablefish (*Anoplopoma fimbria*), and shiner perch (*Cymatogaster aggregata*) by intraperitoneal challenge and in common shiner (*Notropis cornutus*) and flathead minnow (*Pimephales promelas*) by immersion challenge (review by Evelyn 1993). Experimentally infected shiner perch were shown to eliminate *R. salmoninarum* post-inoculation (Kent et al 1998 citing Evelyn, unpublished data). It has been reported that shiner perch are refractory to challenge when cohabiting with infected fish (review by Evelyn 1993).

Based on the information in the literature, AQIS considers that there would be a negligible likelihood that non-salmonid marine finfish would be infected with *R. salmoninarum*.

Unrestricted risk estimate for importation of non-salmonid marine finfish

Taking into account the release assessment documented above, the probability of *R. salmoninarum* becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish would be negligible. Therefore, the probability of establishment of disease would also be negligible.

From to the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of *R. salmoninarum* in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted. A summary of the risk assessment is shown in Box 7.8.

Box 7.8
Risk assessment — *Renibacterium salmoninarum* (bacterial kidney disease)

RELEASE ASSESSMENT (R)

The probability of *R. salmoninarum* entering Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish (farmed or wild-caught) would be negligible.

EXPOSURE ASSESSMENT (E)

Because there is a negligible probablity of *R. salmoninarum* entering Australia as a consequence of unrestricted importation of whole, round, non-salmonid marine fish (farmed or wild-caught) the probability of susceptible fish being exposed to a dose sufficient to cause infection would also be negligible.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

Because there is a negligible probablity of *R. salmoninarum* entering Australia as a consequence of unrestricted importation of whole, round, non-salmonid marine fish (farmed or wild-caught) the probability of disease establishment would be negligible (N).

CONSEQUENCE ASSESSMENT

Because there is a negligible probability of establishment of *R. salmoninarum* in Australia as a consequence of unrestricted importation of whole, round, non-salmonid marine fish (farmed or wild-caught) the consequences of establishment are not considered further.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID, MARINE FINFISH

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because the probability of disease establishment is negligible
- importation risk for *R. salmoninarum* = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of *R. salmoninarum* meet Australia's ALOP; and
- risk management measures are not warranted.

7.2.9 *BROOKLYNELLA HOSTILIS* (BROOKLYNELLOSIS)

Release assessment

Geographic distribution

Brooklynella hostilis has a cosmopolitan distribution, but is more common in warmer waters (Lom 1995). *Brooklynella* spp have been reported in Australia but the presence of *B. hostilis* has not been reported (review by Humphrey 1995). *B. hostilis* has regularly been associated with disease in farmed fish in warmer waters (eg Singapore, Kuwait) (Lom and Dykova 1992) and has recently been reported as causing disease in maricultured fish in the Red Sea (Diamant 1998).

Host range and prevalence

B. hostilis is not host specific and is likely to infest any marine species maintained under intensive conditions in warm water (Lom and Dykova 1992).

The parasite is generally not reported in wild fish populations although disease has recently been reported

in wild-caught ornamental marine fish (Landsberg and Blakesley 1995).

Detection and organs affected

B. hostilis is primarily a gill parasite. In cases of light infestation with *B. hostilis*, fish appear healthy. In cases of heavy infestation, the parasite destroys surface tissues and can cause serious gill and skin lesions (Noga 1996, Stoskopf 1993). Heavily infested fish often die as a result of gill dysfunction (Lom and Dykova 1992, Stoskopf 1993).

Diagnosis is based on identification of the parasite by microscopy in wet tissue specimens or in tissue sections at histopathology (Noga 1996).

Key findings

B. hostilis is not host specific and occurs in a wide range of marine fish species, particularly warm water species. There is no evidence to suggest it occurs more commonly in any particular lifecycle stage of a susceptible host.

Disease generally occurs in cultured marine fish in warm water. These are higher quality fish and are normally imported for human consumption as inspected, eviscerated carcases or as further processed product.

Pathological changes in diseased fish are most prominent in the gills. Externally detectable pathology (eg skin lesions) is generally present in clinically affected fish. Such fish would be detected and rejected in the course of inspection for human consumption.

Lightly infested fish would appear healthy and would not be detected during inspection for human consumption. Infection would be confined to the gills of these fish.

Exposure assessment

Transmission

Transmission is believed to occur horizontally through direct fish-to-fish contact (Lom and Dykova 1992).

Based on the lack of host specificity of *B. hostilis*, most marine fish species present in Australia would be expected to be susceptible. Like other protozoan infestations, clinical disease due to *B. hostilis* is normally reported in 'stressed' marine fish cultured in warm water.

Agent stability

Detailed stability data are not available. Most ciliated protozoa are able to tolerate varied environmental conditions, including fluctuations in temperature and salinity (review by Dykova 1995). At 0° C the contractile vacuole of ciliates becomes enlarged predisposing the cell to rupture (Jones 1974). The development of ice crystals in the protozoan cells would be expected to render the pathogen non-viable in product that was frozen and then thawed. Data on the susceptibility of *B. hostilis* to chilling are lacking.

Key findings

Gill tissues would be the main source of the parasite in infected fish.

Infection is transmitted horizontally.

B. hostilis is not host-specific and most marine species (non-salmonid and salmonid, eg Atlantic salmon) maintained in culture in Australia would be susceptible.

Temperatures conducive to infection (eg 20–25° C) occur in most coastal waters of Australia, including southern waters in summer.

The parasite may persist relatively well in the environment but would not be expected to survive freezing and thawing.

For susceptible fish to become infected with *B. hostilis*, fish of a susceptible species would need to be exposed to a sufficient dose of the pathogen for a sufficiently prolonged period. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. *B. hostilis* would be expected to spread readily between fish under conditions in the Australian marine environment, given the wide host range and the conducive water temperature.

Repeated high level exposure of susceptible fish to a significant titre of *B. hostilis* (for example, from regular discharge of untreated effluent of a fish processing plant or via frequent and extensive use of bait or fish feed) could result in the establishment of infection. However, sporadic or isolated entries of *B. hostilis* into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species being exposed to an infectious dose of the pathogen.

Consequence assessment

Effects on commercially significant finfish species

Based on overseas experience the most significant effect of *B. hostilis* would be in farmed marine species (eg Atlantic salmon, snapper, barramundi), particularly at warm water temperatures. Species maintained in aquaria would also be susceptible. Fish are most susceptible to infection when 'stressed', and inadequate management is usually a predisposing factor to disease. The extent of the impact of disease would depend upon other factors such as water quality, intercurrent disease and nutrition. Significant disease (including mortality) has recently been reported in farmed red sea bream (Diamant 1998) associated with infection by *B. hostilis* but the level of mortality was not reported.

Dipping in fresh water, as undertaken in Atlantic salmon to control amoebic gill disease (*Paramoeba* spp), followed by prolonged immersion in formalin may assist in controlling infection in the early stages (Noga 1996).

The most economically significant mariculture industries in Australia are based on Atlantic salmon, ocean trout and tuna. Other marine farming industries in Australia are at a relatively early stage of development. Some species (eg snapper, barramundi) that are being considered or trialled for potential use in mariculture are in the same taxa as species reported to be susceptible to *B. hostilis*. The establishment of *B. hostilis* in Australia could impede the development of mariculture of snapper or other susceptible species. Given the current stage of development of the mariculture industries based on susceptible species, the consequences of establishment would not be expected to cause significant losses at a national level; however, it could limit the prospects of developing industries.

Many of the species that could be susceptible to infection with *B. hostilis* are also economically significant in commercial and recreational fisheries in Australia.

Taking account of these factors, AQIS considers the establishment of *B. hostilis* could have a significant effect locally or regionally, but not at a national level.

Ecological and environmental effects

There is a single report of disease in wild fish stocks (in an ornamental marine species) due to infection with *B. hostilis* (Landsberg and Blakesley 1995). *B. hostilis* is confined to marine hosts. There is no evidence that the establishment of *B. hostilis* would have a significant impact on wild finfish species, including native fish, in Australia.

Unrestricted risk estimate for importation of non-salmonid marine finfish

For the unrestricted importation for human consumption of whole, round, non-salmonid marine fish the probability of establishment of *B. hostilis* would be very low (for wild-caught) to low (for farmed). For product imported for use as bait or fish feed (ie wild caught) the probability would be very low. The consequences of establishment of *B. hostilis* would be of low significance.

Thus, for *B. hostilis* the risk associated with the unrestricted importation of whole, round, farmed and wild-caught non-salmonid marine fish meets Australia's ALOP and the implementation of specific risk management measures is not warranted. A summary of the risk assessment is shown in Box 7.9.

Box 7.9
Risk assessment — *Brooklynella hostilis*

RELEASE ASSESSMENT (R)

The probability of *B. hostilis* entering Australia as a consequence of the unrestricted importation for human consumption of whole, round, non-salmonid marine fish would be very low (for wild caught) to low (for farmed).

For whole, round, wild-caught non-salmonid marine fish imported for bait or as fish feed (ie wild caught) the probability would be very low.

EXPOSURE ASSESSMENT (E)

Based on the information in Section 1.6 and in this section, if *B. hostilis* entered Australia in whole, round, non-salmonid marine fish for human consumption, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be low.

If *B. hostilis* entered Australia in whole, round, non-salmonid marine fish for use as bait or fish feed, the probability would be moderate.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

Taking into account the release and exposure assessments documented above, the probability of *B. hostilis* becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish for human consumption would be very low (VL) (for wild-caught) to low (L) (for farmed). For whole, round, non-salmonid, marine fish for use as bait and fish feed (ie wild caught) the probability would be very low (VL).

CONSEQUENCE ASSESSMENT

The consequences of the establishment of *B. hostilis* in Australia would be low (L).

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of *B. hostilis* would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID, MARINE FINFISH

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = VL (for wild-caught fish for human consumption or any fish for bait or fish feed) to L (for farmed fish for human consumption)
- significance of consequences = L
- importation risk for *B. hostilis* = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, farmed and wild-caught non-salmonid marine fish meets Australia's ALOP; and
- risk management measures are not warranted.

7.2.10 *MICROSPORIDIUM SERIOLAE* (BEKO DISEASE)

Release assessment

Geographic distribution

Microsporidium seriolae has only been reported from Japan. It causes significant pathological changes in the muscle of juvenile, farmed marine finfish (review by Lom 1995). The disease is not fatal to infected fish but causes lesions that make affected fish unmarketable for human consumption.

Host range and prevalence

M. seriolae has been detected in the musculature of juvenile yellowtail (*Seriola quinqueradiata*) and juvenile gold-striped amberjack (*S. lalandi*) (Yokoyama et al 1996). A similar organism, that has not been speciated, was described in juvenile red sea bream (*Pagrus major*) (Egusa et al 1988).

Prevalence data are limited, however, infection appears to be common in juvenile farmed fish of susceptible species. In a study on beko disease in juvenile farmed yellowtail, groups of fish that were transferred from an indoor tank to sea net pens acquired high rates of infection. When the water temperature was approximately 20°C almost all transferred fish became infected. Those transferred when the water temperature was approximately 30°C had a much lower prevalence of infection. Histological examination showed that the development of *M. seriolae* and host recovery were faster in fish reared at 25°C than those kept at 20°C (Sano et al 1998).

Detection and organs affected

Pathological changes are normally manifested as surface depressions due to liquefaction of affected muscle tissue by the parasite. Lesions are visible to the naked eye (Lom and Dykova 1992).

Confirmatory diagnosis is based on identification of parasitic stages in wet tissue specimens by microscopy, or in tissue sections by histopathology (Dykova 1995). Yokoyama et al (1996) used fluorescence microscopy to detect spores in Uvitex 2B-stained smears of trunk muscle homogenates. This method was found to be more sensitive than the conventional visual inspection for 'cysts' in the trunk muscle. These authors recommended the Uvitex 2B stain for the rapid and sensitive diagnosis of beko disease and also for histopathological studies of microsporan infections.

Spores have been recorded only in the musculature of host fish (review by Dykova 1995).

Key points

M. seriolae is primarily a disease of juvenile, marine-farmed *Seriola* species. It has not been reported in wild-caught fish. Infection has only been reported in Japan. In yellowtail and amberjack, juvenile fish are the lifecycle stage infected and in which disease occurs. Infection has not reported in adult fish of these species and there is evidence to suggest that infected fish recover fully from infection (Sano et al 1998).

Juvenile fish (the lifecycle stage most likely to have clinical disease) are not usually harvested for human consumption. Clinical infection has not been reported in adult fish. Yellowtail are normally imported for human consumption as inspected, eviscerated carcases or as further processed product. Clinically infected fish would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. Some clinically infected fish and subclinically infected fish would not be visibly abnormal and would not be detected at inspection. In covertly infected fish of market size, the number of organisms, if any, would be extremely low.

Exposure assessment

Transmission

The microsporidians that infect fish are known to be transmitted by ingestion and directly between fish (Dykova 1995). Experimentally it is possible to infect fish with microsporans by feeding mature spores suspended in the water or fed shortly before to planktonic organisms (Dykova 1995).

Agent stability

There are limited data on thermal lability and pH stability of this pathogen. The spores would be expected to survive freezing, based on the stability of related

microspora (Amigo et al 1996). Microsporans are known to survive in water for up to one year at 4°C (Dykova 1995).

Of the many chemicals tested for efficacy against microsporans, toltrazuril has been found most promising. Schmahl and Mehlhorn (1989) recommended immersion in 5 or 20 µg toltrazuril per mL of water for 1–4 hours. This treatment should be applied for six days at two-day intervals in well aerated water. This treatment will kill the vegetative stages but not the mature spores.

Key findings

M. seriolae causes a disease that primarily affects juvenile marine-farmed *Seriola* species. The minimum infective dose is unknown and few conclusions can be drawn from available information.

It is expected that *Seriola* species present in Australia would be susceptible to infection. There is little evidence that species other than *Seriola* species would be susceptible to infection.

M. seriolae may be transmitted horizontally via exposure to infective spores in seawater.

It is expected that *M. seriolae* would survive for an extended period in seawater. There is no evidence to suggest that spores would survive in fresh water for a significant period.

For susceptible fish to become infected with *M. seriolae*, fish of a susceptible species and lifecycle stage (ie juvenile *Seriola* species) would need to be exposed to a sufficient dose of the pathogen for a sufficient period of time. Infection would need to be transmitted from the index case of infection to other susceptible hosts to result in the establishment of disease in the population. *M. seriolae* would be expected to spread between fish under conditions in the Australian marine environment.

There would be a negligible probability of the entry of *M. seriolae* into the aquatic environment causing the establishment of disease in finfish other than *Seriola* species. Any infective material entering the aquatic environment and being consumed by fish would most probably be consumed by non-susceptible species, reducing the probability of *Seriola* species being exposed to and becoming infected with *M. seriolae*.

Repeated high-level exposure of susceptible fish to a significant number of *M. seriolae* spores (for example, from regular discharge of untreated effluent from a fish processing plant) could result in the establishment of infection. However, sporadic or isolated entries of *M. seriolae* into the aquatic environment (for example, via the disposal from pleasure craft of infected food scraps) would be expected to have little significance. This is primarily because there would be an extremely low probability of susceptible species at a susceptible lifecycle stage being exposed to an infectious dose of the pathogen.

Consequence assessment

Effects on commercially significant finfish species

There is little information on the economic significance of *M. seriolae*. In outbreaks of acute disease there may be significant pathological effects that render affected fish unmarketable. As clinical infection has only been reported in juvenile fish and fish seem to recover fully from infection, it is likely that the effect of infection with *M. seriolae* would be of minor significance in the long term and of moderate significance in the short term.

There is no registered treatment available for microsporan infection.

The most economically significant mariculture industries in Australia are based on Atlantic salmon, ocean trout and tuna. Other mariculture industries in Australia are at a relatively early stage of development. Gold-striped amberjack (*S. lalandi*) is a potential aquaculture species in Australia. It is expected that this species would be susceptible to infection with *M. seriolae*. The presence of *M. seriolae* in Australia would be an impediment to the development of this industry but would not be expected to have a significant effect at a national level.

The scientific literature reports that *M. seriolae* infects cultured fish only and there are no reports of disease in wild fish. It is likely that this disease would have less significance in wild than in cultured fish. Consequently it is expected that the impact on commercial yellowtail and amberjack fisheries would not be significant. The establishment of *M. seriolae* in Australia would be expected to have less impact than that associated with *Unicapsula seriolae*, a common microsporidian parasite

of yellowtail and amberjack (of all age groups) in Australia.

The establishment of *M. seriolae* in Australia would be unlikely to have a significant effect on any other commercially significant finfish species in Australia.

Ecological and environmental effects

As infection with *M. seriolae* has only been reported in *Seriola* species, it is extremely unlikely that the establishment of disease would significantly affect wild finfish, including native finfish in Australia.

Unrestricted risk estimate for importation of non-salmonid marine finfish

For the unrestricted importation for human consumption of whole, round, non-salmonid marine fish, including juveniles, of susceptible species (*Seriola* spp.) from Japan, the probabilityof establishment of *M. seriolae* would be extremely low (for wild-caught fish) to very low (for farmed fish). The consequences of establishment of *M. seriolae* would be of low significance.

Thus, for *M. seriolae*, the risk associated with unrestricted importation of whole, round, farmed and wild-caught non-salmonid marine fish, including juveniles, of susceptible species from Japan meet Australia's ALOP and the implementation of specific risk management is not warranted. A summary of the risk assessment is shown in Box 7.10.

For the unrestricted importation of whole, round, non-salmonid marine fish of other species and for the importation of non-salmonid marine finfish from countries other than Japan, the probability would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of *M. seriolae* in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

Box 7.10
Risk assessment — *Microsporidium seriolae* (beko disease)

RELEASE ASSESSMENT (R)

The probability of *M. seriolae* entering Australia as a consequence of the unrestricted importation for human consumption of whole, round, non-salmonid marine fish of susceptible species (*Seriola* spp) from Japan would be extremely low (for wild caught fish) to very low (for farmed fish). For other finfish species from Japan and for all finfish from other countries, the probability would be negligible.

Because *M. seriolae* infections are primarily clinically expressed and there is a greater probability of a significant pathogen titre in juvenile fish, the probability associated with the unrestricted importation of juvenile fish of susceptible species from Japan would be low (for wild-caught) to moderate (for farmed).

EXPOSURE ASSESSMENT (E)

If *M. seriolae* entered Australia, the probability of susceptible fish being exposed to a dose sufficient to cause infection would be very low.

PROBABILITY OF DISEASE ESTABLISHMENT (R + E)

For whole, round, non-salmonid marine fish of susceptible species imported for human consumption, including juveniles, from Japan, the probability would be extremely low (EL) (for wild fish) to very low (VL) (for farmed fish).

The probability of *M. seriolae* becoming established in Australia as a consequence of the unrestricted importation of whole, round, non-salmonid marine fish of susceptible speices from countries other than Japan and of all other species would be negligible (N).

Box 7.10 (continued)
Risk assessment — *Microsporidium seriolae* (beko disease)

CONSEQUENCE ASSESSMENT

The consequences of the establishment of *M. seriolae* in Australia would be low, due primarily to the limited effect *M. seriolae* would have on farmed and commercially significant *Seriola* species.

While the effect on the environment cannot be discounted, there is no reason to expect that the establishment of *M. seriolae* would affect the survival of any vulnerable or endangered species in Australia or have any significant effect on the natural environment.

UNRESTRICTED RISK ESTIMATE FOR IMPORTATION OF WHOLE, ROUND, NON-SALMONID, MARINE FINFISH

For susceptible species from Japan

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = EL (for wild-caught fish) to VL (for farmed fish, including juveniles)
- significance of consequences = L
- importation risk for *M. seriolae* = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, farmed and wild-caught non-salmonid, marine fish, including juveniles, of susceptible species from Japan meets Australia's ALOP; and
- risk management measures are not warranted.

For other species and for susceptible species from countries other than Japan

From Figure 1.1 (risk evaluation matrix):

- probability of establishment = N
- significance of consequences = irrelevant because the probability of disease establishment is negligible
- importation risk for M. seriolae = acceptable ('yes' in Figure 1.1)

That is:

- the risk associated with the unrestricted importation of whole, round, farmed and wild-caught non-salmonid, marine fish, including juveniles, of susceptible species from countries other than Japan and of all other species meets Australia's ALOP; and
- risk management measures are not warranted.

7.3 Summary of import risk assessment for non-salmonid marine finfish

A summary of the import risk assessment for non-salmonid marine finfish is shown in Table 7.4.

Table 7.4
Summary of import risk assessment for non-salmonid marine finfish of susceptible species

PATHOGEN	ESTABLISHMENT	CONSEQUENCES	RISK MANAGEMENT
Aquabirnavirus	Human consumption—L Bait/fish feed—M	L–M	Yes Yes
Infectious pancreatic necrosis virus	Human consumption—L–M Bait/fish feed—M	M–H	Yes Yes
Infectious haematopoietic necrosis virus	Negligible	M–H	No
Red sea bream iridovirus	Human consumption—L Bait/fishfeed—L–M	L–M	Yes
Viral haemorrhagic septicaemia virus	Human consumption—L Bait/fish feed—L–M	L–M	Yes Yes
Aeromonas salmonicida (typical)	Wild caught—EL Farmed—VL	M–H	No Yes
Aeromonas salmonicida (atypical)	Wild caught—VL Farmed—L	M	No Yes
Photobacterium damsela piscicida	Human consumption—VL Bait/fish feed—L	M	No Yes
Renibacterium salmoninarum	Negligible	H	No
Brooklynella hostilis	Wild caught—VL Farmed fish—L	L	No No
Microsporidium seriolae	Juveniles—L Adult—EL–VL	L	No No

s Level of probability: H=high, M=moderate, L=low, VL=very low, EL=extremely low, N=negligible.

b Level of significance: C=catastrophic, H=high, M=moderate, L=low, N=negligible.

c Risk categorisation (see Figure 1.1).

Yes = risk management is required.

No = the risk is acceptable and importation can be permitted without further risk management.

Chapter 8
Risk management: non-salmonid marine finfish

8.1 General principles

THIS CHAPTER CONSIDERS THE RISK management measures required to address the quarantine risks associated with disease agents of non-salmonid marine finfish. The risk assessment for the unrestricted importation of whole, round non-salmonid marine finfish (see Chapter 7) showed that the risk associated with the establishment of some disease agents would not meet Australia's appropriate level of protection (ALOP). The next step was to consider how risk management measures could be implemented to reduce the unrestricted risk to a level that would meet the ALOP.

The general principles of risk management, including the use of measures to address quarantine risks, for salmonid product imported for human consumption are discussed in Sections 5.1 and 5.2. These principles also apply for non-salmonid finfish of higher quality and unit cost that are imported for human consumption. Most imported fish of higher quality (such as salmonids, tuna, yellowtail and flounder) will be consumed by humans and inedible parts will be disposed of via the domestic sewerage or solid waste disposal systems. This pathway presents a negligible probability of disease establishment.

However, this import risk assessment (IRA) also considers the importation of non-salmonid marine finfish that are used for purposes other than for human consumption, including for fish feed and fishing bait. Fish imported for such uses are obviously more likely to introduce disease agents (if present in the fish) into the aquatic environment than product imported for human consumption. Risk factors specific to fish imported for purposes other than human consumption are considered in Section 8.2.

The individual disease assessments in Sections 8.3 onwards consider all uses (ie for human consumption and non-edible uses). Section 8.3 describes the risk management measures proposed for the diseases identified in Section 7.1.2 as requiring assessment with high priority (group 1). Section 8.4 shows the overall measures required for import of eviscerated non-salmonid fish to manage the risks associated with the group 1 diseases.

Finally, diseases identified in Section 7.1.2 requiring assessment with lower priority (group 2) are described in Section 8.5. In Section 8.5, an assessment is made of whether the risk management measures identified in Section 8.4 would also ensure that importation would meet Australia's ALOP for these diseases.

For salmonids, the base commodity considered in the risk assessment is eviscerated salmonids. For non-salmonid marine fish, the base commodity is whole, round fish.

In comparison with salmonids, there is little scientific information on disease in most other marine finfish species. The situation is slightly better for cultured species; however, many have only been farmed for a short time and most of the information available is based on observations and studies in wild fish. The lack of information also extends to current knowledge on the susceptibility of Australia's native marine species to exotic pathogens. The Australian Quarantine and Inspection Service (AQIS) has interpreted data conservatively, having regard to the limitations of current scientific knowledge on finfish diseases and the susceptibility of native marine species to exotic pathogens.

Importation of fish for use as fish feed or bait: considerations relevant to risk management

Figures from the Australian Bureau of Agricultural and Resource Economics (ABARE) show that imports of edible non-salmonid marine fish products totalled 57,944 tonnes (including whole fish, fillets, smoked fish and other preparations) in 1997–98, while non-edible imports (eg for commercial fishing bait, mariculture feed, pet food manufacture) totalled 46,771 tonnes.

There is an established history of importation of non-viable non-salmonid marine fish into Australia but there are no substantiated reports that this practice has resulted in the establishment of disease. There is historical information on the use of imported fish as rock lobster bait in Western Australia (Jones and Gibson 1997). Use of thousands of tonnes of imported fish including pilchards, blue mackerel and herring as lobster bait over several decades has not caused any detectable adverse effect on fish health or the aquatic environment. Jones and Gibson determined that it 'cannot be concluded that there is no risk of introducing an exotic disease only that the risk of introducing an exotic disease that is capable of producing a large-scale fish kill is either very low or does not exist at all.'

Using 'pot lift' data from the Western Australian Fishing Industry Council (WAFIC) report (Jones and Gibson 1997), Hawkins (pers. comm.) reported the results of a beta distribution analysis to calculate the most likely number of disease events. He concluded:

> '…for whatever reasons that there have been no reported significant fish disease events in WA waters associated with the rock lobster industry, it is apparent that the risk of fish disease events associated with imported rock lobster bait is small, and that the process through which imported bait passes probably constitutes a sufficient risk mitigation activity.'

A copy of Hawkins' report may be found at Appendix 9.

Beta analysis is a method of statistical analysis that uses historical data to predict the likelihood of an event occurring. Beta analysis can provide useful information about the relative level of risk, but the limitations of the methodology must also be considered. The applicability of the results to the current, or a future, situation assumes that there are no changes to the risk factors. In the current example, if the disease situation in the source fish population changes (eg new disease agents emerge overseas), environmental conditions in Australia change (eg additional pathogens enter the environment from a new fish farming industry), or the factors that influence the risk of disease establishment in the receiving environment change (eg the density of susceptible species changes when a new aquaculture industry establishes), the results of the beta analysis may no longer be applicable. Also, the analysis may fail to detect low-level mortalities or a disease agent that does not cause overt problems in wild populations.

The importation of pilchards for use as fish feed by the tuna industry is a particular case where scientists have raised concerns that exotic disease (pilchard herpes virus) may have become established by this route.

One submission[1] suggested that the use of pilchards by the tuna aquaculture industry presented a significantly higher risk for domestic baitfish stocks than the use of imported pilchards as recreational or commercial bait. This submission reasoned that, when used as mariculture feed, high volume, repetitive applications of imported pilchards deposit a prolonged residue of tissues in that part of the water column usually frequented by domestic baitfish stocks. In contrast, recreational usage results in very low volume inputs over a widely dispersed geographic area, while commercial bait usage is commonly in a different part of the water column to that normally frequented by domestic baitfish species.

Another submission[2] noted that farmed tuna feed on the surface and management practices ensure that wastage of fish feed is minimised and environmental monitoring does not indicate any local degradation. It was also claimed that it was improbable that the initial reports of pilchard mortalities in 1995 and 1998 were linked to the use of imported pilchards by the tuna aquaculture industry.

Recfish Australia[3] commented that the great scale of the pilchard mortality, in volume and area affected, suggests the exposure of a naive pilchard population to an introduced herpes virus.

The consequences of disease in wild fisheries can be severe. This is illustrated by the pilchard mortalities of 1995–96 and 1998–99, which had a significant adverse effect (Section 1.7) through the loss of stock and the temporary closure of fisheries.

The reduction in domestic pilchard catch, as a consequence of the pilchard mortality, has created a high demand for imported pilchards to sustain the operation of domestic industries.[4]

G Morgan (pers. comm.), Chair of the Consultative Committee for Emergency Animal Diseases (CCEAD) Joint Pilchard Scientific Working Group, notes that there is little doubt within the Working Group, and the broader scientific community, that the primary aetiological agent responsible for the 1995 and 1998 pilchard mortality episodes is a herpes-like virus. The Working Group is currently coordinating a national research program on the pilchard mortality events, with one of the objectives being to determine whether the virus is endemic or exotic and, if exotic, the source of the virus. Results to date do not support any definitive conclusions as to whether the virus was exotic and, if so, the means by which it was introduced. In addition, there is currently no confirmation as to whether the herpes-like virus apparently responsible for the 1995 mortality event is the same as the one apparently responsible for the 1998 event.

It is important to continue research to determine the cause of the disease in pilchards and the source of the agent and to determine if any action, including quarantine or stock management measures, is justified. In this IRA, as indicated in Chapter 6, the pilchard herpes virus associated with the disease outbreak is considered endemic to Australia and there is no evidence to suggest that there are exotic strains of the virus overseas. Thus, the implementation of quarantine measures against this agent is not warranted.

The impact of the introduction of a disease agent can also have severe consequences for aquaculture and impede the development of new industries. A pathogen may not cause significant problems in a wild population, but in an aquaculture environment with high stocking densities, susceptible populations and the potential accumulation of the agent in the local area, can cause severe problems.

8.2 Available quarantine measures

The quarantine measures available to reduce the likelihood that the importation of products would lead to the introduction and establishment of exotic disease agents in Australia are described in Section 5.2. The two

1 Denis Brown, Chairman, NSW Purse Seine Industry Committee, submission dated 29 June 1999.

2 The Tuna Boat Owners' Association of Australia, submission dated 10 June 1999.

3 Recfish Australia, submission dated 5 July, 1999.

4 Denis Brown, Chairman, NSW Purse Seine Industry Committee, submission dated 29 June 1999 and John Glaister, NSW Fisheries, submission dated 14 July, 1999.

principal methods reduce the likelihood of disease agents entering Australia in imported product, and reduce the likelihood that susceptible host species in Australia would be exposed to imported product, or derived waste, likely to transmit disease. These measures can be applied in the country of origin before export and/or in Australia after import to reduce quarantine risk.

8.2.1 PRE-EXPORT REQUIREMENTS

Pre-export requirements aim to reduce the likelihood that fish containing pathogens are exported to Australia and/or to reduce the titre of disease agents likely to occur in such fish. Section 1.6 discusses factors that affect the prevalence of disease agents in imported product. Section 5.2 identifies the pre-export measures that may be applied to finfish imported for human consumption.

In contrast with the relatively high-value fish that are used for human consumption, finfish for use as fish feed or bait may be frozen in large blocks without inspection and with minimal sorting. Such frozen blocks may contain more than one species of fish (for example, shipments of pilchards from the United States have been found to contain a small percentage of mackerel) leading to uncertainty about quarantine risk.

The extent and nature of official control and supervision of fish-processing establishments varies from country to country. The processing of fish intended for use as fish feed or petfood may be subject to few controls apart from those imposed commercially to ensure that the product is fit for its intended end use. Commercial requirements may be satisfied by relatively simple measures, such as sorting and rapid freezing of fish individually or in blocks. Product may also be thermally processed (eg by canning) before export. Cooked fish would generally present a very low quarantine risk because cooking would reduce the viability of any pathogens present and because cooked fish is unlikely to be used as fish feed or bait except under exceptional circumstances.

Fish intended for use as an animal feed or bait is not usually processed individually before export. As noted in Section 1.6, many factory trawlers use seawater, which would have some virucidal and bactericidal properties (B Jones, pers comm, quoting Yamamoto et al 1982) for washing. In some cases, freezing and thawing would affect the viability of pathogens present in the imported fish (ADVS 1999). Some pathogens are inactivated (eg metazoans) while for others the presence of viable agents would be reduced, eg *A. salmonicida*, viral haemorrhagic septicaemia virus (VHSV). However, disease agents may survive longer in frozen fish than at higher temperatures. For disease agents such as infectious pancreatic necrosis virus (IPNV), freezing and thawing may have minimal effect on risk, as they can persist and maintain infectivity in frozen fish. Processes such as gamma irradiation may, at an appropriate dose, effectively inactivate pathogens in imported fish. However, there may be consumer objections to feeding farmed fish on irradiated fish. This concern and the cost of such processing means that large-scale irradiation of imported product is not currently feasible.

Sorting and packaging

To ensure that only fish of a given species are included in a consignment and that this can be verified at import inspection, an importing country may require that fish are individually sorted and individually packaged to facilitate inspection (eg if frozen, fish should be individually frozen or packaged in a shatter-pack).

Export certification

Most exporting countries use inspection and/or auditing of the processing system as the basis for the provision of official certification of the requirements of importing countries. However, fish that are not intended for human consumption may not be subjected to inspection or official certification. An importing country may require that fish have been inspected and official health certification provided to ensure that fish bearing visible lesions associated with infectious disease are excluded from the consignment.

8.2.2 POST-IMPORT MEASURES IN AUSTRALIA

The main aim of managing risk after imported product arrives in Australia is to reduce the likelihood that imported product containing pathogens would enter the aquatic environment and result in susceptible fish being exposed to a dose sufficient to cause infection. Section

1.7 discusses factors that affect the probability of disease agents in imported product establishing. Section 5.2.2 identifies the post-arrival measures that may be applied to salmonid finfish imported for human consumption. A similar range of measures could be applied to non-salmonid finfish imported for human consumption, but many of these measures are not applicable to whole, round fish imported for use as fish feed or bait.

Processing of imported fish generates waste products that could present quarantine risks if they enter the aquatic environment without being treated or diluted. Such waste is most commonly disposed of via the domestic sewerage or solid waste disposal systems and presents a negligible probability of disease establishment.

Progressive risk management strategy

Progressive risk management was suggested by the National Task Force on Imported Fish and Fish Products (Higgins 1996) as an approach to improving consistency in quarantine risk management. AQIS has commenced risk analyses of all the items of concern identified by the Task Force, including pilchards for tuna feed, ornamental fish, rock lobster bait and whole fish for human consumption. As recommended by the Task Force, this risk analysis and that for ornamental fish have provided the basis for the development of quarantine policies that deal with risks consistently for the range of aquatic products considered. The importation of non-salmonid freshwater finfish is under review and will be the subject of a specific import risk analysis (as foreshadowed in Animal Quarantine Policy Memorandum 1998/23).[5]

Approximately 16,354 tonnes of North Sea herring (*C. harengus*) were imported into Western Australia in the period 1989–1997 (Jones and Gibson 1997), and there is no evidence that this practice has resulted in any adverse disease developments. However, the Western Australian rock lobster industry has taken steps to manage risk by importing individually inspected and frozen bait fish. Imports of herring have primarily been from Holland which requires herring to be graded fit for human consumption (B. Jones pers. comm., citing Dr. P. van Banning, pers. comm.). Fish showing evidence of disease (ie clinically infected) would not be exported, thus reducing Australia's exposure to disease agents.

The South Australian tuna industry is developing risk management strategies, including the use of manufactured baits and feeds; however, the development of alternative feeds to a commercial stage is judged to be several years away. The ornamental fish industry has implemented an industry code of practice, which addresses disease concerns and other issues. AQIS is working with the industries to help them identify and address matters of quarantine concern.

8.2.3 GENERAL RISK MANAGEMENT MEASURES

For whole, round fish imported for use as fish feed or bait, some or all of the following general risk management measures could be applied to reduce quarantine risk:

- individual sorting to ensure that only fish of a given species are included in the consignment;
- inspection to remove clinically diseased fish;
- packaging to facilitate import inspection;
- cooking or other treatment;
- further processing under control in Australia; and
- certification as to the inspection or other processing applied to the product.

Moreover, risk could be reduced by restricting the usage and/or distribution of imported fish. For example, under natural conditions, the viruses infectious haematopoietic necrosis virus (IHNV) and viral haemorrhagic septicaemia virus (VHSV) have not been reported to be transmitted when the water temperature is above 15°C. It would be possible, using provisions of the Quarantine Act, to restrict the use of imported products to reduce the risk of disease establishment. AQIS might permit the importation of particular fish species that can carry

5 AQIS (Australian Quarantine and Inspection Service) (5 March 1998), Animal Quarantine Policy Memorandum 1998/23, Work program for aquatic animal quarantine policy review.

these viruses for use as bait only in waters above a certain temperature (eg as a result of season and geographical location).

For species that are susceptible to diseases of quarantine concern (as identified in the risk analysis), and in relation to which AQIS will not generally permit importation for use as fish feed or bait, prospective importers could apply for an import permit. This would be based on a detailed submission setting out the conditions that would prevent the establishment of diseases of concern. AQIS would consider the particular circumstances and, if the proposed conditions of export and/or end use would reduce risk sufficiently to meet Australia's ALOP, would approve the application.

The risk analysis has not identified any significant diseases of salmonids or non-salmonid marine fish in relation to which imported pilchards would pose an unacceptable risk of disease establishment. Therefore, no disease-specific risk management is warranted for imported pilchards at this time. AQIS will keep this situation under review and it may be necessary to introduce disease-specific risk management measures at a later time.

8.3 Risk management for high priority disease agents (group 1)

This section considers the risk management measures that could be applied to address the quarantine risks associated with individual high priority (group 1) disease agents. In the risk assessment in Chapter 7, it was shown that for the unrestricted importation of whole, round non-salmonid marine fish, the risk associated with the establishment of some of the group 1 disease agents would not meet Australia's ALOP. The disease agents for which the importation of whole, round non-salmonid marine finfish of susceptible species do not meet the ALOP were identified as:

- aquabirnaviruses (other than infectious pancreatic necrosis virus);
- infectious pancreatic necrosis virus;
- red sea bream iridovirus;
- viral haemorrhagic septicaemia virus;
- *Aeromonas salmonicida* (typical and atypical strains); and
- *Photobacterium damsela piscicida*.

The next step was to consider for each disease how risk management measures could be implemented to reduce the unrestricted risk to a level that would meet the ALOP.

8.3.1 AQUABIRNAVIRUSES

Risk assessment conclusions for susceptible species[6]

For the unrestricted importation for human consumption of whole, round non-salmonid marine fish of susceptible species the probability of the establishment of pathogenic aquabirnaviruses (eg eel virus European (EVE), halibut birnavirus (HBV), viral deformity virus (VDV) or yellowhead ascites virus (YAV)) respectively) would be low. For juvenile fish of susceptible species, the probability would also be low.

The consequences of establishment would be of low to moderate significance. Thus, for pathogenic aquabirnaviruses (eg EVE, HBV, VDV or YAV), the risk associated with the unrestricted importation of whole, round non-salmonid marine fish (farmed or wild-caught) of susceptible species would not meet Australia's ALOP and the implementation of risk management measures is warranted (see Box 7.1).

Risk assessment conclusions for other species

In Chapter 7, AQIS concluded that for the unrestricted importation of whole, round non-salmonid marine fish of other species, the probability of establishment of EVE, HBV, VDV or YAV would be negligible. From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of EVE, HBV, VDV or YAV in Australia, the risk meets Australia's

6 For EVE, susceptible species are *Anguilla* spp from Japan and Taiwan.
For HBV, susceptible species are *Hippoglossus* spp from Norway and the United Kingdom.
For VDV and YAV susceptible species are *Seriola* spp, *Stephanolepis* spp, *Parapristipoma* spp from Japan.

ALOP and the implementation of specific risk management measures is not warranted (see Box 7.1).

Key risk factors for susceptible species

1. Infection of non-salmonid marine fish with pathogenic aquabirnaviruses is associated with clinical disease, primarily in juvenile fish. Clinical disease has not been reported in commercially-harvested, market-size, non-salmonid marine hosts.
2. In covertly infected fish, visceral tissues would be the main source of virus.
3. Aquabirnaviruses could survive in tissues and in the aquatic environment for a significant period.
4. The use of whole, round fish as bait/fish feed or continuous high-level discharge of EVE, HBV, VDV or YAV into the aquatic environment could result in susceptible species being exposed to a dose of virus sufficient to cause infection.

Risk management measures

The following risk management measures would reduce the risk associated with the establishment of aquabirnaviruses via the importation of whole, round non-salmonid marine fish of susceptible species into Australia.

Inspection

- to remove clinically diseased fish.

Individual sorting and packaging to facilitate inspection

- to allow verification of fish species.

Processing

- removal of the viscera;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- a requirement that the fish were processed in a premises approved by a competent authority.

Export certification

- a requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

These measures would normally apply to higher-value, cultured fish imported into Australia for human consumption.

Inspection

Inspection would allow the detection of fish with clinical disease due to EVE, HBV, VDV or YAV and allow for the identification of fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factor 1.

Individual sorting and packaging to facilitate inspection

These measures would normally apply to higher-value, cultured fish imported into Australia for human consumption. These measures would help to ensure that only fish of a given species are included in a consignment and that this can be verified at import inspection.

Processing of the product

As EVE, HBV, VDV and YAV are usually in the viscera, evisceration would significantly reduce risk.

Inspection would not detect covertly infected fish. Pathogenic aquabirnaviruses could be present in the tissues of such fish, particularly in the viscera. Commonly used commercial processes (evisceration and thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with covertly infected fish.

Such processing would not totally eliminate risk as virus could be present in other tissues.

AQIS could require the processing of susceptible species in the country of export to a specified standard, that is, evisceration and thorough cleaning and washing of internal surfaces. This would substantially address risk factors 2, 3 and 4.

The implementation of specific risk management to address risk factor 4 is not warranted because the other risk management measures identified would effectively prevent the entry of EVE, HBV, VDV or YAV into the aquatic environment.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority.

Measures such as evisceration, removal of the head and gills, and inspection and processing in approved premises would normally apply to higher-value, cultured fish imported into Australia for human consumption. Accordingly, AQIS could introduce a requirement for inspection of non-salmonid marine finfish exported to Australia for human consumption and this would not present a significant impediment to trade. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia had been inspected and that they meet relevant conditions of importation.

Bait or fish feed

The importation of whole, round non-salmonid marine finfish of susceptible species for use as bait or as fish feed could present a significant risk of EVE, HBV, VDV or YAV entering Australia. Such consignments would not normally be inspected before export and could contain clinically infected fish. The entry of tissues such as the viscera into the aquatic environment would increase the likelihood of susceptible Australian finfish species being exposed to a dose of EVE, HBV, VDV or YAV sufficient to cause infection.

AQIS has been unable to identify pre-export risk management measures that would reduce the risk of establishment of EVE, HBV, VDV or YAV to the extent required to meet Australia's ALOP. Accordingly, the importation of whole, round finfish of susceptible species will not generally be permitted.

On a case-by-case basis, AQIS will examine proposals for the mitigation of risk using pre-export or post-importation risk management measures.

AQIS will permit importation in accordance with such proposals, providing it can be shown that the proposed risk management measures would reliably reduce risk as required to meet Australia's ALOP.

Conclusions

Species that are susceptible to infection with EVE, HBV, VDV or YAV

To mitigate risks associated with the importation of whole, round non-salmonid marine fish of susceptible species, AQIS will allow the importation of susceptible species subject to the general conditions shown in Box 8.1.

For EVE, HBV, VDV or YAV, the implementation of these measures singly would reduce the risk, but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 8.1 would meet Australia's ALOP; importation of susceptible species will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supporting scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Other species

For EVE, HBV, VDV or YAV, AQIS will permit the importation of whole, round non-salmonid marine finfish of other species.

Box 8.1
Risk management measures for species that are susceptible to aquabirnaviruses (EVE, HBV, VDV or YAV)

PRE-EXPORT REQUIREMENTS

- The viscera must be removed and internal surfaces thoroughly washed.
- The fish must be individually sorted and packaged to facilitate inspection.
- The fish must be inspected under the supervision of a competent authority.
- The product must be free from visible lesions associated with infectious disease.
- The fish must be processed in a premises approved by and under the control of a competent authority in the country of export.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Other conditions for susceptible species

On a case-by-case basis, AQIS may permit the importation of whole, round non-salmonid marine fish of susceptible species under conditions other than as specified above.

Importers proposing to import fish of susceptible species under other conditions should apply to AQIS for an import permit. The importer should provide details of the finfish species to be imported, the waters in which the fish were farmed (if applicable) and harvested and the intended end use of the imported fish. AQIS would assess the application in light of the quarantine risks it would present. If AQIS concluded that the proposed importation would be consistent with Australia's ALOP, it would grant a permit for the importation of single or multiple consignments during a specified time-frame.

8.3.2 INFECTIOUS PANCREATIC NECROSIS VIRUS

Risk assessment conclusions for susceptible species[7]

For the unrestricted importation for human consumption of whole, round non-salmonid marine fish of susceptible species, the probability of establishment of IPNV would be low to moderate. For product imported for use as bait or fish feed the probability would be moderate. The consequences of establishment of IPNV would be of moderate to high significance.

Thus, for IPNV, the risk associated with the unrestricted importation of whole, round non-salmonid marine fish of susceptible species does not meet Australia's ALOP and the implementation of specific risk management measures is warranted (see Box 7.2).

Risk assessment conclusions for other species

In Chapter 7, AQIS concluded that, for the unrestricted importation of whole, round non-salmonid marine fish of other species, the probability of establishment of infectious pancreatic necrosis virus (IPNV) would be negligible. From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of IPNV in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted (see Box 7.2).

Key risk factors for susceptible species

1. Infection of non-salmonid marine finfish with IPNV is rarely associated with clinical disease. Clinical disease is more common in juvenile fish.
2. In covertly infected non-salmonid finfish, IPNV may be present in tissues including the brain and viscera.
3. IPNV could survive in tissues and in the aquatic environment for a significant period.

7 Susceptible species are *Anguilla* spp, *Paralichthys* spp, *Morone* spp and *Brevoortia* spp from all countries.

4. The use of whole, round fish as bait/fish feed or continuous high level entry of IPNV into the aquatic environment could result in susceptible species being exposed to a dose of IPNV sufficient to cause infection.

Risk management measures

The following pre-export risk management measures would reduce the risk associated with the establishment of IPNV via the importation of whole, round non-salmonid marine fish into Australia.

Inspection

- to remove clinically diseased fish.

Processing

- removal of the viscera;
- removal of the head and gills;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- a requirement that the fish were processed in a premises approved by a competent authority.

Individual sorting and packaging to facilitate inspection

- to allow verification of fish species.

Export certification

- a requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Inspection

Inspection would provide for the detection of fish with clinical disease due to IPNV. Inspection would also identify fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factor 1.

Processing of the product

As IPNV usually localises in the viscera, evisceration would significantly reduce risk.

Removal of the head and gills before importation into Australia would reduce risk, as the head is not normally consumed and is (except for pan-size fish) usually removed before the fish is cooked. Disposal of the head by inappropriate means (such as by use as fishing bait) could present a high risk.

Inspection would not detect covertly infected fish. IPNV could be present in the tissues of such fish, particularly in the viscera and head. Commonly used commercial processes (evisceration, removal of the head and thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with these factors.

Such processing would not totally eliminate risk as virus could be present in other tissues.

AQIS could require the processing of susceptible species in the country of export to a specified standard, that is, evisceration, removal of the head and gills and thorough cleaning and washing of internal surfaces. This would substantially address risk factors 2, 3 and 4.

The implementation of specific risk management to address risk factor 4 is not warranted because the other risk management measures identified would effectively prevent the entry of IPNV into the aquatic environment.

Individual sorting and packaging to facilitate inspection

These measures would normally apply to higher-value, cultured fish imported into Australia for human consumption. These measures would help to ensure that only fish of a given species are included in a consignment and that this can be verified at import inspection.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority.

Measures such as evisceration, removal of the head and gills, inspection and processing in approved premises would normally apply to higher-value, cultured fish imported into Australia for human consumption. Accordingly, AQIS could introduce a requirement for inspection of non-salmonid marine finfish exported to

Australia for human consumption and this would not present a significant impediment to trade. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia had been inspected and that they meet relevant conditions of importation.

Bait or fish feed

The importation of whole, round non-salmonid marine finfish of susceptible species for use as bait or as fish feed could present a significant risk of IPNV entering Australia. Such consignments would not normally be inspected before export and could contain clinically infected fish. The entry of tissues such as viscera or brain into the aquatic environment would increase the likelihood of susceptible Australian finfish species being exposed to a dose of IPNV sufficient to cause infection.

AQIS has been unable to identify pre-export risk management measures that would reduce the risk of establishment of IPNV to the extent required to meet Australia's ALOP. Accordingly, the importation of whole, round finfish of susceptible species will not generally be permitted.

On a case-by-case basis, AQIS will examine proposals for the mitigation of risk using pre-export or post-importation risk management measures.

AQIS will permit importation in accordance with such proposals, providing it can be shown that the proposed risk management measures would reliably reduce risk, as required, to meet Australia's ALOP.

Conclusions

Species that are susceptible to infection with IPNV

To mitigate risks associated with the importation of whole, round non-salmonid marine fish of susceptible species, AQIS would allow the importation of susceptible species subject to the general conditions shown in Box 8.2.

For IPNV, the implementation of these measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 8.2 would meet Australia's ALOP; importation of susceptible species will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supporting scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 8.2

Risk management measures for species that are susceptible to infectious pancreatic necrosis virus

PRE-EXPORT REQUIREMENTS

- The viscera, head and gills must be removed and internal surfaces thoroughly washed.
- The fish must be individually sorted and packaged to facilitate inspection.
- The fish must be inspected under the supervision of a competent authority.
- The product must be free from visible lesions associated with infectious disease.
- The fish must be processed in a premises approved by and under the control of a competent authority in the country of export.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Other conditions for susceptible species

On a case-by-case basis, AQIS may permit the importation of whole, round non-salmonid marine fish of susceptible species under conditions other than as specified above.

Importers proposing to import fish of susceptible species under other conditions should apply to AQIS for an import permit. The importer should provide details of the finfish species to be imported, the waters in which the fish were farmed (if applicable) and harvested and the intended end use of the imported fish. AQIS would assess the application in light of the quarantine risks it would present. If AQIS concluded that the proposed importation would be consistent with Australia's ALOP, it would grant a permit for the importation of single or multiple consignments during a specified time-frame.

Other species

For IPNV, AQIS will permit the importation of whole, round non-salmonid marine finfish of other species.

8.3.3 RED SEA BREAM IRIDOVIRUS

Risk assessment conclusions for susceptible species[8]

For the unrestricted importation for human consumption of whole, round non-salmonid marine fish of susceptible species the probability of establishment of RSIV or closely related iridoviruses would be low and importation of such fish for use as bait or fish feed would also present a low probability. The consequences of establishment would be of low to moderate significance.

Thus, for RSIV or closely related iridoviruses, the risk associated with the unrestricted importation of whole, round non-salmonid marine fish of susceptible species for human consumption or for use as bait or fish feed does not meet Australia's ALOP and the implementation of specific risk management measures is warranted (see Box 7.4).

Risk assessment conclusions for other species

In Chapter 7, AQIS concluded that: for the unrestricted importation of whole, round non-salmonid marine fish of other species the probability of establishment of red sea bream iridovirus (RSIV) or closely related iridoviruses would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of RSIV or closely related iridoviruses in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted (see Box 7.4).

Key risk factors for susceptible species

1. Infection of non-salmonid marine fish with RSIV is often associated with clinical disease. Clinical disease is more common in juvenile fish.
2. RSIV is found in visceral organs (predominantly the spleen and kidney) and gills.
3. RSIV could persist in tissues or in the aquatic environment.
4. The use of whole, round fish as bait or continuous high level entry of RSIV into the aquatic environment could result in susceptible species being exposed to a dose of RSIV sufficient to cause infection.

Risk management measures

The following pre-export risk management measures would reduce the risk associated with the establishment of RSIV via the importation of whole, round non-salmonid marine fish of susceptible species into Australia.

Inspection

- to remove clinically diseased fish.

Individual sorting and packaging to facilitate inspection

- to allow verification of fish species.

Processing

- removal of the viscera;
- removal of the gills;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and

8 Susceptible species are *Epinephelus* spp (eg grouper); *Evynnis* spp (eg crimson sea bream); *Lateolabrax* spp (eg Japanese sea bass); *Oplegnathus* spp (eg Japanese parrotfish); *Pagrus* spp (eg red sea bream); *Paralichthys* spp (eg Japanese flounder); *Pseudocaranx* spp (eg striped jack); *Seriola* spp (eg yellowtail); *Takifugu* spp (eg tiger pufferfish) and *Thunnus* spp (eg albacore) from all Asiatic countries.

- a requirement that the fish are processed in a premises approved by a competent authority.

Export certification

- a requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Inspection

Inspection would provide for the detection of fish with clinical disease due to RSIV. Inspection would also identify fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factor 1.

Individual sorting and packaging to facilitate inspection

These measures would normally apply to higher-value, cultured fish imported into Australia for human consumption. These measures would help to ensure that only fish of a given species are included in a consignment and that this can be verified at import inspection.

Processing of the product

As RSIV usually localises in the viscera and gills, evisceration and removal of the gills would significantly reduce risk.

Inspection would not detect covertly infected fish. RSIV could be present in the tissues of such fish, particularly in the viscera and gills. Commonly used commercial processes (evisceration, removal of the gills and thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with these factors.

Such processing would not totally eliminate risk as virus could be present in other tissues.

AQIS could require the processing of susceptible species in the country of export to a specified standard, that is, evisceration, removal of the gills and thorough cleaning and washing of internal surfaces. This would substantially address risk factors 2, 3 and 4.

The implementation of specific risk management to address risk factor 4 is not warranted because the other risk management measures would effectively prevent the entry of RSIV into the aquatic environment.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority.

Measures such as evisceration, removal of the gills, inspection and processing in approved premises would normally apply to higher-value, cultured fish imported into Australia for human consumption. Accordingly, AQIS could introduce a requirement for inspection of non-salmonid marine finfish exported to Australia for human consumption and this would not present a significant impediment to trade. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia had been inspected and that they meet relevant conditions of importation.

Bait or fish feed

The importation of whole, round non-salmonid marine finfish of susceptible species for use as bait or as fish feed could present a significant risk of RSIV entering Australia. Such consignments would not normally be inspected before export and could contain clinically infected fish. The entry of tissues such as viscera or gills into the aquatic environment would increase the likelihood of susceptible Australian finfish species being exposed to a dose of RSIV sufficient to cause infection.

AQIS has been unable to identify pre-export, risk management measures that would reduce the risk of establishment of RSIV to the extent required to meet Australia's ALOP. Accordingly, the importation of whole, round finfish of susceptible species will not generally be permitted.

On a case-by-case basis, AQIS will examine proposals for the mitigation of risk using pre-export or post-importation risk management measures.

AQIS will permit importation in accordance with such proposals, providing it can be shown that the proposed risk management measures would reliably reduce risk as required to meet Australia's ALOP.

Conclusions

Species that are susceptible to infection with RSIV

To mitigate risks associated with the importation of whole, round non-salmonid marine fish of susceptible species, AQIS will allow the importation of susceptible species subject to the general conditions shown in Box 8.3.

For RSIV, the implementation of these measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 8.3 would meet Australia's ALOP; importation of susceptible species will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supporting scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 8.3

Risk management measures for species that are susceptible to red sea bream iridovirus

PRE-EXPORT REQUIREMENTS

- The viscera and gills must be removed and internal surfaces thoroughly washed.
- The fish must be individually sorted and packaged to facilitate inspection.
- The fish must be inspected under the supervision of a competent authority.
- The product must be free from visible lesions associated with infectious disease.
- The fish must be processed in a premises approved by and under the control of a competent authority in the country of export.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Other conditions for susceptible species

On a case-by-case basis, AQIS may permit the importation of whole, round non-salmonid marine fish of susceptible species under conditions other than as specified above.

Importers proposing to import fish of susceptible species under other conditions should apply to AQIS for an import permit. The importer should provide details of the finfish species to be imported, the waters in which the fish were farmed (if applicable) and harvested and the intended end use of the imported fish. AQIS would assess the application in light of the quarantine risks it would present. If AQIS concluded that the proposed importation would be consistent with Australia's ALOP, it would grant a permit for the importation of single or multiple consignments during a specified time-frame.

Other species

For RSIV, AQIS will permit the importation of whole, round non-salmonid marine finfish of other species.

8.3.4 VIRAL HAEMORRHAGIC SEPTICAEMIA VIRUS

Risk assessment conclusions for susceptible species[9]

For the unrestricted importation for human consumption of whole, round non-salmonid marine fish of susceptible species, the probability of establishment of VHSV would be low. For product imported for use as bait or fish feed the probability would be low to moderate. For herring and sprat (*Clupea* spp) the probability would be higher, but still moderate. The consequences of establishment of VHSV would be of low to moderate significance.

Thus, for VHSV, the risk associated with the unrestricted importation of whole, round non-salmonid marine fish of susceptible species does not meet Australia's ALOP and the implementation of specific risk management measures is warranted. (see Box 7.5).

Risk assessment conclusions for other species

In Chapter 7, AQIS concluded that for the unrestricted importation of whole, round non-salmonid marine fish of other species, the probability of establishment of viral haemorrhagic septicaemia virus (VHSV) would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of VHSV in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted (see Box 7.5).

Key risk factors for susceptible species

1. Clinical disease due to VHSV has been reported in market-size non-salmonid marine finfish.
2. Clinically affected fish would have a high titre of VHSV in infected body tissues (eg skin lesions, viscera and possibly the head).
3. In covertly infected fish VHSV may be present in body tissues, particularly the viscera and possibly the head.
4. VHSV could survive in tissues and in the aquatic environment for several weeks.
5. The use of whole, round fish as bait/fish feed or continuous high-level entry of VHSV into the aquatic environment could result in susceptible species being exposed to a dose of VHSV sufficient to cause infection.

Risk management measures

The following pre-export risk management measures would reduce the risk associated with the establishment of VHSV via the importation of whole, round non-salmonid marine fish of susceptible species.

Inspection

- to remove clinically diseased fish.

Individual sorting and packaging to facilitate inspection

- to allow verification of fish species.

Processing

- removal of the viscera;
- removal of the head and gills;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- a requirement that the fish were processed in a premises approved by a competent authority.

Export certification

- a requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Inspection

Inspection would provide for the detection of fish with clinical disease due to VHSV. Inspection would also identify fish that were not processed in accordance with

9 Susceptible species are species in the Families Gadidae (eg Atlantic cod, haddock, blue whiting); Scophthalmidae (eg turbot); Gasterosteidae (eg tubesnout, three-spined stickleback); Embiotocidae (eg shiner perch); Lotidae (eg rockling); Pleuronectidae (eg dab, plaice); *Clupea* spp (eg herring, sprat) and *Merluccius* spp (hake) from all countries.

Australia's import conditions. This would substantially address risk factors 1 and 2.

Individual sorting and packaging to facilitate inspection

These measures would normally apply to higher-value, cultured fish imported into Australia for human consumption. These measures would help to ensure that only fish of a given species are included in a consignment and that this can be verified at import inspection.

Processing of the product

As VHSV usually localises in the viscera, evisceration would significantly reduce risk.

Removal of the head and gills before importation into Australia would reduce risk, as the head is not normally consumed and is (except for pan-size fish) usually removed before the fish is cooked. Disposal of the head by inappropriate means (such as by use as fishing bait) could present a high risk.

Inspection would not detect covertly infected fish. VHSV could be present in the tissues of such fish, particularly in the viscera and possibly the head. Commonly used commercial processes (evisceration, removal of the head and gills, thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with these factors.

Such processing would not totally eliminate risk as virus could be present in other tissues.

AQIS could require the processing of susceptible species in the country of export to a specified standard, that is, evisceration, removal of the head and gills and thorough cleaning and washing of internal surfaces. This would substantially address risk factors 3, 4 and 5.

The implementation of specific risk management to address risk factors 4 and 5 is not warranted because the other risk management measures identified would effectively prevent the entry of VHSV into the aquatic environment.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority.

Measures such as evisceration, removal of the head and gills, inspection and processing in approved premises would normally apply to higher-value, cultured fish imported into Australia for human consumption. Accordingly, AQIS could introduce a requirement for inspection of non-salmonid marine finfish exported to Australia for human consumption and this would not present a significant impediment to trade. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia had been inspected and that they meet relevant conditions of importation.

Bait or fish feed

The importation of whole, round non-salmonid marine finfish of susceptible species for use as bait or as fish feed could present a significant risk of VHSV entering Australia. Such consignments would not normally be inspected before export and could contain clinically infected fish. The entry of tissues such as viscera or brain into the aquatic environment would increase the likelihood of susceptible Australian finfish species being exposed to a dose of VHSV sufficient to cause infection.

AQIS has been unable to identify pre-export risk management measures that would reduce the risk of establishment of VHSV to the extent required to meet Australia's ALOP. Accordingly, the importation of whole, round finfish of susceptible species will not generally be permitted.

On a case-by-case basis, AQIS will examine proposals for the mitigation of risk using pre-export or post-importation risk management measures.

AQIS will permit importation in accordance with such proposals, providing it can be shown that the proposed risk management measures would reliably reduce risk as required to meet Australia's ALOP.

Conclusions

Species that are susceptible to infection with VHSV

To mitigate risks associated with the importation of whole, round non-salmonid marine fish of susceptible species, AQIS will allow the importation of susceptible species subject to general conditions shown in Box 8.4.

For VHSV, the implementation of these measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 8.4 would meet Australia's ALOP; importation of susceptible species will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supporting scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 8.4
Risk management measures for species that are susceptible to viral haemorrhagic septicaemia virus

PRE-EXPORT REQUIREMENTS

- The viscera, head and gills must be removed and internal surfaces thoroughly washed.
- The fish must be individually sorted and packaged to facilitate inspection.
- The fish must be inspected under the supervision of a competent authority.
- The product must be free from visible lesions associated with infectious disease.
- The fish must be processed in a premises approved by and under the control of a competent authority in the country of export.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Other conditions for susceptible species

On a case-by-case basis, AQIS may permit the importation of whole, round non-salmonid marine fish of susceptible species under conditions other than as specified above.

Importers proposing to import fish of susceptible species under other conditions should apply to AQIS for an import permit. The importer should provide details of the finfish species to be imported, the waters in which the fish were farmed (if applicable) and harvested and the intended end use of the imported fish. AQIS would assess the application in light of the quarantine risks it would present. If AQIS concluded that the proposed importation would be consistent with Australia's ALOP, it would grant a permit for the importation of single or multiple consignments during a specified timeframe.

Other species

For VHSV, AQIS will permit the importation of whole, round non-salmonid marine finfish of other species.

8.3.5 *AEROMONAS SALMONICIDA* (TYPICAL AND ATYPICAL)

Risk assessment conclusions for wild-caught non-salmonid marine fish

In Chapter 7, AQIS concluded: for the unrestricted importation of whole, round wild-caught non-salmonid marine fish for human consumption, or for use as bait or as fish feed, the probability of establishment of *A. salmonicida* (typical) would be extremely low. The consequences of establishment of typical *A. salmonicida* would be of moderate to high significance.

Thus, for typical *A. salmonicida*, the risk associated with the unrestricted importation of whole, round wild-caught non-salmonid marine fish for human consumption, bait or fish feed meets Australia's ALOP and the implementation of specific risk management measures is not warranted (see Box 7.6).

For the unrestricted importation of whole, round wild-caught non-salmonid marine fish for human consumption, or for use as bait or as fish feed, the probability of establishment of *A. salmonicida* (atypical) would be very

low. The consequences of establishment of atypical *A. salmonicida* would be of moderate significance.

Thus, for atypical *A. salmonicida*, the risk associated with the unrestricted importation of whole, round wild-caught non-salmonid marine fish for human consumption, bait or fish feed meets Australia's ALOP and the implementation of specific risk management measures is not warranted (see Box 7.6).

Risk assessment conclusions for farmed non-salmonid marine fish

For the unrestricted importation of whole, round farmed non-salmonid marine fish for human consumption, the probability of establishment of *A. salmonicida* (typical) would be very low. The consequences of establishment of typical *A. salmonicida* would be of moderate to high significance.

Thus, for typical *A. salmonicida*, the risk associated with the unrestricted importation of whole, round farmed non-salmonid marine fish for human consumption does not meet Australia's ALOP and the implementation of specific risk management measures is warranted.

For the unrestricted importation of whole, round farmed non-salmonid marine fish for human consumption, the probability of establishment of *A. salmonicida* (atypical) would be low. The consequences of establishment of atypical *A. salmonicida* would be of moderate significance.

Thus for atypical *A. salmonicida*, the risk associated with the unrestricted importation of whole, round farmed non-salmonid marine fish for human consumption does not meet Australia's ALOP and the implementation of specific risk management measures is warranted (see Box 7.6).

Key risk factors for farmed non-salmonid marine fish

1. Infection of farmed non-salmonid marine finfish with typical strains of *A. salmonicida* is rarely associated with clinical disease. Infection with atypical strains of *A. salmonicida* is occasionally associated with clinical disease.
2. Clinically affected fish would have a significant bacterial titre in infected body tissues, particularly skin lesions and occasionally the viscera.
3. In covertly infected fish, *A. salmonicida* may be present in tissues including the gills, skin mucus and viscera. There is no evidence that *A. salmonicida* occurs in the muscle tissue of covertly infected non-salmonid marine fish.
4. *A. salmonicida* could survive in tissues and in the aquatic environment for a significant period.
5. *A. salmonicida* could accumulate in the aquatic environment as a result of the uncontrolled disposal of waste from commercial processing of imported fish.

Risk management measures

The following pre-export risk management measures would reduce the risk associated with the establishment of *A. salmonicida* via the importation of farmed, whole, round non-salmonid marine fish.

Inspection

- to remove clinically diseased fish.

Individual sorting and packaging to facilitate inspection

- to allow verification of fish species.

Processing

- removal of the viscera;
- removal of the head and gills;
- thorough cleaning and washing of internal and external surfaces to remove remnants of the viscera as far as practicable; and
- a requirement that the fish were processed in a premises approved by a competent authority.

Export certification

- a requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Inspection

Inspection would provide for the detection of fish with clinical disease due to *A. salmonicida*. Inspection would also identify fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factors 1 and 2.

Individual sorting and packaging to facilitate inspection

These measures would normally apply to higher-value, cultured fish imported into Australia for human consumption. These measures would help to ensure that only fish of a given species are included in a consignment and that this can be verified at import inspection.

Processing of the product

In clinically infected fish *A. salmonicida* may be present in various tissues such as gills, viscera, muscle or skin mucus. In contrast with the situation in salmonids, there is no evidence that the pathogen would be present in the muscle of covertly infected non-salmonid marine finfish.

Inspection would not detect covertly infected fish. *A. salmonicida* could be present in tissues such as the gills, viscera or skin mucus. Commonly used commercial processes (evisceration, removal of the head and thorough cleaning and washing of internal and external surfaces to remove visceral remnants and external mucus) would substantially reduce risks associated with these factors.

AQIS could require the processing of susceptible species in the country of export to a specified standard, that is, evisceration, removal of the head and gills and thorough cleaning and washing of internal and external surfaces. This would substantially address risk factors 3, 4 and 5.

The implementation of specific risk management to address risk factors 4 and 5 is not warranted because the other risk management measures identified would effectively prevent the entry of *A. salmonicida* into the aquatic environment.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority.

Measures such as evisceration, removal of the head and gills, and inspection and processing in approved premises would normally apply to higher-value, cultured fish imported into Australia for human consumption. Accordingly, AQIS could introduce a requirement for inspection of non-salmonid marine finfish exported to Australia for human consumption and this would not present a significant impediment to trade. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia had been inspected and that they meet relevant conditions of importation.

Bait or fish feed

The importation of whole, round farmed non-salmonid marine finfish for use as bait or as fish feed could present a significant risk of *A. salmonicida* (typical or atypical strains) entering Australia. Such consignments would not normally be inspected before export and could contain clinically infected fish. The entry of tissues such as gills, viscera or skin mucus into the aquatic environment would increase the likelihood of susceptible Australian finfish species being exposed to a dose of *A. salmonicida* sufficient to cause infection.

AQIS has been unable to identify pre-export risk management measures that would reduce the risk of establishment of *A. salmonicida* (typical or atypical) to the extent required to meet Australia's ALOP. Accordingly, the importation of whole, round farmed non-salmonid marine finfish will not generally be permitted.

On a case-by-case basis, AQIS will examine proposals for the mitigation of risk using pre-export or post-importation risk management measures.

AQIS will permit importation in accordance with such proposals, providing it can be shown that the proposed risk management measures would reliably reduce risk as required to meet Australia's ALOP.

Conclusions

Farmed non-salmonid marine finfish

To mitigate risks associated with the importation of whole, round farmed non-salmonid marine fish AQIS will allow importation subject to the general conditions shown in Box 8.5.

For *A. salmonicida* (typical or atypical strains), the implementation of these measures singly would reduce the risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 8.5 would meet Australia's ALOP; importation of susceptible species will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supporting scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 8.5
Risk management measures for *A. salmonicida*

Note: These risk management measures only apply to farmed non-salmonid marine finfish.

PRE-EXPORT REQUIREMENTS

- The viscera, head and gills must be removed.
- The fish must be individually sorted and packaged to facilitate inspection.
- The internal and external surfaces must be thoroughly washed.
- The fish must be inspected under the supervision of a competent authority.
- The product must be free from visible lesions associated with infectious disease.
- The fish must be processed in a premises approved by and under the control of a competent authority in the country of export.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Other conditions for farmed whole, round non-salmonid marine finfish

On a case-by-case basis, AQIS may permit the importation of farmed, whole, round non-salmonid marine finfish under conditions other than as specified above.

Importers proposing to import farmed finfish under other conditions should apply to AQIS for an import permit. The importer should provide details of the finfish species to be imported, the waters in which the fish were farmed and harvested and the intended end use of the imported fish. AQIS would assess the application in light of the quarantine risks it would present. If AQIS concluded that the proposed importation would be consistent with Australia's ALOP, it would grant a permit for the importation of single or multiple consignments during a specified time-frame.

Wild-caught non-salmonid marine fish

For *A. salmonicida* (typical or atypical strains), AQIS will permit the importation of whole, round wild-caught non-salmonid marine finfish.

8.3.6 *PHOTOBACTERIUM DAMSELA PISCICIDA*

Risk assessment conclusions for susceptible species[10]

For the unrestricted importation for human consumption of whole, round non-salmonid marine fish of susceptible species the probability of establishment would be very low. For product imported for use as bait or as fish feed the probability would be low. The consequences of establishment would be of moderate significance.

Thus, for *P. damsela piscicida*, the risk associated with the unrestricted importation for human consumption of whole, round non-salmonid marine fish meets Australia's ALOP and the implementation of specific risk management measures is not warranted. The risk associated with the unrestricted importation for bait and fish feed of whole, round non-salmonid marine fish of susceptible species would not meet Australia's ALOP and the implementation of specific risk management measures is warranted (see Box 7.7).

10 Susceptible species are *Morone* spp, *Brevoortia* spp, *Mugil* spp, *Seriola* spp, *Mylio* spp, *Pagrus* spp, *Navodan* spp, *Epinephelus* spp, *Sparus* spp, *Scophthalmus* spp, *Solea* spp and *Dicentrarchus* spp from all countries.

Risk assessment conclusions for other species

In Chapter 7, AQIS concluded that for the unrestricted importation for human consumption of whole, round non-salmonid marine fish of other species the probability of the establishment of *P. damsela piscicida* would be negligible.

From the risk management matrix presented in Section 1.5.3, regardless of the consequences of establishment of *P. damsela piscicida* in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted (see Box 7.7).

Key risk factors for susceptible species

1. Infection of non-salmonid marine finfish with *P. damsela piscicida* often results in clinical disease, particularly in farmed fish. Except for non-specific signs of generalised septicaemia (ie in moribund fish) there are usually no external signs of disease.
2. In covertly infected fish, *P. damsela piscicida* would primarily be in the viscera.
3. *P. damsela piscicida* would not persist long term in the marine environment; however, this pathogen may enter a viable but non-culturable state. The epidemiological significance of pathogens in this state is unclear.
4. The use of whole, round fish as bait/fish feed or continuous high-level entry of *P. damsela piscicida* into the aquatic environment could result in susceptible species being exposed to a dose of *P. damsela piscicida* sufficient to cause infection.

Risk management measures

The following pre-export risk management measures would reduce the risk associated with the establishment of *P. damsela piscicida* via the importation of whole, round non-salmonid marine fish of susceptible species into Australia:

Inspection

- inspection to remove clinically diseased fish.

Individual sorting and packaging to facilitate inspection

- individual sorting and individual freezing.

Processing

- removal of the viscera;
- thorough cleaning and washing of internal surfaces to remove remnants of the viscera as far as practicable; and
- a requirement that the fish were processed in a premises approved by a competent authority.

Export certification

- a requirement that consignments exported to Australia are accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Inspection

Inspection would provide for the detection of clinically affected fish that show external signs of disease. Inspection would also identify fish that were not processed in accordance with Australia's import conditions. This would substantially address risk factor 1.

Individual sorting and packaging to facilitate inspection

These measures would normally apply to higher-value, cultured fish imported into Australia for human consumption. These measures would help to ensure that only fish of a given species are included in a consignment and that this can be verified at import inspection.

Processing of the product

As *P. damsela piscicida* usually localises in the viscera, evisceration would significantly reduce risk.

Inspection would not detect covertly infected fish. *P. damsela piscicida* could be present in the tissues of such fish, particularly in the viscera. Commonly used commercial processes (evisceration, thorough cleaning and washing of internal surfaces to remove visceral remnants) would substantially reduce risks associated with these factors.

AQIS could require the processing of susceptible species in the country of export to a specified standard, that is, evisceration and thorough cleaning and washing of internal surfaces. This would substantially address risk factors 2, 3 and 4.

The implementation of specific risk management to address risk factor 4 is not warranted because the risk management measures identified above would effectively prevent the entry of *P. damsela piscicida* into the aquatic environment.

Export certification

To support the provision of certification, AQIS could also require that the fish were processed in a premises approved by and under the control of a competent authority.

Measures such as evisceration, removal of the head and gills, inspection and processing in approved premises would normally apply to higher-value, cultured fish imported into Australia for human consumption. Accordingly, AQIS could introduce a requirement for inspection of non-salmonid marine finfish exported to Australia for human consumption and this would not present a significant impediment to trade. An appropriate measure would be for the competent authority of the exporting country to certify that fish exported to Australia had been inspected and that they meet relevant conditions of importation.

Bait or fish feed

The importation of whole, round non-salmonid marine finfish of susceptible species for use as bait or as fish feed could present a significant risk of *P. damsela piscicida* entering Australia. Such consignments would not normally be inspected before export and could contain clinically infected fish. The entry of tissues such as viscera into the aquatic environment would increase the likelihood of susceptible Australian finfish species being exposed to a dose of *P. damsela piscicida* sufficient to cause infection.

AQIS has been unable to identify pre-export risk management measures that would reduce the risk of establishment of *P. damsela piscicida* to the extent required to meet Australia's ALOP. Accordingly, the importation of whole, round finfish of susceptible species will not generally be permitted.

On a case-by-case basis, AQIS will examine proposals for the mitigation of risk using pre-export or post-importation risk management measures.

AQIS will permit importation in accordance with such proposals, providing it can be shown that the proposed risk management measures would reliably reduce risk as required to meet Australia's ALOP.

Conclusions

Species that are susceptible to infection with P. damsela piscicida

To mitigate risks associated with the importation of whole, round non-salmonid marine fish of susceptible species, AQIS will allow the importation of susceptible species subject to the general conditions shown in Box 8.6.

For *P. damsela piscicida*, the implementation of these measures singly would reduce risk but not to the extent required to meet Australia's ALOP. Implementation of all the measures listed in Box 8.6 would meet Australia's ALOP; importation of susceptible species will therefore be permitted subject to these conditions.

Exporting countries seeking to modify any of these requirements should provide a submission for consideration by AQIS. Proposals for the use of alternative risk reduction measures should include supporting scientific data that clearly explain how the alternative measures would reduce risk to meet Australia's ALOP. AQIS will consider such applications on a case-by-case basis.

Box 8.6

Risk management measures for species that are susceptible to *P. damsela piscicida*

PRE-EXPORT REQUIREMENTS

- The viscera must be removed and internal surfaces thoroughly washed.
- The fish must be individually sorted and packaged to facilitate inspection.
- The fish must be inspected under the supervision of a competent authority.
- The product must be free from visible lesions associated with infectious disease.
- The fish must be processed in a premises approved by and under the control of a competent authority in the country of export.
- Consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

Other conditions for susceptible species

On a case-by-case basis, AQIS may permit the importation of whole, round non-salmonid marine fish of susceptible species under conditions other than as specified above.

Importers proposing to import fish of susceptible species under these other conditions should apply to AQIS for an import permit. The importer should provide details of the finfish species to be imported, the waters in which the fish were farmed (if applicable) and harvested and the intended end use of the imported fish. AQIS would assess the application in light of the quarantine risks it would present. If AQIS concluded that the proposed importation would be consistent with Australia's ALOP, it would grant a permit for the importation of single or multiple consignments during a specified time-frame.

Other species

For *P. damsela piscicida*, AQIS will permit the importation of whole, round non-salmonid marine finfish of other species.

8.4 Overall risk management for whole, round non-salmonid finfish

In Section 8.3, AQIS concluded for specified diseases[11] that the importation of non-salmonid marine finfish of specified, susceptible species would be permitted, subject to one of the following groups of risk management measures:

OPTION 1 (no import permit required)

- the fish must be processed in a premises approved by and under the control of a competent authority;
- the fish must be eviscerated;
- the fish must be individually sorted and packaged to facilitate inspection;
- the fish must be subjected to an inspection system supervised by a competent authority;
- the head and gills must be removed and internal and external surfaces thoroughly washed;
- the product must be free from visible lesions associated with infectious disease; and
- consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

11 For whole, round, commercially-harvested, market-size non-salmonid marine finfish, the disease agents which require specific risk management are: aquabirnaviruses, IPNV, red sea bream iridovirus, *Aeromonas salmonicida* and *Photobacterium damsela piscicida*. For *A. salmonicida*, risk management applies to all farmed marine finfish species but not to wild-caught non-salmonid marine finfish. For disease agents other than *A. salmonicida*, risk management applies only to susceptible species (as specified in Chapter 7).

OPTION 2 (no import permit required)

- AQIS will not require an official health certificate for consumer-ready product that has been processed further than the stage described above.

For the purpose of these policies, consumer ready-product is product that is ready for the householder to cook/consume (as for salmonids, above).

OPTION 3 (import permit required)

- if neither option 1 nor option 2 applies, an importer must obtain a permit from AQIS before importing fish.
- the application for the permit should provide details of the finfish species to be imported (scientific and common names), the waters in which the fish were farmed (if applicable) and harvested and the intended end use of the imported fish.
- AQIS will assess the application in light of the quarantine risks it presents. If the delegate concludes that the proposed importation is consistent with Australia's ALOP, a permit for the importation of single or multiple consignments during a specified timeframe would ordinarily be granted.

This risk management regime is generally more restrictive than that applied previously (historically, the importation of non-salmonid marine finfish into Australia was not the subject of specific quarantine measures). Under previous conditions the importation of non-salmonid freshwater finfish was not the subject of specific quarantine measures; however, this is under review and will be the subject of a specific IRA (as foreshadowed in Animal Quarantine Policy Memorandum 1998/23).[12]

8.5 Risk management for lower priority diseases of non-salmonid marine finfish (group 2)

The next step was to consider whether the application of the general risk management strategies outlined above would address the risk associated with the importation of whole, round non-salmonid marine finfish in relation to the establishment in Australia of the pathogens in group 2 (see Section 7.1.1). The following disease agents were identified in Section 7.1.1 to be of lower priority in the import risk analysis of non-salmonid marine fish (group 2):

- erythrocytic necrosis virus;
- viral encephalopathy and retinopathy virus;
- *Pseudomonas anguilliseptica*;
- *Vibrio salmonicida*;
- *Glugea stephani*; and
- *Goussia gadi.*

Sections 8.5.1 to 8.5.6 consider the expected effect of the general risk management strategies on the risk of establishment of these diseases as a result of the importation of whole, round non-salmonid marine finfish.

8.5.1 ERYTHROCYTIC NECROSIS VIRUS (VIRAL ERYTHROCYTIC NECROSIS)

Release assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- Viral erythrocytic necrosis (VEN) caused by ENV[13] has been reported in Europe, the United States, Canada, and Greenland. Reports suggest that it may also occur in Portugal. ENV is not OIE listed.

12 AQIS (Australian Quarantine and Inspection Service) (5 March 1999), Animal Quarantine Policy Memorandum 1998/23, Work program for aquatic animal quarantine policy review.

13 In this chapter, ENV is defined as the iridovirus that causes VEN. The togavirus which causes erythrocytic inclusion body syndrome (EIBS) in salmonids is considered in Chapter 3.

- Erythrocytic abnormalities associated with ENV infection have been recorded in at least 17 families of marine and anadromous fish including Atlantic cod, Atlantic and Pacific herring, Atlantic salmon and Pacific salmon.
- VEN occurs in species from 23 genera of marine and anadromous fish on the Atlantic coast of the United States to Greenland, in three genera in the Pacific north-west of North America and in four genera in Atlantic waters of Europe.
- The clinical signs of VEN include pallor of the gills and internal organs.
- ENV infects erythrocytes and occurs at a significant titre in haematopoietic tissues (kidney, spleen, liver and the intestinal sub-mucosa).
- Infection is detected by observation of characteristic intra-erythrocytic cytoplasmic inclusion bodies.

AQIS considered further information, summarised below.

The prevalence of infection with ENV may range from 1–90%, depending on species and geographical location of fish (review by Dannevig and Thorud 1999).

In a study of wild-caught herring in Canada the prevalence of infection with ENV was found to be low (3%) at capture but became high (70%) in fish maintained in captivity at high density for five months. Haematocrit values were reduced in infected fish but infection was otherwise inapparent. In herring maintained at moderate density, no clear trend was observed in either the prevalence of ENV infection or mean haematocrit values (Traxler and Bell 1988).

In a detailed study of pathogens of wild-caught Pacific herring in Alaska, ENV was not detected (Marty et al 1998).

Key findings

ENV has a diverse host range in marine fish, including Atlantic and Pacific herring.

In infected fish, erythrocytes and blood-rich organs (viscera, brain) would be the main source of virus. Virus could also be present in somatic musculature.

The prevalence of infection in a population may vary widely.

Pathological changes in infected fish are most prominent in the red blood cells. Externally detectable pathology (eg anaemia) may occur in some infected fish. Such fish would be detected and rejected in the course of inspection for human consumption.

Inspection would not detect subclinically infected fish.

Exposure assessment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

Transmission

- VEN may be vertically transmitted; virus has been detected in yolk-sac fry and alevin of chum salmon.
- Horizontal transmission has been achieved experimentally by water-borne challenge.
- Disease has also been transmitted experimentally by injection of blood and also by injection of liver and kidney tissue homogenates.
- Data on minimum infective dose are lacking.
- Natural infections have been reported in Atlantic, chum, pink, coho and chinook salmon.
- Experimental infections have been induced in brook, brown and rainbow trout.

Agent stability

- ENV survives freezing at –70°C but is inactivated at 60°C for 15 minutes.
- ENV has not been isolated in cell culture; data on its resistance to environmental degradation are lacking.

AQIS considered further information on VEN in non-salmonids, and this is summarised below.

Transmission studies have shown that ENV carried by Pacific herring is capable of infecting chum salmon (*Oncorhynchus keta*) and pink salmon (*O. gorbuscha*) by intraperitoneal injection (MacMillan and Mulcahy 1979).

Key findings

VEN could remain infective in frozen product.

Salmonid and non-salmonid species present in Australia would be susceptible to infection.

Data are lacking on the physicochemical characteristics of ENV. Based on the behaviour of epizootic haematopoietic necrosis virus (EHNV), a related iridovirus present in Australia, ENV can be expected to persist in infective form in infected tissues in the aquatic environment.

ENV has not been reported in Australia, despite ongoing importation of herring for use in marine waters as lobster bait and pilchards as feed for caged tuna. It may be concluded that there are factors mitigating the introduction and establishment of the pathogen by this route.

Consequences of disease establishment

The following points are based on information in previous AQIS reports (DPIE 1995, 1996) and the 1997 report of the New Zealand Government (Stone et al 1997b). These reports contain referenced reviews of the relevant literature.

- ENV does not generally cause high morbidity or mortality; rather it is thought to impair fish health and production. Morbidity and mortality rates are low and outbreaks of clinical disease are often associated with intercurrent infection with other pathogens.

AQIS considered further information, summarised below.

The proportion of erythrocytes with inclusion bodies in naturally infected non-salmonid fish varies from about 35% to 60–80% in herring (review by Dannevig and Thorud 1999).

The impact of VEN in susceptible species (eg herring, chum salmon) is unclear. Anaemia occurs in some infected salmonids but has not been documented in experimental studies (Noga 1996). Infection with ENV may increase susceptibility to infection with other pathogens (Meyers and Winton 1995).

Key findings

ENV has been reported in a wide number of fish species, based on identification of the pathogen in erythrocytes.

The impact of ENV infection in susceptible species is uncertain; anaemia has been reported in some infected fish.

The establishment of ENV would be likely to impair fish health and production and would not be likely to result in significant mortality. The most significant impact would be expected in susceptible marine fish maintained in culture. Due to the diverse host range of ENV, most marine fish maintained in culture in Australia (eg Atlantic salmon, barramundi, snapper) would be susceptible to infection.

The significance of the establishment of ENV on wild fish stocks in Australia is uncertain. In North America, occasional epizootic mortality has been reported in Pacific herring (*Clupea harengus*) infected with ENV. The only *Clupea* species present in Australia is the southern sprat (*C. bassensis*). Although southern sprat may have a similar susceptibility to infection with ENV as Pacific herring, there is no reason to expect that the establishment of ENV would have a significant impact on the survival of that species or any vulnerable or endangered finfish species in Australia.

Unrestricted risk estimate

For the unrestricted importation of whole, round non-salmonid marine fish for human consumption (farmed or wild-caught), the probability of the establishment of ENV would be low. For whole, round wild-caught non-salmonid marine fish for use as bait or fish feed, the probability would be moderate. The consequences of establishment would be of low significance.

Thus, for ENV, the risk associated with the unrestricted importation of whole, round non-salmonid marine fish for human consumption (farmed or wild-caught) or wild-caught for bait or fish feed meets Australia's ALOP and the implementation of risk management measures is not warranted.

8.5.2 VIRAL ENCEPHALOPATHY AND RETINOPATHY VIRUS

Infection with viral encephalopathy and retinopathy virus (VERV) causes epizootic disease characterised by high mortality rates in larvae and juvenile fish of several marine species. Infection has been reported, rarely, in older fish. This disease is known as viral encephalopathy

and retinopathy (VER) or viral nervous necrosis (VNN). Fish in fresh water and seawater may be infected. VERV has been reported in turbot (*Scophthalmus maximus*), striped jack (*Pseudocaranx dentex*), redspotted grouper (*Epinepheles akaara*), halibut (*Hippoglossus hippoglossus*), European sea bass (*Dicentrarchus labrax*) and barramundi (*Lates calcarifer*). Farmed fish of these species are higher-quality fish and are normally imported for human consumption as inspected, eviscerated carcases or as further processed product. Larval and juvenile fish are not usually harvested for human consumption, thus, there would be a negligible probability of fish with clinical disease entering Australia via the unrestricted importation of whole, round non-salmonid marine fish. The importation of adult, whole, round non-salmonid marine fish would be very unlikely to result in the entry of fish containing a significant titre of VERV into the aquatic environment in Australia.

VERV has been detected in Australia in association with mass mortality in hatchery-raised larval and juvenile barramundi (Munday et al 1992). There is no national control policy on VER. Some states impose interstate movement restrictions on live barramundi (and other species) with respect to VERV; fingerlings must test negative for VERV before entry. There are no restrictions on the movement of non-viable barramundi or other species.

Unrestricted risk estimate

The probability of additional (exotic) strains of VERV entering Australia as a consequence of the unrestricted importation of whole, round non-salmonid marine finfish would be negligible. The agent occurs rarely in adult fish of susceptible species that are imported for human consumption, and fish of these species are not imported as larvae or fry. Therefore, the probability of establishment of disease would also be negligible.

From the risk management matrix shown in Figure 1.1, regardless of the consequences of establishment of additional (exotic) strains of VERV in Australia, the risk meets Australia's ALOP and the implementation of specific risk management measures is not warranted.

8.5.3 *PSEUDOMONAS ANGUILLISEPTICA*

Infection with *Pseudomonas anguilliseptica* causes serious disease known as 'red spot' (haemorrhagic ulceration) in pond-cultured eels (*Anguilla japonica*) in Japan (Austin and Austin 1993). This pathogen has been isolated from other non-salmonid finfish including Baltic herring (*Clupea harengus membras*) (Lonnstrom et al 1994), sea bream (*Pagrus aurata*), turbot (*Scophthalmus maximus*) and European eel (*A. anguilla*) (review by Humphrey 1995). A recent review (Daly 1999) reported natural infection in grouper (*Epinephelus tauvina*) and barramundi (*Lates calcarifer*). *Pseudomonas anguilliseptica* has also been isolated from Atlantic salmon (*Salmo salar*), sea trout (*Salmo trutta*) and rainbow trout (*Oncorhynchus mykiss*) in Finland (Wiklund and Bylund 1990). Finland seems to be the only country where this bacterium has been frequently isolated from salmonids; however, in 50% of the cases, there were concurrent infections with *Vibrio anguillarum* and *Aeromonas salmonicida* making an assessment of each species' contribution to disease difficult (Wiklund and Lonnstrom 1994).

The pathogen causes characteristic pathological changes in all susceptible finfish, viz, predominantly petechial haemorrhage of the skin, peritoneum and liver. Liquefactive necrosis of the kidney has also been recorded (review by Daly 1999). In clinically infected fish, most pathogens would be located in the viscera.

Disease due to infection with *P. anguilliseptica* has been reported from Finland, France, Malaysia, Japan, Scotland and Taiwan (review by Daly 1999).

All of the marine finfish species mentioned above except herring are higher-value, farmed species that are normally imported for human consumption as inspected, eviscerated carcases or as further processed product. Clinically infected fish would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. Evisceration would reduce the number of *P. anguilliseptica* present in product. Subclinical infection has not been described in naturally infected fish.

Baltic herring could be imported for bait or fish feed. This may present a higher probability of *P. anguilliseptica* entering Australia, since such fish are not normally

individually inspected or eviscerated. However, the single published study of *P. anguilliseptica* in herring reports the isolation of the bacterium from eye lesions of 50% of affected animals, whereas isolation from internal organs was unsuccessful in all cases, suggesting that the titre of bacteria present (if any) was very low.

P. anguilliseptica has not been reported in Australia, despite ongoing importation of herring for use in marine waters as lobster bait and pilchards as feed for caged tuna. It may be concluded that there are factors mitigating the introduction and establishment of the pathogen by this route.

Unrestricted risk estimate

The probability of establishment of disease due to *P. anguilliseptica* as a consequence of the unrestricted importation for human consumption of whole, round non-salmonid marine fish would be very low. For fish imported for bait or fish feed (eg Baltic herring), the probability would also be very low. The consequences of establishment would be of low to moderate significance.

Thus, for *P. anguilliseptica*, the risk associated with the unrestricted importation of whole, round non-salmonid marine fish would meet Australia's ALOP and the implementation of risk management measures is not warranted.

8.5.4 *VIBRIO SALMONICIDA* (HITRA DISEASE)

The following points are based on information in previous AQIS reports (DPIE 1995, 1996), the 1997 report of the New Zealand Government (Stone et al 1997b) and Chapter 6 of this report. These documents contain referenced reviews of the relevant literature.

- Infection with *V. salmonicida* may cause cold-water vibriosis or 'Hitra disease'.
- Natural infections have been found in Atlantic salmon, rainbow trout, Atlantic cod (*Gadus morhua*) and coal fish (*Gadus virens*). Non-salmonids appear to be more resistant to disease than salmonids.
- *V. salmonicida* is widespread in North America, Norway, Scotland, Iceland and the Faroe Islands.
- Disease caused by *V. salmonicida* is characterised by severe haemorrhage and necrosis of the internal organs. Clinically infected fish display deep-seated muscle lesions and tissue necrosis. In fish with chronic infections, muscle lesions may be replaced by scar tissue.
- Outbreaks of clinical disease are restricted to fish in seawater or brackish water.
- In clinically diseased fish *V. salmonicida* may be present throughout the vascular system and may be found in the heart, intestine, blood, liver, kidney, spleen, muscle and faeces.
- This agent has been shown to survive in the marine environment for more than 14 months. It cannot be grown at temperatures above 22°C.
- Mortality rates up to 95% have been recorded. However, the introduction of an effective vaccine (administered by immersion, in food or by injection) and the use of antibiotics has significantly reduced the incidence of disease and associated mortality rates.

V. salmonicida has a limited host range (only reported in *Gadus* spp in the north Atlantic) in non-salmonid fish. The species that are infected are higher quality fish that are normally imported for human consumption as inspected, eviscerated carcases or further processed product.

Clinically and chronically affected fish would be visibly abnormal and would be detected and rejected in the course of inspection for human consumption. Carrier fish would appear normal and would not be detected at inspection. In apparently healthy eviscerated finfish, the titre of bacteria, if any were present, would be extremely low (probably undetectable by standard diagnostic methods).

Unrestricted risk estimate

V. salmonicida is found in similar hosts to those that may be infected with typical *A. salmonicida* and viral haemorrhagic septicaemia virus. The risk management that has been applied to non-salmonid fish in relation to these other diseases would also address the risk associated with *V. salmonicida*.

Accordingly, no additional disease-specific risk management measures are warranted for *V. salmonicida.*

8.5.5 *GLUGEA STEPHANI*

Glugea stephani may cause significant disease in cultured and wild marine finfish stocks (Noga 1996, Sinderman1990). Infection with *G. stephani* has been reported in 11 species of flatfish in the northern hemisphere (Europe and the North Atlantic) (Sindermann 1990, Lom and Dykova 1992). Infection has only been reported in flatfish species that are not present in Australia, although members of the same family (Pleuronectidae; eg greenback flounder, *Rhombosolea tapirina*) are present in Australia.

G. atherinae has been reported in marine fishes (atherinids) in Tasmania (X Su, pers. comm.). Several *Glugea* spp have been reported as incidental findings in pilchards and galaxids in Australia (Langdon 1992); however, *G. stephani* has not been reported in Australia (review by Humphrey 1995). There are no quarantine restrictions on the movement of live or non-viable finfish on account of infestation with *Glugea* spp.

In infested fish the parasite only occurs in the visceral tissues. Members of the Family Pleuronectidae are generally high quality fish that are normally traded as inspected, eviscerated fish for human consumption. As species in the Family Pleuronectidae are not imported as whole, round fish (Food Factotum 1999), the probability of *G. stephani* entering Australia would be extremely low.

Glugea stephani infests a limited range of non-salmonid marine fish. The establishment of *G. stephani* in Australia would be of significantly lower consequence than the establishment of any of the diseases in group 1.

Unrestricted risk estimate

The probability of *G. stephani* becoming established in Australia as a consequence of the unrestricted importation of whole, round non-salmonid marine fish (farmed or wild-caught) would be extremely low. The consequences of the establishment of *G. stephani* in Australia would be of low significance.

Thus, for *G. stephani* the risk associated with the unrestricted importation of whole, round non-salmonid marine fish (farmed or wild-caught) meet Australia's ALOP and the implementation of risk management measures is not warranted.

8.5.6 *GOUSSIA GADI*

Goussia gadi infests the swimbladder of gadoid fish (eg haddock). Heavy infestation may result in death of the host due to dysfunction of the swim bladder (Sinderman 1990, Lom and Dykova 1992).

More than 69 species of *Goussia* have been described; six have been reported in Australia (review by Humphrey 1995). *G. auxidis* has a wide distribution and has been identified in liver, spleen and kidney tissue of pelagic fish in the South Pacific Ocean (Jones 1990). It does not generally cause serious disease. *G. auxidis* has not been reported and may or may not be present in Australian waters.

G. gadi has a relatively wide geographic distribution in the northern hemisphere. Infestation with *G. gadi* has been reported in gadoid fish from the North and Baltic Seas and the North Atlantic Ocean (*Gadus* spp, *Melanogrammus* spp and exceptionally in *Enchelyopus* spp); there are no members of the Family Lotidae and no commercially significant members of the Family Gadidae present in Australia. *G. gadi* has not been reported in Australia.

In infested fish the parasite only occurs in the swim bladder. Members of the Families Gadidae and Lotidae are generally high quality fish that are normally inspected and eviscerated for human consumption. As species in the Families Gadidae and Lotidae are not imported as whole, round fish (Food Factotum 1999), the probability of *G. gadi* entering Australia would be extremely low.

G. gadi has a limited host range in non-salmonid marine fish. The establishment of *G. gadi* in Australia would be of significantly lower consequence than the establishment of any diseases in group 1.

Unrestricted risk estimate

The probability of *G. gadi* becoming established in Australia as a consequence of the unrestricted importation of whole, round non-salmonid marine fish would be extremely low. The consequences of the establishment of *G. gadi* in Australia would be of low significance.

Thus, for *G. gadi*, the risk associated with the unrestricted importation of whole, round non-salmonid

marine fish meets Australia's ALOP and the implementation of risk management measures is not warranted.

8.6 Non-salmonid marine finfish from New Zealand

New Zealand is by far the largest single supplier of non-salmonid marine finfish products for human consumption to Australia. A wide range of species are supplied including snapper, bluenose trevalla, John Dory, black oreo dory, barracouta, sand flounder and hoki (blue grenadier) (Food Factotum 1999).

The New Zealand fish fauna is derived from three faunal groups; a large Australasian component, a small Indo-Pacific component and a minor Antarctic component (McDowall 1979). Tagging studies have confirmed that there is movement between Australia's and New Zealand's continental shelf areas of fish and other aquatic species; thus, it is not surprising that the known marine diseases and parasites of New Zealand fish and shellfish are also very closely related to those found on the Australian continental shelf (Jones 1996). For example, a study by Jones (1988) showed that the New Zealand parasitic copepod fauna was derived from the Australian fauna (Jones 1996).

Many populations of marine fish are common to Australia and New Zealand; others, particularly juveniles, may migrate with prevailing currents between Australia and New Zealand. Pelagic fish that occur in both Australian and New Zealand waters include skipjack tuna, bigeye tuna, yellow fin tuna, striped marlin, blue marlin, black marlin and yellow tail finfish. Pilchards in Australia and in New Zealand are the same species, and have been studied for parasites and diseases in both countries, particularly during the pilchard mortalities of 1995 (Jones 1996).

The risk assessment identified no significant current differences in the health status of non-salmonid marine finfish in the waters of New Zealand and Australia.

AQIS has also considered the possibility that new disease agents, not considered in the risk analysis, may emerge in New Zealand's finfish populations. Australia and New Zealand generally collaborate closely on matters of animal and fish health and quarantine. AQIS recognises the competence of the New Zealand Ministry of Agriculture and Forestry (MAF) in matters relating to fish health, inspection and certification, including surveillance and monitoring of the health of fish populations. In the course of a risk analysis of the importation of non-viable salmonids from New Zealand, AQIS inspected fish processing plants and reviewed the fish health status and monitoring and surveillance of salmonid health in New Zealand (see Appendix 3). MAF has also provided detailed information on the surveillance and monitoring of salmonid health in New Zealand (see Appendix 4), and there is frequent contact between quarantine and health officials of Australia and New Zealand.

Moreover, New Zealand is a member of many Ministerial Councils with overall policy responsibility for fisheries, aquaculture and resource management, and the committees that report to them, such as the Veterinary Committee, the Standing Committee of Fisheries and Aquaculture and the Fish Health and Environment Committee. Thus, Australia would be quickly informed of any significant changes to New Zealand's aquatic animal health status.

AQIS will not require an import permit for non-viable marine finfish caught in or adjacent to New Zealand's Exclusive Economic Zone (EEZ) by fishers approved/registered under controls administered by a government authority of New Zealand. However, consignments of such fish would have to be accompanied by official certification stating that:

- the fish or fish from which the product was derived were caught in New Zealand's EEZ or in adjacent international waters; and
- the consignment is product of New Zealand.

The remainder of the policies set out in this report do not apply to non-salmonid marine finfish from New Zealand.

8.7 Summary of risk management measures for importation of non-salmonid marine finfish

A summary of risk management measured required for importation of non-salmonid marine finfish is shown in Table 8.1.

Table 8.1

Summary of quarantine requirements for disease agents of quarantine concern in non-salmonid marine finfish products[a]

DISEASE AGENT	INDIVIDUALLY SORTED AND PACKAGED	EVISCERATION	INSPECTION	DEHEADING	WASH	CERTIFICATION	APPROVED PREMISES
Aquabirnaviruses	✓	✓	✓	✓	✓	✓	✓
Infectious pancreatic necrosis virus	✓	✓	✓	✓	✓	✓	✓
Red sea bream iridovirus	✓	✓	✓	✓	✓	✓	✓
Viral haemorrhagic septicaemia virus	✓	✓	✓	✓	✓	✓	✓
Aeromonas salmonicida	✓	✓	✓	✓	✓	✓	✓
Photobacterium damsela piscicida	✓	✓	✓	×	✓	✓	✓

✓ = risk management measure applies;

× = risk management measure not required.

a These requirements do not apply to product of New Zealand origin. Non-salmonid product of NZ origin may be imported into Australia provided it is accompanied by a certificate of origin from the competent authority . Where product is not of NZ origin and does not meet these requirements importers will need to apply to AQIS for an import permit.

Part 4
Conclusions

Chapter 9
General conclusions

9.1 Outcome of the risk analyses

THIS REPORT DEALS WITH THE IDENTIFICATION, assessment and management of quarantine risks associated with the importation from all countries of eviscerated non-viable salmonids and non-viable, uneviscerated, marine finfish. The importation of eviscerated salmonids was considered in Part 2 (Chapters 3, 4 and 5) and the importation of whole, round non-salmonid finfish was covered in Part 3 (Chapters 6, 7 and 8). This chapter sets out the general conclusions of this report, notes the conclusions of a parallel import risk analysis (IRA) of live ornamental finfish and explains how the two reports together address the outcome of the World Trade Organization (WTO) salmon case (see Chapter 1, Section 1.1, Introduction).

The objective of the Australian Quarantine and Inspection Service (AQIS) is to adopt quarantine policies that provide the animal and plant health safeguards required by government policy in the least trade-restrictive way. Wherever appropriate, measures are based on relevant international standards. In developing quarantine policies, the disease risks associated with importations are analysed using a structured, transparent and science-based process of import risk analysis (IRA).

As prescribed in the *Quarantine Act 1908*, the Director of Animal and Plant Quarantine may permit the entry of products on an unrestricted basis or subject to compliance with conditions, which are normally specified on a permit. A risk analysis provides the scientific and technical basis for quarantine policies that determine whether an import may be permitted and, if so, the conditions to be applied. In practice, specific protocols have been established for a minority of imported aquatic animal products; most enter under standard conditions based on decisions of long standing.

The matters to be considered when deciding whether to issue a permit include the quarantine risk, and whether the imposition of conditions would be necessary to limit the quarantine risk to an acceptably low level consistent with Australian Government policy.

These risk analyses (this report and the ornamental fish import risk assessment report: AQIS 1999) provide a scientific and technical basis for AQIS to amend the

conditions that currently apply to the importation of non-viable salmonids, non-viable non-salmonid marine finfish and live ornamental finfish. In keeping with the scope of the *Quarantine Act 1908*, only the factors relevant to the evaluation of quarantine risk (ie the risk associated with the entry, establishment and spread of unwanted pests and diseases) are considered in the risk analyses.

Equivalent approaches to managing risk may be accepted, generally or on a case-by-case basis. Exporting countries seeking to use alternative risk reduction measures should provide a submission for consideration by AQIS; such proposals should include supporting scientific data that clearly explain the degree to which alternative measures would reduce risk.

9.1.1 IMPORT RISK ANALYSIS FOR NON-VIABLE SALMONIDS AND NON-SALMONID FINFISH

The IRA for non-viable salmonids and non-salmonid finfish concluded that the importation of non-viable finfish would be permitted, subject to risk management measures to reduce the probability of entry and establishment of specified diseases to an acceptably low level. The diseases of concern are those identified in the risk analysis as requiring risk management to meet Australia's appropriate level of protection (ALOP). For eviscerated, commercially-harvested, market-size salmonids,[1] the disease agents that require specific risk management are:

- infectious haematopoietic necrosis virus (IHNV);
- infectious salmon anaemia virus (ISAV) (for Atlantic salmon);
- *Aeromonas salmonicida* (not for wild, ocean-caught Pacific salmon);
- *Renibacterium salmoninarum*; and
- *Myxobolus cerebralis* (for rainbow trout).

As these diseases are either not reported in New Zealand or (for *M. cerebralis*) occur at extremely low prevalence in New Zealand Pacific salmon, these measures would not apply to imports of Pacific salmon from New Zealand.

For whole, round, commercially-harvested, market-size non-salmonid finfish, the disease agents that require specific risk management are:

- aquatic birnaviruses (aquabirnaviruses);
- infectious pancreatic necrosis virus (IPNV);
- viral haemorrhagic septicaemia virus (VHSV);
- red sea bream iridovirus;
- *Aeromonas salmonicida*; and
- *Photobacterium damsela piscicida*.

For *A. salmonicida*, risk management applies to all farmed (but not to wild-caught) non-salmonid marine finfish species. For all other disease agents, risk management applies only to the susceptible species specified in Chapter 7.

Measures affecting the importation of non-viable salmonids and non-salmonid marine finfish into Australia

As this analysis considers the risks associated with the establishment of individual disease agents, the status of each exporting country (or source of fish if not exported from the country of origin) with respect to the diseases of concern will determine the risk management measures to be applied to fish exported from that country.

Exporting countries may provide an official statement of freedom from one or more of the disease(s) of concern, based on the results of a program of monitoring and surveillance of the health of farmed fish that is recognised by AQIS. A competent authority that is recognised by AQIS should provide this statement.

If the exporting country does not provide certification attesting to the freedom of source populations from the disease(s) of concern, countries may still export non-viable salmonids and non-salmonid marine finfish to Australia by complying with the following risk management measures, as appropriate to the health

1 AQIS will not generally permit the importation of juvenile salmonids and sexually mature adult salmonids (spawners) as this would present an unacceptably high quarantine risk for certain disease agents (specified in Chapter 5).

status of relevant fish populations in the exporting country. The implementation of these measures would mitigate the risk of establishment in Australia of diseases of quarantine concern and so maintain consistency with Australia's ALOP.

Measures for non-viable salmonids

As stated in Chapter 1, the starting point of the risk analysis for salmonid products is the product that is traded internationally; ie eviscerated salmon.
As warranted by the analysis in Chapters 4 and 5, non-viable salmonid fish may be imported subject to the following risk management measures:

- the fish must be derived from a population for which there is a documented system of health monitoring and surveillance administered by a competent authority;
- the fish must not be derived from a population slaughtered as an official disease control measure;
- for countries in which infectious salmon anaemia (ISA) occurs[2] Atlantic salmon must not come from a farm known or officially suspected of being affected by an outbreak of ISA;
- the fish must not be juveniles;
- the fish must not be sexually mature salmonids (spawners) (not from New Zealand);
- the fish must be processed in premises approved by and under the control of a competent authority;
- the head and gills must be removed and internal and external surfaces thoroughly washed;
- the product must be free of visible lesions associated with infectious disease;
- the fish must be subjected to an inspection and grading system supervised by a competent authority; and
- consignments exported to Australia must be accompanied by official certification confirming that the exported fish fully meet Australia's import conditions.

Product derived from non-viable salmonids meeting these conditions will be released from quarantine if imported in consumer-ready form.

In this risk analysis, the following products are considered to be 'consumer-ready':

- cutlets — including central bone and external skin but excluding fins— of less than 450g in weight;
- skinless fillets — excluding the belly flap and all bone except the pin bones of any weight;
- skin-on fillets — excluding the belly flap and all bone except the pin bones — of less than 450g in weight; and
- eviscerated, headless 'pan-size' fish of less than 450g in weight; and
- product that is processed further than the stage described above.

Imported head off, gilled and gutted salmonids or skin-on salmonid product of greater than 450g in weight (ie not consumer-ready) must be processed to consumer-ready form in premises approved by AQIS before release from quarantine.

These conditions cover the importation of uncooked salmonids from any country that meets Australia's quarantine requirements. Previously, cooked (smoked or canned) salmonids or salmonid roe could be imported. The former conditions for smoked salmonids will be withdrawn, but imports under these conditions will be permitted until further notice. The conditions for salmonid roe (AQIS currently requires washing and pasteurisation of such products) will be maintained pending validation of the time/temperature of the thermal treatments currently required. The conditions for canned salmon remain unchanged.

Measures for non-viable non-salmonid marine finfish

As stated in Chapter 1, for non-viable, non-salmonid marine finfish, the starting point of the risk analysis is the product that is traded internationally; ie whole, round (uneviscerated) fish. AQIS is introducing new restrictions that reflect quarantine risk associated with this

2 As at July 1999, ISA has been reported from Scotland, Norway and Canada.

commodity. As warranted by the analysis in Chapters 7 and 8, non-viable non-salmonid marine finfish may be imported subject to one of the following groups of risk management measures.

OPTION 1 (no import permit required):

- the fish must be processed in a premises approved by and under the control of a competent authority;
- the fish must be eviscerated;
- the fish must be individually sorted and packaged to facilitate inspection
- the fish must be subjected to an inspection system supervised by a competent authority;
- the head and gills must be removed and internal and external surfaces thoroughly washed;
- the product must be free from visible lesions associated with infectious disease; and
- consignments exported to Australia must be accompanied by official certification confirming that the exported fish meet Australia's import conditions in full.

OPTION 2 (no import permit required):

- AQIS will not require an official health certificate for consumer- ready product that has been processed further than the stage described above.
  (For the purpose of these policies, consumer ready-product is product that is ready for the householder to cook/consume; as for salmonids, above.)

OPTION 3 (import permit required):

- if neither option 1 nor option 2 applies, an importer must obtain a permit from AQIS before importing fish;
- the application for the permit should provide details of the finfish species to be imported (scientific and common names), the waters in which the fish were farmed (if applicable) and harvested and the intended end use of the imported fish; and
- AQIS will assess the application in light of the quarantine risks it presents; if the delegate concludes that the proposed importation is consistent with Australia's ALOP, a permit for the importation of single or multiple consignments during a specified timeframe would ordinarily be granted.

The importation of non-salmonid marine finfish products will be permitted to continue in the interim under the existing conditions, pending the completion of administrative arrangements to provide for implementation of the new policies. As the opportunity arises, AQIS may conduct technical consultations with exporting countries to confirm compliance with the new policies.

This risk management regime is generally more restrictive than that applied previously (historically, the importation of non-salmonid marine finfish into Australia was not the subject of specific quarantine measures). Under previous conditions the importation of non-salmonid freshwater finfish was not the subject of specific quarantine measures; however, this is under review and will be the subject of a specific IRA (as foreshadowed in Animal Quarantine Policy Memorandum 1998/23).[3]

Measures for non-viable non-salmonid marine finfish from New Zealand

AQIS will not require an import permit for non-viable marine finfish caught in or adjacent to New Zealand's Exclusive Economic Zone (EEZ) by fishers approved/registered under controls administered by a government authority of New Zealand. However, consignments of such fish would have to be accompanied by official certification stating that:

- the fish or fish from which the product was derived were caught in New Zealand's EEZ or in adjacent international waters; and
- the consignment is product of New Zealand.

3 AQIS (Australian Quarantine and Inspection Service) (5 March 1998), Animal Quarantine Policy Memorandum 1998/23, Work program for aquatic animal quarantine policy review.

The remainder of the policies set out in this report do not apply to non-salmonid marine finfish from New Zealand.

9.1.2 IMPORT RISK ANALYSIS FOR LIVE ORNAMENTAL FINFISH

The risk analysis on live ornamental finfish concluded that the importation of live ornamental finfish on Schedule 6 should be permitted, subject to risk management measures to mitigate the probability of entry and establishment in Australia of diseases of quarantine concern. Diseases of quarantine concern, those identified as requiring specific risk management, include goldfish haematopoietic necrosis virus, iridoviruses of freshwater ornamental finfish, spring viraemia of carp virus, *Aeromonas salmonicida* ('typical' strains and exotic 'atypical' strains), *Dactylogyrus vastator* and *D. extensus, Argulus foliaceus* and *A. coregoni,* and *Lernaea elegans*.

Live animals generally present a greater risk than product and there are significant gaps in knowledge of the diseases of ornamental finfish. Accordingly, AQIS will supply baseline risk management measures to all ornamental finfish imported. The measures for goldfish are consistent with the higher risks presented by that species.

As warranted by the conclusions of the risk analysis, each consignment of ornamental finfish must be accompanied by:

- an animal health certificate from the competent authority attesting to the health of the fish in the consignment and the health status of the source population;
- certification from a competent authority that the premises of export or exporter are currently approved for export to Australia; and
- certification from a competent authority attesting that the fish had not been kept in water in common with farmed food fish.

Each consignment must also be subject to:

- visual inspection of all fish on arrival to identify overtly diseased consignments and to ensure that the fish are of a species listed on Schedule 6;
- post-arrival quarantine detention for a minimum period in approved private facilities under quality assurance arrangements agreed with AQIS (the minimum period of quarantine will be three weeks for goldfish and one week for all other Schedule 6 listed finfish); and
- quarantine security over procedures in quarantine premises, including the disposal of sick and dead fish, transport water, packaging materials and other waste.

In addition to these baseline requirements, AQIS will apply the following risk management measures either singly or in combination, to address specific disease concerns associated with the importation of ornamental finfish:

- health certification from the competent authority that the source of the fish was free of specified disease agents;
- treatment either of the source population of the fish or of the fish for export, to address the likelihood that unwanted disease agents may be present;
- testing of imported fish during quarantine detention, either on an ad hoc or routine basis, to validate the certification provided by overseas competent authorities, and/or to provide additional data to improve the targeting of risk management measures on imports generally;
- treatment of imported fish during quarantine detention by appropriate means if the presence of specific disease agents is suspected or confirmed following diagnostic testing; and
- increased post-arrival quarantine detention over the minimum required (eg due to concerns over the risks posed by iridoviruses, the minimum quarantine period for gouramis and cichlids will be two weeks).

Equivalent approaches to managing identified risk may be accepted, generally or on a case-by-case basis. Parties seeking to use alternative risk reduction measures to those listed in the new conditions — for

example, an extended period of quarantine detention or a specified testing regimen — should provide a submission for consideration by AQIS. Such proposals should include supporting scientific data that clearly explain the degree to which alternative measures would reduce risk. AQIS will consider such applications on a case-by-case basis.

The implementation of these conditions will provide for the continued importation of ornamental finfish. The new importation conditions are more restrictive than the current conditions in that health certification is required for each consignment and post-arrival quarantine will be applied to all imports of live ornamental finfish.

Under previous conditions for the importation of live ornamental freshwater finfish listed on Schedule 6, AQIS required that:

- the pre-export premises for freshwater finfish were approved by AQIS;
- each consignment of freshwater fish was accompanied by an exporter's certificate attesting to the health of the fish in the consignment and that, for goldfish, the farms of origin were free from goldfish ulcer disease;
- each consignment of goldfish was accompanied by a health certificate from the appropriate government authority that the goldfish had been examined and showed no clinical evidence of disease, and that they originated either from a country free from spring viraemia of carp or from premises at which there had been no evidence of spring viraemia of carp for the three months before export; and
- the freshwater fish were held in post-arrival quarantine of two weeks (four weeks for gouramis) in approved premises, during which time they could be subjected to tests and treatment required by AQIS.

The established trade in live ornamental finfish will be permitted to continue under transitional arrangements until the new conditions are fully implemented

9.2 Addressing the findings of the WTO report

As stated in Chapter 1, the WTO found in November 1998 that Australia had not complied with its obligations under the WTO Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement) with regard to the measures applying to salmon. In short, the key findings were:

- Australia's IRA on uncooked, wild-caught Pacific salmon from Canada did not fulfil all the requirements of the SPS Agreement in relation to a risk analysis, and there was no risk analysis to support the restrictions on the importation of other uncooked salmon products; and
- there were arbitrary or unjustifiable distinctions in the level of protection applied by Australia in relation to salmon and other fish, and these distinctions resulted in a disguised restriction on international trade.

AQIS conducted accelerated risk analyses on non-viable salmonids and non-viable non-salmonid marine finfish and on live, ornamental finfish to address the WTO findings. The reports of the risk analyses together address the WTO findings.

9.2.1 WTO FINDING OF INADEQUATE RISK ANALYSIS

AQIS has ensured that the measures to be applied to the importation into Australia of non-viable salmonids, non-viable non-salmonid marine finfish and live ornamental finfish are based on a risk analysis that is consistent with the provisions of the *Quarantine Act 1908*, and that meets international obligations, by adopting the following approach to the risk analyses:

- the risk analyses contain a scientific evaluation of disease risks associated with the probability and consequences of establishment of individual pathogens;
- the scientific validity of the risk analyses was strengthened by making arrangements for independent scientific experts to review draft papers and advise AQIS on the accuracy of scientific information and the rigour and balance of the analyses;

- the risk analyses were conducted in accordance with relevant international standards,[4] which were taken into account as appropriate throughout the analysis;
- for each pathogen, the unrestricted risk of establishment was compared with Australia's ALOP. Where it was concluded that the unrestricted risk would be consistent with the ALOP, AQIS will permit importation and will not require disease-specific risk management measures against that pathogen;
- for each pathogen, where AQIS concluded that the unrestricted risk would not meet the ALOP, AQIS considered the effect of applying disease-specific risk management measures. Where AQIS judged that the implementation of disease-specific measures against that pathogen would have the effect of reducing risk to meet the ALOP, AQIS will permit importation subject to appropriate disease-specific risk management conditions;
- for each disease agent, the trade-restrictive effects of available measures were considered, and (where options are available) the least trade-restrictive option available was adopted;
- AQIS has taken into account the presence or absence of pathogens in countries in determining the measures to be applied for individual pathogens, including, as appropriate, the existence of populations that have a low prevalence of disease (eg VHSV in pilchards, *Sardinops sagax*). AQIS has also undertaken to consider submissions from exporting countries regarding the absence of specified diseases from populations that are the subject of monitoring and surveillance of fish health;
- the analyses are broad in scope to cover quarantine issues relevant to the entire range of measures applied to the importation of aquatic species that carry the diseases of concern; and
- AQIS has undertaken to apply the principles of equivalence and national treatment in considering the systems that countries use to provide guarantees on fish health or other measures.

9.2.2 WTO FINDING OF INCONSISTENCY

The level of protection applied to the importation of aquatic animals (finfish) and their products will be made consistent through the implementation of the conclusions of the two risk analyses, thus addressing the second WTO finding, regarding inconsistency.

AQIS has ensured consistency in the level of protection applied to non-viable salmonids, non-viable non-salmonid finfish and live ornamental finfish by adopting the following approach to the risk analyses.

- The risk analyses are based on a scientific evaluation of quarantine risk arising from the probability and consequences of establishment of individual pathogens.
- In examining the consequences of establishment, AQIS assumed that the consequences would be similar regardless of the pathway of pathogen entry and establishment. Measures were thus applied to bring the probability of establishment of pathogens into consistency by applying measures as appropriate to the different commodities.
- AQIS compared the effect of measures that could be used to mitigate the probability of establishment of each pathogen via the different imported commodities, with the objective of ensuring that, for all disease agents, importation would be permitted only if the measures applied to imported goods would have the effect of reducing risk to meet Australia's ALOP.
- For each disease agent, trade-restrictive effects of available measures were considered, and (where appropriate) the least trade-restrictive option available was adopted.

A key consideration in the risk analyses was the comparison of probability of establishment (before and after the application of risk management) via the different pathways presented by the importation of live finfish in contrast to the importation of non-viable finfish. The pathways and, therefore, the risk management options are quite different for these two groups of

4 The international standards used were Section 1.4 of the *International Animal Health Code* (OIE 1999, available on the internet at http://www.oie.int/norms/mcode/a_summry.htm), as reviewed by the International Committee of the Office International des Epizooties (OIE, or World Organisation for Animal Health) in May 1999; the OIE *International Aquatic Animal Health Code* (OIE 1997a) and the OIE *Diagnostic Manual for Aquatic Animal Diseases* (OIE 1997b).

commodities; thus, the comparison of the effect of risk management presents some complexities.

As stated in Section 1.6, the importation of live fish and viable products (eg eyed ova) presents greater quarantine risk than the importation of non-viable finfish and their products. This relates to the propensity for infectious agents to persist in live fish and the fact that live fish will be introduced into an aquatic environment (albeit a closed system, such as an aquarium), where any pathogens they carry may multiply in fish that commingle with fish of a similar kind. In recognition of the greater quarantine risks associated with viable fish and genetic material, Australia has not permitted the importation of live salmonids or their genetic material into Australia since the 1970s. In respect of live fish, only ornamental finfish on Schedule 6 of the *Wildlife Protection (Regulation of Exports and Imports) Act 1982* may be imported into Australia.

Non-viable products imported for human consumption would not generally be introduced into the aquatic environment, so the opportunity for transmission of infectious organisms would be greatly reduced. The commercial processing of imported fish in Australia could generate a significant volume of solid or liquid waste at the premises' point of discharge. For historical reasons, many fish processing plants are located near or on waterways. Large-scale discharge (deliberate or accidental) into the aquatic environment of untreated waste from imported fish would increase the risk of establishment of pathogens, if present in imported product.

Imported non-viable fish or products that are used for fishing bait, or for feeding to farmed fish, would enter the aquatic environment. For certain pathogens, the quarantine risks associated with this practice may be at least as high as those associated with the importation of live fish and gonadal products.

AQIS has taken into account the differing probabilities of entry and establishment of pathogens via the pathways associated with the different commodities and the factors relevant to these pathways in reaching a consistent outcome to the risk analyses.

9.3 Next steps: implementation of the conclusions of the risk analyses

With effect from the publication of AQIS's findings, these conditions will apply to countries that wish to export fish and aquatic products to Australia. The necessary arrangements are being set in place for recognition of:

- the competent authorities of exporting countries in relation to fish health, and control of fish processing plants and live fish exporting plants; and
- the system of surveillance and monitoring of health of populations from which salmonids for export to Australia are sourced.

9.3.1 RECOGNITION OF THE COMPETENT AUTHORITY

In some countries (such as New Zealand) a single government agency is responsible for all functions; in others, responsibility may lie with different government agencies, which may be at national or subnational level, or with a government-authorised body.

For Canada, the United States and New Zealand there is an established history of exporting a large range of terrestrial animals, fish, and animal and fish products to Australia. Appendix 2 provides an overview of the responsibilities of the competent authorities for fish health, inspection and certification in Canada, the United States, New Zealand and the European Union. Appendixes 3 and 4 contain detailed information on New Zealand's regulatory systems for fish health monitoring and surveillance, the approval and control of fish processing plants and the provision of export certification in New Zealand.

On the basis of current information, AQIS recognises the following government agencies as competent authorities in relation to fish health (monitoring and surveillance) and the approval and control of fish processing plants (provision of export certification) as appropriate: the United States Food and Drug Administration; the National Marine Fisheries Service of the United States Department of Commerce; the Canadian Food Inspection Agency; the Canadian Department of Fisheries and Oceans; and the New Zealand Ministry of Agriculture and Forestry.

For other countries, AQIS would enter into discussions with competent authority(ies) to satisfy itself as to controls over salmonid health and systems for approval and control of fish processing plants and for monitoring and surveillance of salmonid health. Animal Quarantine Policy Memorandum 1999/41[5] provides guidelines for the approval of countries to export animals (including fish) and their products to Australia. The requirements set out in this memorandum are based on the provisions of the OIE *International Aquatic Animal Health Code* (Chapter 1.4.3, Evaluation of competent authority) (OIE 1997a).

9.3.2 SYSTEMS FOR SURVEILLANCE AND MONITORING OF THE HEALTH OF SALMONIDS

AQIS will assess an exporting country's system for surveillance and monitoring of salmonid health as part of the implementation of the measures identified in this risk analysis.

AQIS will base its assessment of systems for monitoring and surveillance of the health of salmonids on the provisions of the OIE *Diagnostic Manual for Aquatic Animal Diseases* (Chapter 1.1, General information) (OIE 1997b). The OIE refers to the general basis for fish health surveillance and control measures with reference to the health of cultured fish as follows.

A comprehensive approach for animal health control in fish culture requires:

- Assessment of the health status of animals in a production site based on inspections and standardised sampling procedures followed by laboratory examinations conducted according to instructions given in this *Manual*.
- The constraint of restocking open waters and farming facilities only with aquatic animals having a health status higher than or equal to that of animals already living in the considered areas.
- Eradication of disease when possible, by slaughtering infected stocks, disinfecting and restocking with pathogen-free fish.
- Notification by every Member Country of its particular requirements, besides those provided by the *Code*, for importation of aquatic animals and animal products.

If the above procedures are followed, it becomes possible to give adequate assurance of the health status of aquaculture products for specified diseases, according to their country, zone or site of origin.

The OIE manual (1997b) goes on to state that the issuance of a health certificate, based on a health status report and examinations of aquatic animals, provides assurance that the aquaculture products in a consignment originate from a farm (or other defined zone) free of one or more of specified diseases.[6]

In considering the minimum requirements for fish health surveillance and monitoring, AQIS will take into account the provisions of the OIE *Diagnostic Manual for Aquatic Animal Diseases* (1997b). However, the primary focus of this manual is on trade in live fish and genetic material; moreover, the provisions relate to fish diseases listed by the OIE. Australia is free of many serious pathogens that are not listed by the OIE, and government policy is to maintain freedom from these pathogens. The importation of non-viable salmonids under the conditions recommended by the OIE as a minimum acceptable safeguard (ie evisceration of the carcase) would not meet Australia's ALOP with regard to several serious diseases (such as infectious salmon anaemia), only some of which are OIE-listed. It is important to ensure that the system of surveillance and monitoring of the health of fish in exporting countries is sufficiently comprehensive to address the issues of concern in this risk analysis. Accordingly, in conducting its assessment, AQIS will also take into account relevant general provisions of the OIE *International Animal Health Code* (1998) contained in Chapter 1.4.5, Surveillance and monitoring of animal health.

In considering 'minimum requirements' for disease surveillance by exporting countries, Australia has an obligation to consider the principles of equivalence and

5 AQIS (Australian Quarantine and Inspection Service) (11 June 1999), Animal Quarantine Policy Memorandum 1999/41. Guidelines for the approval of countries to export animals (including fish) and their products to Australia.

6 The reference in the OIE manual to the provision of assurances regarding disease freedom specifically relates to live fish and viable product, not non-viable fish. For the OIE, provided that fish for human consumption have been eviscerated, they are considered to be safe for the purposes of international trade.

national treatment in the SPS Agreement. In this regard, it is relevant that most of the information on aquatic disease in Australia is based on the results of salmonid health surveillance in Tasmania (see Appendix 6). The Tasmanian system for farmed Atlantic salmon is far more rigorous than that of other Australian States and Territories. Moreover, surveillance and monitoring of salmonid health is far more intensive than that for non-salmonid marine finfish in Australia. It would be inconsistent with our international obligations if Australia were to require countries to conduct a significantly more intensive national surveillance program to demonstrate the absence of specified diseases than that deemed sufficient to support Australia's claims to freedom from the same diseases (all other technical issues being equal).

9.4 Summary

The risk analyses have been conducted according to an accelerated timetable necessitated by the WTO decision on implementation of the WTO findings on salmon. AQIS has completed these analyses within three months from 23 April, when the Australian Government announced that the risk analyses would be accelerated.

The findings of the risk analyses are based on a comprehensive analysis of relevant scientific literature, including scientific information in previous reports of the Australian Government (DPIE 1995, 1996) and a report of the New Zealand Government on the importation of non-viable salmonids into New Zealand (Stone et al 1997b). AQIS also discussed disease issues with experts in fish health and quarantine in Australia and overseas. AQIS took several steps to ensure the scientific validity of the risk analyses, including considering the reports of consultancies (most of which were commissioned in 1998) on identified gaps in information relating to these risk analyses. AQIS also made arrangements for 14 independent scientists (in Australia or overseas) to review one or both of the drafts of the new IRA reports as they were being prepared. AQIS asked the independent reviewers to advise on:

- the completeness and accuracy of scientific information in the IRA reports;
- the balance and objectivity with which scientific information was treated;
- the extent to which the exercising of professional judgment in the reports was supported by and consistent with relevant scientific information; and
- the consistency of professional judgments on scientific issues that were common to each risk analysis report (where appropriate).

AQIS did not ask the independent reviewers to advise on Australia's ALOP, as this is the responsibility of the Australian Government, having regard to the broad range of quarantine decisions and precedents within AQIS's purview.

To ensure that the process fulfilled the Government's commitment to an open and consultative approach to IRA, AQIS held public meetings in five capital cities and held two meetings of key stakeholders in Canberra. AQIS also made each chapter of the draft reports available to the public for comment by posting them on the Internet.

In the course of the risk analyses, AQIS received 35 submissions on scientific issues. AQIS also received a large number of representations, most of which restated the importance of maintaining the current prohibition on importation of uncooked salmon, but which presented no scientific issues requiring consideration in the risk analyses.

AQIS considered all scientific issues raised in the submissions of respondents and sought the advice of the independent scientific reviewers on significant points in the submissions. For each risk analysis, AQIS reviewed each part of the report in the light of stakeholder submissions.

The scientific information reviewed in these reports is comprehensive and up to date, and the independent scientific reviewers have agreed that the scientific analysis is accurate, objective and balanced. On this basis, the conclusions in the risk analyses will be incorporated (where appropriate) into legal instruments and procedures for the importation of non-viable salmonids and non-salmonid marine finfish and live ornamental finfish in accordance with the conditions set out in these reports.

Appendixes

Appendix 1
Taxonomic details of salmonids

THE 'SALMONIDS' DISCUSSED IN THIS IMPORT risk analysis include all members of the family Salmonidae and *Plecoglossus altivelis.* Taxonomic details are as follows.

Superorder Protacanthopterygii

Order Salmoniformes

Family Salmonidae (salmonids)

Genus *Acantholingua*
Acantholingua orhidana

Genus *Brachymystax*
Brachymystax lenok

Genus *Coregonus* (whitefish, ciscoes, vendace)
Coregonus albula
Coregonus artedi
Coregonus autumnalis (Arctic cisco)
Coregonus lavaretus

Genus *Hucho* (huchen or taimen)
Hucho hucho

Genus *Oncorhynchus* (Pacific salmon)
Oncorhynchus clarki (cutthroat trout)
Oncorhynchus clarki clarki
Oncorhynchus clarki henshawi
Oncorhynchus clarki utah
Oncorhynchus gilae
Oncorhynchus gorbuscha (pink salmon)
Onorhynchus keta (chum salmon)
Oncorhynchus kisutch (coho salmon)
Oncorhynchus masou (cherry salmon)
Oncorhynchus masou ishikawai
(Ishikawa's cherry salmon)
Oncorhynchus mykiss (rainbow trout)
Oncorhynchus nerka (sockeye salmon)
Onorhynchus rhodurus (amago)
Oncorhynchus tshawytsha (chinook salmon)
Oncorhynchus spp (salmon, trout)

Genus *Parahucho*
Parahucho perryi (taimen)

Genus *Prosopium* (whitefishes)
Prosopium spilonotus

Genus *Salmo* (salmon, trout)
Salmo carpio
Salmo fibreni
Salmo macrostigma
Salmo marmoratus

Salmo salar (Atlantic salmon)
Salmo trutta (brown trout)
Salmo spp (salmon, trout)

Genus *Salvelinus* (chars)
Salvelinus alpinus (Arctic char)
Salvelinus confluentus (bull trout)
Salvelinus fontinalis (brook trout)
Salvelinus leucomaenis (whitespotted char)
Salvelinus leucomaenis pluvius (whitespotted char)
Salvelinus malma (Dolly Varden)
Salvelinus namaycush (lake trout)

Genus *Stenodus*
Stenodus leucichthys (inconnu)

Genus *Thymallus* (grayling)
Thymallus arcticus
Thymallus thymallus

Order Osmeriformes

Family Plecoglossidae

Genus *Plecoglossus*

Plecoglossus altivelis (ayu or sweetfish)

Appendix 2
Fish inspection and grading systems

THIS APPENDIX PROVIDES AN OVERVIEW of procedures for inspection and certification of non-viable fish for human consumption in New Zealand, the United States, Canada and the European Union. Detailed information on New Zealand's procedures for monitoring and surveillance of fish health and for inspection and certification of fish for human consumption are given in Appendixes 3 and 4.

New Zealand

RESPONSIBLE AUTHORITY

The New Zealand Ministry of Agriculture and Forestry (MAF) is responsible for food safety standards, including those that relate to the export of wild and aquacultured fish and fish products.

The MAF Regulatory Authority (MAF Reg) is the controlling authority for seafood. It develops policy and standards (including certification standards) for seafood and makes arrangements for independent audit of seafood processing and standards.

The MAF Verification Agency (MAF VA) validates inspection processes, and verifies and certifies inspection services at premises that produce seafood products for export. This ensures compliance with the MAF Reg-approved standards and with requirements of overseas trading partners.

The MAF Reg Compliance Group has the following objectives:

- to verify that delivery organisations implement and maintain the agreed standards;
- to ensure that corrective action is taken if needed;
- to provide information to the chief meat veterinary officer and national managers on the efficacy and state of compliance of the relevant specifications; and
- to provide delivery organisations with specifications and technical advice on achieving compliance with the specifications.

LEGISLATIVE REQUIREMENTS

MAF Reg administers legislation relating to the safety and wholesomeness of seafood produced for domestic consumption and for export.

The *Meat Act 1981* and accompanying regulations apply to the licensing of seafood processors, including those involved in the processing, packaging, preservation, handling, holding and storage of fish products. The Act also provides regulatory controls over the export of seafood (products and byproducts) with regard to safety, wholesomeness and labelling accuracy.

The Fish Export Processing Regulations 1995 include legal requirements relating specifically to fish, including:

- requirements for the construction of and maintenance of standards for plant and equipment in fish processing premises;
- obligations of the licensee to maintain the hygiene, quality and fitness of product for human consumption;
- requirements for operation of premises, including those used for storage and transportation;
- requirements that companies carry out regular checks, record results and take corrective action on compliance with respect to MAF requirements;
- requirement that no fish or shellfish are exported from New Zealand unless they are accompanied by an export certificate;
- provision of powers to inspectors to examine and sample fish, to remove and dispose of unfit fish and to prohibit the use of equipment or premises, and
- provisions for the exemption from licensing of whole fish processing premises and limited processing fishing vessels.

The Fish Export Processing Regulations 1995 require that all fish and fish products exported from New Zealand should have an export certificate, confirming that each consignment is a product of New Zealand, processed and packed under hygienic conditions in licensed premises in accordance with the regulations.

STANDARDS AT PROCESSING PLANTS

Companies processing salmon for export must have documented programs on sanitation and hygiene of premises, as well as standards for storage, transport, water quality, waste management and staff training. Checks must be carried out on product quality, condition and labelling. Salmon must be processed according to an approved standard. It is currently recommended that companies use the 'hazard analysis critical control point' (HACCP) system to ensure that satisfactory public health standards are maintained, and this will soon become mandatory.

MAF Reg requires that companies conduct pre-operational, daily and weekly inspections of the processing area, product, personnel, sanitation, equipment, refrigeration and water/ice to ensure compliance with standards. Routine microbiological testing is not required but ready-to-eat products must be tested for *Listeria monocytogenes*.

MAF VA travelling meat inspectors inspect and test fish and fish products during processing to enable official certification on behalf of MAF Reg. This involves delivery of quality assurance services, including system design, inspection, laboratory analysis, audit and certification. The inspector also audits the company compliance checksheets, notes any non-compliance, sets time limits for corrective action and determines further action. MAF VA conducts its own internal compliance audits, to ensure satisfactory compliance is achieved on a national level and to ensure the requirements of trading partners have been met for all seafood products exported from New Zealand.

The Fish Export Processing Regulations 1995 also allow MAF to issue circulars known as Industry Agreed Implementation Standards (IAI Standards). These circulars provide a means of achieving compliance with the standards required by the regulations. Companies can apply for approval for various methods of compliance with the standards.

All vessels licensed to harvest and transport wild or aquacultured fish must meet IAI Standards for the proper handling of fish. All fishing vessels are required to preserve the catch in ice or in an ice/seawater slurry at a temperature no greater than 4°C. Live salmonids must be

pithed and bled, inspected, then placed in a sea-ice slurry in plastic bins for transport to the processing plant.

Fish intended for export must be processed in registered processing plants, which must meet the requirements of the IAI Standards. All fish landed are inspected by company staff before processing to ensure that the fish meet the minimum acceptable public health and quality standards. During online processing of fish, company inspectors monitor critical control points throughout the process. The fish are inspected after dressing to ensure that viscera have been completely removed and that the body cavity has been thoroughly washed to remove all slime and blood. The fish are individually inspected and graded before freezing, icing and boxing or before further processing.

Fish containing remnants of viscera (except small remnants of the kidney, mainly anterior kidney) or which have been insufficiently washed are withdrawn and reprocessed. Quality grading is based on the appearance of the fish; unblemished fish go into the highest grade, while fish with blemishes are downgraded. Blemishes include loss of scales, and physical damage incurred during processing and handling. Fish that are downgraded to grades other than first grade are trimmed and/or processed into other products. Fish found to have visible lesions or major blemishes during harvest or processing inspections are rejected from human consumption. The reject fish are incorporated into pet-food or into fishmeal (for later use in stockfeed for land-based animals).

PRODUCT CERTIFICATION

Export certification for fish and fish products from New Zealand is based on fish inspection and grading systems as outlined above, and is supported by ongoing fish disease monitoring and surveillance.

MAF has been actively involved in monitoring fish health for over 20 years and has well-developed health surveillance programs for fish and shellfish. These programs focus on detecting unwanted organisms and maintaining accurate information on endemic diseases, under the terms of the *Biosecurity Act 1993*. Disease testing is mainly carried out by a central fish disease diagnostic laboratory. MAF has an ongoing program of health certification for salmonid fish, supported by diagnostic testing, routine disease monitoring and active surveillance testing, including surveys for specific diseases. The Ministry of Fisheries is responsible for authorisation of fish transfers under the Freshwater Fish Farming Regulations 1983 and the *Marine Farming Act 1971*, and controls the movement of fish stock across the boundaries of the *M. cerebralis* and enteric redmouth disease control areas. It also monitors diseases of wild marine fish and shellfish under the *Fisheries Act 1983*, in cooperation with the MAF.

INTERNATIONAL AGREEMENTS

The Australia New Zealand Closer Economic Relations Trade Agreement 1983 (CER) has as its objective the removal of unnecessary barriers to trade in goods and services between Australia and New Zealand. It endorses the modification or removal of quarantine barriers between the two countries, subject to technical analysis of the risk of introduction of disease or pests, and commits both countries to ensure that quarantine is not used as a means of creating a technical barrier to trade.

The *Trans-Tasman Mutual Recognition Act 1997* provides for recognition in Australia of regulatory standards in New Zealand, but specifically exempts quarantine. The Australia New Zealand Food Authority (ANZFA) has been established to administer public health food processing standards. ANZFA recognises processing standards in both Australia and New Zealand for food products (including fish), but can exempt certain products on public health and quarantine grounds.

Canada

RESPONSIBLE AUTHORITY

The Canadian Food Inspection Agency (CFIA) has legal responsibility for food safety standards, including those relating to the export of fish and fish products.

The Department of Fisheries and Oceans has legal responsibility for licensing vessels to harvest and transport wild salmon and must ensure that they meet regulated construction and operating standards to ensure that the fish are handled properly.

LEGISLATIVE REQUIREMENTS

The *Fish Inspection Act* is the legislative basis for standards relating to the export and import of fish and containers and authority of inspectors, including for inspection and detention of fish and containers.

The Regulations Respecting the Inspection of Processed Fish and Processing Establishments (also known as the Fish Inspection Regulations) cover imported and exported fish and define the inspection requirements and fees, registration of vessels and processing establishments, code marking and labelling of products, packaging, product specifications, quality and grading. Schedules under the Fish Inspection Regulations provide:

- construction and equipment requirements for establishments;
- operating requirements for establishments with regard to hygiene;
- requirements for vessels used for fishing or transporting fish for processing, including equipment, storage, refrigeration and freezing;
- requirements for establishments storing frozen fish;
- requirements for vehicles and equipment used for unloading, handling, holding and transporting fresh fish for processing;
- requirements respecting quality management programs.

Section 15 of the Fish Inspection Regulations requires that federally registered fish processing establishments that process fish and fish products for export have quality management programs (QMP) for each type of fish processing carried on at their establishment. QMP are mandatory food inspection programs largely based on HACCP inspection principles.

New regulations amending the Fish Inspection Regulations were promulgated and came into force in April 1999. The new regulations fully incorporate HACCP principles into the QMP and strengthen and clarify requirements for operational standards and record keeping.

STANDARDS AT PROCESSING PLANTS

Under the Regulations Amending the Fish Inspection Regulations, as a part of a QMP, operators must conduct hazard analyses and formulate HACCP inspection plans in accordance with the *Facilities Inspection Manual* and *Canadian Shellfish Sanitation Program Manual*. Plants must also meet guidelines for fish and fish products.

In the regulations, QMP has three components:

- Prerequisite Programs, which cover design, construction and maintenance of plant facilities including maintenance of records to ensure that food is produced under sanitary conditions, contamination is prevented and there are procedures for product recall;
- Regulatory Action Point Programs, which establish minimum standards for input materials, product, labelling and coding of products, sanitary controls over fish handling, ingredients, packaging, corrective actions and record keeping;
- HACCP plans, involving hazard analysis, establishment of critical points and critical limits, monitoring procedures, corrective action systems, verification procedures and records, and integration of these into an overall documented HACCP inspection plan for the establishment.

CFIA has produced draft QMP plans for various fish processing operations to assist industry in developing QMP for products and processes.

Plants are audited periodically by CFIA inspectors who check that the QMP is functioning effectively and the operations comply with the regulations. These inspections cover plant conditions, hygiene and employee practices. Processing plants are graded on the basis of the number of deficiencies detected at inspection. The grade is used to determine the frequency of QMP audits. Plants that consistently maintain excellent or good ratings gain access to a streamlined certification process for exported products, and can display a 'Canada Inspected' logo on their products.

CFIA publishes an *Approved List of Exporters,* which identifies Canadian processing plants that, having satisfied CFIA requirements, can export fish and seafood to the United States and the European Union.

All vessels licensed to harvest and transport fish must meet regulated construction and operating standards to ensure that the fish are handled properly and not subject to temperature abuse or contamination. These requirements are enforced by CFIA inspectors, and the level of compliance of the vessels is high. Holds of most vessels are constructed of aluminium or fibreglass, designed to facilitate the cleaning and sanitisation of fish contact surfaces. All fishing vessels are required to preserve the catch by chilling in ice at or below 4°C or in chilled water at –1°C, or by freezing. Fish are required to be iced or chilled during transport to processing plants.

Salmon processed in registered processing plants must meet the requirements of the QMP. All fish landings are sampled and inspected before processing to ensure that the fish meet the minimum acceptable safety and quality standards. The catch is hand-sorted before processing, and any fish with visible lesions are rejected for human consumption.

CFIA inspectors monitor critical control points during the online processing of salmon. Under the QMP, fish are inspected to ensure that viscera have been cleanly removed and the body cavity has been thoroughly washed free of slime and blood before the freezing of fish or the icing and boxing of fresh fish, and the fish are individually inspected and graded to ensure that they meet the final product standards for safety and quality. Fish product that does not meet company specifications because it is tainted, decomposed or unwholesome is rejected.

Quality grading is based on the presentation of the fish. Fish are classed as 'grade A' if whole, properly cut and cleaned and free of all entrails, physical damage, bellyburn and signs of advanced sexual maturity. Blemishes include loss of scales, traumatic lesions such as those from predation, and physical damage incurred during processing and handling. Fish with remnants of viscera (except remnants of the anterior kidney) or which have been poorly washed are culled from the line and reprocessed. Fish that do not meet grade A may be downgraded to 'standard' or 'utility' grades, and may be retrimmed and/or processed into other products such as canned fish, fillets, fish mince etc.

PRODUCT CERTIFICATION

The export certification for fish and fish products exported from Canada is based on fish inspection and grading systems as outlined above, and is supported by ongoing fish disease monitoring and surveillance.

The Department of Fisheries and Oceans (DFO) is responsible under the Fisheries Act for protecting fisheries resources in Canada. The DFO implemented Fish Health Protection Regulations (FHPR) in 1977, under section 43b of the Fisheries Act, and produced a manual of compliance with guidelines for producers, inspecting officials and administrative officers, as well as diagnostic procedures. These regulations have been upgraded and include marine and freshwater salmonids. Fish health officers are appointed from both federal and provincial agencies to administer the FHPR and a National Registry of Fish Diseases is maintained in Ottawa. The regulations list a number of scheduled fish diseases, though they do not currently require mandatory notification of these diseases. Proposed amendments will extend the regulations to include non-salmonid finfish, and include mandatory reporting of selected fish disease agents. The provincial governments are responsible for health surveillance within their provincial aquaculture industries, under local provincial legislation. There are a number of fish diagnostic laboratories scattered across Canada, providing a range of diagnostic services. Provincial disease surveillance is particularly strong in British Columbia and in the maritime provinces, which have substantial support services for their large wild fisheries and aquaculture industries.

INTERNATIONAL AGREEMENTS

Australia negotiated a memorandum of understanding with Canada in 1993 that provides for the mutual recognition of processing standards for fish and fish products in both countries but does not include quarantine issues.

United States

RESPONSIBLE AUTHORITY

The United States Food and Drug Administration (FDA) is the competent authority for post-harvest practices and is legally responsible for food safety standards, including those relating to export of saltwater and freshwater finfish and finfish products.

The National Marine Fisheries Service (NMFS) of the United States Department of Commerce has delegated authority for the inspection and certification of establishments and fishery products for human consumption.

The individual states regulate fisheries and aquaculture production and harvest practices, including vessel sanitation and fish storage.

LEGISLATIVE REQUIREMENTS

The authority of the FDA arises from the United States Federal Food, Drug and Cosmetic Act (FFD&C Act), the Public Health Services Act (PHS Act), and the Fair Packaging and Labelling Act. These laws are implemented by regulations in Titles 21 and 42 of the United States Code of Federal Regulations.

The Lacey Act, as amended, is a significant law enforced by both the United States Department of the Interior and the United States Department of Commerce (USDC) and prohibits interstate commerce in fish or fishery products if any law, treaty, or regulation of the United States, or of the place of shipment or receipt (eg the laws of a foreign country) is violated.

The FFD&C Act includes powers to regulate the safe and sanitary processing and importing of fish and fish products, including powers to:

- appoint inspectors;
- inspect factories and records;
- impose fees; and
- impose regulations covering sanitation, packaging, labelling, marking, and collection of samples for examination and analysis.

Title 50: Regulations Governing Processed Fishery Products establishes procedures for the inspection and certification of establishments and fishery products for human consumption, with provision to delegate any or all such functions to the NMFS. All fish processors in the United States are required under the laws and regulations enforced by the FDA to produce safe, wholesome, properly labelled products. The processing and storage of these products must comply with current FDA Good Manufacturing Practice Regulations.

The FDA, under Federal Register 60 FR 65095, published new procedures for safe and sanitary processing and importing of saltwater and freshwater finfish and fishery products in December 1995, which were trialled by a number of companies. These new procedures became law in December 1997, and require that saltwater and freshwater finfish processors, repackers and warehouses further ensure food safety by following a HACCP program. These laws are implemented by regulations in Titles 21, 42 and 50 of the United States Code of Federal Regulations (CFR).

STANDARDS AT PROCESSING PLANTS

The Federal Standard 369 (Sanitation Standards for Fish Plants) established sanitation standards for fish product processing plants operating under the USDC Fishery Products Inspection and Safety Program. Compliance with provisions of this standard is mandatory for listing in the *USDC Approved List of Sanitarily Inspected Fish Establishments*. The standard covers cleaning and sanitation requirements for raw materials, buildings, processing rooms, water supply, waste disposal, cleaning and sanitisation treatments, packaging, storage and personnel.

While the FDA operates an oversight compliance program for fishery products, NMFS has the delegated authority at federal level to inspect and grade fish and fishery products and issue certification. Anyone using the NMFS Seafood Inspection Program must comply with all the regulations governing the program, including all regulations pertaining to seafood promulgated by the FDA. If a company chooses to export to another country, all the import requirements of that country must be met

before an official USDC Export Health Certificate may be issued.

Firms that wish to use official USDC 'Packed Under Federal Inspection' or 'United States Grade' marks, or firms that require certification of processing conditions to meet buyer or foreign country requirements, must pass NMFS sanitation evaluations using Federal Standard 369 — Federal Sanitation Standard for Fish Plants or applicable foreign requirements. This standard is used by NMFS to evaluate production, storage, and distribution facilities.

The NMFS publishes semi-annually an *Approved List of Fish Establishments and Products* that identifies United States firms that satisfy NMFS inspection service requirements and participate in its program for in-plant inspection services. In order to maintain this status, facilities under the HACCP-based inspection service undergo a systems audit that includes sanitation and product evaluation, and that records review of critical control points at a frequency based on their level of compliance. The FDA also regularly inspects seafood processors and warehouses, using company records to enable it to determine how well a company is complying.

The new regulations require each processor to conduct a hazard analysis for each kind of fish and fishery product to identify hazards that can occur before, during and after harvest.

Every processor is required to submit and implement a written HACCP plan which identifies all hazards and critical control points, sets limits for each critical control point, lists the frequency and type of monitoring procedures, identifies appropriate corrective actions, lists the frequency and type of verification procedures and defines the necessary records to ensure compliance. The regulations also require processing plants to monitor and record eight areas of sanitation.

The safety features of the HACCP regulations are now incorporated into the NFMS National Seafood Inspection Program. NFMS inspects seafood processors, checking vessels and processing plants for sanitation and examining products for quality. The agency certifies seafood plants that meet federal standards and rates products with grades for quality. Seafood processors in good standing with the program are free to use official marks on products to indicate that the product has been federally inspected.

The fee-for-service inspection services provided by NMFS include sanitation inspections of vessels, shore-based processing facilities, warehouses, distribution and retail facilities, product evaluation in-process and by end item examination for compliance with minimum safety, wholesomeness, and labelling criteria, processor or buyer specifications, federal or state procurement specifications, United States standards for grades, and foreign country requirements. Products inspected and certified by NMFS for export must be at least in compliance with the requirements of the country to which they will be exported, and the buyer's requirements, when known.

The Official USDC Export Health Certificate has been used for many years. It is a controlled document (ie each bears a unique number and the embossed seal of the United States Department of Commerce, and can only be issued by an inspector of NMFS or authorised cross-licensed state or federal government inspector). The certificate identifies the individual products as to type, package size and count, and the official findings of the inspector. Analytical results may appear in the remarks section of the certificate or noted as an attachment to the certificate. Analytical reports from non-governmental laboratories, which are noted on the certificate as attachments, are recognised by NMFS as providing credible results. United States exporters use this service to comply with regulations of those importing countries which require certification by a competent authority of the exporting country.

INTERNATIONAL AGREEMENTS

AQIS is currently negotiating the mutual recognition of fish processing standards with the United Sates with regard to food safety issues.

European Union

RESPONSIBLE AUTHORITY

The European Commission (EC) is responsible for setting and auditing food safety standards on catching vessels and in processing plants, in cooperation with the competent authorities of the Member States, so as to ensure uniform application of standards and requirements within the single European market.

The Member States are responsible for control of production circumstances and requirements, including statutory inspections and issuing health certification to the agreed standards and requirements.

LEGISLATIVE REQUIREMENTS

Most veterinary legislation is in the form of 'Directives'. These laws are binding on the Member States with regard to the result to be achieved, but leave each Member State free to determine the means to implement the measure in national legislation within a given time-frame.

Council Directive 90/675/EEC lays down the principles governing the organisation of veterinary checks on products entering the European Union from third countries, including Norway.

Council Directive 91/67/EEC, amended by Council Directives 93/54/EEC and 95/22/EEC, gives the health conditions for the marketing of aquaculture animals and products. Article 3 specifies that aquacultured animals placed on the market must show no signs of disease, must not be intended for destruction or slaughter under a disease eradication scheme, and must not come from or have had contact with a farm subject to a prohibition because of health reasons.

Council Directive 91/493/EEC covers saltwater and freshwater finfish, whether wild or aquacultured, and requires competent authorities of the Member States to carry out checks and inspections to ensure that producers and manufacturers comply with specified requirements for the hygienic handling and marketing of fish and fish products on vessels and processing establishments.

Commission Decision 93/144/EEC gives protective measures to be taken following recognition of infectious salmon anaemia in Norway. It prohibits importation of salmon from Norway, whether live or slaughtered in the non-eviscerated state, though this was later modified by Commission Decision 95/118/EC, which authorises the importation of slaughtered and non-eviscerated salmon from fish farms located within specified regions of Norway.

STANDARDS AT PROCESSING PLANTS

The EC is responsible for setting food safety standards on catching vessels and in processing plants, utilising processing standards based on HACCP principles, covering cleanliness and hygiene, storage conditions, inspection, packaging, labelling, transport and health checks. The EC is also responsible for auditing processing plant standards, in cooperation with Member State competent authorities, so as to ensure uniform application of standards and requirements within the single European market.

Under Council Directive 91/493/EEC, fish caught in their natural environment must be handled in accordance with hygiene rules established by the EC covering the bleeding, heading, gutting and removal of fins, chilling or freezing, health checks, packaging, transportation and storage on board vessels in accordance with set conditions.

This directive also requires Member States to ensure that persons responsible for processing establishment must take all necessary measures to ensure that the specifications of this directive are complied with at all stages of production of fishery products. The responsible persons must carry out their own checks based on the following principles:

- identification of critical points in the establishment on the basis of the manufacturing process used;
- establishment and implementation of methods for monitoring and checking such critical points;
- taking samples of analysis in an approved laboratory by the competent authority for the purpose of checking cleaning and disinfection methods and compliance with the standards established by this directive;

- keeping a written record of the preceding points to allow submission to an appropriate competent authority; and
- taking appropriate measures, under official supervision, in the event of suspicion or detection of a health risk.

The competent authority for each Member State is required to inspect and monitor establishments regularly, approve establishments once they have verified that these establishments meet the requirements of this directive, and take necessary measures if the requirements cease to be met. The authority is required to draw up a list of approved establishments, each of which should have an official number. The Member State is then required to notify the EC of its list of approved establishments. Experts from the EC may, in cooperation with the competent authority of the Member States, make on-the-spot checks to ensure the uniform application of this directive.

The Annex to Council Directive 91/493/EEC establishes conditions for the design and equipment for factory vessels, with regard to work areas, storage areas, general hygiene, water quality, waste disposal and if appropriate, preparation, processing and freezing areas, and establishes hygienic requirements for packaging, storage and landing requirements for factory vessels and staff. The Annex also establishes general conditions concerning processing premises and equipment, covering structure, equipment, pest control, hygiene (including staff hygiene), waste disposal, and storage. It requires hygienic processing, avoidance of contamination, container hygiene, freezing equipment, visual inspection to detect parasites, and rejection of fish parts infested with parasites.

The Annex requires regular audit of the production process by the competent authority, to check health control measures, and to check monitoring of production conditions on fishing vessels, at landing and at establishments, so as to verify that approval conditions are still fulfilled. These conditions cover handling, cleanliness of premises and equipment, staff hygiene, identification marks, parasite checks, storage and transport conditions, microbiological analyses (sampling plans and methods), and packaging, storage and transport hygiene. The identification marks are required to include country of dispatch and official approval numbers.

The EU regulates fish disease monitoring and surveillance and other disease control activities in its Member States, through the issuance of Council Directives which are binding on the Member States. Council Directives, including 91/67/EEC and 93/54/EEC, set out the health conditions for marketing animals from aquaculture and list three categories of disease agents. These Directives allow the establishment and recognition of approved disease control zones for specific disease agents and specify the certification requirements for the movement of live fish, eggs and fish products between zones and for imports from third countries. Council Directive 93/53/EEC defines minimum EC measures for the control of specified fish diseases. Commission Decision 92/532/EEC prescribes sampling plans and detailed diagnostic methods for the detection and confirmation of specified fish diseases. Other Directives regulate disease control programs for specified diseases and imports from third countries.

Disease monitoring and surveillance is carried out under the above Directives by agencies of the individual Member States, under individual Member State legislation and through the activities of the member state ministries of agriculture and fisheries.

INTERNATIONAL AGREEMENTS

The European Union has signed agreements with both Canada and New Zealand establishing mechanisms for the mutual recognition of the equivalence of sanitary measures applicable to trade in live animals and animal products, consistent with the protection of public and animal health. These measures include legislation, inspection, disease control, hygiene and certification systems.

Appendix 3
Review of salmonid health in New Zealand

REPORT PREPARED BY DR PJK DURHAM, Senior Veterinary Officer, Animal Quarantine Policy Branch, on the salmonid aquaculture industry of New Zealand (December 1997).

Summary

The New Zealand salmon export market is largely supplied by two companies, the New Zealand King Salmon Co Ltd and Sanford Co Ltd, whose facilities were inspected during this visit. The salmon hatcheries, seacage production and processing facilities appeared to be well organised integrated facilities, managed by technically knowledgeable and competent staff. Good records of stocking rates, feed consumption and mortality rates were kept by the hatcheries and seacage facilities, and used to provide baseline statistics, allowing easy and prompt monitoring of any departures from normal. The processing plants are stated to run along ISO9000 and HACCP lines and are regularly monitored by Ministry of Agriculture Quality Management inspection staff.

There are very few disease problems reported in New Zealand salmon. Losses during production are stated to be low, the main problems relating to seal bite damage causing carcase rejection. It was reported that while infectious disease organisms have occasionally been detected in the past, they are now rarely reported, presumably as a result of improved management practices which minimise environmental stresses. The companies expressed a strong commitment to regular and ongoing health monitoring programs, utilising a Ministry of Agriculture health monitoring and surveillance plan which provides many years of disease testing records for all the disease agents of concern (*Myxobolus cerebralis, Vibrio ordalii*, *Yersinia ruckeri*, IPN-like birnavirus), as well as for several major diseases currently considered exotic to New Zealand.

The visit gave a valuable overview of salmonid health issues, with regard to the standards of operation, quality of the product, level of disease, and quality of disease records. It also facilitated contact with New Zealand government officials, the acquisition of detailed disease testing records, and provided insight into the methodology and reliability of testing. The visit clarified

information on production processes, allowed AQIS to identify further information requirements and facilitated access to additional data.

Detailed comments

SANFORD CO LTD

Sanford seacages in Big Glory Bay

The aquaculture area in Big Glory Bay appeared to be well sheltered, the cages being located in deep water about a half kilometre off shore. The operations consist of two large sets of seacages located about one kilometre apart, with attached moored barges, and serviced by ancillary boats and barges. They are reported to currently produce about 1100 tonnes of King salmon per annum. There is some transfer of fish stocks between the two sets of seacages. The only other aquaculture operations nearby are mussel farms, located some kilometres away nearer the mouth of the bay. The seacage operation is operated under a Marine Farm Licence issued by the New Zealand Ministry of Fisheries and is subject to the Resource Management Act 1991. The stock for the seacages is supplied from two hatcheries located at Waitaki and Kaitangata (a previously utilised third hatchery located in Marlborough is no longer in use).

During inspection of the seacages, managment provided explanation of their production and harvesting procedures. The seacages are suspended in double rows of rectangular steel pontoons, connected by walkways and served by boat berthing areas. Cages are approximately 15 metres deep, and are located in 25 metre deep water. The layout of the cages facilitates easy inspection and assessment of the various stocks, and easy movement to different size groups. Fish stocks are reported to be regularly graded for size and reallocated into uniform size groups, moving the stock along towards the harvest area. The seacages containing young stock are covered by netting to stop bird predation, and have peripheral electric fencing to deter entry of seals.

Production stock are all female. The fish are fed from bins using imported extruded pelleted feed (sourced from Gibson's, Tasmania) distributed by spinners at set times, though automated dispensing is now being trialed. Breeding stock are selected from production stock, and supplied to the hatcheries for use as broodstock, breeding males being produced by sex reversal with testosterone. Harvesting is mainly carried out in the spring (October to December) at about 3 kg live weight, though action is currently being taken to prolong the harvest season.

Market size fish are starved for 3–4 days prior to harvest, crowded into a holding pen, and transferred into pens containing Aqui-S® anaesthetic. Aqui-S® is approved in New Zealand for use as a food additive, and is registered as a fish anaesthetic under the Animal Remedies Act 1967. It does not have any withholding period. (Aqui-S® is also registered with the National Registration Authority in Australia for use as a fish anaesthetic). Once anaesthetised, the fish are then brailled onto the harvest table where they are manually pithed, bled by incising the gills, then graded by weight. Fish showing blemishes, deformities, lesions (including seal bites) or signs of overmaturity (skin darkening) are rejected at this stage. The fish are immediately placed in a sea ice slurry in bins and despatched by barge to the Sanford processing plant located at Bluff, about a 3 hour journey.

Senior staff have attended fish health and related courses at the Cawthron Institute in Nelson which included instruction from the Ministry of Agriculture (MAF) Fish Pathologist.

Records are kept of stocking rates, feed, and mortality data for each cage and compiled into monthly cage summaries, being used with growth data to determine baseline cage food conversion rates and mortality statistics, allowing detection of any deviations from normal. The production and mortality records are held in the on-site office and appear comprehensive. Records of results of periodic batch health testing by the MAF Fish Pathologist are also held on site, together with results of any other laboratory testing. Based on the above, there appeared to be a good working relationship between the managerial staff and the MAF Fish Pathologist.

Dead fish are collected into weighted areas at bottom of the nets. Apart from normal daily visual observation, the bottoms of the sea cages are inspected by contract

divers twice weekly for mortalities and net damage. Dead fish are collected either by removal via 'fish hoist' from the bottoms of the nets or directly by divers. Total mortality rates in the seacages are reported to be low, between 5–10% over the growout period. Fish receive no antibiotic treatment or vaccination during grow out.

Local health issues were identified as mainly seal bite damage, blockage of nets by jelly fish causing suffocation, some dorsal skin damage attributed to net damage and overcrowding, and sunburn. Although *Paramoeba* spp are present and can affect the gills, this organism causes little damage and is not subject to any control measures. Occasional algal blooms (*Heterosigma*) are reported to occur. Predation of smaller fish by birds is minimised by netting over the cages. Seals are claimed to be the biggest problem, their effects are minimised by using tight nets and electric fencing to deter access to cages and entry to walkways.

Staff advised that batch samples (60 fish per batch) are submitted for health monitoring checks to the MAF Fish Pathologist during the growout phase, sometimes from stock under one year, and regularly from one and two year old stocks, prior to harvest. As the two sites are close together, they are treated as a single site for purposes of sampling. Organ samples are collected on ice and sent by air courier to the Central Animal Health Laboratory (CAHL) for testing by the MAF Fish Pathologist. Annual on-site inspections are made by MAF field veterinary officer. This inspection involves discussion with the farm manager and staff on health issues, and inspection of the fish stocks and mortality records. The veterinary officer also records a summary of the mortality findings and stock movement, and comments on stock health. Completed records are sent to the MAF Fish Pathologist at the CAHL. These inspections and documents complement the information gained by routine laboratory testing. Information regarding the disease status of the inspected/tested properties is provided by the MAF Fish Pathologist to the Ministry of Fisheries, to assist with decisions on issuing salmonid movement permits.

I was advised that there is provision for further *ad hoc* testing of diseased fish, should problems arise.

Sanford processing plant (Bluff)

The plant receives the containers of bled fish carcases held in seawater-ice slurry from the previous days harvest, and immediately processes the fish on two chains. The larger fish are placed on an endless belt and the heads and gills removed with an automated guillotine. The fish are collected, the abdomen incised and the viscera removed into bins. The fish are then placed belly up on a flat conveyor belt and the kidneys removed by scraping with a long handled spoon. Residual tissues and blood are then removed by aspiration, and the carcase is then checked for lesions and blemishes. Unacceptable fish are not used for human consumption, but go to the petfood industry. The carcases are then frozen at -30^{O}C overnight, given a starch glaze to prevent freezer burn, packaged in plastic inside cardboard cartons and labelled with grade, weights, dates packed, and source.

The smaller fish were processed similarly on a second chain, except that the heads were not removed.

Plant hygiene appeared to be good, staff wearing protective gumboots, headware, gloves, body clothing, and aprons. Visitors were also required to wear similar protective clothing. Plant management advised that they maintain ISO 9000 standards, and are currently introducing Hazard Analysis Critical Control Point (HACCP) procedures. The plant is subject to periodic audit by MAF inspectors.

Fish carcases are examined at the factory on receipt, and again at grading after processing. The main reasons for rejecting salmon were seal bites. There is little opportunity to check for abnormality inside the carcase due to speed of processing chain. A check of viscera in bins is conducted at least weekly, and if abnormalities are found organ samples are sent to MAF for testing and the product is detained until laboratory results are known. Knives are washed in sanitised water as required, during work breaks and at the end of processing.

Production is currently reported to be about 1100 tonnes, with most exported to Japan, the USA and other countries around the Pacific Basin. The plant does not smoke any fish. Product is despatched within 36 hours if chilled and within 3 weeks if frozen.

Sanford Kaitangata hatchery

This property operates under a Marine Farm Licence issued by the Ministry of Fisheries and is subject to the Resource Management Act 1991. It comprises 5 partly in-ground raceways, two lined with plastic and the remainder lined with concrete. Water supply is from the Clutha River where the water is subjected to initial coarse screening to remove larger debris followed by finer screening at the ponds to remove smaller debris. Water temperature is about 12°C. The ponds cleaned out every week, and are left empty for 3–6 months of the year. Outflow is via concrete channels and coarse screens back into the river. The fish feed is currently imported from Chile, as this product has been found to be of a more consistent quality than product from other sources.

Eggs are derived from local broodstock which are selected from production seacages at Big Glory Bay on Stewart Island. The broodstock are transported back to the Kaitangata hatchery and held in plastic lined tanks pending collection of eggs and milt. Testosterone is used for sex reversal. The eggs are fertilised and hatched in plastic upwelling incubators (using water cartridge filtered down to 5µm). The hatched larvae are then grown in shallow raceways within the hatchery building before being placed in outside raceways. The fish held in outside raceways are manually fed, and periodically sorted by size. Bird netting is used to protect fish in some ponds.

The raceways are cleaned in early January and left to 'sunbake' until mid-March. Rubber raceways and all gear and nets are chlorinated. After brood holding (mid March to mid May), the broodstock ponds and races are thoroughly cleaned and immersed or sprayed with chlorine disinfectant. The incubators are disinfected after shut down with hypochlorite solution, followed by thiosulphate neutralisation.

The fish are generally dispatched to Big Glory Bay at about 20g weight (sometimes heavier) prior to Christmas, when they are ready for smoltification. Extra salt is added to the diet pre transfer to assist the salt water adaptation process. No photoregulation is used to regulate growth.

There are reported to be few health problems. Survival is quoted as 85% from stripping to hatch, and 95% plus from hatch to smolt. No antibiotics are used, though malachite green is used to control *Saprolegnia* fungus infection of eggs, stopping just before hatching. *M. cerebralis* has not been detected in the hatchery. White spot (*Ichthyophthirius* infestation) and *Flexibacter columnaris* infection have been seen occasionally in the past.

Organ samples from 6 month old fish are sent to the MAF Fish pathologist in batches of 60, and tested for cytopathic viruses, *Y. ruckeri*, *Aeromonas salmonicida* and *M. cerebralis.* The broodstock are tested for these viruses and bacteria as well as *R. salmoninarum*. There is provision for samples to be collected and forwarded to MAF for appropriate testing if unusual mortalities occur, though this has not been necessary over the past 5 seasons. Records are kept of feed, numbers of fish, mortalities, sizes and laboratory tests results.

Annual on-site inspections are made by a MAF field veterinary officer. This inspection covers discussion with the farm manager and staff with regard to stock health, inspection of the stock and the mortality records. The veterinary officer completes records summarising mortalities, stock movement records and comments on stock health. Completed forms are sent to the MAF Fish Pathologist at the CAHL. These inspections and records complement the laboratory testing program. Information regarding the disease status of the inspected/tested properties is provided by the MAF Fish Pathologist to the Ministry of Fisheries, to assist with decisions on issuing salmonid movement permits.

NEW ZEALAND KING SALMON (NZKS)

NZKS Tentburn hatchery

This hatchery operates under a Marine Farm Licence issued by the Ministry of Fisheries and is subject to the Resource Management Act 1991. It was originally started as a commercial sea release salmon farm, but sea-return salmon numbers were too low for this to be economically viable, though anglers benefited significantly. The hatchery still releases small numbers of smolt of which about 2–3000 return, amounting to less than a 1% return of released fish. The returning fish return via their own channel close to the main farm outflow and progress up a fish ladder to a holding area located at a distance of about 50 metres from the

nearest part of the main rearing raceways. The sea-run stocks are not used for broodstock, and have their own separate equipment. Boots are required to go through a footbath. Harvest equipment such as knives are washed and disinfected with Halamid™ after use. The Manager reported that the sea-return fish are of variable size and quality, and are only utilised for the local market; there is no intention to use these for export. The main activity of the hatchery is now to supply smolt to the NZKS seacages in the Marlborough Sounds.

The hatchery is a large operation with 60 concrete raceways, producing about 1.6 million smolt a year. The stock are fed local (NRM Aquafeed) compressed and extruded feed pellets, using handfeeding as well as self-feeder (tickle feeder) bins. The ponds are extensively covered by bird netting to minimise predation by local birds. The raceways are disinfected by scrubbing, air drying and exposure to sunlight. Disinfection of equipment is with Chloramine T, though hypochlorite has been used on occasions.

Water for general pond use is collected from the river via screens to remove surface weed, and pumped into the head ditch for distribution to the various ponds. Water temperatures range from 8°C to 15°C. Incubation of eggs and alevins is carried out in sandfiltered bore water — the bore water has a temperature and water quality advantage over river water. The raceways are cleaned out 1–2 times per week using a vacuum system, the raceways being dried out once per season. Pond effluent waters are discharged via unlined channels to the sea, however effluent from cleaning operations is discharged via a settling pond before discharge into the sea.

Anglers are prohibited from fishing within a 100 metre of the farm outfall. During the salmon return season, screen barriers are placed to divert all sea-run salmon to the fish ladders leading to the sea-return ponds.

Production stock in the seacages are all female. The growth performance of these stock is used as a basis for selection of sister stock in the hatchery program, these being individually identified by microchip. The brood fish are crowded using a screen, caught by hand, knocked on the head and bled, then placed onto a processing rack where eggs are taken. Milt is then added, being produced from sex reversed female stock following testosterone treatment. Following fertilisation, eggs are incubated in upwelling incubators, being sorted when eyed using salt baths and mechanical egg pickers. Generally no disease problems are reported to occur at this stage, though Tentburn has sometimes used Chloramine T or Halamid™ to batch treat fry for gill disease. No triploidy is used in the production process. Following hatching, the larvae are later transferred to larger incubators, all supplied by bore water. Chilling is used for some stock to control rate of development and hence extend the season.

Smoltification is carried out naturally, smolts tending to commence smoltification at around 6 months of age when they have grown to weights of 5–10g. Tentburn is now able to obtain smolting weights of at least 40–50g at this age. The smolt are transferred to seacages at from 40–200g at from 6 to 14 months of age (depending on the time of year), being transported in compartmented tankers. The hatchery also uses chilling to extend the incubation stage where necessary.

Tentburn stated that it gets a 85–90% hatching rate, with 80% hatch-to-release survival (includes culls). Diseases diagnosed in the past in freshwater stock include *Saprolegnia* infection of unhatched eggs, and *Y. ruckeri* infection (enteric redmouth, ERM). ERM was diagnosed in 1990 causing 2–2.5% mortality over 3 weeks. ERM again caused minor mortality problems in 1991, and again in 1993, which were reported to respond well to oxytetracycline treatment. It was considered that the disease was a consequence of stresses induced on the fish by excess silt in the supplying stream following heavy rain. There have been no further problems with *Yersinia* infection. In 1993, the parasite *M. cerebralis* was demonstrated at low levels in a clinically normal 3rd generation sockeye salmon. Sockeye salmon are no longer stocked on the farm, production being limited to chinook salmon.

IPN-like virus was recovered from wild sea-run salmon in 1984, and was recently recovered from a batch of sea-run salmon in 1997. The fish appeared clinically normal.

In the past, redspot has been occasionally seen in the eyes of returning sea-run salmon, and is suspected to be due to mechanical injury, stress or possibly *Y. ruckeri*.

As indicated earlier, sea-return stocks are held in separate ponds to the freshwater stock.

Batch samples from all 'lots' are submitted at 6–9 months of age for routine health monitoring tests. The sea-run stocks are also sampled as a single lot, as a group of particular interest. 'Ad hoc' testing is carried out as required (eg to investigate the cause of runting) at least 3 to 4 times per year, as determined by the perceived general health of the stock.

Annual on-site inspections are made by a MAF veterinary officer. This inspection covers discussion with the farm manager and staff with regard to stock health, inspection of the stock and the mortality records. The veterinary officer fills out a form detailing mortalities, stock movement records and comments on stock health. Completed forms are sent to the MAF Fish Pathologist at the CAHL. These inspections and records complement the laboratory testing program. Information regarding the disease status of the inspected/tested properties is provided by the MAF Fish Pathologist to the Ministry of Fisheries, to assist with decisions on issuing salmonid movement permits. The local veterinary officer acts as a local health contact, to facilitate coordination with Fish Pathologist.

Good records are kept of feed utilisation, growth rates, mortalities, laboratory test results and compiled on computer, allowing comprehensive records for each pond.

Staff have attended salmon aquaculture and health courses organised by the New Zealand National Institute of Water and Atmospheric Research (NIWA).

NZKS Marlborough Sounds seacages

These were inspected with Mr Paul Steere, Chief Executive of The New Zealand King Salmon Co Ltd. The company manages four seacage operations on the Marlborough Sounds, three in Queen Charlotte Sound and one in Pelorus Sound. The seacage operation is operated under a Marine Farm Licence issued by the Ministry of Fisheries and is subject to the Resource Management Act 1991.

The seacages are suspended on square floats constructed of tubular steel, and are arranged in rows connected by walkways, being serviced by barges and boats. Water temperatures range from 11–19°C. The cages are 10–15 metres deep, and are located in water at least 30 metres deep (Otanerau is over 40 m deep) a short distance offshore in sheltered bays, the water depth and tidal flow being reported to be sufficient to ensure adequate flow of oxygenated water into the cage as well as to prevent water fouling from uneaten food and faeces. Nets are pulled frequently to remove biofouling. No antifouling is used on the nets. The seacages are sometimes resited within the licence area, though one seacage is permanently sited as it is located in an area of high tidal flow. The nature of the construction facilitates easy inspection and assessment of the various stocks, as well as easy stock movement. Fish stocks are periodically graded for size and reallocated into uniform size groups, gradually moving the stock along towards the harvest area. Problems of seal bites are controlled by keeping the nets in a tight state, assisted by acoustic warning devices — these latter only keep out 'inexperienced seals' — the habituated seals ignore it.

The fish are fed from bins using locally produced extruded pelleted feed (NRM Aquafeed) distributed by spinners. Automated dispensing is now being trialed, using a feedback system from Tasmania. Some imported extruded feed is used occasionally. Production stocks are all female, a record being kept of growth performance for use in selection of microchipped sister stock at the hatcheries for use for broodstock purposes. Harvesting is carried out throughout the year at approximately 3 kg weight (ranging from 2.1 kg to 3.6 kg).

Fish for harvest are crowded together using nets, anaesthetised with Aqui-S®, and harvested by fish pump, following which the fish are mechanically pithed and manually bled out by incising the gills. Fish showing blemishes, deformities, lesions (including seal bites) or signs of overmaturity (skin darkening) are rejected at this stage, being used for the petfood industry. The fish are immediately placed in a sea ice slurry in bins and despatched by barge to Picton, for transport by truck to the processing plant at Nelson, about a 3 hour journey.

Senior staff have attended fish health and related courses at the Cawthron Institute in Nelson, which included instruction from the MAF Fish Pathologist.

The company maintains comprehensive computerised records of stock levels and sizes, feed consumption, and mortalities by cage and date. Company divers check 2–3 times a week for mortalities. Two sites also use weighted rings to collect mortalities to facilitate collection, but the other site (Tory Sound) has too much current to allow this. The salmon are harvested when about 3kg in weight.

Survival rates over the growout period vary, but are currently about 85%. There are reported to be very few disease problems in the cages, and blemishes were rarely seen on the skin, reportedly because lower stocking rates are used compared to stock at Big Glory Bay. Other occasional problems are reported to include sunburn, amoebic gill disease, and algal blooms (*Heterosigma*). Predation of smaller fish by birds is minimised by use of netting. The main local problems were seals which caused considerable stress to fish stocks as well as carcase damage. This was well demonstrated at the time of the visit, when a seal caused nearby cage stocks to school violently.

Batch samples (60 fish per batch) from each age class on each sea-cage site are submitted for routine health monitoring checks to the MAF Fish Pathologist twice during the growout phase, sometimes from stock under one year, and regularly from one and two year old stocks, prior to harvest. Organ samples are collected on ice and sent by air courier to the MAF Fish Pathologist at the CAHL for testing. This program is supplemented by *ad hoc* testing of any diseased fish if needed.

Annual on-site inspections are made by a MAF field veterinary officer. This inspection covers discussion with the farm manager and staff with regard to stock health, inspection of the stock and the mortality records. The veterinary officer fills out a form detailing mortalities, stock movement records and he comments on stock health. Completed forms are sent to the MAF Fish Pathologist at the CAHL. These inspections and records complement the laboratory testing program. Information regarding the disease status of the inspected/tested properties is provided by the MAF Fish Pathologist to the Ministry of Fisheries, to assist with decisions on issuing salmonid movement permits.

NZKS Pupu Springs hatchery and freshwater growout facility

This combined hatchery and growout facility uses the outflowing Pupu Springs. The operation is operated under a Marine Farm Licence issued by the Ministry of Fisheries and is subject to the *Resource Management Act 1991*.

This facility is mainly geared to produce smolt for the Marlborough Sounds seacage operation, but also grows freshwater salmon to market size. It previously produced 500 tonnes per annum of total biomass, but now proposes to discontinue production of freshwater growout as management wishes to concentrate on seacage fish, allowing focus to be on smolt production.

The water supply from Pupu Springs is extremely clear, and has a high calcium content from its passage underground. Water temperature is 12°C. Grids are use to hold back vegetation at the point of entry, and are followed by mesh screens to hold out any fish located in the Springs (as a small number of trout exist in the Springs and an associated stream).

The hatchery contains a large number of concrete raceways, and reuses waters as it passes through the farm complex. Despite this, the used effluent waters are still nearly crystal clear. No silt was seen in the raceways, just residual food and faeces. The raceways are cleaned out twice a week, mainly to remove weed and algal growth. The stock are hand fed until used to spinners and self feeders.

The effluent water is discharged through an unlined channel into a settling pond, and thence over a liftable gate out to a stream. The settling pond is cleaned out once a year, and the residue held locally, though some dried residue is used as a fertiliser.

Broodstock are selected from sisters of monitored seacage production stock. The females are slaughtered to collect the eggs, which are then mixed with milt from sex reversed females. The company uses metal upwelling incubators to hatch and grow its young stock, and also uses newer plastic incubators, which appeared to be well designed. Equipment is disinfected with Chloramine T. The unhatched eggs are treated with malachite green twice daily up to 3 days prior to hatch, to control *Saprolegnia* infection. The larvae are held in

the incubators for up to 6 weeks then taken to outside ponds.

The company maintains comprehensive records of stocking rates, feed utilisation and mortalities, and stated it aims for mortalities of less that 3% over the growout period, usually achieving a lower mortality rate than this. No photoregulation or triploidy is used. The fish are sorted for size using a sizing machine. Mortalities are checked twice a day, this being easy to do because of the layout and exceptionally clear water.

The freshwater stock are crowded and harvested by fish pump, anaesthetised with Aqui-S® in a tank, then pithed by hand and the gills incised to allow bleeding, and the fish placed in an ice slurry in plastic vats (0–1°C). The fish are then trucked to the Nelson processing plant as soon as possible the same day, for processing next morning.

Survival rates from stripping to hatch are reported to average 75–85%, while from hatch to smolt they average 80% (97% exclusive of culls). There is stated to be little problem with disease, and no antibiotics or vaccines are used. The company places a high priority on biosecurity and hygiene (it required us to wear disinfected gumboots while on the property). *Saprolegnia* infection of unhatched eggs is controlled with malachite green, while sunburn (due to the sunny climate and exceptionally clear water) is controlled by use of sun shades over the ponds to protect the fish. Some fish are reported to develop calcification of the kidney due to the high calcium intake and older poorer diets, though this is no longer of concern.

Organ samples from year lots of fish are sent to the MAF Fish Pathologist in batches of 60, at about 6–9 months of age, one year and two+ year brood stock for routine health monitoring. There is provision for 'ad hoc' testing, especially if there are perceived problems with the performance of the stock (eg to investigate the cause of runting). Staff stated that ad hoc testing is conducted at least 3–4 times per year. Records are kept of feed, numbers of fish, mortalities, sizes and laboratory tests results.

Annual on-site inspections are made by a MAF field veterinary officer. This inspection covers discussion with the farm manager and staff with regard to stock health, and inspection of the stock and the mortality records. The veterinary officer completes record summaries of mortalities and stock movements and comments on stock health. Completed forms are sent to the MAF Fish Pathologist at the CAHL. These inspections and records complement the laboratory testing program. Information regarding the disease status of the inspected/tested properties is provided by the MAF Fish Pathologist to the Ministry of Fisheries, to assist with decisions on issuing salmonid movement permits.

NZKS processing plant (Nelson)

The bled fish are received packed in bins in an ice slurry, placed by forklift on a table, then manually placed on a near-vertical processing chain. The abdomen is then manually incised, viscera removed, the blood vessels along the backbone incised, and the residual blood, kidneys and tissues removed with a vacuum aspirator. The fish are then dispatched by endless belt to a grading table, where they are manually graded to first grade (no blemishes), second grade (minor blemishes or cuts), or rejected (major blemishes, cuts, overmaturity etc) to reject bins. They are then mechanically graded by size, placed in bins, and dispatched to a chiller or to a –30°C freezer. They are then given a water glaze to prevent freezer burn. Carcases can be rejected at any stage where blemishes are detected, the rejects being diverted for use in pet food. Any fish that fall to the floor are stated to be reinspected and either recycled back to the beginning of chain, or rejected and sent for pet food. Pin bone removal from fillets is not routine, and is only carried out specifically to order.

Fish are then despatched to the packaging area for sale, if sold as chilled or frozen head-on or head-off gilled gutted fish, or are dispatched to separate areas of the factory for further processing such as hand filleting, mechanical skinning and slicing, or hot or cold smoking. We were unable to view these latter areas in operation as they had shut down for the day, however the facilities appeared clean and well equipped. There are separate smoking and packing areas, and we were not permitted entry to these, though we could view staff working in these areas through a viewing window.

Factory personnel stated they carry out periodic checks for abnormalities in the viscera, but that these are rarely

seen. Any abnormalities detected would initially shown to experienced management staff, and where appropriate, fresh and fixed organ samples would be sent to MAF for testing. The factory considers its staff to have sufficient experience to be able to detect any abnormalities. The company can identify the origin of any fish with abnormal viscera right back to the growout cage and thence to the hatchery through its data management system.

All the operations appeared very clean and efficient, with a strong emphasis on hygiene. The company required protective clothing and hats on entry, together with washing of hands prior to entry, and washing of gumboots in disinfectant. Disinfectant footbaths were present in all separate areas of the plant, the disinfectant being reported to be replaced several times daily. Knives are stated to be washed at least every 2 hours (hosed with cold water, scrubbed in warm water and detergent, and stored in a knife sanitiser until next use). Production is stated to be to ISO9000 standards, and the company uses a HACCP process audited by MAF inspectors, currently at a rate of 7 hours per fortnight.

The New Zealand King Salmon Company is stated to produce about 5000 tonnes per year, much of it for export to countries around the Pacific Basin. Management stated it preferred to market gilled and gutted fish, for commercial reasons. Product is sold on basis of customer preference and includes a wide range of sliced product, pieces, hot or cold smoked product, gravulax etc.

The main reasons for carcase rejection are stated to be for 'bloat' or seal bites, plus cosmetic reasons such as cuts, blemishes.

GENERAL INFORMATION

Role of industry

Industry is responsible for monitoring fish health, and utilises MAFQual scientists to investigate on-farm disease problems. The National Institute of Water & Atmospheric Research (NIWA) and MAF Quality Management (MAFQual) provide training to industry in support of this approach. There is a legal obligation to report detections of notifiable aquatic animal diseases.

The New Zealand Government funds specific health surveillance projects based on MAF advice.

There are industry Codes of Practice for the production of mussels and rock lobsters, but not for salmon production.

The Seafood Industry Council (SEAFIC) provides general assistance to the wild and cultured salmonid-growing industry, including:

- training (with government funding);
- research (mainly in relation to wild fish). This has joint government and industry funding, and mainly relates to catch limits, devolution of responses to industry;
- business services — advice on managing change, setting standards, etc;
- provision of trade information eg on APEC, WTO non-tariff barriers;
- provision of material for public awareness/publicity.

Role of Ministry of Fisheries

The Ministry of Fisheries is responsible for supervising management of marine fish and freshwater fish (including aquacultured fish). The Ministry of Fisheries responsibilities include licensing, movement of sea fish and freshwater fish, regulation of salmon production under a quota management system, and management of disease control areas. The Ministry is responsible for regulation of fish movement under the Freshwater Fish Farming Regulations 1983, the *Conservation Act 1987*, the Fisheries Act 1983 and the Marine Farming Act 1971, including authorisation of fish transfer, and guidelines for salmonid transfers (with regard to *M. cerebralis* and *Y. ruckeri*). Under this legislation, the movement of live salmonids out of disease control areas is regulated within the South Island, and live salmonid fish are not allowed to move from the South to North Islands.

Training courses on salmon aquaculture and diseases are run by the NIWA, with assistance from other staff including the MAF Fish Pathologist. Manuals contain brief description of disease and sampling procedures.

Role of Ministry of Agriculture and Forestry

The Ministry of Agriculture and Forestry (MAF) is responsible for fish health issues, export testing, and import/export issues. Diagnosis of any fish disease that is notifiable under the *Biosecurity Act 1993* must be reported to the New Zealand Chief Veterinary Officer and where appropriate to the OIE. Subsequent action would depend on the nature and severity of the disease. Any health surveillance requirements negotiated with New Zealand's trading partners are included in export certification for specific commodities. MAF provides the services potential exporters need to meet these requirements.

All farm sites of the large New Zealand salmon companies are now stated to be under a routine disease inspection and testing program which has been running for two years for two of the companies and one year for the other. The companies involved, the New Zealand King Salmon Co Ltd, Sanford Ltd, and Amuri Salmon Co Ltd supply almost all New Zealand's salmon exports.

Annual on-site inspections are made by MAF veterinary officers. Inspections include discussion with the farm manager and staff with regard to stock health, inspection of the stock and the mortality records. The veterinary officer completes records detailing mortalities, stock movement records and comments on stock health. Completed forms are sent to the MAF Fish Pathologist at the CAHL. These inspections and forms complement the laboratory testing program. Information regarding the disease status of the inspected/tested properties is provided by the MAF Fish Pathologist to the Ministry of Fisheries, to assist with decisions on issuing salmonid movement permits.

This inspection procedure is stated to be in place for all freshwater and marine salmon farm sites included in the Salmon Farm Disease Freedom Assurance Program, and is also in place for the North Island sport fish hatcheries.

Exports

Health certification requirements are negotiated with trading partners and included in export certification for specific commodities. MAF will provide services to exporters to meet these requirements.

The New Zealand Chief Veterinary Officer would certify country freedom from *Henneguya salminicola*, *Kudoa thrysites* and *Ceratomyxa shasta,* although other species of *Henneguya* and *Kudoa* have been detected in eels and cod respectively.

MAF confirmed that the disease monitoring program involves testing once in hatchery, twice in sea cages (at 4–6 months and at harvest). The current level of testing is considered adequate to support current export certification requiements.

The export of head-on, gilled and gutted product is preferred, for commercial reasons.

Testing of New Zealand salmon farms for US certification purposes commenced in 1985 and continued for 9 years until the USA changed its certification requirements. The disease agents included in this program were VHS virus and *M. cerebralis*. The disease testing program was reduced in intensity following relaxation of US requirements in 1994, but it was increased again after 1996, with inclusion of all cytopathic viruses, *M. cerebralis, A. salmonicida, Y. ruckeri* and *R. salmoninarum.* Routine testing protocols would also detect *Vibrio* species if present.

MAF has, in the last two years, conducted projects which included testing for the OIE listed finfish viruses (EHN, IHN, OMV, SVC,VHS), and for *Y. ruckeri, A. salmonicida,* and *R. salmoninarum.* These programs covered a broad range of properties, aquacultured and sea-run salmon and sport fish.

Quarantine policy

MAF advised that head-off, gilled and gutted salmon are allowed entry into New Zealand, also that trout and salmon caviar are also allowed entry if pasteurised and packed in sealed containers. Other marine finfish for human consumption are allowed entry, with no specified conditions. Marine shellfish are allowed entry as food if dead and removed from the shell. Preserved and mounted fish are allowed entry if formalin fumigated. Fishing rods are allowed entry subject to treatment with hypochlorite. Fish meal is allowed entry, subject to heat treatment at 85°C for 15 minutes. The protocols for importation of fish products for use as fish bait and in animal feed are still evolving. Imported salmon is

processed at one approved plant; this product is used for local consumption only.

Public health controls

MAF has legal responsibility for food safety standards, including those that relate to the export of fish or fish products from New Zealand. MAF Regulatory Authority (MAFReg) provides policy, specification and independent audit, is the controlling body for sea food, and has accountability and responsibility for food safety standards, branding and certification of products and byproducts. MAF Quality Management (MAFQual) has responsibility for inspection of product and byproduct, ensuring compliance with standards, and providing certification on behalf of MAFReg. The performance of MAFQual in these roles is audited by MAFReg.

Processing and packing of salmon for export is carried out in fish packing houses. These premises are licensed under the Meat Act 1981 for the processing, packaging, preservation, handling, or storage of fish and their products, the Act and its regulations providing appropriate regulatory controls for export seafood (products and byproducts). Primarily, concerns are for the safety and wholesomeness of food, as well as for truth in labelling. The specific legal requirements relating to fish are contained in the Fish Export Processing Regulations 1995, which cover plant construction standards, hygiene requirements, compliance checks, export certification, and powers to examine, sample and reject fish. It is recommended that companies use the HACCP system as a tool for process control for ensuring food safety. Adequate records must be kept to demonstrate compliance with the approved process.

Frozen fish and fish products must be stored and transported at –18°C or colder. Chilled fish must be stored and transported at –1°C to +1°C.

General laboratory procedures

The MAF Fish Pathologist (Dr C Anderson) reaffirmed that the program for health surveillance involves annual testing of all year groups (*M. cerebralis* — first year stock only) at Amuri Salmon Co and all hatcheries supplying Sanford and New Zealand King Salmon seacages. It also involves testing of two year groups at the same sea cages, one at approximately 3 months after entry to seawater and the other at harvest. Lots of 60 fish are selected for sampling by age (by year groups), the poorest fish in hte group being taken first. There is also provision to receive samples from diseased fish at other times from the seacages and hatcheries in the event of unusual disease problems. Tissue samples are selected for testing as follows:

viruses	spleen, kidney, sometimes brain from pre-spawning classes, and ovarian fluid from spawning stock
M. cerebralis	heads
R. salmoninarum	kidney
Y. ruckeri	lower intestine
A. salmonicida	lower intestine

The CAHL supplies bacterial culture plates to salmon farms/hatcheries on occasions, but usually salmon farms and hatcheries send organ specimens (or whole fish if small) on wet ice direct via air courier. The farm and hatchery staff have manuals giving brief descriptions of diseases and sampling procedures, and have attended training courses in health management and fish diseases, to assist the diagnostic process.

Bacteriological examinations are based on direct plating of tissue material, without any broth culture step. Use of direct plating minimises costs and is considered adequate for disease diagnostic purposes for all named bacteria. Plate cultures are conducted individually for each fish submitted.

Routine testing for cytopathic virus was previously based on two passages in CHSE and BF2 cell cultures, but is now based on a single passage of two weeks (the USA only requires one passage to comply with its requirements). The CAHL's quality assurance program requires regular testing of fish cell lines currently used for virus isolation (including most of the OIE-listed viruses) and for work on mycoplasmas. The QA program has been running for more than 12 years.

Health surveillance and monitoring

The MAF National Manager, Disease Surveillance (Dr R Poland) provided details of a major viral disease survey which was carried out over a 21 month period

from March 1996 to December 1997. The survey involved a testing program designed to detect the five OIE listed finfish viruses (EHN, IHN, OMVD, SVC and VHS). The survey covered 3 North Island trout hatcheries, and 18 South Island salmon farms and salmon sea-return sites. Most salmon farms were sampled twice. Brain, kidney and spleen were sampled at all sites, while ovarian fluids were collected from 5 South Island salmon farm sites and 3 North Island wild trout sites. Samples were pooled in groups of 5. Test protocols were based on those of the OIE Diagnostic Manual for Aquatic Animal Diseases, ie two 14 day passages in BF2, CHSE-214 and EPC cells. The only virus isolated was an aquatic birnavirus, found in sea-run quinnat salmon in the Hakataramea River and at one sea-run farm site (previously isolated from this site). See adjacent table for details.

		NUMBERS OF FISH SAMPLED	
FISH TYPE	SOURCE	TISSUES	OVARIAN FLUID
Salmon	Farmed	3026	361
	Wild	466	431
Trout	Farmed	675	–
	Wild	–	75
Total		**3722**	**868**

IPN-like birnavirus has only been isolated from Tentburn sea-run salmon, and from sea-run salmon in South Canterbury in the Rakaia river tributaries. This organism is occasionally reported and always in the absence of clinical disease.

Between 1993–1997, diagnostic tests for *Y. ruckeri* and *A. salmonicida* were carried out on 1128 salmon and 695 trout. The trout (mainly rainbow trout) were from the North Island and the salmon were from the South Island. *Y. ruckeri* was only isolated (on 5 occasions) from South Island salmon, located in the Rakaia and Waitaki catchments and near Blenheim. *A. salmonicida* was not detected.

Y. ruckeri has been confirmed only in the South Island from Blenheim to the Waitaki River (a reported North Island isolation is considered to be a laboratory error, as further investigations failed to confirm this finding).

Between 1986 and 1995, a total of 1173 freshwater, seacage and searun salmon were tested for *R. salmoninarum,* using histopathological and bacteriological techniques. During 1997, a survey was carried out using the enzyme-linked immunosorbent assay (ELISA) for *R. salmoninarum.* The survey involved 919 quinnat salmon and 47 sockeye salmon at farm sites and sea-return locations, as well as 23 rainbow trout from the central North Island. All results were negative.

M. cerebralis infection occurs in the South Island only and is considered to be limited in its distribution to a zone demarcated by the east coast of the South Island, the Waimakariri River (in the north) and the Clutha River (in the south). During the visit, data was provided on recent testing of rainbow trout from the North Island, and of wild brown trout and salmon were from the South Island for *M. cerebralis.* A total of 5700 salmonids were tested between 1992 and 1996 using digestion/differential centrifugation techniques. Additional locations where *M. cerebralis* has been detected include the Kuriwao and Waiwera streams, on the southern edge of the known infected area.

V. ordalii has been detected in marine waters and fish at Stewart Island, Owaka (Dunedin) and Marlborough Sounds, but has not been detected in the last 8–10 years.

V. anguillarum infection has not been diagnosed in fish, but this organism has been isolated from the environment.

The presence of VHS has not been reported during a period of 7–8 years surveillance and *ad-hoc* studies, although the program was discontinued for 2 years.

*Aeromona*s spp, *Vibrio fischeri* and *Flexibacter* spp have been reported occasionally, mainly in gold fish, and rarely in freshwater salmon.

'Bloat' is a occasional problem at sea cages. It is due to a distended and inflamed swim bladder, cause unknown. It is apparently associated with head of bay locations, poor flushing of waters, and possibly stress. The distended swim bladder is filled with sea water and is inflamed though this is not associated with any particular bacteria or viruses. The condition appears to be less common in fish fed imported fish meal. Gastric distension and inflammation is also seen occasionally at Stewart Island sites.

Regarding health issues of galaxid fish, the MAF Fish Pathologist (Dr C Anderson) advised that there has been no specific investigation of disease in galaxids in New Zealand. A 1991–93 survey for *Y. ruckeri* and *A. salmonicida* did include 55 galaxid fish which tested negative for these two disease agents. Dr Anderson stated there is no evidence in New Zealand that galaxids are susceptible to *M. cerebralis* infestation. Galaxid fish have been examined a number of times, but mortalities could not be attributed to any recognised pathogens of salmonid fish.

Pathogenicity testing

The pathogenicity of strains of *Y. ruckeri, V. anguillarum* and *V. ordalii*, found in New Zealand has not been determined partly because of a lack of overseas isolates for comparative purposes. Information on pathogenicity is based on field experience and data obtained from vaccine trials. The LD_{50} for *V. ordalii* and *Y. ruckeri* by intraperitoneal injection in young quinnat salmon was determined to be 10^4–10^5 colony forming units in laboratory trials.

By use of standard bacteriological identification methods and restriction endonuclease analysis (REA), it has been shown that *Y. ruckeri* isolates in New Zealand belong to a single strain (typed as serotype 1 by the United Kingdom). Similar studies have shown that one of the two Australian isolates of *Y. ruckeri* had an identical REA profile to that of the strain which occurs in New Zealand.

IPN-like birnavirus: In a preliminary transmission test, involving intraperitoneal inoculation of six young rainbow trout, there was no clinical or histopathological evidence of disease. This preliminary study has not, apparently, been followed up. Clinical disease has not been associated with natural infection of sea-run salmon, nor has it been seen in young quinnat salmon reared from their eggs.

Acknowledgment

I would like to thank Dr Matthew Stone and Mr Mark Gillard for their help in organising the itinerary, meetings and accommodation for this visit. Thanks are also due to MAF and numerous other government and industry staff for their very willing cooperation and assistance during the visit.

Dr PJK Durham
Senior Veterinary Officer
Animal Quarantine Policy Branch

26 June 1998

Appendix 4

Health status and health regulation of the New Zealand salmon aquaculture industry

PAPER PREPARED BY THE NEW ZEALAND MAF Regulatory Authority to summarise the health status and health regulation of the salmonid aquaculture industry of New Zealand. The information was provided by various persons from The New Zealand King Salmon Company (Paul Steere, Mark Gillard, Greame Davidson), Sanfords (Tommy Foggo, Peter Buxton), the New Zealand Salmon Farmers Association (Mark Gillard), MAF (Colin Anderson, Roger Poland, Judy Barker, John Lee) and the Ministry of Fisheries (Peter Todd).

The information was presented to Dr Peter Durham, Australian Quarantine Inspection Service, during his visit to New Zealand in November 1997 while preparing a technical report for a risk analysis examining the disease risks posed by imports into Australia of New Zealand salmon products. Dr Durham prepared an early draft of the technical report before the risk analysis process was halted by AQIS. That draft was used during the preparation of this paper.

Matthew Stone
National Adviser International Animal Trade
MAF Regulatory Authority

Overview of salmon aquaculture industry

NEW ZEALAND SALMONIDS

New Zealand has seven species of salmonids, all of which were introduced. Importations were mainly imported as ova late in the last century and up to about 1930 (McDowall 1990). The sports fishery is based on rainbow, brown and brook trout. These species are classed as sports fish under the Freshwater Fisheries Regulations 1983 and are not allowed to be farmed or sold commercially. Other species of minor importance include Atlantic salmon and mackinaw trout. The commercial fishery is based on quinnat salmon, with a small amount of sockeye salmon.

The salmonid species known to be present in New Zealand are the following:

- *Oncorhynchus mykiss* (rainbow trout)
- *Oncorhynchus tshawytscha* (chinook, quinnat or king salmon)
- *Oncorhynchus nerka* (sockeye salmon)
- *Salmo trutta* (brown trout)
- *Salmo salar* (Atlantic salmon)
- *Salvelinus fontinalis* (brook char)
- *Salvelinus namaycush* (mackinaw)

THE NEW ZEALAND SALMON AQUACULTURE INDUSTRY

Originally, salmonids were introduced for the purposes of recreational fishing, which has developed into a substantial industry. Enhancement programs for wild salmonids are currently operated by the New Zealand Fish and Game Council and the Department of Conservation.

The New Zealand Conservation Act 1987 prevents the farming and sale of all sports fish, but farming of Atlantic, quinnat and sockeye salmon is permitted under the New Zealand Fisheries Act 1983 and the Freshwater Fish Farming Regulations 1983. Management of aquaculture, in particular designation of approved areas for farming, is also covered by the Resource Management Act 1991.

The New Zealand salmon aquaculture industry began with stocks sourced from the wild in the 1970s. The aquaculture method was initially ocean ranching. Sea cage culture was set up in the 1980s in Big Glory Bay, Stewart Island. The industry is now largely based on sea-cage culture of quinnat salmon, which accounts for over 94% of New Zealand salmon production.

Studies of the salmon aquaculture industry (Wildman 1992; Commerce Commission 1995), company profiles and information generated through reviews and submissions during market access negotiations for salmon imports and exports provide a significant information resource for the industry in New Zealand.

National industry associations

The New Zealand Salmon Farmers Association (NZSFA) is the national body representing the salmon aquaculture industry.

The Seafood Industry Council (SeaFIC, previously the Fishing Industry Board of New Zealand) is the national body representing the fisheries industries of New Zealand, and in that role also represents the salmon aquaculture industry.

Size and value of the industry

The domestic salmon industry is small by international standards. There are 23 salmon farms rearing quinnat and sockeye salmon in the South and Stewart Islands.

In 1991 New Zealand produced about 2,000 tonnes of salmon products. In 1995 New Zealand exported 4,183.5 tonnes of product worth $27.2 million (statistics on New Zealand production provided by the Fishing Industry Board of New Zealand). In 1996 New Zealand produced 5,986.3 tonnes of salmon products worth $34.7 million.

About 21% of production goes to the domestic market, and 79% is exported. The major export markets for New Zealand aquacultured salmon products are Japan, Taiwan, Australia, the USA, the United Kingdom, France, Germany, Belgium, Italy, Guam, Hong Kong and Singapore.

The New Zealand King Salmon Co Ltd

In 1996 the New Zealand King Salmon Company Ltd was formed following purchase by Southern Ocean Seafoods Ltd of the assets and business of Regal Salmon Ltd. The company is a wholly owned subsidiary of Karamea Holdings Ltd, which is ultimately owned by the Tiong Group of Malaysia (King Salmon Company of New Zealand Ltd 1996). The estimated 5,000 tonne harvest of king salmon in the 1996 year comprises 80% of the total New Zealand production, and makes the company one of the largest producers of farmed king salmon in the world.

New Zealand King Salmon Co Ltd operates two hatcheries located at Tentburn (south of Christchurch) and Takaka (which has until recently also been used for freshwater grow-out). The company has six sea cage operations located in the Marlborough Sounds, sourcing its stock from both hatcheries. It also has a broodstock

research centre located at Kaituna, and a full processing facility located at Nelson including a large smokehouse.

All salmon feed is purchased from a dedicated local plant or imported from Chile or Tasmania in accordance with New Zealand government regulations. Feeds are independently verified according to company specifications before use.

The company maintains extensive monitoring and testing of livestock and product to ensure quality compliance with statutory requirements, internal company standards and customer request, including independent analysis of livestock, environmental issues, processing equipment and surfaces for pathogens. It uses hazard analysis at critical control points (HACCP) as its quality process standard.

No vaccines or antibiotics are used during the production process. Disease surveillance programs have been conducted in cooperation with MAF. Between 1985 and 1998 sampling occurred 2–3 times a year, covering *Myxobolus cerebralis* and cytopathic viruses at Tentburn hatchery. There is also a 14 year record of testing at the Takaka hatchery/grow-out facility. Testing is periodically carried out on sea cage stock. Bacterial testing for *Yersinia ruckeri, Aeromonas salmonicida* and *Renibacterium salmoninarum* has been undertaken routinely since 1997, and during clinical disease investigations in previous years. The only disease events recorded to date are at Tentburn, which recorded *Myxobolus cerebralis* in 1994 in 2 smolts from the 1993 year-class sockeye salmon, and mortalities in quinnat smolt in 1993 associated with *Y. ruckeri*.

Sanfords Ltd

Sanfords Ltd produces most of the remaining New Zealand salmon production (1100 tonnes). The company operates two hatcheries located in Waitaki and Kaitangata (a third hatchery at Marlborough is no longer in use), and grows-out in two sea cage operations located in Big Glory Bay on Stewart Island. Product is processed at facilities located at Bluff.

The fish are fed extruded pelleted feed imported from Tasmania. No vaccines or antibiotics are routinely used during the production process. The company also participates in MAF fish health surveillance programs, and has experienced no significant disease problems.

The processing plant at Bluff maintains a high standard of operation under ISO 9000. It is currently introducing HACCP procedures.

Other companies

Production from the Amuri Salmon Co. and the other remaining 20 companies is relatively small, amounting to about 150 tonnes.

Health status of salmon in New Zealand

New Zealand salmonids have an excellent health status, though they are not free of all disease. The introduction of salmonids into New Zealand appears to have been achieved without the introduction of many of the serious diseases that occur elsewhere. This claim is based on passive surveillance (the long history of observation of the species in New Zealand and the lack of disease occurrences that have been observed) and testing for the presence of specific disease agents for purposes including disease monitoring, research and export certification.

Hatchery-bred fish, whether for commercial aquaculture or recreational enhancement, are routinely monitored for the presence of disease. Fish-kills in the wild populations are investigated by MAF and the Ministry of Fisheries.

The following salmonid pathogens/organisms have been recorded in New Zealand.

VIRUSES

IPN-like birnavirus

A marine birnavirus has on occasion been reported from quinnat salmon returning to South Island rivers (Tisdall and Phipps 1987). The virus has had no impact on salmon farming.

Bacteria

Yersinia ruckeri

A strain of *Y. ruckeri*, causing enteric redmouth disease (ERM), has been isolated from salmonid farms and hatcheries here. The New Zealand strain has not been definitively serotyped but reacts strongly with type 1 and weakly with type 2 antisera. The REA profile of New Zealand isolates are very similar to Australian isolates. On this basis these isolates are suspected to be type 1 (pers. comm. Colin Anderson, MAF Wallaceville Animal Health Laboratory, Jan. 1997).

Vibrio ordalii

Vibriosis caused by *V. ordalii* has been an intermittent problem in salmon farming in New Zealand. Studies have shown all New Zealand isolates to be very similar to the type strain (Wards et al 1991). That study examined fish suffering clinical vibriosis from seven outbreaks in five geographically distinct marine areas comprising all marine and brackish water salmon-rearing areas in New Zealand.

Vibrio anguillarum

A study of *Vibrio anguillarum* isolates from sites around New Zealand, including salmon farms, has demonstrated that these isolates differ from pathogenic Northern Hemisphere strains (Powell and Loutit 1990).

Bacterial gill disease

Bacterial gill disease occurs in New Zealand (Boustead 1985). However, the bacteria associated with BGD infections in New Zealand have not been precisely defined.

Flexibacter columnaris

Flexibacter sp. and *F. columnaris* occur in New Zealand (Boustead 1982; Anderson 1996). They have been reported to cause disease in cultured elvers in warm water (noted by McDowall 1990). Bacterial gill disease and columnaris disease were the most common infectious diseases referred to Fisheries Research Division of MAF prior to 1982 (Boustead 1982). The relative pathogenicity of New Zealand strains compared to overseas strains has not been determined.

Aeromonas hydrophila

A. hydrophila has been isolated several times from the kidneys of dead or moribund fish in New Zealand, including from salmon, trout, ornamental fish, and eels (Boustead 1982).

Hafnia alveii

Hafnia alveii has been isolated from salmonids in New Zealand (Anderson 1996).

Nocardiosis

Losses of quinnat salmon in 1972 at one fresh water salmon farming location in New Zealand were attributed to nocardiosis (Boustead 1985).

Mycobacterium sp.

Mycobacterium marinum has been identified in imported tropical fish, as have other acid-fast bacteria (Boustead 1982).

Fungi

A variety of fungi have been isolated from gill, integumentary and egg mycoses of fresh water fish including salmonids in New Zealand. The species recorded are *Saprolegnia* sp., *Aspergillus* sp., *Trichoderma* sp., *Peyronellaea glomerata*, *Botrytis* sp., and *Fusarium merismoides* (Boustead 1982). Fungal infections in fish in New Zealand are typically opportunistic infections in stressed fish.

Protozoa

Paramoeba sp. (amoebic gill disease)

Paramoeba sp. occurs in New Zealand, and has caused mild disease in salmon farms in the Marlborough Sounds and Stewart Island (Anderson 1996).

Myxobolus cerebralis

M. cerebralis occurs in New Zealand (Hewitt and Little 1972). The means of introduction has not been conclusively determined. The distribution and impacts of *Myxobolus cerebralis* in New Zealand have been documented (most recently by Boustead 1996).

Ichthyophthirius multifiliis

I. multifiliis has been recorded on the gills and skin of sockeye and quinnat salmon, as well as a wide range of other fish species including eels, in New Zealand (McDowall 1990; Anderson 1996). The parasite has occasionally caused major outbreaks of disease in cultured and feral fish in New Zealand.

Metazoa

The metazoan parasites which have been recorded from fin fish, including salmonids, in New Zealand have been reviewed (Hewitt and Hine 1972; Boustead 1982).

An up-to-date record of metazoan parasites from salmonids in New Zealand is held by the National Institute of Water and Atmospheric Research (pers. comm. Mike Hine, NIWA, January 1998).

Health regulation of salmon aquaculture

MINISTRY OF FISHERIES

The Ministry of Fisheries is responsible for administering the Fish Transfer Authorisation and Guidelines for Salmonid Transfers under the Freshwater Fish Farming Regulations 1983 and the Marine Farming Act 1971. The Ministry restricts movement of salmonids from the South to the North Island, and controls movement of stock across the boundaries of the *M. cerebralis* and enteric redmouth disease control areas. The Ministry of Fisheries is responsible for handling marine fish disease issues, though in practice it closely cooperates with and often utilises the resources of the Ministry of Agriculture and Forestry. The Biosecurity Act 1993 provides the legal basis for movement controls during a disease emergency.

It is illegal to transfer diseased fish under the Freshwater Fisheries Regulations 1983.

Whirling disease (*Myxobolus cerebralis*)

The control area for this agent is defined as that area east of the main divide of the South Island from the Waiau River in the north to the Taieri River in the south, and includes the adjacent seas.

Where *M. cerebralis* has been found within this area, transfer of salmonids to natural waters within the Control Area or to facilities within the Control Area require assessment of the implications of the translocation.

Where *M. cerebralis* has been found in a facility following a disease outbreak or routine surveillance, transfer of live salmonids from the infected stocks will not be approved.

Transfers to facilities outside the disease Control Area from facilities within the Area will be authorised provided the facility has participated in the MAF health surveillance program within the preceding 12 months, and all tests were negative.

Exemptions apply to salmonid eggs or fish that are water hardened and/or eyed and/or reared entirely in isolation on waters free of *M. cerebralis*.

Transfers of salmonids to the North Island from any South Island or Stewart Island source may be approved only for salmonid ova and milt or salmonid eggs or fish that are water hardened and/or eyed and/or reared entirely in isolation on waters free of *M. cerebralis*.

Enteric redmouth (ERM)

The control zones for ERM are the Rakaia, Tentburn and Waitaki catchments.

No salmonid transfers from facilities within the control area will be approved to areas outside of the control area or to areas isolated by dams or weirs where there is no record of previous transfer of potentially ERM affected fish, unless the facility has undertaken ERM testing within the previous 12 months or it is anticipated after consultation and a critical examination of factors such as the age and species of fish and the stock mortality records that such testing would prove negative.

Provisions for transfer of fish to the North Island or West Coast parallel those for whirling disease and recognise the natural barriers to the movement of salmon at sea.

MINISTRY OF AGRICULTURE AND FORESTRY

The Ministry of Agriculture and Forestry (MAF) retains close links with the Ministry of Fisheries in dealing with aquaculture issues. MAF provides policy advice to government on international trade in animal and plant

products, biosecurity, sector performance, sustainable resource use. MAF is primarily responsible for national animal health surveillance, import technical policy for animal and plant products, market access negotiation for animal and plant products, and emergency response for animal and plant disease outbreaks.

MAF Regulatory Authority (MAF Reg) develops and specifies standards for the agricultural, horticultural and forestry biosecurity, domestic and export food safety (including seafood), pest and disease management, agricultural compounds and animal welfare. MAF Reg comprises five chief technical officers (CTO), including a chief veterinary officer, chief meat veterinary officer, chief plants officer, chief dairy officer and chief forestry officer, and their staff, as well as generic groups for Compliance and Enforcement

MAF Operations (MAF Ops) consists of three subgroups, the MAF Verification Agency, the MAF Quarantine Service, and the New Zealand Animal Health Laboratory and Exotic Disease Response Centre.

The MAF Verification Agency (MAF VA) validates inspection processes, verifies and certifies export and domestic meat, dairy, and seafood products, including aspects relating to their preparation, packaging and storage.

The MAF Quarantine Service (MAF QS) provides inspection and clearance services for incoming passenger and cargo and vessels, ensuring imports of animal and plant products comply with import health standards.

The New Zealand Animal Health Reference Laboratory (NZAHRL) and Exotic Disease Response Centre (EDRC) have responsibility for national reference and diagnostic services, and for management and contingency planning of responses to exotic diseases and pests. These two organisations operate in association as the MAF National Centre for Disease Investigation (NCDI).

On 1 November 1998 the former MAF Quality Management became two state owned enterprises (SOE), Assure New Zealand and AgriQuality New Zealand. The SOEs continue to provide specific accredited services in animal, plant and public health according to standards set by MAF. Assure New Zealand is a MAF-accredited supplier of meat inspection services, including public health inspection of meat, fish and game animals, and including hygiene and quality management in food and other product processing industries. AgriQuality New Zealand is a MAF-accredited supplier of field veterinary livestock services, disease control, and services relating to exports of live animal and animal products.

COMPLIANCE

The MAF Reg Compliance Group provides services to the Chief Technical Officers (CTOs). The Compliance Group audits delivery agencies approved by the CTOs, ensuring that corrective action is taken where necessary; provides information to the CTOs and their staff on the efficacy and state of compliance of the relevant specifications; and provides technical advice and adjudication to assist with compliance with the specifications.

The Compliance Group has the authority to take any necessary action to obtain compliance. This may include restrictions on processing, removal of exports to certain markets, and suspension or cancellation of the licences held by the premises.

IMPORTS OF AQUATIC ANIMALS AND PRODUCTS

MAF Reg regulates entry of animals and animal products into New Zealand, and assists with market access for New Zealand products overseas. The relevant legislation controlling importation of risk goods into New Zealand is the Biosecurity Act 1993, particularly Part III Section 22 which describes the issue of import health standards. MAF Reg has incorporated the process of risk analysis into the development of all new import health standards, consistent with New Zealand's obligation under the GATT WTO-SPS agreement.

There are currently no import health standards for the importation of live salmonids or their viable genetic material into New Zealand.

Cooked salmon products are allowed into New Zealand from a number of countries. Following two risk analysis on the subject (MacDiarmid 1995; Stone et al 1997), fresh or frozen salmonid products may be imported under the conditions outlined in an import health standard for these products.

All imports of fish food and fish meal, including aquaculture feeds, must comply with an import health standard for these products.

AQUATIC ANIMAL HEALTH SURVEILLANCE

MAF Reg has developed standards for health surveillance programs in fish and shellfish. These focus on detecting unwanted organisms and maintaining an accurate knowledge of the endemic diseases. It is designed to give an early warning of exotic, new and emerging diseases and it uses the information generated from existing systems such as industry disease control programs, monitoring during processing, and cases sent to veterinary diagnostic laboratories.

The centre of animal health surveillance is the NZAHRL, which specialises in virology, serology, fish disease diagnosis, and exotic disease testing. The NZAHRL undertakes export health testing and active surveillance to support market access programs. Surveillance data also arises from diagnostic testing within a country-wide network of private veterinary diagnostic laboratories operating to standards set by the Chief Veterinary Officer and Chief Meat Veterinary Officer. This network gives New Zealand the capacity to gather information, to confirm animal health status and to respond to any suspected exotic disease in a nationally consistent and comprehensive manner.

In 1995 MAF commissioned a report on fish and shellfish surveillance in New Zealand (Hine 1995).

Testing of NZ salmon farms for US certification purposes commenced in 1985 and continued until the USA changed their certification requirements in 1993. The disease agents tested under this program were VHS virus and *M. cerebralis*.

The former MAF Central Animal Health Laboratory (now NZAHRL) compiled data during routine disease surveillance testing of salmonids. Bacteriological testing of 1173 freshwater, seacage and searun salmon for *R. salmoninarum* between 1986 and 1995 yielded negative results. A total of 5700 salmon were tested for *M. cerebralis* between 1992 and 1996, yielding one positive sample in a clinically normal farmed South Island sockeye salmon. Between 1993 and 1997, 1128 salmon and 695 trout were tested for *Y. ruckeri* and *A. salmonicida*, from which 5 isolates of *Y. ruckeri* were obtained. (Data supplied by Colin Anderson, Fish Pathologist, NZAHRL).

All farm sites of the larger New Zealand salmon companies (NZ King Salmon, Sanfords Ltd and Amuri Salmon) are now under a routine disease inspection and testing program which has been running for between 1–2 years. The test procedures are based on those of the OIE Diagnostic Manual for Aquatic Animal Diseases 1995, and cover all cytopathic viruses, *M. cerebralis*, *A. salmonicida*, *Y. ruckeri* and *R. salmoninarum.* The procedures will also detect *Vibrio* spp.

In addition to routine disease surveillance and monitoring, the Chief Veterinary Officer commissions specific projects to confirm the presence or absence of particular causative agents or clinical conditions. Recent fish disease surveys include surveys for cytopathic viruses and *Renibacterium salmoninarum,* both of which have yielded negative results (unpublished data, Colin Anderson, NZAHRL).

A major viral disease survey was carried out over a 21 month period from March 1996 to December 1997 which involved a testing program designed to detect the five OIE listed finfish viruses (EHN, IHN, OMVD, SVC and VHS). The survey covered three North Island trout hatcheries, and 18 South Island salmon farms and salmon sea-return sites. Most salmon farms were sampled twice. Brain, kidney and spleen were sampled at all sites, while ovarian fluids were collected from five South Island salmon farm sites and three North Island wild trout sites. Samples were pooled in groups of five. Test protocols were based on those of the OIE Diagnostic Manual for Aquatic Animal Diseases. The testing involved 3387 farmed salmon, 897 wild salmon and 750 farmed trout. The only virus isolated was an aquatic birnavirus, found in sea-run quinnat salmon in the Hakataramea River and at one sea-run farm site where it had been previously isolated (unpublished data, Colin Anderson, NZAHRL).

A survey for *Y. ruckeri* and *A. salmonicida* was undertaken between 1991 and 1993, and covered 799 farmed and wild salmonids and 78 other fish, most fish being from the South Island (Anderson et al 1994). *Y. ruckeri* was isolated from clinically normal salmon on two

South Island salmon farms, however the survey yielded no evidence of *A. salmonicida* infection.

In 1997, the New Zealand MAF also undertook a survey for *R. salmoninarum* utilising an enzyme-linked immunosorbent assay (ELISA) test. The survey involved 451 freshwater salmon at seven farm sites and 515 sea-run salmon at six salmon return locations in the South Island, and also included 23 rainbow trout from the North Island. All the fish tested negative for *R. salmoninarum.*

Further surveillance data are provided by occasional private research, such as the survey confirming the absence of infectious haematopoietic necrosis (IHN) virus from New Zealand sockeye salmon (Boustead et al 1993).

New Zealand is a member of the Office International des Epizooties (OIE), so reports quarterly and annually on its animal and fish health status.

EXOTIC DISEASE AND PEST RESPONSE

MAF Reg standards for exotic animal and plant disease response ensure capability to investigate and diagnose suspect cases of exotic disease, and response to a confirmed incursion of nominated unwanted organisms.

The EDRC is responsible for investigating and diagnosing suspect exotic disease outbreaks, and for coordinating and executing response plans. The MAF EDRC would contract external expertise during an investigation and response as required. For example, EDRC has worked closely with experts from NIWA to prepare for any pilchard mortality event in New Zealand waters following the Australian mortalities of 1998–1999.

RELEVANT LEGISLATION

The Resource Management Act 1991 introduced legislation to control the use, distribution and preservation of natural and physical resources. It places the emphasis on the effect a proposed activity will or might have on the environment. It also provides for the community to become involved in making decisions about resource management.

The Biosecurity Act 1993 replaced and consolidated several previous Acts. It shifts the responsibility for the control of pests (weeds, pests, and diseases of animals and plants) from direct Government control and funding to those responsible for the introduction and spread of the pest and those that will benefit most from its control. Central Government would be justified in funding pest management only when it represents 'public good output' or when pests on crown lands are causing problems to the crown or landholders. Notifiable organisms are declared notifiable under section 45 of the Biosecurity Act in the Biosecurity (Notifiable Organisms) Orders.

The Biosecurity Act 1993 introduced the term 'unwanted organism'. MAF Reg has developed a policy which lists the criteria for the various categories of unwanted organisms. Aquatic animal pathogens appear on two categories of the unwanted organisms list, namely 'notifiable organisms', for which every person has a duty to report to the relevant MAF Reg Chief Technical Officer, and 'other exotic organisms'. The unwanted organisms affecting aquatic animals within both the notifiable and other exotic organims categories are listed in Table 1.

Health regulation during salmon processing

REGULATORY SYSTEMS

MAF has the legal responsibility and accountability on behalf of the New Zealand Government for the food safety standards which relate to the export of meat (including seafood) from New Zealand. MAF is also responsible for control over the slaughter and dressing of animals for a multitude of end uses for both the domestic and international market.

The Ministry of Health has the legal responsibility for food safety once product is released onto the domestic market.

MAF Reg (Meat and Seafood) has the accountability and responsibility for food safety standards, branding and certification of products (edible) and by-products (inedible). MAFReg maintains documented standards/specifications that implement both the requirements of New Zealand legislation and the market access requirements of New Zealand's trading partners. The working procedures are formed into manuals of procedures. The manuals are the practical working

documents for both inspection and company staff. They contain instructions and guidelines for implementing the various Acts and Regulations. Because of the importance and necessity for all inspection staff to operate to the same standards, a national communication network exists, consisting of Technical Directives, Ministry of Agriculture Circulars and Technical Bulletins.

MAF Reg accredited suppliers of food inspection services are contracted by meat and seafood processing companies to provide food inspection services. The performance of accredited suppliers in these roles is audited by the MAF Reg Compliance Group. The MAF Verification Agency (VA) supervises meat and seafood processing premises which are producing products for export. The MAF VA is responsible for ensuring the requirements of trading partners have been met for all meat and seafood products exported from New Zealand.

LEGISLATION

MAF administers the legislation relating to the safety and wholesomeness of seafood.

The Meat Act 1981 and its regulations provide general regulatory controls for the slaughter, processing and sale of meat, venison, wild game and seafood (products and by-products) for human consumption. Primarily, concerns are for the safety and wholesomeness of food, as well as for truth in labelling. The Act controls appointment and powers of Inspectors, requirements for licensing of premises, inspection, production and prerequisites for the sale of meat and seafood and their products for human consumption, and requirements for the export of meat and seafood.

The Fish Export Processing Regulations 1995 provide specific legal requirements for construction and standards of fish export processing plant and equipment, obligations to maintain hygiene and quality, fitness for human consumption, storage and transportation, checks on compliance and corrective action, export certification, powers of Inspectors to examine, sample and remove unfit fish, and to prohibit the use of equipment or premises.

LICENSING AND OPERATION OF FISH PROCESSING PREMISES

The processing and packing of salmon for export is carried out in fish packing houses. These premises are licensed under the Meat Act 1981 for the processing, packaging, preservation, handling, or storage of fish and their products. Before a license is issued to a premises, an application must be made which provides information on the type of processing to be carried out, and detailed plans on the construction and operation of the premises. A license is granted provided the completed construction of the premises, its equipment and product flows meet the requirements of the legislation. Licenses may be suspended or cancelled by MAF if a premises is considered no longer fit for purpose or if the licensee has failed or refused to comply with any legislative requirements.

Premises processing and packing salmon must meet detailed construction and hygienic requirements. General requirement for operations in the premises are also specified. Provisions relating to the risk of contamination during processing, movement of appliances, movement of personnel are covered. specific attention must be paid to prevent cross contamination from raw to cooked product and from one process to another. Companies are required to have documented programs to cover sanitation and hygiene requirements.

INDUSTRY AGREED IMPLEMENTATION STANDARDS

The Fish Export Processing Regulations 1995 provide for the Director-General MAF to issue Industry Agreed Implementation Standards which provide means of achieving the standards of the regulations. These standards are typically developed by MAF and industry, through negotiations with the Seafood Industry Council or another industry representative.

SALMON PROCESSING PROCEDURES

Each company is required to check the salmon on arrival at the fish premises to determine that the salmon is fit for human consumption, that the salmon have been chilled or frozen since harvesting, and that he salmon is labelled or identified in the correct manner. Records must be kept of the checks carried out.

All salmon is required to be processed in accordance with an approved process. Processing is required to be carried out so that the possibility of contamination or deterioration of the fish is minimised. It is recommended that companies use the HACCP system as a tool for process control for ensuring food safety.

The standards specify a number of requirements that must be met when limited processing (eg filleting, gutting etc) is carried out. Where further processing (eg canning, smoking, drying) is carried out each process is required to have specific approval from the Inspector. The process approval must contain the critical control points, the checks carried out, and the action taken to correct any non-compliance. Adequate records must be kept to demonstrate compliance with the approved process.

Changes have recently been made to the processing standard to align the requirements more closely with the seven principles of HACCP (previously not all the principles were included). All processors will be required to undertake a hazard identification and where necessary develop a HACCP plan. Competent people are required to be involved in the development of the HACCP plan and the review of process records.

Companies are required to carry out daily and weekly checks of premises and of the product produced, classify defects and record actions taken to rectify any defects. The emphasis of the program is on the corrective action for any defect found being carried out in the minimum time.

The carrying out of pre-operative and daily checks on any day does not exempt a company from the responsibility of continuous control of processing throughout the day. Any defects found should be recorded in the normal manner and acted upon immediately.

The results of the inspection undertaken by the company checker are recorded on the Company Compliance Checklist. Where defects are found these must be recorded as well as the action taken and time allowed to resolve the defect. When the defect is corrected this must be recorded on the checklist.

COMPLIANCE WITH PROCESSING STANDARDS

The inspection of fish facilities is the responsibility of MAF VA Travelling Meat Inspectors (TMIs). The personnel involved in this function were formerly employed by MAF Quality Management, but were transferred into MAF VA on 1 November 1998 following restructuring of MAF. The MAF VA TMIs perform surveillance and inspection of fish and fish products during processing to enable official export certification to be given on behalf of MAF Reg. This involves delivery of quality assurance services including system design, inspection, laboratory analysis, audit and certification.

Inspections of fish premises are made in accordance with a Performance Based Verification Standard prescribed by MAF Reg. The frequency and duration of the visit will depend on the standard of processing operations, size of operation, quality assurance status of the company, and type of processing carried out.

The MAF requirements are divided into two parts. The first involves inspection of the premises to ensure that the industry agreed implementation standards relating to construction, hygiene, and sanitation of the premises, soundness of the product, and certification for export are complied with.

The second part involves ensuring that the company is carrying out the required daily and weekly checks, recording the details and action is taken to correct the defects. Details on the procedures to be followed when non-conformance's arise are given. Included in this are penalties to be used if compliance is not achieved. At each visit the MAF VA TMI audits the Company Internal Compliance Programme to ensure they have been completed by the company. Where non-compliance with the standards are found a target time to correct the non-compliance is determined. If further action is required this is done according to the procedures in the standards. In all instances, significant issues are followed up.

In addition to the audits by MAF Reg Compliance, MAF VA has its own internal compliance audit program. The MAF VA Technical Manager receives a direct report from a quality assurance group with responsibility for ensuring satisfactory compliance is achieved on a national level.

The quality assurance group also addresses national consistency of application of MAF Reg requirements.

EXPORT CERTIFICATION

The Fish Export Processing Regulations 1995 requires that all fish and fish products exported from New Zealand be accompanied by an export certificate. The MAF export fish certificate states that the fish are a product of New Zealand and were processed and packaged under hygienic conditions in premises licensed and inspected by MAF in accordance with the Fish Export Processing Regulations 1995. The certificate also states that the fish or fish product is fit for human consumption. MAF VA is responsible for providing export certification for all meat and seafood products exported.

If necessary, specific health requirements which are set by an importing country will also be certified by MAF VA.

TRUTH IN LABELLING

Under the New Zealand Fish Export Processing Regulations 1995, 'all containers of fish, fish products or fish by-products intended to be exported from New Zealand shall be labelled in an approved manner'. The regulations also state 'No container of fish or fish product shall be labelled with any false or misleading statements, words, pictures or marks'.

The Fishing Industry Agreed Implementation Standards lay down the specific requirements for the labelling of outer containers and retails packs. Specific labelling requirements can be set by the importing country.

Table 1
Unwanted organisms affecting aquatic animals

SCIENTIFIC NAME OF ORGANISM	COMMON NAME OF DISEASE OR ORGANISM	TYPE OF ORGANISM	REF. TO MAF POLICY
Notifiable organisms			
Organisms affecting fish			
Aeromonas salmonicida	Furunculosis	Bacteria	9.2.1
Epizootic haemorrhagic necrosis virus (EHNV)	EHN	Virus	9.2.1
Infectious haematopoietic necrosis virus (IHNV)	IHN	Virus	9.2.1
Infectious pancreatic necrosis virus (IPNV) (exotic strains)	IPN	Virus	9.2.1
Myxobolus cerebralis	Whirling disease	Myxosporea	9.2.4
Oncorhynchus masou virus (OMV)		Virus	9.2.1
Renibacterium salmoninarum	Bacterial kidney disease	Bacteria	9.2.1
Spring viraemia of carp virus (SVCV)	Spring viraemia of carp (SVC)	Virus	9.2.1
Viral haemorrhagic septicaemia virus (VHSV)	VHS	Virus	9.2.1
Yersinia ruckeri (exotic strains)	Enteric redmouth (ERM)	Bacteria	9.2.1
Organisms affecting molluscs			
Bonamia ostreae	Bonamiosis	Protozoa	9.2.1
Marteilia refringens	Marteiliosis	Protozoa	9.2.1
Marteilia maurini	Marteiliosis	Protozoa	9.2.1
Marteilia sydneyi	Marteiliosis	Protozoa	9.2.1
Mikrocytos mackini	Mykrocytosis	Protozoa	9.2.1
Mikrocytos roughleyi	Mykrocytosis	Protozoa	9.2.1
Perkinsus olseni	Perkinsosis	Protozoa	9.2.1
Organisms affecting crustacea			
Aphanomyces astaci	Crayfish plague	Fungi	9.2.1

continued on next page

Table 1 (continued)
Unwanted organisms affecting aquatic animals

SCIENTIFIC NAME OF ORGANISM	COMMON NAME OF DISEASE OR ORGANISM	TYPE OF ORGANISM	REF. TO MAF POLICY
Other exotic organisms			
Organisms affecting fish			
Ceratomyxa shasta	ceratomyxosis	Myxosporea	10.1.1
Channel catfish virus disease (CCV)	Channel catfish virus disease (CCVD)	Virus	10.1.1
Edwardsiella ictaluri	Enteric septicaemia of catfish	Bacteria	10.1.1
Enterocytozoon salmonis		Microspora	10.1.1
Epizootic ulcerative syndrome	Epizootic ulcerative syndrome (EUS)	Various contributing organisms	10.1.1
Erythrocytic necrosis virus	Viral erythrocytic necrosis (VEN)	Virus	10.1.1
Gyrodactylus salaris	Gyrodactylosis	Monogenea	10.1.1
Henneguya salminicola		Myxosporea	10.1.1
Infectious salmon anaemia virus (ISAV)	Infectious salmon anaemia (ISA)	Virus	10.1.1
Kudoa thyrsites		Myxosporea	10.1.1
Loma salmonae	Loma	Microspora	10.1.1
Pancreas disease of salmon virus	Pancreas disease	Virus	10.1.1
Parvicapsula sp.		Myxosporea	10.1.1
PKX	Proliferative kidney disease (PKD)	Myxosporea	10.1.1
Piscirickettsia salmonis	Piscirickettsiosis	Rickettsia	10.1.1
Rosette agent		Fungi (?)	10.1.1
Salmon anaemia virus	Erythrocytic inclusion body syndrome	Virus	10.1.1
Salmon leukaemia virus	Plasmacytoid leukaemia	Virus	10.1.1
Vibrio anguillarum (exotic strains)	Vibriosis	Bacteria	10.1.1
Vibrio salmonicida	Cold water vibriosis or Hitra disease	Bacteria	10.1.1
Viral encephalopathy and retinopathy virus	Viral encephalopathy and retinopathy	Virus	10.1.1
Organisms affecting molluscs			
Gill necrosis virus (and other pathogenic exotic iridoviruses)	Iridovirus	Virus	10.1.1
Haplosporidium spp.	Haplosporidiosis	Protozoa	10.1.1
Perkinsus marinus	Perkinsosis	Protozoa	10.1.1
Minchinia spp.		Protozoa	10.1.1
Organisms affecting crustaceans			
Penaeus monodon -type baculovirus (MBV)	Nuclear polyhedrosis baculoviroses	Virus	10.1.1
Yellowhead virus (YHV)	Yellowhead disease	Virus	10.1.1
Baculovirus penaei (BP)	Nuclear polyhedrosis baculoviroses	Virus	10.1.1
Baculoviral midgut gland necrosis virus (BMNV)	Baculoviral midgut gland necrosis	Virus	10.1.1
Infectious hypodermal and haematopoietic necrosis virus (IHHNV)	Infectious hypodermal and haematopoietic necrosis (IHHN)	Virus	10.1.1
White spot disease baculovirus (WSBV)	White spot disease	Virus	10.1.1
Taura syndrome virus	Taura syndrome	Virus	10.1.1

Appendix 5
Taxonomy and distribution of Australian native fish related to salmonids

Taxonomy

Order Osmeriformes

Family Aplochitonidae

Lovettia sealii	Tasmanian whitebait

Family Galaxidae

Galaxias auratus	Golden galaxias
Galaxias brevipinnis	Short-fin galaxias
Galaxias cleaveri	Tasmanian mudfish
Galaxias fontanus	Swan galaxias
Galaxias fuscus	Brown galaxias
Galaxias johnstoni	Clarence galaxias
Galaxias maculatus	Common jollytail
Galaxias occidentalis	Western galaxias
Galaxias olidus	Marbled galaxias
Galaxias parvus	Swamp galaxias
Galaxias pedderensis	Pedder galaxias
Galaxias rostratus	Murray jollytail
Galaxias tanycephalus	Saddled galaxias
Galaxias truttaceus	Spotted mountain trout
Galaxiella munda	Mud minnow
Galaxiella nigrostriata	Black-stripe minnow
Galaxiella pusilla	Dwarf galaxias
Paragalaxias dissimilis	Shanon paragalaxias
Paragalaxias eleotroides	Great lake darter
Paragalaxias julianus	Julian paragalaxias
Paragalaxias mesotes	Arthur's paragalaxias

Family Lepidogalaxidae

Lepidogalaxias salamandroides	Salamanderfish

Family Prototroctidae

Prototroctes maraena	Australian grayling

Family Retropinnidae

Retropinna semoni	Australian smelt
Retropinna tasmanica	Tasmanian smelt

Distribution of native fish related to salmonids

The following distributional data are derived from Allen (1989), and Wager and Jackson (1993).

RETROPINNIDAE

Retropinna semoni *(Australian smelt)*

Coastal drainages from the Fitzroy river system in south-east Queensland to eastern South Australia. Also occurs inland over the south-eastern area of the Murray-Darling system, and Coopers Creek in the Lake Eyre drainage, central Australia.

Retropinna tasmanica *(Tasmanian smelt)*

Coastal creeks and rivers of Tasmania.

APLOCHITONIDAE

Lovettia sealii *(Tasmanian whitebait)*

Coastal seas and rivers of Tasmania.

LEPIDOGALAXIDAE

Lepidogalaxias salamandroides *(Salamanderfish)*

Coastal creeks and rivers of south-western Australia, between Albany and the Blackwood River.

GALAXIDAE

Galaxias auratus *(Golden galaxias)*

Restricted to Lake Sorell and Lake Crescent, on the central plateau of Tasmania, and two tributaries of Lake Crescent, one of which is the Clyde River.

Galaxias brevipinnis *(Short-fin galaxias)*

Coastal drainages in south-eastern Australia, between the Hunter River, New South Wales, to Kangaroo Island and the Fleurieu Peninsula in South Australia. Also found on King and Flinders islands in Bass Strait, and widespread throughout Tasmania.

Galaxias cleaveri *(Tasmanian mudfish)*

Occurs at Wilsons Promontory in Victoria, Flinders Island, Bass Strait, and along the northern, western and south-eastern coasts of Tasmania.

Galaxias fontanus *(Swan galaxias)*

Restricted to several streams that are tributaries of the Swan and Macquarie river drainages.

Galaxias fuscus *(Brown galaxias)*

Distribution not well known. Specimens have been taken from the Rubicon River, Victoria, inland of the Great Dividing Range, 75 kilometres north-east of Melbourne (part of the Murray River drainage), and from Woods Point, Victoria.

Galaxias johnstoni *(Clarence galaxias)*

Restricted in Tasmania to Clarence Lagoon and its tributaries, the upper parts of the Clarence River, two unnamed lagoons, one of which is in the Wentworth Hills, and the headwaters of Dyes rivulet and Dyes Marsh.

Galaxias maculatus *(Common jollytail)*

Well distributed throughout the southern hemisphere, including Lord Howe Island and New Zealand. In Australia it occurs in coastal streams throughout south-eastern Australia, between Brisbane (Queensland), in the north, New South Wales, Victoria and Tasmania (including Flinders and King islands in Bass Strait), and Port Lincoln (South Australia).to the west. A separate population occurs in Western Australia, in coastal streams between Esperance and Albany.

Galaxias occidentalis *(Western galaxias)*

Coastal drainages in south-western Australia, between Waychinnicup Creek 80 kilometres east of Albany, to Winchester 250 kilometres north of Perth.

Galaxias olidus *(Marbled galaxias)*

Found at higher altitudes and subalpine areas of south-eastern Australia from southern Queensland to eastern south Australia. Occurs in river systems draining to both the east and west of the Great Dividing Range.

Galaxias parvus *(Swamp galaxias)*

Restricted in south-western Tasmania, to the headwaters of the Gordon and Huon rivers.

Galaxias pedderensis *(Pedder galaxias)*

Restricted in Tasmania to several streams flowing into Lake Pedder. Allen (1989) also lists it as occurring in Lake Gordon.

Galaxias rostratus *(Murray jollytail)*

Occurs in the Murray River and its tributaries (including the Lachlan, Murrumbidgee, Loddon, Goulburn, Ovens and Mitta Mitta rivers) in South Australia, Victoria and New South Wales.

Galaxias tanycephalus *(Saddled galaxias)*

Restricted to Arthur's Lake and Woods Lake on the central plateau of Tasmania.

Galaxias truttaceus *(Spotted mountain trout)*

Occurs in Victoria from Wilsons Promontory, west to the Glenelg River. Also occurs in lowland coastal streams around the Tasmanian coast, and in several lakes on its central plateau (including Great Lakes, Julian Lakes, and Bronte Lagoon), and on the King, Flinders and Clark islands in Bass Strait. A second mainland population occurs in the south-west corner of Western Australia, in a few streams around Albany.

Galaxiella munda *(Mud minnow)*

Occurs in the south-western corner of Australia, in coastal streams between Albany and Ellen Brook 50 kilometres north of Perth.

Galaxiella nigrostriata *(Black-stripe minnow)*

Restricted to coastal streams in south-western Australia, between Albany and Northcliffe.

Galaxiella pusilla *(Dwarf galaxias)*

Occurs in coastal streams from the Mitchell River in Victoria west to Mount Gambier in South Australia. Also occurs in the north-eastern corner of Tasmania, and on Flinders Island in Bass Strait.

Paragalaxias dissimilis *(Shannon's paragalaxias)*

Restricted to several lakes on the central plateau of Tasmania: Great Lake and Shannon Lagoon, the river that connects them, and in Penstock Lagoon.

Paragalaxias eleotroides *(Great lake darter)*

Restricted to Great Lake and Shannon Lagoon on the central plateau of Tasmania.

Paragalaxias julianus *(Julian paragalaxias)*

Restricted to several lakes on the central plateau of Tasmania: Julian Lakes, Carters Lake, Lake Dudley, Lake Ada, and the Ada and Talinah lagoons.

Paragalaxias mesotes *(Arthurs' paragalaxias)*

Restricted to the eastern central plateau of Tasmania, in Arthurs Lake, Woods Lake, and the river beneath Woods Lake dam.

PROTOTROCTIDAE

Prototroctes maraena *(Australian grayling)*

Coastal drainages of south-eastern Australia, from the Grose River near Sydney, New South Wales, through Victoria and Tasmania (including King Island, Bass Strait), to the eastern part of South Australia. Patchily distributed throughout this range.

Finfish species listed as endangered or vulnerable under the *Endangered Species Protection Act 1992*

SPECIES	COMMON NAME	TYPE
Brachionichthys hirsutus	Spotted-hand fish	Endangered
Chlamydogobius micropterus	Elizabeth Springs goby	Endangered
Galaxias fontanus	Swan galaxias	Endangered
Galaxias fuscus	Barred galaxias	Endangered
Galaxias johnstoni	Clarence galaxias	Endangered
Galaxias pedderensis	Pedder galaxias	Endangered
Maccullochella ikei	Clarence River cod	Endangered
Maccullochella macquariensis	Trout cod	Endangered
Maccullochella peeli mariensis	Mary River cod	Endangered
Melanotaenia eachamensis	Lake Eacham rainbow fish	Endangered
Scaturiginichthys vermeilipinnis	Red-finned blue-eye	Endangered
Carcharodon carcharias	Great white shark	Vulnerable
Carcharias taurus	Grey nurse shark	Vulnerable
Craterocephalus fluviatilis	Murray hardyhead	Vulnerable
Galaxias tanycephalus	Saddled galaxias	Vulnerable
Galaxiella pusilla	Dwarf galaxias	Vulnerable
Nannoperca obscura	Yarra pygmy perch	Vulnerable
Nannoperca oxleyana	Oxleyan pygmy perch	Vulnerable
Nannoperca variegata	Ewens pygmy perch	Vulnerable
Prototroctes maraena	Australian grayling	Vulnerable
Pseudomugil mellis	Honey blue-eye	Vulnerable

More detailed information on the conservation status of finfish in Australia may be found in the report *Baseline Environmental Data Relevant to an Evaluation of Quarantine Risk Potentially Associated with the Importation to Australia of Ornamental Finfish* (Arthington et al 1999; copies are available from AQIS).

Appendix 6
Surveillance and monitoring of fish health in Australia

Fish disease surveillance and monitoring in Victoria

THE STATE DERIVES SUBSTANTIAL BENEFIT from wild fisheries and aquaculture, fisheries production (including fish caught in Commonwealth-controlled offshore waters) being valued at approximately A$129 million (1997–98). Finfish species caught include pilchards, bream, King George whiting, orange roughy, blue grenadier, ling, tiger flathead, redfish, warehou, gemfish, jackass morwong and shark. There are also substantial crustacean fisheries for rock lobster, prawns and crabs, and molluscan fisheries for abalone, scallops, squid and octopus. Victoria is the main blue mussel-producing state in Australia.

Substantial populations of rainbow and brown trout and smaller populations of chinook salmon are maintained by one hatchery that supplies the Victorian Department of Natural Resources and Environment with fish for release for recreational purposes in the cooler waters of high-country lakes and rivers. There is also a number of large freshwater trout farms supplying trout product to the local market, as well as some smaller farms producing small volumes for the tourist trade. Victoria has a small number of farms supplying Atlantic salmon to the domestic market. Smaller numbers of eels are harvested from Victorian lakes and dams, the numbers being maintained by artificial stocking. There is an increasing interest in the intensive culture of two species of fish; that is, shortfinned eel and Murray cod for the supply of product to both the domestic market and for export.

LEGISLATION

The *Livestock Disease Control Act 1994* provides powers to manage exotic animal disease emergencies, declare protected and quarantine areas, and to control the movement, disinfection and destruction of animals and products for proclaimed diseases. The Act and its regulations cover fish and other aquatic animals (including fish products and reproductive material) under its definition of 'livestock', and prohibits importation of 'diseased' animals. The Act lists two schedules of fish diseases, classified as notifiable and exotic:

Notifiable diseases of finfish
(to be notified within 7 days)

- *Aeromonas salmonicida* infections (other than in goldfish)
- Epizootic haematopoietic necrosis
- Epizootic ulcerative syndrome
- Viral encephalopathy and retinopathy, (including Barramundi nodavirus)

Exotic diseases of finfish
(to be notified immediately)

- Bacterial kidney disease (*Renibacterium salmoninarum*)
- Infectious haematopoietic necrosis
- Herpes-virus of salmonids type 2
- Spring viraemia of carp
- Viral haemorrhagic septicaemia
- Whirling disease (*Myxobolus cerebralis*)

The *Fisheries Act 1998* establishes conditions for stocking fish in Victorian waters, introduction of noxious fish species, possession and release of protected aquatic biota, requires aquaculture licence holders to ensure freedom from notifiable diseases as specified in Schedule 16 of Fisheries Regulations 1998, and allows measures to protect any fishery, ecosystem or habitat.

Schedule 16 (finfish)

- *Aeromonas salmonicida* (atypical strains)
- *Aeromonas salmonicida* var *salmonicida* (Furunculosis)
- *Aphanomyces invaderis* (Epizootic ulcerative syndrome)
- Channel catfish disease
- *Edwardsiella ictaluri* (Enteric septicaemia of catfish)
- Epizootic haematopoietic necrosis virus
- *Gyrodactylus salaris*
- Infectious haematopoietic necrosis virus
- Infectious salmon anaemia virus
- Infectious pancreatic necrosis virus
- *Myxobolus cerebralis* (Whirling disease)
- *Oncorhynchus masou* disease
- *Renibacterium salmoninarum* (Bacterial kidney disease)
- Spring viraemia of carp
- Viral encephalopathy and retinopathy
- Viral haemorrhagic septicaemia
- *Yersinia ruckeri* (Enteric redmouth)

The *Wildlife Act 1975* deals with control of noxious aquatic species but does not cover disease control issues.

DISEASE ZONING

There are no fish disease control zones established in Victoria.

FISH DISEASE DIAGNOSTIC SERVICES

The first fish health services in the State were provided by a fish health officer situated at the central government salmonid hatchery at Snob's Creek for over 25 years. Pathology examinations were conducted at Snob's Creek until the inception of the Australian Fish Health Reference Laboratory (AFHRL) in 1980 at Benalla. This facility was relocated to the CSIRO Australian Animal Health Laboratory (AAHL) in Geelong in 1991 and is now known as the AAHL Fish Diseases Laboratory (AFDL).

The Victorian Institute of Animal Science (VIAS) located in Melbourne is the veterinary diagnostic and research facility for the State of Victoria and has provided fish disease diagnostic facilities for the Victorian Fish Health Service (VFHS) since 1991. It has all the major diagnostic disciplines, including pathology, histopathology, virology, bacteriology, parasitology and biochemical services. Limited holding facilities are available for freshwater fish. The bacteriology laboratory is mainly involved in testing for *Nocardia, Yersinia ruckeri* and *Mycobacteria.* The virology section routinely uses two passages in RTG, FHM, BF2 cells to check for viruses, and identifies any suspect viruses by electron microscopy, referring on if necessary to AFDL. There is a

transmission electron microscope on-site. An epizootic haematopoietic necrosis (EHNV) antigen enzyme-linked immunosorbent assay (ELISA) is also available. A fish pathologist is responsible for coordinating fish disease diagnostic services at the laboratory, while field support is provided by a field officer at the Marine and Freshwater Resources Institute at Snob's Creek.

The laboratory receives submissions from a variety of sources. The submissions involve suspected fish diseases and fish kills. Samples may be submitted directly from aquaculturists, although most are received via visiting fish health professionals, Department of Natural Resources and Environment (DNRE) officers, the Environmental Protection Authority (EPA), AQIS (import quarantine) and private individuals. Fish kills in wild stocks are routinely sampled and assessed by the VFHS when reported to officers of the EPA or DNRE.

DISEASE SURVEILLANCE AND MONITORING

Fish health problems on salmonid farms are investigated using routine procedures. Pathology, histopathology and bacteriology examinations are performed on most fish accessions, while parasitology and virology are performed if clinical signs or histopathology indicate a parasitic or viral agent.

Specific disease monitoring and surveillance schemes are as follows.

- *The Victorian Fish Health Accreditation Scheme* was introduced in 1993, and is based on periodic inspection of fish stocks. It is a voluntary scheme available to native fish and salmonid farms. The scheme involves six clinical inspections of each farm per year for the first year, then four per year (three of which are during the summer period) by a government fish health official. As part of this scheme the farmer is required to report disease occurrences to a fish health professional. Disease problems are then investigated with the involvement of the VFHS.

The scheme provides information on the disease status of fish in Victorian aquaculture farms, introduces fish health professionals to the farms and enables a two-way flow of information. Farmers receive continual reinforcement in the principles of quarantine and the scheme is often used as a benchmark for movement of stock between farms.

- A *Virological Survey of Trout Hatcheries* is currently in progress. It involves sampling ova and milt at spawning from 60 fish per hatchery, storage at –80^{o}C and subsequent virological testing. Samples have been collected from 10 trout farms and one Atlantic salmon farm. The samples are passed twice through RTG, FHM, BF2 cells lines, and checked for development of cytopathic effects; electron microscopic examination is used if indicated.
- *Bacteriological Survey of Salmonid Hatcheries*: Victorian Fisheries are currently organising a disease survey of trout and salmon, sampling about 30 fish per property. Sampling will take place in the hotter months when there is more stress on the fish, and will involve gross examination, histopathology and bacteriological testing. The intention is to collect samples on a regular basis (6-monthly) from individual farms within the different river catchment areas.

HEALTH CERTIFICATION TESTING

Apart from the Victorian Fish Health Accreditation Scheme, which was instigated to allow live fish to be sold into NSW, ACT and SA, there is no other health certification testing carried out on Victorian farms.

SIGNIFICANT FINFISH DISEASE AGENTS DETECTED IN VICTORIA

Finfish disease agents detected in Victoria include herpes virus associated with pilchard mortalities, *reovirus* (in *Perca fluviatilis*), epizootic haematopoietic necrosis (in *Perca fluviatilis* and *Oncorhynchus mykiss*), *Aeromonas hydrophila, Aeromonas salmonicida* (atypical strains in goldfish), *Yersinia ruckeri*, *Flexibacter columnaris*, *Mycobacteria* sp, *Nocardia* sp, *Enterococcus seriolicida, Saprolegnia* sp, *Chilodonella cyprini*, *Goussia* sp, *Ichthyophthirius multifiliis*, *Kudoa thyrsites*, *Myxobolus gadopsi*, *Triangula percae*, and *Trichodina* sp.

Bacterial kidney disease was diagnosed at Snob's Creek in 1980 based on pathology, histopathology and fluorescent antibody test (FAT); however, later follow-up

involving a large number of specimens confirmed that the original diagnosis was wrong and that the organisms involved were *Nocardia sp* and *Mycobacterium sp*.

Fish disease surveillance and monitoring in New South Wales

The State derives substantial benefit from wild fisheries and aquaculture production (including fish caught in Commonwealth-controlled offshore waters) amounting to a value of over A$138 million (1997–98). Finfish species caught include sea mullet, black and yellowfin bream, snapper, tuna, whiting, orange roughy, blue grenadier, ling, tiger flathead, blue and silver warehou, gemfish and redfish. There are also substantial crustacean fisheries for prawns, rock lobster, and crabs, and molluscan fisheries for abalone, pipi, squid and octopus.

Rainbow and brown trout and small populations of brook trout are located in the cooler waters of lakes and rivers of the highlands of NSW and maintained by hatcheries production. There are also a number of freshwater trout farms supplying trout product for export, for the local market and for the tourist trade. Kuruma prawns are farmed in northern NSW and there is a considerable number of yabby farms of various sizes. Sydney rock oysters are farmed in central coastal NSW.

LEGISLATION

The *Stock Diseases Act 1923* provides powers to declare quarantine and protected areas, control movement of stock into the State and order destruction of stock. Fish, molluscs and crustaceans are proclaimed as stock under this Act, though no diseases of aquatic animals have been proclaimed. It is proposed to remove fish from this Act and cover them under the *Fisheries Management Act 1994.*

The *Exotic Diseases of Animals Act 1991* provides powers to manage emergency situations and to control outbreaks of suspected exotic disease. The Act includes fish, molluscs, crustaceans including eggs and gametes, but does not list aquatic animal diseases. It is proposed to remove aquatic animals from the scope of this Act and transfer them to the Fisheries Management Act.

The Fisheries Management Act includes a number of disease control measures such as obligations to report disease and powers to order destocking, and provides controls on the importation and release of live fish. The Fisheries Management Act and Regulations provide a number of requirements for health testing and control over the movement of finfish, oysters and prawns.

The following diseases of finfish are listed under the Fisheries Management Act (Aquaculture) Regulations 1995.

- *Aeromonas salmonicida* infection
- Bacterial kidney disease
- Enteric redmouth disease/Yersiniosis (*Yersinia ruckeri*)
- Infectious haematopoietic necrosis
- Infectious pancreatic necrosis
- Epizootic haematopoietic necrosis
- Epizootic ulcerative syndrome
- Viral haemorrhagic septicaemia
- Viral nervous necrosis (see below)
- Whirling disease (*Myxobolus cerebralis*)

Proposed additional diseases and changes

- *Oncorhynchus masou* virus disease
- Spring viraemia of carp
- Infectious salmon anaemia
- Piscirickettsiosis
- Gyrodactylosis (*Gyrodactylus salaris*)
- Furunculosis (*Aeromonas salmonicida* subsp *salmonicida*)
- Viral encephalopthy and retinopathy

DISEASE ZONING

NSW has proclaimed a disease control zone in the south-east region of the State where EHN is endemic. The movement of live salmonids to or from this region and to the designated EHN-free remainder of the State are controlled (see certification testing).

FISH DISEASE DIAGNOSTIC SERVICES

Between 1985 and 1990, NSW Agriculture and Fisheries provided a free diagnostic service to capture fisheries and aquaculture industries through its five Regional Veterinary Laboratories (RVLs). Since 1990, NSW Fisheries has provided this service State-wide through its staff at RVL Wollongbar, and NSW Agriculture has also offered a diagnostic service to aquaculture industries through the then existing four RVLs on a full cost-recovery basis. In 1996, two RVLs (Wagga Wagga and Armidale) were closed and accessions from their service areas were diverted to Menangle and Wollongbar, respectively.

NSW Fisheries currently offers a free diagnostic service in relation to disease outbreaks in farmed and wild aquatic animals in NSW, through the Regional Veterinary Laboratory, Wollongbar. This service is managed by a veterinary pathologist specialising in fish diseases and provides necropsy, histopathology, bacteriology, and parasitology services. Virology is referred to the virology section of the Elizabeth Macarthur Agricultural Institute (EMAI) (a reference laboratory of the Office International des Epizooties (World Organisation for Animal Health, or OIE) which has several fish cell lines, including BF-2, RTG cell lines, and has electron microscopy facilities and an EHNV ELISA. Toxicology is referred to appropriate public or private sector testing facilities.

SURVEILLANCE AND MONITORING

Health problems on salmonid farms are investigated using routine procedures. Pathology, histopathology and bacteriology examinations are performed on most fish accessions, while parasitology and virology are performed if clinical signs or histopathology suggest the presence of a parasitic or viral agent. Diagnostic accessions of finfish submitted to the laboratory in 1997 totalled 76, each accession comprising 1–20 fish.

Specific disease surveillance and monitoring schemes are as follows.

NSW Fisheries salmonid hatcheries disease monitoring program: NSW Fisheries conducts a disease monitoring program for salmonid disease at Gaden trout hatchery, Jindabyne (introduced in 1988), and at Dutton Trout Hatchery, Ebor (introduced in 1990). The program's objectives are to:

- minimise the risk of introducing major pathogens to the hatchery;
- minimise the risk of disseminating major pathogens via translocated fish; and
- maintain the health of the fish populations at the hatchery.

The sample sizes taken from the populations provide for 95% certainty of detecting at least one positive animal at a disease prevalence of 2%. The program is as follows.

Rainbow trout

March	Prior to release, test 150 fingerlings for EHNV infection using the RTG-2 cell line only, and for both *Y. ruckeri* and *A. salmonicida* infection, using standard plate culture methods.
August	Test samples of all milt and ovarian fluid collected from wild fish as above for evidence of EHNV infection. Strict precautions are taken to prevent the spread of infection from the resultant progeny to other stocks at the hatchery until negative test results are obtained.
November	Prior to release, test 150 fry, as in March, for evidence of EHNV and *Y. ruckeri* infection.

Brown trout

March	Release fry, provided tests on rainbow trout fry for March are negative.
May	Test all samples of milt and ovarian fluid collected from wild fish as for rainbow trout. Similarly, eyed ova released to other hatcheries will not be tested but should be disinfected at the receiving hatchery.

Atlantic salmon

November	Prior to release, test 150 fry for evidence of *A. salmonicida* infection. The submitted specimens are examined using conventional microbiological techniques. The program as described was used until mid-1996, when

an ELISA test for EHNV was substituted for the cell culture method. For the ELISA test, 150 mortalities amongst the fish proposed for translocation, or from contiguous populations if required to reach the required sample size, are collected in the period immediately prior to the proposed movement date. Collected fish are held frozen at –20°C until tested. The testing laboratory is an OIE reference laboratory for EHNV testing.

Annual submissions under the monitoring program are summarised below.

Rainbow trout

- 2x150 fry ex Gaden and 1x150 fry ex Dutton for EHNV testing
- 1x150 fry ex Gaden and 1x150 fry ex Dutton for *Y. ruckeri* testing
- 1x150 fry ex Gaden and 1x150 fry Dutton for *A. salmonicida* testing

Atlantic salmon

- 1x150 fry ex Gaden for *A. salmonicida* testing

As an adjunct to the monitoring program, all stock at both hatcheries are regularly examined for evidence of clinical disease, with the intention that affected fish will be submitted for laboratory examination. Fish must be submitted for examination whenever the morbidity or mortality rates rise significantly above background levels.

Since the program began, no significant outbreaks of clinical disease have been detected at Gaden or Dutton, with the exception of an outbreak of streptococcosis associated with abnormally high water temperatures, at Dutton. No EHNV infection, other viral infection, *A. salmonicida* infection or *Y. ruckeri* infection has been detected at either hatchery.

Other schemes with a fish health component include the following.

- A River Health Program was undertaken by NSW Fisheries and the CRC in Freshwater Ecology and entailed structured surveillance of finfish stocks in freshwaters throughout NSW. The program included a health component involving recording gross abnormalities. The total number of fish surveyed amounted to approximately 23,000.
- Goldfish are checked periodically by NSW Fisheries inspectors for goldfish ulcer disease.

HEALTH CERTIFICATION TESTING

- *Salmonid translocation program*: Salmonid fish imported from EHN endemic zones in other States for grow-out in NSW outside the designated 'endemic area' must test free of EHNV by ELISA. Salmonid fish translocated for grow-out from the EHN endemic area of NSW (defined catchments in the south-east) to other parts of the State must test free of EHNV by ELISA. Salmonid fish translocated for restocking into public waterbodies in NSW from both government-operated hatcheries must test free of EHNV by ELISA, and *Yersinia ruckeri* and *Aeromonas salmonicida* infection by conventional bacteriological methods.

DISEASE AGENTS DETECTED IN NSW

Finfish disease agents include: EHN, lymphocystis, herpes virus (pilchards), *Aeromonas hydrophila*, *Aeromonas salmonicida* (atypical strains), *Vibrio anguillarum*, *Vibrio cholerae*, *Vibrio harveyi*, *Edwardsiella tarda* (farmed rainbow trout), *Yersinia ruckeri*, *Lactobacillus piscicola*, *Mycobacterium* sp, *Nocardia*, *Streptococcus* sp, epizootic ulcerative syndrome (EUS) (*Aphanomyces invadans*), *Saprolegnia* sp, *Chilodonella* sp, *Eimeria* sp, *Goussia* sp, *Ichthyobodo necator*, *Ichthyophthirius multifiliis*, *Kudoa* sp, *Myxobolus* sp, *Trichodina* sp. There are a range of parasites including *Anasakis* sp, *Contracaecum* sp.

Fish disease surveillance and monitoring in Tasmania

The State derives substantial benefit from wild fisheries and aquaculture, with production (including fish caught in Commonwealth-controlled offshore waters) valued at nearly A$261 million (1997–98).

The main species caught include orange roughy, blue grenadier, ling, silver warehou, tiger flathead, redfish, gemfish, squid, shark, rock lobster, abalone, oysters and scallops.

The most significant aquaculture industry is based on Atlantic salmon.Trout (mainly rainbow trout), oysters, mussels, abalone and eels are also farmed.

LEGISLATION

The *Living Marine Resources Management Act 1995* controls the introduction of live fish into the State, the introduction of fish into State waters, and the purchase or possession of noxious fish species. Fish are included in a broad definition that includes all living marine resources and their parts and breeding materials, but excludes marine mammals and birds.

The *Animal Health Act 1995* is a comprehensive Act containing provisions in relation to exotic disease and endemic disease. It includes aquatic animals and products in its definition of animals. The Act provides for control over movement of aquatic animals and aquatic animal products within, into and from Tasmania. It also provides mandate to declare protected, restricted and control areas and to order into quarantine or to order the destruction, cleaning and/or treatment of materials or buildings in contact with diseased aquatic animals or products.

The Act lists a range of notifiable diseases, including finfish diseases. Under section 17 of the Act all imports of animals, disease agents, and restricted materials must be authorised by special or general authority. There are penalties for importing animals or animal materials that may be infected with List A, List B, new or unknown disease, without prior permission. The Animal Health Act does not replace import controls enacted under other Tasmanian legislation.

Carcases or parts of carcases from all scale fish, and fresh or frozen aquatic invertebrates consigned to a seafood wholesaler for human consumption, are generally permitted unrestricted entry. The introduction of live freshwater crayfish (*Cherax* spp) is banned by the *Inland Fisheries Act 1995.* Goldfish for open sale are permitted entry but only from approved interstate hatcheries under specified conditions and certification.

Other conditions under the Animal Health Act and the Inland Fisheries Act restrict entry of live aquarium fish, pond fish (other than goldfish), and aquarium molluscs, and set out certification requirements for approved entry of such species.

The special authorities set out import conditions for goldfish spawned in Australia, New Zealand and overseas countries, for aquatic animals for research purposes and for importation of live finfish for human consumption.

Under the Fish Health Surveillance Program, industry is required to investigate disease and submit diagnostic samples. The Animal Health Act requires fish farms to report or investigate all diseases of known aetiology which have not previously been reported in the State, and diseases of unknown aetiology. The Surveillance Program and industry's contribution to health research funding reflects the close cooperation of the industry in health matters. For a number of years this has been fostered by industry, the Department of Primary Industries, Water and Environment (DPIWE) and the Tasmanian Advisory Committee of Fish Health (recently revised as the Tasmanian Fish Health Planning and Advisory Group, with an increased role in planning for and responding to fish health emergencies).

Notifiable diseases of finfish under the Animal Health Act include the following.

List A diseases

- Bacterial kidney disease
- Epizootic haematopoietic necrosis
- Epizootic ulcerative syndrome
- Furuncolosis (*Aeromonas salmonicida salmonicida*)
- Goldfish ulcer disease (*Aeromonas salmonicida* atypical strain)
- Infectious haematopoietic necrosis
- Infectious pancreatic necrosis
- *Oncorhynchus masou* virus disease
- Piscirickettsiosis
- Spring viraemia of carp

- Viral encephalopathy and retinopathy
- Viral haemorrhagic septicaemia

List B diseases

- Streptococcosis of salmonids (*Lactococcus garviae*)

DISEASE ZONING

As a result of the discovery of an aquabirnavirus in salmon and other fish species in Macquarie Harbour on the west coast of Tasmania, this area has been proclaimed a disease control zone. Restrictions on movement of live farmed salmonids from the zone and protocols for processing (removal of viscera and gills) of non-viable salmonids and treatment of nets have been developed. There are no restrictions on the movement of eviscerated fish for human consumption.

FISH DISEASE DIAGNOSTIC SERVICES

The State Government Veterinary Laboratory of the DPIWE, Tasmania, has provided a diagnostic service to the salmonid farming industry since its inception in 1984. The Salmonid Surveillance Program was initially provided at no cost. It was later replaced in 1993–94 by an industry-funded Fish Health Surveillance Program, which provides services to the industry at no additional charge on submission of samples.

Field investigation and research services were initially provided by the industry; however, veterinary field services are now provided by the DPIWE through industry funding. Research activities and industry funding are now channelled through research organisations such as the Cooperative Research Centre (CRC) for Aquaculture and the Fisheries Research and Development Corporation (FRDC), which provide for the integration of research priorities and programs throughout Australia.

The state veterinary laboratory provides competent necropsy, histopathology, bacteriology, parasitology and chemistry services to livestock, including commercial fish, shellfish and crustaceans. The fish diagnostic facilities now include two full-time fish pathologists, a full-time fish bacteriologist, three research officers (temporary) and two technicians. Samples requiring virology testing are referred to the AFDL. The service to the salmonid industry includes diagnosis, surveillance, certification, diagnosis of diseases and kills of wild fish, disease investigation and research. Vaccine production and vaccine research services are also provided. Field support for investigation of diseases in finfish, especially salmon, flounder and eels, is provided by a full-time field veterinary officer.

Disease investigation and surveillance by farm staff have been encouraged through a number of measures including the provision of field kits for bacteriological and histological sampling on remote farms, backed by training programs in disease awareness and sampling, and recently by increased assistance through regular visits by DPIWE field staff. This has also been facilitated by the employment of Aquaculture Diploma and Associate Diploma graduates on fish farms, enabled by the formation in Tasmania in mid-1980s of a Technical/University School of Aquaculture, now part of the University of Tasmania. All farms are encouraged to submit samples regularly as well as during disease outbreaks.

DISEASE SURVEILLANCE AND MONITORING

The diagnostic service has been available since the inception of trout farming in 1963. There were an average of 21 fish diagnostic submissions per year between 1972 to 1980, involving both wild and cultured trout (rainbow trout and some brown trout), and a few other fish species. With the establishment of the Australian Fish Health Reference Laboratory (AFHRL) in 1981, resulting in availability of standard fish cell lines for virology, the Tasmanian laboratory participated in several virological, bacteriological, and parasitological surveys, published in the early 1980s. The number of diagnostic submissions from farmed and wild fish (based on finfish, excluding aquarium fish) was 71 in 1985, 114 in 1986, 255 in 1988, and has remained near that level since. There was some decline in diagnostic submissions after 1991 as understanding and management of endemic diseases improved. However, the number of submissions increased by 1997, as the industry expanded.

The on-farm investigation of diseases is assisted by field veterinary services, which are currently provided by both

private specialist fish veterinarians, and DPIF. In 1994 there were 44 farm visits by DPIF veterinarians. During the first 6 months of 1998, there were 69 farm visits by DPIF staff and 30 visits in response to fish health incidents.

The state government, University of Tasmania, and industry have had active research programs in salmonid health over this period.

A joint histological and haematology survey was carried out over 7 months by the commercial grower Saltas in 1990–91. This was the largest specific survey of salmonid blood. Other relevant surveillance programs are a free diagnostic service for lesions suggestive of goldfish ulcer disease, proactive sampling for this disease in goldfish, and surveillance of experimental culture of new aquaculture species such as flounder. This resulted in formal extension of the surveillance program to all finfish aquaculture in 1998–99, the type of surveillance varying according to the stage of industry development.

There are a number of specific disease monitoring and surveillance schemes, as follows.

- *Goldfish ulcer disease surveillance:* Goldfish farms are registered in Tasmania, and goldfish sold or imported into the State must be certified free of goldfish ulcer disease, by visual inspection, and culture of suspect lesions. The disease has been diagnosed once in Tasmania in imported stock in quarantine, all stock being destroyed. A further case was diagnosed in the late 1970s in goldfish that were sold to the public. GUD has not been subsequently isolated in Tasmania.
- *Aquarium fish diagnostic and advisory service:* This service is provided to aquarium fish producers. Over 120 cases have been examined since 1990, 46 over the last two years.
- *Wild fish monitoring:* Based on investigation of fish kills as they occur.
- *Water Quality Program:* This is a structured surveillance program aimed at the detection of faecal contaminants, biotoxins and heavy metal contamination. This is funded by the shellfish industries.

TASMANIAN FISH HEALTH SURVEILLANCE PROGRAM

This major program continues and extends the original Tasmanian Salmonid Health Surveillance Program, first initiated in 1993. The aim of the program is to ensure a coordinated and effective health monitoring and surveillance program is operating throughout the Tasmanian finfish aquaculture industry.

The program includes commercial business operations, not-for-profit enterprises, private companies, government organisations and educational facilities involved in the aquaculture of salmonids and other finfish in Tasmania. The program encompasses surveillance and monitoring for disease states due to viruses, bacteria, parasites and fungi, as well as conditions of nutritional, environmental and neoplastic origin. The program is also intended to assist finfish aquaculture industries with strategic exotic disease preparedness and other policy advice as required. The test program has been designed to deliver comprehensive monitoring for the following diseases/disease agents, as shown below.

- Viral diseases which would be detected by the virological culture methods employed since 1982:
  - infectious pancreatic necrosis (IPN)
  - infectious haematopoietic necrosis (IHN)
  - viral haemorrhagic septicaemia (VHS)
  - herpes virus salmonis disease
- Other virus diseases or presumed viral diseases not readily detected by these means, but which would be expected to be recognised through clinical and histopathology findings:
  - plasmacytoid leukaemia
- Virus diseases less likely to be detected by routine histological means, but detected by more specific methods such as haematology:
  - erythrocytic inclusion body syndrome
  - viral erythrocytic necrosis
- Bacteria and bacterial diseases detectable by routine bacteriological cultures:
  - *Aeromonas salmonicida* (typical and atypical strains)

- enteric redmouth (*Yersinia ruckeri*)
- Edwardsiellosis (*Edwardsiella tarda*)
- vibriosis (*Vibrio ordalii* and *V. anguillarum*)
- Hitra disease, coldwater vibriosis (*Vibrio salmonicida*)

- Bacterial diseases not detectable through routine culture, but which would be detectable on Gram stained smears or clinical and histopathology findings:
  - bacterial kidney disease (*Renibacterium salmoninarum*)
  - salmonid rickettsial septicaemia
- Other diseases and agents, including protozoa, likely to be detected by routine pathology and histopathology examination:
  - *Loma salmonae*
  - *Enterocytozoon salmonis*
  - pancreas disease
  - proliferative kidney disease
  - *Ceratomyxa shasta*
  - *Kudoa thyrsites*
  - *Henneguya salminicola*
  - parvicapsular disease
- Other agents including protozoa which are detected with more difficulty or are unlikely to be detected unless specific examinations are carried out:
  - Rosette agent
  - whirling disease (Myxobolus cerebralis)

The program collects information in the following ways:

- strategic and routine sampling by freshwater and marine salmonid farm technical staff;
- strategic and routine virology testing conducted by fish health officers from DPIWE and the Inland Fisheries Commission (IFC); and
- investigations of disease incidents and other unusual situations.

Technical staff at hatcheries, marine farms, IFC hatcheries, DPIWE and university facilities are accredited by the DPIWE fish veterinarian and are responsible for collecting samples on a routine basis from moribund or diseased stock. This is a key element of the program. The ability of technical staff to collect samples competently is very important, and requires a good knowledge of fish anatomy and sample collection techniques. Training in this area is ongoing.

Farms and hatcheries have individual annual programs prepared in consultation with a DPIWE fish health specialist to ensure monthly samples are submitted from most 'at risk' stock. Such a program will take into account when fish are coming onto the farm, when broodstock are near being returned to the hatchery, when fish are to be moved and so on. This will not preclude the submission of extra samples should this be warranted, but will ensure the farm knows in advance where at least 12 of their sample submissions are to come from. Samples will also be taken during harvest and processing, where there is an ideal opportunity to look closely at many fish. It is vitally important that farmers are continually using acquired skills in the area of fish sampling. The submission of at least one sample per farm per month is an important way of achieving this, as well as providing continued background data on disease status. To have such sampling written into their on-farm management program is a more formal and effective way of overcoming the uncertainty of ad hoc submissions, as has been the practice in the past.

It is also important that technical staff have access to suitable equipment and facilities to facilitate the collection of samples. A dedicated laboratory area, and suitable dedicated dissecting equipment for each farm is absolutely essential.

The supply of fish health kits to farms by the DPIWE Fish Health Unit is designed to further increase the ability of staff to collect such samples. These kits are currently supplied once monthly to all salmonid farms, or more frequently on request. The kits contain bacterial culture plates, sterile swabs and microscope slides. The collection of samples onto such equipment aids in the identification of bacterial microbes affecting fish.

Farms are also supplied with adequate fixative (formalin) and sample jars to facilitate collection of specimens for histopathology. The collection of such samples helps to ensure that most disease processes that affect organ function (including such diseases caused by parasites and viruses) are detected.

Bacterial plates supplied include blood agar and plates for *Vibrio* species and surface myxobacteria, which in conjunction with Gram stains of tissue smears detect most acute bacterial pathogens of fish. Fixed samples for histopathological examination include tissues where gross abnormalities are detected or suspected on clinical grounds, as well as gills and a standard range of internal organs.

VIROLOGY TESTING

This work is conducted by DPIWE fish health officers, with IFC fish health officers conducting work at the Salmon Ponds hatchery. Routine visits to marine sites occur on a regular basis or at least two times per year as per the OIE Code for farms that have been monitored regularly free of major disease for 2 years. During such visits the officer collects samples from moribund or freshly dead fish if available, plus an additional 30 of the most susceptible fish on the farm (eg fish one month after transfer). Hatcheries are also visited routinely or as required; however, with the increased use of photo-manipulation and other methods to alter transfer time, there is an increased number of strategic visits to hatcheries to ensure all classes of fish are tested before transfer to sea.

Generally, virology samples are pooled (each pool comprises samples from 10 fish), and sent to AFDL for testing. At least 30 fish are sampled per marine group per year, once site status has been defined. A minimum of 60 fish are sampled from marine farms supplying broodstock to hatcheries. These may include fish culled from the broodstock so costs associated with loss of valuable fish can be minimised.

Farmed salmonid populations have generally been tested for viruses twice yearly since the mid-1980s or earlier. All salmonid stocks have been under similar test regimes for a substantial part of that time. Since late 1993, all farms have been included in the virus sampling program of the Salmonid Health Surveillance Program. The test involves passaging appropriate samples twice in susceptible cell cultures, with checks for cytopathic effect and electron microscopic examination as appropriate.

These cell lines (EPC cells were only tested recently) have been fully tested for susceptibility to the relevant virus.

Virus	Cell lines susceptible
infectious pancreatic necrosis (IPN)	BF 2, CHSE 214, RTG 2
infectious haematopoietic necrosis (IHN)	CHSE 214, EPC, FHM, RTG 2
viral haemorrhagic septicaemia (VHS)	BF 2, CHSE 214, EPC, FHM, RTG 2
herpes virus salmonis disease CHSE	214, RTG 2

DISEASE INVESTIGATION

Health incidents on farms are investigated by a DPIWE fish veterinarian or a suitably qualified fish health officer. In such investigations, samples are collected to determine the cause of the problem and to confirm or rule out the involvement of serious pathogens. For statistical purposes, the results of such sampling are included in the program, but details may remain confidential to the farm if the incident is not reportable.

GENERAL SURVEILLANCE

There are additional ad hoc submissions from fish farms and other sources throughout the year. Such submissions may be collected by farm technical staff in response to a request by a fish health officer, or may be, for example, specimens collected for bacterial and histopathological investigation from a DPIWE fish health officer while on a routine virology visit.

New finfish aquaculture farms are ideally incorporated into the program during the developmental stage of such farms. Individually tailored fish health surveillance and monitoring programs are developed by the DPIWE fish health unit in consultation with the owner or company responsible for the farm, and in consultation with the Marine Resources Division of DPIWE, or the IFC, depending on the nature and location of the farm. Such individual programs ensure that the fish health surveillance for the farm is in keeping with principles outlined in this program.

NUMBERS OF SPECIMENS EXAMINED

Finfish are mainly subject to pathological, virological and bacteriological examinations. The pathology examinations generally involve histological examination of multiple tissues, while bacteriology generally involve routine kidney cultures, though some involve skin culture only. There has been a large amount of virological testing of Tasmanian salmonids, as all four commercial hatcheries and many of their recipient sea farms have been under virus test programs for export certification for substantial periods of time. Most farmed fish populations have been tested twice yearly for viruses of interest. Virological sampling under the Salmonid Surveillance Program has included at least annual samples from all growing regions. From hatcheries a minimum of 60 ovarian fluids and 60 pre-smolts or fingerlings from each species are tested, 30 fish from each Marine Farm Group, and 60 fish from sites supplying broodstock to a hatchery or fish for distribution to other sites.

For the period 1995 to June 1998, a total of 693 salmonid submissions (comprising variable numbers of individual animals) were received for routine testing, including 595 submissions of Atlantic salmon and 70 submissions of rainbow trout. Of these, 417 submissions were subjected to pathology tests, 149 to virology tests, and 379 to bacteriology tests.

Further details of the number of fish examined by pathology, bacteriology and virology are shown below:

Salmonids

	NUMBER OF SALMONID FISH EXAMINED BY PATHOLOGY, BACTERIOLOGY & VIROLOGY		
YEAR	PATHOLOGY	BACTERIOLOGY	VIROLOGY
1994	216 (195 salmon, 21 trout)	782 salmonids	1945 salmonids
1995	81 (68 salmon, 13 trout)	369 salmonids	1779 salmonids
1996	95 salmonids	325 (216 salmon, 109 Rb trout)	1302 salmonids
1997	122 salmonids	930 (648 salmon, 282 Rb trout)	2388 salmonids
1998 (to June)	108 salmonids	908 (693 salmon, 215 Rb trout)	1790 salmonids

Non-salmonid fish: pilchard, yellowtail, seahorse, wild fish etc

	NUMBERS OF:		NUMBER OF FISH EXAMINED BY:			
YEAR	ACCESSIONS	FISH EXAMINED	H/PATHOLOGY	BACTERIOLOGY	VIROLOGY	PARASITOLOGY
1995	49	266	142	94	10	20
1996	26	108	66	40	0	4
1997	51	189	108	66	15	0
1998	51	287	143	63	75	6

Aquarium fish

	NUMBERS OF:		NUMBER OF FISH EXAMINED BY:			
YEAR	ACCESSIONS	FISH EXAMINED	H/PATHOLOGY	BACTERIOLOGY	VIROLOGY	PARASITOLOGY
1995	18	80	60	20	0	0
1996	30	129	74	55	10	0
1997	5	26	20	6	0	0
1998	39	282	109	173*	10	0

* Includes 150 goldfish for certification

RESULTS OF DISEASE TESTING PROGRAM

Virus diseases detected by the virological culture methods employed since 1982

As stated above, the majority of farmed fish have been under twice yearly virus test since at least the mid-1980s, and all fish stocks have been under such test regimes for substantial periods over that time. The tests until recently have given negative results, and as there was no clinical or histological evidence of disease, these diseases were considered exotic to Tasmania. Negative results to a limited number of serological tests for IHN also supported this.

Reoviruses have been recovered from salmon (three submissions in 1994, five submissions in 1997 and 13 submissions in 1998). An aquabirnavirus was isolated in 1998 from a pool of farmed Atlantic salmon pinhead smolt in Macquarie Harbour in western Tasmania. Follow-up testing yielded a total of eight isolates from smolt, rainbow trout (*Oncorhynchus mykiss*), flounder (*Rhombosolea tapirina*), cod (*Pseudophycis* sp*),* spiked dogfish (*Squalus megalops*) and ling (*Genypterus blacodes*) in the same area. There was no histological evidence of significant pathological changes in fish except for pancreatic lesions in two fish in the index submission. Experimental testing conducted at the AFDL in susceptible rainbow trout, brook trout and Atlantic salmon indicates that the aquabirnavirus is of low pathogenicity, causing no clinical disease or mortality and only minor lesions of pancreatitis.

Other virus diseases or presumed viral diseases not readily detected by these means, but which would be expected to be recognised through clinical and histopathology findings

Plasmacytoid leukaemia

The neoplastic condition diagnosed as lymphosarcoma in Atlantic salmon in Tasmania may be the same as plasmacytoid leukaemia reported overseas; however, there is no evidence to support this. Information that suggests the two diseases are separate and distinct include: the apparently widespread distribution of fish retroviruses and retrovirus-like neoplastic conditions; the relatively species- (or genus-) specific nature of these viruses; and the long period of isolation of Australian salmonids. The tumours found in Tasmanian Atlantic salmon are typically solid focal tumours, rather than diffuse leukaemic infiltrates. In some cases the whole kidney may eventually be involved. Frank leukaemia is rare.

Lymphoid-like neoplasms have been recognised for several years as a consistent sporadic finding. The prevalence of this condition is stable but varies between populations. Industry records of gross kidney lesions in harvested fish show that 0.05% to 0.27% of market-size fish may be affected. Lesions are most commonly reported in the kidneys. Lesions may also occur in the liver and in muscle. Lesions have been seen at least once in the choroid of the eye and the spleen, and a leukaemic pattern in blood is rarely seen. Neoplastic infiltrates of gut and pancreas, as reported in chinook salmon in Canada, have not been reported except for a caecal and pancreatic infiltrate in one fish in 1998.

There have been no transmission studies of this tumour in Tasmania. Mortality is only occasionally reported, most cases of disease being detected at harvest.

An estimate of the prevalence of such tumours in muscle from a limited number of populations is being made as part of the histological survey of muscle being carried out this year to determine the incidence of Kudoa infection in Atlantic salmon at harvest (see notes on Kudoa for details.).

Histological examination of so-called lymphosarcoma or lymphoma has been carried out on 23 occasions since 1989, sometimes involving multiple cases. The exact cell type involved has not been determined. Renal tumours were diagnosed in Atlantic salmon by histopathological examination in 1994–95, none were diagnosed in 1996 or 1997. The condition has not been seen in rainbow trout.

Plasmacytoid leukaemia in Canadian chinook salmon appears to be exacerbated by concurrent diseases such as bacterial kidney disease and Enterocytozoon infection, neither of which have been reported in Australia. The absence of these diseases in Australia further complicates efforts to compare these two conditions.

Virus diseases less likely to be detected by routine histological means, but detected by more specific methods such as haematology

Erythrocytic inclusion body syndrome (EIBS)

Detection is by visualisation of the intracytoplasmic inclusions in erythrocytes during haematological examination. As for VEN, haematology records are of most value in determining the presence of the virus, as well as the incidence of anaemia. It should be noted that in the survey reported by Cameron in 1991, fish with anaemia were carefully examined for inclusions, with negative results. Also see notes on haematology examinations of salmonids given for viral erythrocytic necrosis (VEN).

Viral erythrocytic necrosis (VEN)

As this virus has not been isolated on cell culture and does not produce characteristic histopathology findings, the best means of detection is by visualisation of characteristic intra-cytoplasmic inclusions in erythrocytes during direct haematology examination.

Blood smear examination is commonly performed in the course of salmonid disease investigations. Detailed records are not available to indicate how many times this has been done. Haematology is routinely performed where possible, on less commonly studied species in which the status of blood parasites is not known. Blood of salmonids of different ages has been examined to establish a reference collection of normal values (mostly collected in 1989), for comparison with diseased fish. Numerous trout bloods were examined during experimental transmission trials with *Enterococcus seriolicida* in 1990 and an extensive salmon haematology survey was conducted by Cameron in 1991. In 1995, in addition to routine diagnostic blood smear examinations, blood smears were examined from approximately 60 rainbow trout and 80 Atlantic salmon during experimental toxicology studies.

Other species in which significant numbers of blood smears have been examined by pathologists in specific trials either by DPIWE, or at the University of Tasmania (Dr Barry Munday, personal communication to Dr Judith Handlinger) include banded morwong, green back flounder, striped trumpeter, eels, lampreys, *G. maculatus* (Jollytail), blennie, yellow eyed mullet, southern blue fin tuna. This includes a number of families from which the infection has been diagnosed elsewhere.

Though a number of parasites have been detected, no blood cell pathology suggestive of VEN has been seen.

Bacteria and bacterial diseases detectable by routine bacteriological cultures

Two strains of *Aeromonas salmonicida*, one known strain of *Yersinia ruckeri*, and *Vibrio anguillarum* have been detected. There have been two isolations of *Edwardsiella tarda* from marine mammals (from a sperm whale in 1987, and a sub-adult fur seal with an abscess in 1993). DPIWE has not isolated *E. tarda* or *Vibrio salmonicida* from any fish species.

Aeromonas salmonicida

The only strains of *A. salmonicida* which have been detected in Tasmania are the atypical goldfish ulcer disease strain, and a recently described new marine atypical strain from greenback flounder and associated species.

The goldfish ulcer disease strain has been isolated from two groups of goldfish which had been recently introduced to Tasmania, and both of which were destroyed, plus one putative detection by indirect fluorescent antibody test (IFAT) smear from an aged goldfish of longer Tasmanian residency, which could not be confirmed by culture. The remaining fish from this aquarium were also destroyed. The entry of goldfish to Tasmania is prohibited except from sources certified free of this disease for at least 2 years.

The newly described flounder strain of *A. salmonicida*, which has been tentatively termed *A. salmonicida 'Ierunnica'*, was first detected in two groups of experimentally farmed flounder in 1993, both of which had contact with wild stocks. Low levels of infection were found in contact striped trumpeter and one Atlantic salmon (but not from in contact black bream) from one source, but there was no evidence of infection of rainbow trout resident on the other farm. Neither strain was seen during 1994 or 1995. The only other laboratory isolations of *A. salmonicida* have been from experimental

infections and pathogenicity trials using isolates from the above-mentioned cases.

In addition to diagnostic submissions, testing of larger groups of fish (generally 60 per submission) on routine media for bacterial pathogens including *A. salmonicida* has been carried out on stocks derived from all of the four commercial hatcheries for the purpose of export certification. This has included rainbow trout and Atlantic salmon. During 1989 there were 16 such submissions involving kidney cultures of 910 fish from four sea farm sites, and a further 1,470 fish were tested by culture in 1990–93. These cultures would have detected the pathogens listed above if present in a culturable state. In 1994 there were 173 submissions involving bacterial cultures of 782 fish. (Most of these included routine kidney cultures as above, but a few involved only skin culture on media which would not detect this organism). Of these, 112 were of Atlantic salmon from marine sites, one of rainbow trout from a marine site, 42 were of Atlantic salmon from freshwater hatcheries, and 11 from rainbow trout from freshwater hatcheries. In 1995 there were 68 salmonid submissions, involving culture of 369 fish, plus 40 submissions involving culture of 221 finfish of other species.

Yersinia ruckeri

Y. ruckeri is sporadically isolated from fish in Tasmania, mainly from Atlantic salmon, though a few isolates have been from rainbow and brown trout. In total, 258 Atlantic salmon, five rainbow trout and four brown trout were found infected between 1995–98. (In 1994, *Y. ruckeri* was isolated from 14 submissions (37 fish), in 1995, from six submissions (24 fish), in 1996 from seven submissions (15 fish), in 1997 from 28 submissions (86 fish), and in 1998 from 24 submissions (105 fish).

A study of strain serotype of Australian isolates (135 isolates, including 99 from Tasmania) showed all were of the one serotype, serovar 3, which is the so-called 'Australian' strain. It is distinct from the Hagerman strain. This study included clinical cases in stressed fish and hatchery survey isolates.

Vibrio anguillarum

This pathogen was detected in salmonids in the initial years of marine culture, with 38 isolations from salmonids from 1987 to 1989. There have been no further isolations from salmonids since vaccination against the local strain was routinely adopted.

Vibrio ordalii

This was detected in the water column and sediment of Tasmanian waters in 1995–96, but has not been isolated from nor associated with disease in fish.

Bacterial diseases not detectable through routine culture, but which would be detectable on Gram stained smears or clinical and histopathology findings

Renibacterium salmoninarum

If present in Tasmania, infection of salmonids would have been detected, given the large number of fish examined by farm inspections at harvest during disease investigations and during collection of virology samples for virus certification and systematic bacteriological examinations. In addition, two of the four commercial hatcheries have been regularly tested for the presence of this pathogen to satisfy export requirements. Testing in 1989 included four sea farm sites, including stock derived from all four hatcheries. In 1990–93, DPIWE conducted 1170 IFAT tests for bacterial kidney disease. An additional 910 kidney smears were referred to AFDL for IFAT during 1989. In 1994, 210 kidney smears were examined by IFAT, and 120 in 1995.

As part of the routine surveillance, all farms have been advised to examine fish for abnormalities during processing (and to submit samples to the laboratory at no additional cost), and processing factory staff have been included in training sessions on fish sampling procedures to encourage this practice. Farms have been examining and recording the incidence of kidney lesions to determine the incidence of the renal lymphoma, and histological examinations at this laboratory have confirmed swollen kidneys as renal lymphoma or more rarely as nephrocalcinosis of tubular origin, and once as a fungal granuloma.

The number of occasions in which kidneys have been examined during routine disease investigations is not recorded. The number of occasions in which renal lesions have been detected between 1983–93 is 90. Renal lesions were detected on three occasions in 1994

(two in rainbow trout and one in Atlantic salmon) and on nine occasions during 1995 (seven in Atlantic salmon, two in rainbow trout), on 10 occasions in 1996, 11 in 1997, and 10 between Jan-June 1998, with no suspicious lesions.

Piscirickettsiosis

Both DPIF and the Tasmanian salmonid industry have been well aware of this condition since it was described, and efforts have been made to ensure that any lesions which remotely resemble the characteristic lesions are examined for the presence of the organism, including Giemsa stains of sections on a number of occasions where slightly similar lesions were seen. In addition to the substantial amount of histological examinations, samples have once been submitted for cell culture specifically for Rickettsia (without antibiotics) during investigation of a previously unknown gill condition, because pale gill lesions had been reported with rickettsial infections. (The gill condition was later considered to be algal bloom related, though other potential environmental influences are still being investigated.) There has been no histological, haematological, or Gram stained smear evidence of rickettsial septicaemia in Tasmanian fish.

Other diseases and agents, including protozoa, likely to be detected by routine pathology and histopathology examination

Loma salmonae is unlikely to have gone undetected in Tasmania due to the very large number of gills of sea-caged salmonids examined, the high level of monitoring for amoebic gill disease, and the more recently recognised algal bloom associated clubbed/necrotic syndrome, plus the large number of experimental studies carried out on gills. The latter include substantial studies, undertaken over a number of years, of the experimental effect of various potential treatment for amoebic gill disease, the timing of the smolt window, experimental transmission of amoebic gill disease, the effects of various suspected deleterious algae on the gills, the effect of temperature on gills, and the effects of benzalconium chloride and peroxide treatment on gills. The number of occasions in which gills were examined is not recorded. The number of occasions in which gill pathology has been found in farmed or wild fish between 1983–93 is 464. Gills were examined from 281 fish (243 salmon and 38 trout) during routine diagnosis in 1994, and 385 fish (250 salmon, 135 trout) in 1995. Gills from 1300 additional fish per year have been examined in a three-year study monitoring the influence of environmental conditions on the gills of salmonids.

The low incidence of clinically and haematologically detectable anaemia suggests that *Enterocytozoon salmonis* is not in Tasmania. Large numbers of organs have been examined in routine pathological examinations of freshwater and marine salmonids. A large number of kidney samples from failed smolts have been studied as well as the examination of kidneys for haematopoietic tissue depletion due to trimethoprim treatment, and in studies of renal lymphoma. In addition to routine diagnostic examinations, at least 60 trout and 80 salmon kidney samples were tested haematologically in 1995 in the course of short-term toxicity trials.

The number of kidney samples examined is not available. Ninety cases of kidney pathology were reported in 1983–93. Renal lesions were detected on three occasions in 1994 and on nine occasions in 1994.

Pancreas disease: Lesions typical of pancreas disease have not been reported in routine pathological examination or in surveys. Heart lesions and skeletal muscle lesions are less characteristic of this disease. Heart lesions of the severity reported for pancreas disease overseas have not been seen; nor is there a significant incidence of heart lesions in Tasmanian salmonids.

The number of heart and muscle samples examined is not available. Pathological findings in the pancreas were reported six times. Cardiovascular lesions have been reported 27 times and musculo-skeletal (predominantly muscular) lesions were recorded 72 times until 1993 and 12 times in 1994–95.

Proliferative kidney disease: This disease has not been reported. If present in Tasmania, it is likely that it would have been detected, in view of the number of kidney samples histologically examined (see bacterial kidney disease and plasmocytoid leukaemia, above), and the lack of endemic diseases producing such lesions in Tasmanian salmonids. Proliferative-type lesions are

occasionally reported in association with fungal infections. Tubular pathology is seen in association with nephrocalcinosis).

Parvicapsular disease: Myxosporean spores or developmental stages typical of this organism have not been detected in the course of examination of kidneys of marine salmonids. As stated above, proliferative nephritis has not been reported.

Ceratomyxa shasta: Spores of this pathogen are of a characteristic shape. No similar spores have been reported in Tasmanian salmonids; nor have other myxosporean stages (or other myxosporean spores except as recorded here). Two of the three commercial rainbow trout hatcheries have conducted routine testing programs for this parasite according to methodology of the Canadian Fish Health Protection Regulations Manual of Compliance. The purpose of this testing is to support export certification. These stocks have a common origin with the other commercial stocks. Four marine sites have also been tested for this parasite. At least 60 fish were examined from each site, including Atlantic salmon and rainbow trout and some fish derived from all four hatcheries. A total of 2080 fish were specifically tested for *C. shasta* in 1989–93 and 150 were examined in 1994.

Kudoa thyrsites can be readily detected, if present at significant levels, by gross and histological examinations. Muscle tissue is examined in routine examinations of sick fish, and in submissions of muscle with melanosis or tumours. Muscle is also included in full investigations of new or unusual incidents, such as the summer stress syndrome, and all the larger surveys such as the copper toxicity trials conducted in 1995. The number of muscle samples tested is not recorded. Muscle lesions have been reported 72 times prior to 1993, and 12 times in 1994–95. This represents a very low proportion of the number of muscle examinations.

Muscle lesions containing myxosporean spores resembling *K. thyrsites* as a single focus of a small number of spores within one muscle fibre were reported once. Because of the small number of spores, these could not be identified to species level.

A survey of the prevalence of *K. thyrsites* or other myxosporeans in muscle of Tasmanian salmonids was carried out in 1994 by the University of Tasmania. Muscle samples from 1606 Atlantic salmon and 120 rainbow trout from marine farms were examined. *Kudoa thyristes*-like myxosporea were seen in one sample from Atlantic salmon (B Munday personal communications).

Henneguya salminicola: This parasite was not reported in routine histological examination of muscle, or during the 1994 *Kudoa* survey. Salmonid processors have not reported the problem of 'milky flesh'.

Other agents, including protozoa, which are more difficult to detect or are unlikely to be detected unless specific examinations are carried out

Rosette agent may be difficult to detect. However, the normal rate of mortality of salmonid fish is low. The intensity of surveillance is high, providing confidence that all episodes of significant mortality are investigated. There are no endemic conditions that closely mimic disease due to Rosette agent histologically. Liver lesions are rare but were reported in 90 cases in 1983–93, and 10 times in 1994–95.

Whirling disease (Myxobolus cerebralis). Since 1988, more than 4000 fish-heads have been examined for whirling disease, all with negative results.

Other disease agents

Other disease agents detected include: *Mycobacteria* spp, *Vagococcus salmoninarum*, atypical marine strains of *Aeromonas salmonicida* (in greenback flounder), *Trichodina* spp (in greenback flounder), *Gyrodactylus* sp (one report in greenback flounder), *Ichthyophonus hoferi, Sphaerospora* spp (in guppies), *Lactococcus garvieae* (not seen since 1992), systemic iridovirus (isolated from imported dwarf gouramis and Ramirez dwarf cichlids, all stocks being destroyed), *Bonamia* sp (in oysters, 1996).

Fish disease surveillance and monitoring in Western Australia

Western Australia has a large fish production based on marine and aquacultured fish, with production (including fish caught in Commonwealth-controlled offshore waters) valued at nearly A$560 million in 1997–98.

Fish species caught include shark, snapper, Spanish mackerel, pilchards, dhufish, Australian salmon, snapper, grouper, boarfish, and orange roughy as well as rock lobster, prawns, crabs, abalone, and scallops.

The pearl oyster fishery is the dominant aquaculture industry; however, yabbies and maron are also cultured. Rainbow trout are the main salmonid species cultured commercially, although there is a small production of brown trout. Most trout production occurs at the Fisheries WA hatchery at Pemberton.

LEGISLATION

The *Stock Diseases Regulations Act 1996* provides for control over suspected cases of disease to order stock into quarantine, requires testing of stock prior to entry to the State and limits movement within the State. The Act applies to fish gazetted as 'stock' under the Act, including most aquacultured species. Notifiable diseases are listed in an 'exotic disease list' and a list of diseases of 'special significance to the State'.

The objective of the *Exotic Disease of Animals Act 1993* is to control exotic diseases in the State. It provides powers to declare any disease of animals (including fish and shellfish) to be an exotic disease, and covers aquatic diseases.

The *Fisheries Resources Management Act 1994* is not intended for disease control, but provides powers to restrict movement of aquatic animals in the State and to require cleansing of fishing equipment.

Finfish diseases listed under the *Stock Diseases Regulations Act 1996*:

- Anguillicola
- Bacterial kidney disease
- Channel catfish disease (ictalurid herpes virus type 1)
- Edwardsiellosis
- Goldfish ulcer disease (*Aeromonas salmonicida*)
- Epizootic haematopoietic necrosis
- Epizootic ulcerative syndrome
- Furunculosis (*Aeromonas salmonicida* var *salmonicda*)
- Infectious haematopoietic necrosis
- Infectious salmon anaemia
- Infectious pancreatic necrosis
- *Oncorhynchus masou* virus disease (herpes virus 2)
- Piscirickettsiosis
- Spring viraemia of carp
- Viral haemorrhagic septicaemia
- Viral encephalopathy and retinopathy

DISEASE ZONING

All imports of live salmonids and redfin perch from other Australian States have been banned since 1988. Imports of live silver perch, golden perch and Murray cod (which may be infected with EHNV) have been restricted since 1987 to prevent the entry of EHNV into the State. There are no restrictions on the entry of non-viable marine finfish for human consumption.

There are no disease control zones within the State.

FISH DISEASE DIAGNOSTIC SERVICES

The WA Department of Fisheries employs two specialist fish pathologists to provide a diagnostic service for commercial fish, shellfish (including pearl oysters), crustacea and aquaculture (including imported fish inspected by AQIS) through the Animal Health Laboratory, South Perth. Samples may be submitted directly from the farm, though they are usually received via a visiting animal health professional.

The facilities have good general diagnostic resources and skills, and specialist fish diagnostic skills in pathology, histopathology, bacteriology, parasitology and virology. The virology laboratory routinely conducts two passages in appropriate cell cultures (which include FHM, RTG,

CHSE, BB, BGF cells), checking for cytopathic effect and backed by transmission electron microscopy as needed (on site). The antigen-ELISA for EHNV is available, positive control samples being held in a secure facility. A scanning electron microscopy and backup transmission electron microscopy are available at Murdoch University Veterinary School.

DISEASE MONITORING AND SURVEILLANCE

There has been a steady increase in diagnostic submissions since 1995. In 1998, most diagnostic cases were fish samples. There have been 26 salmonid submissions since 1995.

Annual fish health monitoring and investigation of unusual mortalities have been conducted at the Pemberton trout hatchery since 1988. Samples are tested using the standard diagnostic techniques for bacteria and parasites; viruses have been tested for since 1995. Screening for EHNV began at Pemberton in 1996, based on techniques recommended by the Elizabeth Macarthur Agricultural Institute, NSW. Samples collected in 1996–97, 1997–98 and 1998–99 tested negative for EHNV.

There is ongoing monitoring of trout hatcheries for EHNV. All results to date have been negative.

Marine fish hatchery surveillance: All shipments of black bream (*Acanthopagrus butcheri*) and snapper (*Pagrus auratus*) fingerlings from marine hatcheries are sampled (150 fingerlings) and examined by histology for lesions prior to movement.

Tests for VHS on marine fish submissions since 1995 have all been negative.

Targeted investigation of goldfish ulcer disease: Publicity in a farming magazine was used to increase public awareness and reporting of ulcers in cyprinids and goldfish. Reports were investigated by histopathology and culture.

Salmonid hatchery program: There is one salmonid hatchery in the State, which is subjected to surveillance and monitoring for EHNV.

CERTIFICATION TESTING

In 1998, 26% of all submissions were submitted for the purpose of certification.

One submission of 150 silver perch was tested for EHNV exclusion prior to import into the State.

Numbers of finfish examined

YEAR	FISH HEALTH / DIAGNOSTIC	HEALTH CERTIFICATION
1994	50	
1995	92	
1996	108	
1997	105	
1998	131	3
1999 (to 8/3/99)	36	3

Fish health and diagnostic submissions usually comprise 10–50 fish per case, while health certification tests (including tests before interstate movement) usually comprise 60 finfish.

SIGNIFICANT FINFISH DISEASE AGENTS DETECTED IN THE STATE

Since 1995 there have been about 30 cases of infections of tropical fish in quarantine with iridovirus, EUS, systemic amoebae and systemic *Tetrahymena*-like flagellates. Other infectious agents diagnosed include herpes virus in pilchards, epitheliocystis, *Flexibacter* sp, *Mycobacterium marinum*, *Vibrio mimicus* and ubiquitous external parasites such as *Ichthyophthirius*, *Trichodina, Chilodonella hexasticha and C. cyprini.* The Australian atypical strain of *A. salmonicida* has been isolated from salmonids in Western Australia but not since 1992.

Fish disease surveillance and monitoring in South Australia

South Australian production from wild fisheries (including fish caught in Commonwealth-controlled offshore waters) and aquaculture was valued at about A$291 million in 1997–98.

Main species caught include King George whiting, snapper, garfish, tuna, ling, jack mackerel, deepwater flathead, Bight redfish, shark, ocean leatherjacket, yellow-spotted boarfish, jackass morwong, western gemfish, orange roughy, rock lobster, prawns, and abalone.

The main aquaculture enterprise involves farming of wild-caught tuna in sheltered offshore waters; other aquaculture enterprises include oysters, yabbies, and marron. Small numbers of Atlantic salmon are cultured in cages offshore and rainbow trout are cultured onshore in ponds.

LEGISLATION

The *Livestock Act 1998* includes fish in the definition of livestock; lists notifiable fish diseases; and provides controls over the importation, treatment and movement of farmed fish.

Notifiable diseases of finfish under the *Livestock Act 1998* include the following.

- *Aeromonas salmonicida* var *salmonicida*
- *Aeromonas salmonicida* (atypical strains)
- *Aphanomyces invadans*
- Channel catfish virus
- *Edwardsiella ictaluri*
- Epizootic haematopoietic necrosis virus
- *Gyrodactylus salaris*
- Infectious haematopoietic necrosis virus
- Infectious pancreatic necrosis virus
- Infectious salmon anaemia virus
- *Myxobolus cerebralis*
- *Oncorhynchus masou* virus
- *Piscirickettsia salmoninarum*
- *Renibacterium salmoninarum*
- Spring viraemia of carp virus
- Viral encephalopathy and retinopathy (nodavirus)
- Viral haemorrhagic septicaemia virus
- *Yersinia ruckeri*

The *Fisheries Act 1982* provides for the control of exotic fish species; regulates fish farming and processing; and gives powers to declare protected and quarantine areas. Section 51 of the Act provides control of disease in fish and empowers inspectors to investigate and control declared diseases. There are also regulations to prohibit, control and regulate possession and sale of fish. The legislation does not control the movement of non-viable fish or fish products.

Schedule 4 of the Fisheries Act lists a number of notifiable diseases of finfish.

- *Aeromonas salmonicida*
- Cichlid virus
- Epizootic haematopoietic necrosis (EHN)
- Infectious haematopoietic necrosis (IHN)
- Infectious pancreatic necrosis *(IPN)*
- *Myxosoma cerebralis* (Whirling disease)
- Viral haemorrhagic septicaemia (VHS)

FISH DISEASE DIAGNOSTIC SERVICES

Prior to 1997, the state veterinary laboratory (VETLAB) provided diagnostic, disease certification and disease investigation services (including necropsy, histopathology, bacteriology, parasitology, virology, biochemistry and toxicology) to commercial fisheries and aquaculture (including shellfish and crustaceans). It also investigated environmental fish kills and provided limited services to ornamental fish hobbyists. Species examined included barramundi, salmon, rainbow trout, snapper, pilchards, perch, Murray cod, tuna, mulloway, mullet and various aquarium fish species.

From 1997 a private laboratory service (Veterinary Pathology Services, VPS) was contracted by the State of South Australia to provide veterinary diagnostic services.

These services include diagnostic services to commercial fisheries (finfish, shellfish and crustacean) and aquaculture, and investigation of wild fish kills. VPS diagnostic services include necropsy, histopathology, bacteriology, parasitology, and biochemistry. VPS has professional and technical staff with experience in fish and shellfish diseases. A molecular biologist has been employed by VPS to assist with development of a PCR for barramundi nodavirus. Samples requiring virological testing are referred to Victoria or to the AFDL This diagnostic service receives specimens from many sources, including aquaculturists, governmental officers and private individuals, and the samples include many species of fish. The laboratory is accredited under the National Australian Testing Authorities (NATA) and ISO systems.

A state Fish Health Manager was appointed in 1998. Aquatic animal health surveillance and monitoring programs are consequently expected to be expanded considerably.

DISEASE SURVEILLANCE AND MONITORING

The numbers of finfish subjected to laboratory examinations are as follows.

YEAR	TOTAL FISH	PATHOLOGY	BACTERIOLOGY	VIROLOGY	PARASITOLOGY
1997	980	974	20		
1998	1,104	457	96	6	5

The Tuna Boat Owners Association's Monitoring Program is a baseline health survey of southern bluefin tuna (*Thunnus maccoyii*), involving monthly sampling of farmed tuna on two farms between the months of March and October. Under the program, 15–30 tuna per farm were subjected to gross pathology examinations, and 10 tuna per farm to histology, haematology and serum biochemistry studies. Up to 20 April 1999, 62 tuna had been subjected to gross pathology examinations, 25 tuna to histology examinations, and 32 tuna to haematology and blood biochemistry. Additionally, similar testing was carried out on 10 transferred tuna, and 76 cases of tuna mortality were examined by gross pathological examinations, with histopathology and microbiology examinations as appropriate.

There is ongoing monitoring of disease in European carp (*Cyprinus carpio*) and golden perch (*Pectroplites ambiguus*) in the Murray-Darling river system. Losses due to *Exophiala* spp in farmed King George whiting (*Sillaginodes punctata*) are also being investigated.

CERTIFICATION TESTING

Salmonids must be certified free of notifiable diseases by relevant authorities in Victoria, NSW and Tasmania before import into South Australia.

Barramundi eggs and larvae imported into South Australia must be from healthy stock and are disinfected within 10 hours of fertilisation to ensure freedom from barramundi picorna-like (nodavirus) virus before or after entering the State.

Barramundi fingerlings exported from the State are screened for viral encephalopathy and retinopathy virus (VERV) by histopathology. A polymerase chain reaction (PCR) for VERV is being developed at the VPS laboratory.

Atlantic salmon and their ova entering South Australia must be certified free of the diseases of concern prior to entry into the State. Investigations of problems in Atlantic salmon are conducted at industry request.

SIGNIFICANT DISEASE AGENTS OF FINFISH DETECTED IN THE STATE

Barramundi: *Aeromonas hydrophila*, *Vibrio vulnificus*, *Saprolegnia* sp, *Ichthyophthirius multifilis, Klebsiella oxytoca*, *Chilodonella*, *Epitheliocystis*. Nodavirus infection has been detected in imported barramundi fry in 1992, 1994 and 1998, affected stock being destroyed.

Snapper: *Pseudomonas stutzeri, V hollinae*, gill flukes, *Epitheliocystis, Acinetobacter haemolyticus, Flavobacterium sp. Lymphocystis* disease (diagnosed in 1994), amoebae.

Pilchards: A herpes virus was demonstrated in association with high mortalities in 1995 and 1998.

Whiting: *Exophiala salmonis*, gill flukes, *V. alginolyticus*.
Salmon: *Ichthyophthirius multifilis*.
Rainbow trout: *Ichthyophirius multifilis*, *Aeromonas* sp.
Mulloway: *Dactylogyrus* sp flukes.
Mullet: *Myxobolus* sp.
Tuna: *Uronema nigricans*, *Caligus elongatus*.

Redfin perch: EHNV was first detected in 1991 in South Australia. Clinical cases of the disease continue to occur in the summer months in reservoirs near Adelaide.

Ornamental fish: *Mycobacterium sp, Aeromonas hydrophila, A. sobria, Enterobacter* sp. *Aeromonas salmonicida* (atypical strain, detected in 1994 in imported goldfish; subsequently eradicated).

Kudoa thyrsites has not been detected in South Australia, though evidence from nearby States suggests the possibility that some local non-salmonid marine fish may be infected.

Fish disease surveillance and monitoring in Queensland

Queensland production (including fish caught in Commonwealth-controlled offshore waters) from wild fisheries and aquaculture amounted to over A$360 million in 1997–98.

This was mainly based on mullet, Spanish and grey mackerel, coral trout, barramundi, whiting, red throat emperor, snapper, yellowfin tuna and shark, as well as prawns, crabs, and lobster, and scallops. Aquacultural production is mainly centred on farmed prawns and barramundi.

Salmonid species do not occur naturally. There is an Atlantic salmon and rainbow trout facility near Stanthorpe, which is stocked from interstate hatcheries. These fish are used for recreational fishing on site.

LEGISLATION

The *Queensland Fisheries Act 1994* includes a broad definition of live and dead aquatic animals, and provides control over the possession and sale of noxious or non-indigenous fisheries resources, and the release of such resources into Queensland waters. The Act provides for the imposition of quarantine in response to disease events; defines responsibility to report fish disease; prohibits actions causing the spread of disease; prohibits sale of diseased fish or fish products; and gives power to order destruction or treatment of fisheries resources. Live fish imported into Queensland must be certified free of declared diseases. It is prohibited to import or sell fish products if they contain a declared disease agent. Any disease seen in wild or cultured finfish must be immediately reported to the department.

The Fisheries Regulations 1995 have not yet declared any fish diseases.

The list of declared finfish diseases under the *Queensland Fisheries Act 1994* include the following.

- *Aeromonas salmonicida* var *salmonicida*
- *Edwardsiella ictaluri*
- Channel catfish virus
- Epizootic haematopoietic necrosis virus
- Enteric redmouth disease (*Yersinia ruckeri* serovar I and II)
- Infectious pancreatic necrosis virus
- Infectious haematopoietic necrosis virus
- Infectious salmon anaemia virus
- *Oncorhynchus masou* virus
- Spring viraemia of carp
- *Piscirickettsia salmonis*
- *Renibacterium salmoninarum*
- Viral haemorhhagic septicaemia virus

DISEASE ZONING

There are currently no disease control zones for finfish.

FISH DISEASE DIAGNOSTIC SERVICE

The Queensland Department of Primary Industries (QDPI) Fish Health Services (FHS) team provides a free diagnostic service to all aquaculture operators and will assist in the investigation of fish kills and wild fishery diseases. Diagnostic services are provided from the Oonoonba veterinary laboratory (OVL) for northern Queensland and the Yeerongpilly veterinary laboratory (YVL) for southern Queensland. The fish health service members at the OVL also undertake research on high-priority aquatic animal disease problems.

OONOONBA VETERINARY LABORATORY

The fish health service professional staff include two veterinary pathologists (one of whom acts as fish health services team coordinator), one veterinary officer (laboratory and extension), a microbiologist, and a virologist. Three full-time technicians provide further support to the aquatic animal health services. The facilities at the OVL include an isolation building, fresh and saltwater tank capacity and on-site ponds.

This laboratory is well equipped to provide necropsy, histopathology, microbiology, parasitology, and virology services to aquatic animals, including finfish, mollusc and crustaceans, and is also active in aquatic animal health research. Virology services include barramundi spleen, barramundi kidney, Australian bass head kidney, RTG, BF-2 and FHM cell lines. Suspect tissues are passed twice and checked for CPE and by electron microscopy as appropriate. Samples are sent to YVL in Brisbane for electron microscopy or to the AFDL if indicated.

Special interests at OVL include diseases of all aquaculture species (including barramundi, eels and silver perch) and wild species. Scientific expertise covers columnaris disease and streptococcosis in barramundi; bacterial diseases of live reef fish in holding facilities; bacterial vaccines for tropical marine finfish; development of tropical finfish cell culture isolation systems; barramundi nodavirus; and identification of pathogenic *Vibrio* species by PCR.

YEERONGPILLY VETERINARY LABORATORY

The YVL at Brisbane provides basic aquatic animal pathology, microbiology and parasitology services, which are coordinated by veterinary officers specialising in fish diseases (laboratory and extension). Fish virology is referred to OVL or the AFDL.

Specialist technical support staff for toxicology, clinical chemistry and electron microscopy present in the Animal Research Institute (Yeerongpilly, Brisbane) are utilised by the Yeerongpilly and Oonoonba staff as required.

Other aquaculture and fishery extension officers with basic training in aquatic animal health are also available for field visits.

DISEASE SURVEILLANCE AND MONITORING

There are no structured disease monitoring and surveillance programs for finfish in Queensland.

CERTIFICATION TESTING

Barramundi fingerlings are examined routinely by histology prior to interstate movement.

NUMBERS OF SPECIMENS EXAMINED

Laboratory resources are mainly applied to tropical finfish, prawn, freshwater crayfish and pearl oyster diseases.

Queensland Department of Primary Industries had 1152 accessions of finfish or groups of finfish in the 1990–98 period.

Significant diseases/disease agents of finfish reported in Queensland include the following.

Barramundi (*Lates calcarifer*): *Vibrio harveyi*, *Streptococcus iniae*, *Flexibacter marinum*, *Flavobacterium* sp., *Ichthyophthirius multifiliis*, Epitheliocystis, Lymphocystis disease.

Eels (*Anguilla* sp): *Trichodina* sp, *Ichthyophthirius mulifiliis*

Sooty grunter (*Hepahaestrus fuliginosus*): *Exophiala* sp.

Perch: *Chilodonella cyprini.*

Mangrove Jack (*Lutjanus argentimaculatus*): *Mycobacterium* sp.

Dwarf gourami (*Colisa lalia*): Iridovirus infection and amoebiasis in imported stock during quarantine.

Goldfish (*C. auratus*): *Citrobacter freundi*, *Aeromonas salmonicid* (atypical strains isolated from ulcerated goldfish held at ornamental fish wholesalers).

Northern Territory

Fisheries production in State and adjacent Commonwealth-controlled offshore waters was valued at over A$135 million in 1997–98.

The wild fisheries in Territory waters are mainly based on snapper, barramundi, shark, mackerel, crab and pearl oysters, while aquaculture predominantly involves pearl oysters. There are no salmonids in the Northern Territory.

LEGISLATION

The *Stock Disease Act 1954* does not specifically apply to fish, though it could be used if fish are declared an animal under the Act.

Regulations under the *Fisheries Act 1998* contain provisions to control exotic disease in fish and fish product through movement and importation restrictions, power to declare quarantine and protected areas, power to order treatment or destruction of diseased fish or associated equipment or water, and by prohibiting the importation of diseased fish. There are currently no declared diseases of aquatic animals.

Holders of an aquaculture licence under the Fisheries Act and its regulations (Fisheries Regulations, Part 3, Divisions 1, 2 and 3) require a permit to import live fish or aquatic life into a body of water, must provide the Director of Fisheries with certification declaring the product to have been tested and found to be disease-free prior to shipment, and are required to report above-average mortalities and/or disease outbreaks to the Director of Fisheries.

The 'National List of Reportable Disease of Aquatic Animals' was incorporated into the Fisheries Act by June 1999.

DISEASE ZONING

There are no disease control zones.

FISH DISEASE DIAGNOSTIC SERVICES

A fish disease diagnostic service is provided by the Department of Primary Industry and Fisheries Berrimah laboratory. If needed, diagnostic material can also be referred to Queensland Veterinary Laboratories and to the AFDL.

DISEASE SURVEILLANCE AND MONITORING

There is no formal health monitoring or surveillance in place for wild fish. Lymphocystis disease has been detected in barramundi (*Lates calcarifer*).

CERTIFICATION TESTING

A coastwatch program has been instituted to monitor and report significant disease events in fish.

Australian Capital Territory

The Australian Capital Territory has no commercial fisheries but it has a recreational fishery based on introduced species such as trout.

LEGISLATION

The *Stock Act 1993* does not cover fish. The *Animal Diseases Act 1993* includes 'fish' (vertebrates and invertebrates including eggs and gametes) but no aquatic animal diseases. It is proposed to include notifiable diseases of aquatic animals under the *Animal Diseases Act 1993*.

Wild fisheries resources are controlled under the *Nature Conservation Act 1980* and the *Fishing Act 1967*, which are not designed for disease control. The Nature Conservation Act requires a licence to import live fish into the ACT, requires transport water to be treated in holding tank prior to release, requires containers, bags and so on to be sterilised or burnt, and requires any disease to be reported within 24 hours.

FISH DISEASE DIAGNOSTIC SERVICES

Because of the small size and population of the ACT, field investigation services are very limited and local veterinary diagnostic facilities do not exist. Consequently, disease problems are referred to the EMAI or to the Wollongbar regional veterinary laboratory in NSW for diagnosis and further investigation.

DISEASE SURVEILLANCE AND MONITORING

This is limited to investigation of disease problems as they arise.

CERTIFICATION TESTING

No certification testing is carried out.

AAHL Fish Diseases Laboratory

The AAHL Fish Diseases Laboratory (AFDL) was previously known as the Australian Fish Health Reference Laboratory (AFHRL), and is located at the CSIRO Australian Animal Health Laboratory (AAHL) at Geelong, Victoria, which has high microbiological security facilities for undertaking exotic disease research. AFDL acts as a national referral laboratory for aquatic animal diseases, and AAHL is the OIE reference laboratory for the epizootic haematopoietic necrosis virus (Dr Alex Hyatt, nominated as expert). AFDL is staffed by four scientists and a number of technical officers, with further scientific and technical support being provided by other AAHL scientific staff.

FACILITIES

The laboratory has excellent pathology, bacteriology, parasitology and virology facilities, including high security freshwater and seawater aquarium facilities. The procedures used at the laboratory for the detection and identification of fish pathogens are based on standard protocols from the United States, Canada, the United Kingdom and the European Union. In addition, in conjunction with these standard protocols, other standardised procedures have been developed to assist pathogen identification, such as PCR, *in situ* hybridisation, ELISA, immunoperoxidase tests and Western blotting.

The pathology facilities include immunohistochemistry for IPN, VHS, IHN, EHN viruses and *Aeromonas salmonicida.* Virology facilities include a wide range of fish cell lines including CHSE-214, BF-2, RTG-2, Snakehead, CAR, FHM, EPC, which can be used for the isolation of a wide range of fish viruses. In addition, various reagents such as polyclonal and monoclonal antibodies are available for the identification of the major exotic viruses of farmed fish. There are excellent transmission and scanning electron microscopy facilities applying techniques such as immunogold staining for the major fish virusesóEHNV, IPNV, VHSV and IHNV, amongst others. The fish bacteriology facilities include selective media and reagents for *Aeromonas salmonicida, Flexibacter maritimus, Yersinia ruckeri, Enterococcus seriolicida, Renibacterium salmoninarum,* a range of diagnostic antibodies, an extensive collection of reference and clinical isolates, and a number of PCR-based tests with access to automated sequencing facilities.

The laboratory is actively involved in health surveillance and certification services to assist export of salmonid products. Hence many of the accessions received by AFDL are for export certification and health surveillance rather than diagnosis of disease, and involve virological examination only. Examinations not requiring the special (microbiologically secure) facilities at AAHL (eg bacteriology, parasitology) are undertaken by local state laboratories with specific capabilities. All procedures used for export certification and health surveillance have been validated using exotic bacterial and viral pathogens available at AAHL.

The laboratory is actively involved in developing and applying new diagnostic techniques, and in carrying out research into fish and prawn diseases (in conjunction with other national, state and university authorities). Some of the earlier investigations by AFDL involved survey work, the methods and results of which have been published.

1 Amos KH (ed) (1994) *Suggested Procedures for the Detection and Identification of Certain Finfish and Shellfish Pathogens*. 4th edition. Fish Health Section, American Fisheries Society. Corvallis, Oregon.

2 Fisheries and Marine Service (1977). *Fish Health Protection Regulations Manual of Compliance*. Miscellaneous Special Publication No. 31. Ottawa, Canada.

3 Ministry of Agriculture (Amended 1982). *Test Requirements for Health Certification of Fish Egg Imports. Imports of eyed Eggs from Fish-Farm Brood Stocks*. DOF 6. Ministry of Agriculture, Fisheries and Food. London, UK

4 Commission Decision of 19 November 1992 laying down the sampling plans and diagnostic methods for the detection and confirmation of certain fish diseases (92/532/EEC). *Official Journal of the European Communities* No. L 337/18, 21.11.1992.]

Appendix 7
Review of literature on *Aeromonas salmonicida*: typical (furunculosis) and atypical strains

IN VIEW OF THE SIGNIFICANCE OF THIS DISEASE, AQIS has undertaken a review of the literature as a basis for the release, exposure and consequence assessments. The review draws upon information in AQIS's previous reports and the 1997 report of the New Zealand Government (Stone et al 1997b) as well as information presented in Bernoth et al (1997), a recent and extensive compilation of data on furunculosis.

DESCRIPTION OF DISEASE / DISEASE AGENT

The classification of *Aeromonas salmonicida* subspecies is unresolved; most recently it has been proposed that this species would be best considered as three subspecies, which may infect various freshwater and marine fish. Typical strains of the organism are known as *A. salmonicida* subspecies *salmonicida*. Atypical strains isolated from salmonids are classified as *A. salmonicida* subspecies *achromogenes*, while atypical strains isolated from non-salmonid fish are classified as *A. salmonicida* subspecies *nova*. However, this does not adequately categorise all known isolates; for practical purposes isolates may be classified as typical or atypical strains of *A. salmonicida*, both of which are considered in this risk analysis (DPIE 1996, Stone et al 1997b).

Furunculosis in salmonids may manifest as follows.

- Peracute infection typically occurs in fry and fingerlings. The fish are dark and there is a high rate of mortality. Internally the gross pathological changes are similar to those seen in acute disease.
- Acute infection may occur in any age or size of fish. Signs of disease (darkening, anorexia) are often noted 2–3 days before fish start to die. Internal signs include haemorrhage of the viscera, softening of the kidney tissues, enlargement of the spleen, pallor or mottling and petechial haemorrhage of the liver.
- Subacute infection is typified by the formation of skin lesions and, in some cases, lesions in the viscera. The mortality rate gradually increases.
- Chronic infection has a similar course to subacute infection but there is evidence of healing of lesions.

In cases of latent infection there are no clinical changes or increases in mortality.

Histopathological examination of acutely infected fish may reveal focal accumulations of bacteria in the heart, kidneys and spleen and in the vasculature of other organs. Other changes include necrosis of the haematopoietic tissues and liver and degenerative changes in the myocardium and renal tubular tissues. In chronic disease, the heart and spleen are the most consistently affected organs. The presence of large bacterial colonies in the myocardial trabeculae is virtually pathognomonic for furunculosis in salmonids in fresh water. The 'furuncles' in the skeletal musculature of chronically infected fish comprise necrotic tissue, tissue exudate and macrophages (Shotts 1997).

Atypical strains of *A. salmonicida* appear to be less invasive than typical strains. In contrast to the marked pathological effects of clinical infection with typical *A. salmonicida,* infection with atypical *A. salmonicida* usually causes less severe skin ulceration and internal lesions are generally limited to minor visceral haemorrhage and splenomegaly. More recently, atypical *A. salmonicida* has caused significant but sporadic outbreaks of disease in salmonids.

Fish that are clinically infected with typical *A. salmonicida* may display visible external signs, including haemorrhage, especially around the bases of the fins and vent, and furuncles. Postmortem findings include internal haemorrhage, especially over the swim-bladder. Visibly abnormal fish would be unlikely to pass inspection and grading. However, fish that are not clinically infected with typical *A. salmonicida* and fish infected with many of the atypical strains of *A. salmonicida* would be expected to appear normal and pass inspection.

Geographical distribution

TYPICAL *A. SALMONICIDA*

A. salmonicida salmonicida causes furunculosis in fish in North America, South America, Europe, Asia and Africa (Shotts 1994).

Furunculosis was diagnosed for the first time in farmed Atlantic salmon in Norway in 1964. It was probably introduced with a consignment of smolts from Denmark. Attempts to eradicate the disease have been ineffective (Gjedrem et al 1991). The disease is now considered to be endemic in major fish breeding areas along the coast and to have caused significant problems in Norwegian fish farms for the last decade (cited in Husevag and Lunestad 1995). In recent years, the incidence of furunculosis has reduced in freshwater hatcheries and the disease is regarded mainly as a problem of farmed fish in seawater (Jarp et al 1993). However, wild salmon and trout may also be affected (Wiklund et al 1992).] The prevalence of furunculosis in sea-reared salmonids in Norway has been historically high (Jarp et al 1993). However, the application of sanitary measures and widespread use of vaccine have contributed to effective management of furunculosis in Norway, and mortality rates due to this disease are now low (AQIS 1996).

Furunculosis was first reported in Britain in 1906–07, with large mortalities in wild fish in 1911. By 1935, disease had been reported in Atlantic salmon and brown and sea trout in 28 rivers flowing to the east and south-west coasts of Scotland. Furunculosis infection is considered to be endemic in wild spawned salmon from these rivers. There is still a high prevalence of infection in wild spawned salmon. Epidemic furunculosis is rarely seen in wild salmon in Scotland, but it is a serious infectious disease affecting farmed salmon (cited in Johnsen and Jensen 1994). Since the introduction of efficacious vaccines in the early 1990s, furunculosis has been of less concern. Currently, survival figures from smolt to harvest are well above 90%, this figure including losses due to all causes (eg storm damage, escapes, other disease) (A McVicar pers. comm.). Because of the success in control, furunculosis has now dropped well down the ranking in importance among diseases currently affecting the Scottish salmon farming industry (A McVicar pers. comm.).

Furunculosis has caused epizootic disease in salmon and trout at two coastal farms in Northern Finland since 1986, with a mortality rate of 1–29% in affected pens(Rintamaki and Valtonen 1991). Historically, furunculosis has been reported as a serious disease of Spanish aquaculture (Sanz et al 1993, Ortega et al 1993, Real et al 1994). Furunculosis also occurs in Russia (Wiklund et al 1992).

There is less information available on the progression of disease due to *A. salmonicida* in North America. Furunculosis continues to be problem in all Atlantic Canadian provinces with the exception of Prince Edward Island (Hammell 1995). In British Columbia, furunculosis has caused disease in Atlantic salmon and, to a lesser extent, in chinook salmon. In Washington state, USA, *A. salmonicida* occurs primarily in wild adult salmon in fresh water (K Amos pers. comm.).

Furunculosis has been reported in mature chum salmon, pink salmon and masou salmon in Japan (Nomura et al 1993). In South Africa, *A. salmonicida* was isolated from trout reared in seawater in the Cape Province and rainbow trout in Transvaal. Furunculosis has not been reported from Natal (Bragg 1991).

Disease due to infection with typical *A. salmonicida* does not occur in New Zealand (Anderson et al 1994) or Australia.

ATYPICAL *A. SALMONICIDA*

Infection of farmed fish with atypical *A. salmonicida*, causing an epidermal ulcer disease, has been reported since the 1960s. The prevalence of infection with atypical *A. salmonicida* has increased in northern Europe during the last 10 years (Hanninen and Hirvela-Koski 1997). Atypical strains of *A. salmonicida* cause a variety of disease conditions in salmonids and non-salmonids. In Nova Scotia and Newfoundland (Groman et al 1992), mortality rates as high as 25% have been recorded in Atlantic salmon over three-year classes (DPIE 1996, Stone et al 1997b).

A recent review of the occurrence of atypical *A. salmonicida* in non-salmonid and salmonid fish (Wiklund and Dalsgard 1998) concluded that atypical strains of *A. salmonicida* infect a large number of species worldwide. Generally these strains cause more disease in farmed fish than wild fish, and are of increasing importance due to their propensity to develop antibiotic resistance and the failure of diagnostic programs to detect them. Atypical strains of *A. salmonicida* appear to be less invasive than typical strains and generally cause skin infection but not septicaemia. Infection with atypical *A. salmonicida* usually has relatively minor pathological effects, compared with disease due to typical strains of *A. salmonicida*.

While records of ulcer disease in goldfish at a Victorian farm date back to 1974 (Trust et al 1980), an atypical strain of *A. salmonicida* was not isolated in Australia until 1980. When inoculated intraperitoneally, this organism caused the development of skin lesions and septicaemia in Atlantic salmon and in rainbow, brown and brook trout. Bath exposure caused disease in trout. Exposure via cohabitation caused infection in five out of 195 trout; one fish became a carrier of infection. It was concluded that this pathogen poses a significant threat to the salmonid farming industry and wild salmonid fisheries in Australia (Whittington and Cullis 1988). Accordingly, the government of Tasmania introduced legislative controls over the movement of live goldfish into Tasmania. In 1990, the government of Western Australia gazetted disease due to atypical *A. salmonicida* as notifiable in 1990, and goldfish and koi carp as 'stock' in 1991 to provide legislative control of 'atypical furunculosis' (B Jones pers. comm.).

A virulent, atypical strain of *A. salmonicida* was also isolated from juvenile hatchery-reared and wild-caught greenback flounder held in tanks in Tasmania. This strain was also associated with infection in in-contact Atlantic salmon and striped trumpeter in Tasmania. This pathogen has not been associated with disease under natural conditions (Whittington et al 1995).

Host range and prevalence

Furunculosis may occur in various species of marine finfish and may affect fish of every age. However, fish of the family Salmonidae, particularly the brook trout (*Salvelinus fontinalis*), brown trout (*Salmo trutta*) and Atlantic salmon (*Salmo salar*), are most susceptible to infection. Rainbow trout are relatively resistant to infection (McCarthy 1977). In addition, susceptibility may vary in a species of different genetic lines (Dahle et al 1996, Marsden et al 1996) or with different histories of exposure to *A. salmonicida* (St Jean 1992). In salmonids, susceptibility to furunculosis may be age-related. In Finland, infection was reported in Atlantic salmon and brown trout, with higher mortality rates in

yearling sea trout and brood fish than in salmon (Rintamaki and Valtonen 1991).

While typical *A. salmonicida* is usually isolated from diseased salmonids, it has also been associated with clinical and unapparent disease in non-salmonid species in fresh, brackish and seawater. Typical *A. salmonicida* has been isolated from nine non-salmonid marine finfish species, including goldsinny wrasse (*Ctenolabrus rupestris*), turbot (*Scophthalmus maximus*), Atlantic cod (*Gadus morhua*) and coalfish, (*Pollachius viriens*) and 25 species of freshwater fish including carp (*Cyprinus carpio*), minnow (*Phoxinus phoxinus*), tench (*Tinca tinca*) and yellow perch (*Perca flavescens*). In non-salmonids, clinical disease due to typical *A. salmonicida* has typically been recorded under conditions of stress or in fish penned with infected salmonids.

Information in previous reports (AQIS 1996, Stone et al 1997b) indicate that the prevalence of typical *A. salmonicida* in wild Pacific salmon is generally in the range of 0–10%. Numerous studies have demonstrated a higher prevalence of *A. salmonicida* infection in anadromous wild salmonids entering fresh water and maturing sexually. Sexually mature fish returned to fresh water to spawn are likely to have a higher prevalence of infection. There is little evidence that fish returning to fresh water are latently infected; rather, data suggest that few if any wild salmonid fish are infected when entering fresh water at an early stage of sexual maturation.

Olivier (1992) presented the most comprehensive study of prevalence in farmed salmonids. In the Atlantic provinces of Canada (1983–91) 17 of 291 (5.8%) sea cages tested positive for typical *A. salmonicida*. In 1984–91, 15 of 218 lots (6.8%) of juvenile Atlantic salmon from hatcheries tested after the application of stress tests gave positive results for typical *A salmonicida*.

Data on the prevalence of *A. salmonicida* must be interpreted with caution, as a number of factors (which may interact) will affect the detected rate of prevalence. These factors include age, stage of sexual maturity and location (fresh water or seawater) of the population surveyed. Factors such as the sensitivity of diagnostic methods and the use of antibiotics in farmed fish may confound accurate reporting of prevalence. Smith (1997) advised that the effect of seemingly simple factors, such as the age of fish or water temperature, complicate accurate reporting on the prevalence of infection. A McVicar (pers. comm.) stated that for highly susceptible species, such as *Salmo trutta*, it is unlikely that disease would be observed in the wild because of the rate of mortality when infected and the infection would be difficult to detect.

In a recent review of atypical *A. salmonicida* in non-salmonid and salmonid fish, Wiklund and Dalsgaard (1998) concluded that atypical strains of *A. salmonicida* infect a large number of species worldwide. Generally these strains cause more disease in farmed fish than wild fish, and are of increasing importance due to antibiotic resistance and unsuccessful testing programs. With regard to non-salmonid wild fish, atypical *A. salmonicida* has been isolated occasionally from cases of ulcerated or otherwise diseased fish in the field and in wild fish in aquaria (cited in Wiklund and Dalsgaard 1998). In a few cases, atypical *A. salmonicida* has been associated with disease epizootics in wild fish (cited in Wiklund and Dalsgaard 1998).

Detection methods

A. salmonicida may be detected by culture of kidney or gut; however, false negatives occur commonly. The bacterium may be isolated from the skin or gills of carrier fish but the presence of other bacteria in these tissues may make it difficult to detect low numbers of *A. salmonicida*. Failure to recover the organism may be attributed to the presence of *Pseudomonas* spp and other non-glucose fermenting, gram-negative bacteria that overgrow the more fastidious *A. salmonicida* (Cipriano et al 1996). *A. salmonicida* may be cultured on tryptone-soya agar (TSA) or brain-heart infusion agar incubated at temperatures of 15–25°C. It has been suggested that some strains do not grow readily on TSA.

Many investigators have reported that routine bacteriological examination of kidney samples may fail to detect *A. salmonicida* in carrier fish. Pre-incubation of pathological material for 24–48 hours in tryptone-soya broth followed by the use of Coomassie Brilliant agar improves the rate of recovery (Cipriano et al 1996, Daly and Stevenson 1985). It has been suggested that

enrichment of samples in tryptone-soy broth enhances detection (Daly and Stevenson 1985), but subsequent experiments do not support this. The spleen and heart should also be cultured as these organs were most commonly infected in studies on furunculosis in brown trout (Daly and Stevenson 1985).

Bacteria isolated in culture can be identified rapidly by serological procedures such as fluorescent antibody test (FAT). Serological methods (such as enzyme-linked immunosorbent assay (ELISA) and latex agglutination) may be used on pure and mixed cultures and with pathological material to confirm the presence of *A. salmonicida*. However, there are serologically distinct strains of *A. salmonicida*. For example, in a study of hatcheries in the USA, each of the four types of epidemics recorded in a year was caused by a serologically distinct strain of *A. salmonicida* (Klontz and Wood 1972).

DNA probes can be used to detect *A. salmonicida* in samples of clinical and environmental material. Polymerase chain reaction (PCR) using specific DNA probes has been used to detect *A. salmonicida* in effluent, water, faeces and sediment from a farm in Ireland (O'Brien et al 1994 and Mooney et al 1995 from Austin 1997). In contrast, culture isolated *A. salmonicida* only from clinically diseased fish (Austin 1997).

ELISA and PCR have detected *A. salmonicida* when culture methods did not detect the presence of bacteria (Austin 1997). It is difficult to distinguish cells that have entered the 'viable but non culturable' (VBNC) state from cells that are non-viable and non-infectious (Morgan et al 1992). Cells that are viable but non-culturable may have pathological significance (Austin 1997). Methods that detect bacterial DNA or antigen do not provide information about the viability/infectivity of the bacteria.

The detection of *A. salmonicida* in unapparently infected fish and in the aquatic environment is difficult, even when an outbreak of furunculosis is occurring. Latent furunculosis may be diagnosed using FAT in combination with culture of intestinal material and kidney (Shotts 1994). [Detection rates in carrier populations may be improved by the use of 'stress tests' (Munro and Hastings 1993). However, other studies have suggested that ELISA may be a viable alternative to the stress test for the detection of fish populations that contain carriers of furunculosis (Rose et al 1989).

A study by Dalsgaard et al (1994) reported that 130 strains of typical *A. salmonicida* isolated in Denmark, Norway, Scotland, Canada and the United States were consistent in general culture and biochemical characteristics. While antibiograms could be used as epidemiological markers, typical strains of *A. salmonicida* show little variation in biochemical and antigenic characteristics, so traditional typing methods offer little value. The plasmid profiling technique has been considered useful (Dalsgaard et al 1994).

Culture of atypical isolates tends to require a modified approach, for example, the inclusion of blood or serum in the isolation medium (Austin and Austin 1993). It has been noted that the onset of disease due to infection with atypical strains may be so rapid at temperatures greater than 10°C (particularly in Atlantic salmon parr) that screening for carriers and monitoring of mortality rates are poor predictors of an impending epizootic (Groman et al 1992).

Tissue distribution

In peracute infection, colonies occur in a number of organs with no inflammatory infiltration and only limited necrosis. In acute furunculosis, the development of furuncles is unusual, because of the rapid course of this form of disease. However, diseased fish show haemorrhagic septicaemia and skin lesions on the side or dorsal body surface. Hiney and Olivier (1999) reported that furuncles and exophthalmia were often observed in chronic infection; however, in practice furuncles may be observed rarely (EM Bernoth pers. comm.). In cases of clinical disease, the kidney usually contains the highest number of bacterial cells. Muscle lesions yielded on average fewer culturable cells per gram. However, titres up to 10^8 CFU/g have been recovered from furuncles in the muscle of Atlantic salmon (McCarthy 1977).

Despite almost 80 years of speculation, there is no certainty as to the location of typical *A. salmonicida* in covertly infected fish. It is likely that the pathogen is located externally (on the skin mucus), on the gills and in the intestine. Titres of 10^3 CFU/g were reported in the skin mucus of apparently healthy brown trout; no

organisms were isolated from the kidney (Hiney and Olivier 1999). Titres of 10^6 CFU/g in mucus were found in apparently healthy Atlantic salmon in fresh water immediately prior to the onset of disease (Cipriano et al 1992).

STABILITY OF THE DISEASE AGENT

Information in previous reports (DPIE 1996, Stone et al 1997b) indicated that *A. salmonicida* is stable for up to 28 days in kidney tissue and for up to 32 days in muscle tissues at 4°C. Freezing infected salmon flesh for 5–7 days at –20°C reduced the titre by 99%. The organism was culturable after 48 hours at 35°C, 3 hours at 40°C, 10 minutes at 45°C, 2 minutes at 50°C and was not detectable after heating to a temperature ≥55°C. *A. salmonicida* was resistant to pH 4 at 22°C.

A. salmonicida has been reported to survive in fresh water for 17 days, in brackish water for 24 days and in seawater for 8 days at 11–13°C. The pathogen may survive for up to 29 days in sediment. It has also been postulated that *A. salmonicida* can enter a dormant period and survive as a viable but non-culturable organism; however, there is little evidence as to the viability, infectivity or epidemiological significance of organisms in a VBNC state.

A. salmonicida would be capable of proliferation in the nutrient-rich environment of a dead host, given appropriate conditions of temperature (ie failure to maintain chiller temperatures) (A McVicar pers. comm.).

Susceptibility of finfish species in Australia

All salmonids present in Australia would be expected to be susceptible to infection with typical *A. salmonicida* and some atypical strains. Non-salmonid fish in fresh water are more likely to be infected with atypical *A. salmonicida* than with typical *A. salmonicida*. However, of the species that have been recorded as being infected with typical *A. salmonicida* overseas, many are members of the family Cyprinidae (including goldfish, tench, roach and carp). Infection does not normally cause serious disease in these species. The family Cyprinidae does not occur naturally in Australia, but several members have been introduced and are now widespread throughout freshwater habitats. These species are expected to be susceptible to infection with *A. salmonicida*, especially atypical strains. Given the minor pathogenic significance of *A. salmonicida* infections in non-salmonid freshwater finfish overseas, AQIS considers that the most significant aspect of the establishment of infection in non-salmonids would be the potential for these fish to serve as a reservoir of the pathogen for freshwater salmonids.

It has been suggested that the establishment of *A. salmonicida* would threaten the survival of native finfish in Australia. However, there is little evidence that Australian native fish (which are not closely related to the family Cyprinidae) would be particularly susceptible to infection with typical or atypical strains of *A. salmonicida*. A single case of disease due to the goldfish ulcer disease biovar of *A. salmonicida* was reported in native fish (silver perch) at a farm where goldfish had been infected. Further, atypical *A. salmonicida* was detected (by indirect fluorescent antibody test) but not isolated in roach with ulcerative dermatitis in a Victorian lake (cited by Whittington et al 1995). However, the presence of the goldfish ulcer disease variant of *A. salmonicida* in Australia has had no apparent effect on the status of threatened or endangered native fish on the mainland.

Very few of the marine species in which typical *A. salmonicida* has been recorded overseas occur in Australia. However, given the expanding list of susceptible hosts it is likely that some marine species present in Australia would be susceptible to infection with typical and atypical *A. salmonicida*.

Modes of transmission

A. salmonicida can spread horizontally via water, contaminated equipment and food, and contact between fish. Furunculosis may be transmitted via the entry of the pathogen into the gills, mouth, anus and/or surface injury through contact with infected fish or contaminated water (Austin 1997). Vertical transmission on the surface of eggs is theoretically possible but is not thought to be a significant route of transmission (McCarthy 1977). International regulations do not recognise any significant risk from pseudo-vertical transmission of *A. salmonicida*. The surface disinfection of eggs is normal practice in salmonid farming worldwide

and this is considered to provide sufficient health safeguard. Effendi and Austin (1995) showed that the most effective route of entry leading to mortalities in Atlantic salmon was via the gills.

The minimum infective dose for Atlantic salmon in seawater by short duration (1–3 days) bath exposure was estimated at 10^4 CFU/mL, and by long duration (3 weeks) immersion at 10^2 CFU/mL. Immersion in water containing 10^2 CFU/mL *A. salmonicida* for a period up to 1 week failed to cause infection. Intragastric intubation with a dose $>10^5$ CFU/fish established infection in Atlantic salmon (Rose et al 1989). In a lake trout hatchery, 0.1–0.01 *A. salmonicida* per millilitre in the inflow water initiated infection (DPIE 1995). A similar situation was reported in a freshwater brown trout farm (AQIS 1996). In both instances, the low dose of pathogens would have been present for a long period. Given that brown trout are very susceptible to infection, these findings would have limited, if any, application to other, more resistant species.

Appendix 8
Review of literature on infectious salmon anaemia

IN VIEW OF THE SIGNIFICANCE OF THIS DISEASE and the substantial increase in knowledge on infectious salmon anaemia (ISA) in recent years, AQIS has undertaken a review of the literature as a basis for the release, exposure and consequence assessments. The review covers information in the report of the New Zealand Government (Stone et al 1997b) and more recently published literature.

DESCRIPTION OF DISEASE / DISEASE AGENT

ISA is a disease affecting farmed Atlantic salmon in Norway, Canada and Scotland. It causes high mortalities and significant economic loss (Getchell 1997, Hastein 1997, Bricknell et al 1998). The implementation of effective management strategies may significantly lessen the impact of disease (B Hill pers. comm.). ISA is listed by the Office International des Epizooties (World Organisation for Animal Health, OIE) as an 'other significant' disease.

Disease due to ISA is characterised by lethargy, severe anaemia, leukopaenia, congestion of the liver, spleen and foregut, haemorrhagic necrosis of the liver, petechial haemorrhage of the viscera and ascites (Thorund and Djupvik 1988, Evensen et al 1991). The disease is transmitted horizontally and spreads relatively slowly (Thorund and Djupvik 1988). Vertical transmission has not been demonstrated (Hastein 1997). The causative virus, ISAV, may be shed via skin mucus, faeces and urine. It has been suggested that the most likely routes of entry are the gills and skin lesions (Totland et al 1996). Infection has also been transmitted via a homogenate of liver, kidney, spleen and blood plasma (Dannevig et al 1994).

To date there has been no effective method of treatment for ISA. However, in advice to the Tasmanian Salmonid Growers Association (TSGA 1999), A Munro (pers. comm.) stated that nearly all smolts going to sea this year in Canada are being vaccinated. The efficacy of vaccination in the field remains to be proven; however, there is good experimental evidence to suggest that vaccination may provide some protection. Jones (1999) demonstrated that fish vaccinated with ISAV antigen in an oil emulsion showed increased survival when challenged with ISAV. Mortality rates for fish treated with

the most effective vaccine preparations were 1.4% and 4.8%, in contrast to mortality rates of 72% and 85.7% respectively in fish injected with saline.

Munro (cited by TSGA 1999) further advised that the widespread use of vaccination could increase the number of apparently healthy fish carrying ISAV. In a personal communication to AQIS, A McVicar stated that European Union legislation bans vaccination against listed diseases (including ISA), based on concern that vaccinated fish will become carriers of infection. However, in a personal communication to AQIS, E-M Bernoth advised that EU restrictions on vaccination related to approved zones and approved farms in non-approved zones. Moreover, the rationale for this restriction relates more to the potential to confuse differentiation between infected and vaccinated fish in surveillance programs rather than to concern at the risk of fish becoming carriers (which would not be affected by the use of inactivated vaccines).

Management controls, including prohibition of the movement of live fish between regions and eliminating the use of untreated seawater in hatcheries, have significantly reduced the number of new outbreaks in Norway (Binde 1997). Other management controls include the adoption of protocols for the treatment of wastewater from processing plants. Fallowing of infected areas and implementation of movement restrictions on fish and equipment have been used with success in Canada to prevent the spread of ISA (Anon 1998). Similar methods of control and management have been put in place in Scotland.

Although ISAV has not yet been fully classified, the morphological, functional and genomic properties of the virus are consistent with those of the Orthomyxiviridae (Falk et al 1998). Blake et al (1999) believe that, based on virion morphology and the number and size of genome segments, ISAV appears to resemble members of the Orthomyxiviridae.

Geographical distribution of ISA

ISAV has a limited geographical distribution. It has been recorded in Norway, Canada and Scotland (Getchell 1997, Hastein 1997, Bricknell et al 1998). ISAV is listed by the OIE and is the subject of EU directives, with which Norway complies. This pathogen is the subject of concern and a focus of scientific research, surveillance and monitoring in the countries of Europe and North America that have a significant farmed Atlantic salmon industry.

NORWAY

ISA was first reported in a Norwegian hatchery in 1984 in association with increased mortality in Atlantic salmon parr. The outbreak lasted for several months, with an increasing mortality during 1984 and spring 1985 resulting in the death of approximately 80% of the parr in the hatchery (Hastein 1997). In 1986–87 the disease was reported in a fish farm with hatchery, brood fish and grow-out fish. Clinical disease and extensive mortality occurred in several year-classes of fish. The disease spread rapidly and became endemic in many regions of Norway (Hastein 1997).

During the 1980s and early 1990s there was an exponential rise in the incidence of disease, with the number of newly affected farms increasing to 101 in 1990 (Jarp and Karlsen 1997). Later, the annual incidence of disease decreased to a minimum of one in 1994. The occurrence of two and five new cases in 1995 and 1996, respectively, shows that the disease has not been eradicated in Norway (Hastein 1997).

In 1998, 13–15 outbreaks and/or new infections occurred with wide distribution in Norway. Given that most salmon farming areas in Norway have at some time been affected by ISA it is evident that the 1998 outbreaks were in areas previously affected. What is more significant is that the distribution of sites affected covered most of the country and included areas where there had been no recorded outbreaks for several years. This could suggest that the origin of some was not through proximity to other cases but as a consequence of another source, for example a natural or established local reservoir (A McVicar pers. comm.). The origin of ISAV in Norway has not been determined (A Munro cited by TSGA 1999).

There is evidence of seasonal variation in the number of outbreaks of ISA in Norway, with the peak incidence occurring in May (spring) to July (early summer) and a further minor peak in November (Dannevig and Thorud 1998).

SCOTLAND

ISA was first reported in Scotland in May 1998. From this initial infected site, the disease was spread to other farms on the Scottish west coast, Skye and the Shetland Isles. By June 1999, 11 sites had been confirmed as being infected, with a further 18 suspected as being infected.

Under European Commission regulations (93/53/EEC) and United Kingdom legislation (*The Diseases of Fish (Control) Regulations 1994*), a farm is placed under suspicion of infection with ISA when fish show clinical signs or postmortem lesions, or laboratory results provide evidence of infection. A farm is considered to be infected when fish show clinical and postmortem signs of ISA. Laboratory results should be used to support this diagnosis. The same disease containment measures are placed on ISA-infected or suspect farms except that all stocks in the confirmed ISA-infected farm are removed as quickly as practically possible. All farms in the area surrounding infected or suspect farms are subject to rigorous conditions of containment (A McVicar pers. comm.).

Of the 18 suspect farms, none have progressed to confirmed infected status (for a period of up to 11 months for two of the farms), although some farmers have chosen to harvest stocks early and approximately half are now fallow. The latest farm designated as suspect in Loch Broom is outside the previous affected area and has increased the area under control and surveillance (A McVicar pers. comm.).

A McVicar (pers. comm.) stated that in Scotland investigations into the source of ISA include the consideration of the following hypotheses: that there is a natural reservoir of infection in coastal waters of Scotland or that illegally imported infected fish or contaminated equipment was the source of infection. There is evidence that ISA spread from the single point source through one or more of the following routes:

- transfer of live fish between sea sites for growing and harvest;
- activities of divers in Loch Nevis removing dead fish without adequate disinfection;
- use of equipment (boats, graders etc) on different farms owned by one company;
- use of 'bus stop' deliveries of smolts where a well boat visited one site subsequently shown to be infected, discharged a part load of smolts and delivered the remaining smolts to another site;
- discharge of untreated effluent from factories processing salmon from infected farms;
- via water, from an infected farm to adjacent farms.

These risk factors are similar to those identified in Norway and Canada.

The primary outbreak in Scotland was in a remote area with no salmonid processing plants. All subsequent cases were associated with poor hygiene practices and movements between farms, processing units or farms in close proximity to each other. A McVicar (pers. comm.) considers that until such time as wild populations of salmonid and non-salmonid fish are shown to be carriers of infection, the role of wild fish as a reservoir of infection remains to be established.

CANADA

ISAV was first diagnosed in Canada in 1997 (Getchell 1997). The condition described as haemorrhagic kidney syndrome in 1996 was subsequently shown to be due to infection with ISAV (Lovely et al 1999). By the time this was recognised, ISA had spread to 80 cages at 16 sites (Anon 1998).

The first outbreak in New Brunswick (Bay of Fundy) is spreading towards the border of the United States. A second outbreak recently occurred in broodfish in the north of Nova Scotia. The origin of ISAV in Canada has not been determined (A Munro cited in TSGA 1999).

UNITED STATES OF AMERICA

In a personal communication to AQIS, L Chaves (National Marine Fisheries Service) advised that United States authorities have implemented a management program to prevent the entry of ISAV into Maine (the state closest to the ISA outbreaks in Canada). A surveillance program has been set up to confirm the effectiveness of management controls. Surveillance of 21 marine net-pen

sites in Maine during 1998, using virus isolation on CHSE-214 and SHK cell lines, ISA-specific indirect fluorescent antibody test (IFAT) and reverse transcriptase polymerase chain reaction (RT-PCR), has not reported the presence of ISAV in Maine.

CHILE

ISA has not been reported in Chile.

AUSTRALIA AND NEW ZEALAND

None of the signs recommended by OIE as necessary findings for the diagnosis of ISA have been observed in listed susceptible species, ie Atlantic salmon, in Australia or New Zealand.

Relationship between strains of ISAV in different geographic regions

Current data indicate that the New Brunswick isolate differs considerably from the Nova Scotian and Norwegian isolates but that the latter two are closely related. The Scottish isolates are similar to the Norwegian isolate but show some consistent differences. A. Munro (cited in TSGA 1999) stated that the considerable difference in sequence homology between the New Brunswick and the Norwegian and Nova Scotian strains ruled out any possibility of transfer of virus, for example via trade in carcases, and suggested that there might be a local reservoir of infection. Munro further noted that the Nova Scotian strain was probably of native origin, but until a wild fish species was identified as a reservoir of infection there remained the possibility that ISAV had been introduced into Canada.

HOST RANGE AND PREVALENCE

ISAV has been reported only in Atlantic salmon exposed to seawater (Nylund 1997), except for a single instance in juvenile Atlantic salmon in fresh water, where the route of infection was not determined (Nylund et al 1999). Under natural conditions, Atlantic salmon may display clinical and subclinical infection.

Under experimental conditions, infection was induced in brown trout and rainbow trout via intraperitoneal inoculation (Totland et al 1996, Nylund et al 1997). Experimentally infected brown trout did not develop clinical signs of disease or mortality, but ISAV was present in these fish at up to 7 months post-challenge (Nylund et al 1995). Rolland and Nylund (1998) found that brown trout that had cohabited with infected Atlantic salmon (based on gross lesions in 'indicator' Atlantic salmon injected with material form the trout) apparently became infected with ISA.

There is no report of ISAV causing clinical disease or significant mortality in experimentally infected rainbow trout (Nylund et al 1997).

Hjeltnes (1993) [challenged wrasse (*Ctenolabrus rupestris*, *Ctenolabrus exoletus*), turbot (*Scophthalmus maximus*), and charr (*Salvelinus alpinus*) with ISAV-infected material. None of the challenged fish developed disease or became infected with the virus. Similarly, Thorud and Torgensen (1994) could not demonstrate the presence of ISAV in challenged sea bass (*Dicentrarchus labrax*). The herring (*Clupea harengus*) is being tested for susceptibility to infection with ISAV (Nylund 1997). The OIE states that the ISA agent has not been shown to survive in turbot (*Psetta maxima*), ballan wrasse (*Labrus berggylta*), sea bass (*Dicentrarchus labrax*) or cod (*Gadus morhua*).

A Munro (cited in TSGA 1999) and Nylund (1997) suggested that the emergence of ISA (including unrelated strains of virus) in many countries and the wide distribution of outbreaks in Norway may be explained by the hypothesis that fish in coastal waters are natural reservoirs of the virus. Endemically infected wild populations could have a higher prevalence of infection as a consequence of outbreaks in farmed fish.

A McVicar (pers. comm.) stated that the appearance of ISA in Norway in the early 1980s and the subsequent recurrence every year since, particularly in areas without close connection to existing outbreaks, indicates that there is a natural occurrence or established reservoir of ISA in that country. Similar speculation has been made for Canada where no contacts can be established with

Norway and the strain of virus present in New Brunswick shows major differences.

A McVicar (pers. comm.) further stated that no evidence has been presented in Canada or Norway for the source of ISAV, although speculation has focused on the role of sea trout and herring.

Detection methods

Until recently, diagnosis was based on clinical and pathological signs and haematological findings. In the absence of more sensitive and specific tests, Norwegian authorities and industry have successfully used these diagnostic methods to reduce the number of ISA outbreaks (Binde 1997). While this approach is satisfactory for diagnosis of ISA in an outbreak, it has limitations in the diagnosis of carriers and of subacute and chronic cases of ISA (Dannevig and Thorud 1998).

The establishment of a new Atlantic salmon head kidney cell line and the subsequent isolation of ISAV provided for the development of an integrated diagnostic method (Dannevig et al 1995, A McVicar pers. comm.). Because of the technical difficulties associated with the method (eg long incubation time) and lack of sensitivity, it is not routinely used in ISA screening, but ideally should be integrated, particularly in the diagnosis of primary outbreaks. Isolation in cell culture remains the gold standard for detection of ISAV (A McVicar pers. comm.). Growth of ISAV (from Atlantic salmon in New Brunswick) in CHSE-214 cell cultures was recently reported (Bouchard et al 1999).

A fluorescent antibody test (FAT) was the first established diagnostic method for ISAV. Although time-consuming and cumbersome, this method has proven to be useful and reliable for the detection of ISAV in diseased fish (Falk 1997). It has greater sensitivity than clinical and pathological evaluation, providing for earlier diagnosis and the detection of ISAV in fish with diffuse symptoms or multiple infection (Falk 1997). New, more specific and more sensitive methods including IFAT (Falk et al 1998) and RT-PCR (Lovely 1999)) allow scientists to detect ISAV in salmon in the absence of clinical disease. The IFAT and RT-PCR have been shown to be robust in the hands of Scottish diagnosticians (A McVicar pers. comm.).

The OIE recommends that macroscopic, histological and haematological findings (now supported by the laboratory diagnostic tests) be used in the diagnosis of ISA.

Distribution of virus in, and infectivity of, tissues

Nylund et al (1994) described ISA as a multi-organ disease. By transmission electron microscopy (TEM), the virus was shown to be present in the integument, kidney, urinary bladder, gut, somatic muscle, and many hormone-producing tissues including the pituitary gland, thymus, thyroidea and gonad. The virus has also been detected in the liver, spleen, erythrocytes, leukocytes, epidermal mucus, gut contents, urine, faeces and gills (Dannevig et al 1994, Hjeltnes et al 1994, Nylund et al 1994, Totland et al 1996).

Torgersen (1997) reported that viscera and trimmings from the slaughter process, including from apparently healthy fish, were highly contagious. Muscle was less infective than internal organs and material from the head. This author stated that salmon from farms where ISA has been diagnosed should be considered as infective and contagious, even if the fish do not show clinical or macroscopic signs of disease. While most attention must be paid to bleeding and slaughtering, further processing of eviscerated fish, filets (sic) and handling of offal from the slaughter and processing plant must be considered a risk for spreading the infectious agent. In commenting on this report, A McVicar (pers. comm.) noted that most of the risk is removed by the bleeding and primary processing and that the EU (including the UK) would have taken this into account when placing restrictions on uneviscerated Norwegian salmon.

Stability of disease agent

Virus replication in salmon head kidney cells was observed at a temperature range of 10–15°C. At 20°C the production of infective virus was reduced by more than 99%. No virus replication was detected at 25°C (Falk et al 1997).

Torgersen (1997) treated infective material with chemical disinfectants and physical treatments, and tested infectivity by means of transmission trials. ISAV lost

infectivity at temperatures ≥50°C for 2 minutes, formic acid (≤pH 4.0) for 8 hours, sodium hydroxide (at pH 11.5 for 48 hours or pH 12.0 for 24 hours), sodium hypochlorite (100 mg/L for 15 minutes) and UV doses ≥4 mJ/cm^2. Similarly, Falk et al (1997) found that ISAV was sensitive to treatment with chloroform, heat and low pH. Complete inactivation of ISAV infectivity was observed after 5 minutes at 56°C, while 80% of the infectivity was lost after 6hours at 37°C. The virus was stable at pH 5, 7 and 9; however, infectivity was reduced by more than 90% at pH 11. Complete inactivation was demonstrated at pH 4 for 30 minutes. Five cycles of freezing at –80°C followed by thawing at 20°C or sonic disruption at 50–60 watts for 90 seconds did not reduce infectivity.

A study by Nylund et al (1994) showed that ISAV maintained infectivity after 20 hours in seawater, but there is a reduction in infectivity after 24 to 48 hours (A Munro cited in TSGA 1999) and 4 days in blood and kidney tissue at 6°C.

There have been no published reports on the efficacy of iodophores, the most commonly used group disinfectant in fish farms (A McVicar pers. comm.).

Munro (cited in TSGA 1999) reported that storage of muscle, head and visceral tissues on ice resulted in an initial increase in virus infectivity after 3 days, followed by a decrease after an additional 3 days (6 days in total). This could be explained by assuming that in the initial period the tissue decomposed, releasing virus, but after 6 days the virus was inactivated. From the scientific literature it can be concluded that ISAV survives in tissue stored at –20 to –30°C. Munro advised that on current information, it is not possible to distinguish the risk posed by fresh chilled and frozen carcases.

Susceptibility of host species in Australia

Under natural conditions, Atlantic salmon is the only species that is susceptible to infection with ISAV. Brown trout and rainbow trout have been shown to be susceptible to infection experimentally. These salmonid species occur in Australia.

Modes of transmission

Horizontal transmission has been demonstrated in cohabitation experiments, indicating that water-borne transmission is effective for the spread of ISAV (Thorud and Djupvik 1988).

Totland et al (1996) suggested that clinically and subclinically infected fish may shed ISAV (most likely through the urine and faeces). These workers showed that short-term exposure of healthy Atlantic salmon smolts to ISA-inoculated cohort smolts led to near 100% mortality. Skin mucus, faeces, urine and blood samples from ISA-inoculated fish transmitted infection to healthy cohort smolt with variable efficiency. All sources were infectious and resulted in viral transmission via intraperitoneal inoculation. Administration of skin mucus to the gills transmitted infection as efficiently as intraperitoneal injection. Introduction of infective inocula into the stomach did not result in transmission of ISAV and these authors concluded that gastrointestinal passage rendered ISAV non-infective. However, Rolland and Nylund (1998) demonstrated transmission of ISAV via the introduction of infected faeces into the gastrointestinal tract. These authors also induced 100% mortality in Atlantic salmon smolts by intraperitoneal injection of 0.04 mL of mucus from ISA-infected fish. Hjeltnes et al (1994) also demonstrated the highly infectious nature of natural excretions and secretions from ISA-infected fish.

Totland et al (1996) suggested that ISAV was more likely to be absorbed by the mucus from the surrounding water than to be secreted or produced in the skin.

Experimental studies have demonstrated the infectivity of preparations of liver, kidney, spleen, plasma, erythrocytes and head kidney leukocytes inoculated into salmon parr. Generally, the infectivity of kidney was higher than spleen, head kidney leukocytes, erythrocytes and plasma. On a per-gram basis, head kidney leukocytes contained more infectious material than erythrocytes (Dannevig et al 1994).

The most likely portal of entry for ISAV is via the gills. Totland et al (1996) demonstrated that the viral particles

initially appeared in the pillar cells of the gills. Other thin organs such as the olfactory or lateral line organs may also be sites of infection.

Factors influencing the spread of disease

A study by Vagsholm et al (1992) showed that the chances of a farm in Norway becoming infected with ISAV was 13 times higher if the number of ISAV-infected sites within less than 5 kilometres was increased from one to six. The risk also increased with increasing numbers of slaughterhouses/production sites less than 5 kilometres from a farm. A study (also of Norway) by Jarp and Karlsen (1997) demonstrated similar risk factors. These reports suggest that untreated wastewater from slaughterhouses or production sites may transmit infection to susceptible fish.

A retrospective analysis of outbreaks of ISA in Norway concluded that ISAV may spread a maximum distance of 5–6 kilometres via seawater. Eide (1992) reported that transmission over this distance took 6–12 months.

While vertical infection of ISAV is not thought to occur, Nylund et al (1999) reported an outbreak of ISA in first-feeding Atlantic salmon fry. As seawater was not used in the hatchery (according to the farmer), these authors suggested that there may be natural reservoirs of ISAV in fresh water, or that infection may be transmitted vertically. It is also possible that ISAV entered the hatchery via a route yet to be discovered.

ISA-related restrictions on international trade

Restrictions imposed by the United States, France, Spain and Italy on Norway in relation to the importation of salmonids for human consumption were subsequently lifted in relation to eviscerated fish. Restrictions on the importation of uneviscerated salmon from Norway have been harmonised in the EU for about 6 years via a series of European Commission decisions. In accordance with these decisions, EU Member States prohibit the importation from Norway of uneviscerated fish except from farms on the south coast. Decision 98/450/EC lists the individual farms from which importation of dead, non-eviscerated salmon may be dispatched to the European Community.

Appendix 9

Estimating the disease risk associated with bait used by the Western Australian rock lobster industry

IMPORTATION OF FISH, PARTICULARLY WHOLE FISH, for use as bait in lobster pots is considered to pose a higher risk of introducing disease to domestic fish stocks, than the import of fish for human consumption[1]. However, the quantification of this risk is somewhat problematic, because of the relatively unknown status of fish imported for bait. Some retrospective data is available, and has been used in the evaluation of risk.

The Western Australian Rock Lobster Industry has been importing bait since 1976, and records of bait use are available back to the1964–65 season. In 1997, the Western Australian Fishing Industry Council conducted a risk assessment of frozen baitfish[2]. In that assessment, a deterministic risk model was developed using beta distributions, and the number of years for which zero disease events had been reported. The beta distribution model was also extended to include an estimate of disease frequency from 'pot lifts' or the number of times baited pots were placed into the ocean. Data were available which indicated that 47% of all pot lifts used imported bait. This model resulted in the expectation that the risk of a disease event over all seasons was in the order of 7.54×10^{-9}, with an upper 95% interval of 4.01×10^{-8}. These risks are exceedingly small, and would accord with most assessments of 'acceptable'. The assumptions behind the risk assessment are clearly stated[3].

The data presented in the baitfish risk assessment also allows an extension of the model, to compute in a stochastic manner, the most likely number of disease events, for any given risk estimate. This brief report documents these computations.

Method

Data presented in the baitfish document were transferred to a computer spreadsheet (Microsoft Excel) to which the risk package, @Risk (Palisade Corp.) had been added. Because @Risk has upper limits to the size

1 Humphrey, JD (1995) *Australian Quarantine Policies and Practices for Aquatic Animals and their Products: a review for the Scientific Working Party on Aquatic Animal Quarantine*. Bureau of Resource Sciences, Canberra. Part 1: Review and Annexes, p123

2 Jones, JB and Gibson AP (1997) *Risk Analysis for the Practice of Importing Frozen Fish as Bait*. Western Australian Fishing Industry Council (Inc.) 188pp.

3 Jones and Gibson Op Cit. p33

of numbers it will handle, data were expressed in pot-lifts per day. From the baitfish document, the number of pot lifts using imported bait were calculated. These data were then used to compute beta distributions through the @Risk package which expressed the risk of a disease event on a daily basis for each season. This procedure for computation of risk has been well recognised[4]. It should be noted that computation using the beta distribution on a daily basis results in an overestimate of the risk, by a factor equating to the number of days for the particular season (about 250). The impact of this overestimate was compared with a more realistic risk figure, computed by dividing by the number of days in each season. Assumptions for the risk model were those presented in the baitfish document.

This risk, expressed as a decimal, was then used as the probability parameter for a binomial distribution, which estimated the likely number of disease events for each fishing season. When totalled over all the years for which data were available, this number represents the expected number of disease outbreaks.

Results

The model was run for 1000 iterations. The mean and median for total expected disease events over 32 years was 0 (zero). The upper 95% confidence interval was 1 disease event in 32 years. When this overestimate was corrected for the number of days in the season, the mean and median remain the same, but the upper 95% confidence interval is 0 (zero).

Comments

These outputs agree closely with those of the published baitfish model. The use of retrospective data in this way results in a non-zero risk, and hence a possible non-zero number of disease events. The model suggests that with the overestimated prevalence calculations, one disease event would be expected (95% confidence limit), over the 32 years for which data were available, assuming 47% of all bait used were imported. However, as stated elsewhere, there have been no indications of significant exotic disease events in the waters of Western Australia associated with the rock lobster industry[5].

In the face of recognised risks, for which a probability can be calculated, the likelihood of actual disease events occurring is small. As pointed out in the baitfish document, the fact that bait was imported from 1976 onwards provides a 'before and after' comparison, in which the actual risks of imported bait could be assessed. There were no reported disease events (such as large-scale fish kills) in either period, although there is the computed possibility that such an event could have occurred.

Questioning why a disease event did not occur, although speculative, may be useful. The freezing-thawing of imported bait may reduce pathogens to sufficiently low levels to prevent infective doses being released; the small size of baits (about 2 kg) similarly reduces the infective dose; pots are spread over a wide area, thereby reducing the likelihood of sufficient infectious material being available at any one site; the water into which baits are placed is relatively warm, compared to that from which the bait fish originated, thereby reducing the likelihood of survival of disease agents; appropriate intermediate hosts are not present in local waters; appropriate definitive hosts are not present in local waters.

For whatever reasons that there have been no reported significant fish disease events in WA waters associated with the rock lobster industry, it is apparent that the risk of fish disease events associated with imported rock lobster bait is small, and that the process through which imported bait passes probably constitutes a sufficient risk mitigation activity.

Based on this analysis of historical data, there would seem to be little urgency in taking action regarding the importation of rock lobster bait, either in preventing its import, or requiring further risk mitigation activities.

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4 Vose, D (1996) Quantitative Risk Analysis. A Guide to Monte Carlo Simulation Modelling. Wiley and Sons, Chichester. Pp138-139

5 Jones and Gibson Op Cit. p41

	YEAR	BAIT FISH ZONE A	ZONE B	POT LIFT MODEL (2) BIG BANK	ZONE C	DAYS	DATA FROM WAFIC RA 1997 TOTAL POT LIFTS	POT LIFTS PER DAY	POT LIFTS PER DAY WITH IMPORTED BAIT	REPORTED DISEASE INCIDENTS	BETA DISTRIBUTION EXPECTED DAILY DISEASE RISK (OVERESTIMATE)	BINOMIAL DISTRIBUTION EXPECTED EVENTS PER SEASON	
	62/63	n/avail.	n/avail.	0	n/avail.	274							
	63/64	n/avail.	n/avail.	0	n/avail.	275							
1	64/65	1154317	2558023	0	3049301	274	6761641	24677	0	0	0	0	
2	65/66	1333434	2558094	0	3359116	274	7250644	26462	0	0	0	0	
3	66/67	1200563	2636874	0	3592251	274	7429688	27115	0	0	0	0	
4	67/68	1279365	2597806	0	3971296	275	7848467	28539	0	0	0	0	
5	68/69	1326099	3283959	0	4201186	274	8811244	32157	0	0	0	0	
6	69/70	1385875	2880000	0	4091916	274	8357791	30502	0	0	0	0	
7	70/71	1436663	3217567	0	4884292	274	9538522	34812	0	0	0	0	Bait was not imported prior to 1976
8	71/72	1443842	3629073	0	4832934	274	9905849	36152	0	0	0	0	
9	72/73	1547694	3310286	0	4182665	273	9040645	33115	0	0	0	0	
10	73/74	1462705	3365655	0	4985892	273	9814252	35949	0	0	0	0	
11	74/75	1518702	3359611	0	5327167	273	10205480	37382	0	0	0	0	
12	75/76	1442896	3417007	0	5292204	274	10152107	37051	17410	0	5.74317E-05	0	
13	76/77	1592239	3552353	0	5666431	273	10811023	39600	18610	0	5.37288E-05	0	
14	77/78	1379772	3535754	0	5628575	228	10544101	46246	21730	0	4.60151E-05	0	
15	78/79	1380315	3569197	0	5842612	228	10792124	47333	22250	0	4.49398E-05	0	
16	79/80	1347669	3558899	0	5767541	229	10674109	46611	21910	0	4.56371E-05	0	
17	80/81	1303834	3648627	0	5894115	228	10846576	47572	22360	0	4.47187E-05	0	
18	81/82	1367531	3569114	0	6268767	228	11205412	49146	23100	0	4.32863E-05	0	
19	82/83	1337017	3977877	0	6260396	228	11575290	50768	23860	0	4.19076E-05	0	
20	83/84	1318466	3984080	0	5861877	229	11164423	48752	22910	0	4.36453E-05	0	
21	84/85	1313124	4045841	0	6192501	228	11551466	50664	23810	0	4.19956E-05	0	
22	85/86	1242770	4171829	0	5367380	228	10781979	47289	22220	0	4.50005E-05	0	
23	86/87	1424471	4180329	179856	5567899	228	11352555	49791	23400	0	4.27314E-05	0	
24	87/88	1457533	4738872	191667	6495961	229	12884033	56262	26440	0	3.78186E-05	0	
25	88/89	1378517	4536616	145833	6257987	228	12318953	54030	25390	0	3.93825E-05	0	
26	89/90	1454945	4572770	66667	5998356	228	12092738	53038	24920	0	4.01252E-05	0	

continued...

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	YEAR	BAIT FISH ZONE A	ZONE B	BIG BANK	POT LIFT MODEL (2) ZONE C	DAYS	DATA FROM WAFIC RA 1997 TOTAL POT LIFTS	POT LIFTS PER DAY	POT LIFTS PER DAY WITH IMPORTED BAIT	REPORTED DISEASE INCIDENTS	BETA DISTRIBUTION EXPECTED DAILY DISEASE RISK (OVERESTIMATE)	BINOMIAL DISTRIBUTION EXPECTED EVENTS PER SEASON
1	64/65	1154317	2558023	0	3049301	274	6761641	24677	0	0	0	0
27	90/91	1461876	4315049	153846	6094705	228	12025476	52743	24790	0	4.03356E-05	0
28	91/92	1475885	4495181	142857	6579266	229	12693189	55428	26050	0	3.83848E-05	0
29	92/93	1495387	3726270	168844	6343611	228	11734112	51465	24190	0	4.1336E-05	0
30	93/94	1199906	3613149	192661	5250917	228	10256633	44985	21140	0	4.72992E-05	0
31	94/95	1141737	3765686	435393	5213649	228	10556465	46300	21760	0	4.59517E-05	0
32	95/96	1219619	3771803	124658	5347169	229	10463249	45691	21470	0	4.65723E-05	0
											32-year expectation	0

Glossary of terms

Appropriate level of protection (ALOP)	Annex A of the SPS Agreement states that the appropriate level of protection is the level of protection deemed appropriate by the Member establishing a sanitary or phytosanitary measure to protect human, animal or plant life or health within its territory. Note: many Members refer to this concept as the 'acceptable level of risk'.
Aquatic Code	The OIE International Aquatic Animal Health Code, 1997
Biodiversity	A measure of the variety of the Earth's animal, plant and microbial species; of genetic differences within species and of the ecosystems that support those species.
Biofilm	A thin film of bacteria that forms on a surface and is difficult to remove.
Biological oxygen demand	The amount of organic pollution in the water, measured as the amount of oxygen taken up from a sample containing a known amount of oxygen kept at 20°C for five days. A low BOD indicates little pollution while a high BOD indicates increased activity of heterotrophic microorganisms and thus heavy pollution
Carrier fish	An apparently healthy fish that is infected with a pathogenic agent and capable of transmitting infection to another individual.
Commensal	An organism, usually the one that benefits, in a commensalism. Hence, commensal bacteria are those that live within another animal species without normally causing disease.
Commensalism	The association between two organisms of different species that live together and share nutrient resources, one species benefiting and the other being unharmed by the association.
Competent authority	The National Veterinary Services or other authority of a country having the responsibility and competence for aquatic animal health measures within the country and for export certification.
Consequence assessment	An assessment of the adverse consequences that would result from the establishment of a disease in a previously free country.
Endemic disease	A disease that is present within a defined region or country.
Evisceration	Removal of the viscera (does not include brain and gills).
Exotic disease	A disease that is not present within a defined region or country.
Export certification	Official certification that accompanies goods in international trade.

Exposure assessment	An assessment of the probability of susceptible hosts being exposed to pathogens in a dose sufficient to cause infection.
Finfish	Members of the Teleostomi, which includes the bony fishes. It does not include sharks, rays or invertebrates.
Fish product	Non-viable fish or parts of fish.
Fry (salmonid)	The salmonid lifecycle stage between hatching and parr.
Grading	A classification of product according to defined criteria. 'A' grade or 1st class is normally the product of highest quality.
Hazard	In the context of this import risk analysis, a hazard is a biological agent that may have an adverse effect.
Hazard identification	In the context of this import risk analysis, hazard identification is the process of identifying the biological agents that could be carried by the commodity being considered in the risk analysis.
Host	Species that the pathogen of interest can infect.
Idiopathic diseases	Diseases for which the aetiology has not been defined.
Import risk analysis	The process through which quarantine policy is developed or reviewed, incorporating risk assessment, risk management and risk communication.
Incidence	The number of new cases of a disease that occur in a population at risk in a particular geographical area within a defined period of time.
Index case	The first case of infection in a population previously free of the disease agent.
ID_{50}	The median infective dose of a pathogen (ie the dose at which 50% of the test units become infected).
Juvenile fish (salmonid)	A fish that weighs less than 200g in eviscerated, head-off presentation.
Metazoan	A phylum of multicellular animals with cells organised into tissues and possessing nervous tissue.
Health surveillance and monitoring system	Systematic process of investigating the health status of a given population.
Native species	Species that originated in Australia (ie not introduced).
Non-salmonid marine finfish	In this import risk analysis, this includes finfish, except salmonids, that are caught or cultured in brackish or marine waters.
Non-viable	Dead; incapable of propagation.

Notifiable diseases (OIE)	The list of transmissible diseases that are considered to be of socioeconomic and/or public health importance within countries and that are significant in the international trade of aquatic animals and aquatic animal products. Diseases notifiable to the OIE were previously known as listed diseases.
Pathogen	An organism that causes disease.
Parr (salmonid)	The freshwater stage of the salmonid lifecycle (before transfer to sea).
Sanitary (quarantine) measure	A measure used to prevent the establishment of pests and diseases.
Prevalence	The total number of cases or outbreaks of disease that are present in a population at risk, in a particular geographical area, in a specified time period.
Protozoan	A phylum of unicellular heterotrophic, generally non-photosynthetic, eukaryotes, lacking cell walls. Protozoans are often now classified with algae and other simple eukaryotes in a separate kingdom, Protista.
Quarantine risk	The combination of the probability and the consequences of establishment of a new disease or pest in Australia.
Regionalisation	The recognition of a part of a country or countries having a different pest or disease status, due to epidemiological reasons or because of sanitary controls.
Release assessment	An assessment of the probability of viable pathogens being present in the commodity at the time of entry into a country.
Risk assessment	The processes of identifying and estimating the risks associated with the importation of a commodity and evaluating the consequences of taking those risks (OIE International Animal Health Code).
Risk management	The identification, documentation and implementation of the measures that can be applied to reduce the risks and their consequences (OIE International Animal Health Code).
$TCID_{50}$	A measure of infectivity for viruses, ie the dose at which 50% of tissue cultures become infected and show degeneration
Salmonid fish (salmonids)	Species of finfish that belong to the families Salmonidae and Plecoglossidae.
Sexually mature fish (spawner) (salmonid)	Fish in milt or in spawn (ie with developed gonads).
Smolt	The stage of the salmonid lifecycle that immediately precedes transfer to sea.
Spawners	see sexually mature fish
SPS Agreement	The WTO Agreement on the Application of Sanitary and Phytosanitary Measures.

Unrestricted risk estimate	An estimate of the risk associated with the importation of a commodity in the absence of quarantine measures.
Wild-caught fish	Fish that are captured in a natural environment (ie not maintained in an enclosure before slaughter)
Whole, round fish	Fish that are whole (ie viscera and all other organs intact).
Zoning	*see* Regionalisation.

Abbreviations and acronyms

AAHL	Australian Animal Health Laboratories
ABARE	Australian Bureau of Agricultural and Resource Economics
ADVS	Aquaculture Development and Veterinary Services
AFDL	AAHL Fish Diseases Laboratory
AFFA	Agriculture, Fisheries and Forestry ó Australia
AFMA	Australian Fisheries Management Authority
AFZ	Australian fishing zone
ALOP	appropriate level of protection
AQIS	Australian Quarantine and Inspection Service
AQPM	Animal Quarantine Policy Memorandum
AQUAPLAN	Aquatic Animal Health Plan
BKD	bacterial kidney disease
BOD	biological oxygen demand
BRS	Bureau of Resource Sciences later termed the Bureau of Rural Sciences
CCEAD	Consultative Committee on Emergency Animal Diseases
CFU	colony forming units
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CVO	Chief Veterinary Officer
DMSO	dimethylsulfoxide
DNA	deoxyribonucleic acid
DPIE	Department of Primary Industries and Energy
DPIF	Department of Primary Industries and Fisheries
DPIWE	Department of Primary Industries, Water and Environment
EA	Environment Australia
EHN	epizootic haematopoietic necrosis
EHNV	epizootic haematopoietic necrosis virus
EIBS	erythrocytic inclusion body syndrome
ELISA	enzyme-linked immunosorbent assay
EM	electron microscopy
EMAI	Elizabeth Macarthur Agricultural Institute
ENV	erythrocytic necrosis virus
ERM	enteric redmouth

EU	European Union
EUS	epizootic ulcerative syndrome
EVE	eel virus European
FAO	Food and Agriculture Organization
FAT	fluorescent antibody test
FRDC	Fisheries Research and Development Corporation
GATT	General Agreement on Tariffs and Trade
GIT	gastrointestinal tract
GIV	grouper iridovirus
GUD	goldfish ulcer disease
HACCP	hazard analysis critical control point
HBV	halibut birnavirus
HKS	haemorrhagic kidney syndrome
HPV	herpes virus salmonis
HRI	hotel, restaurant or institution
IFAT	indirect fluorescent antibody test
IHN	infectious haematopoietic necrosis
IHNV	infectious haematopoietic necrosis virus
ID	infectious dose
IPN	infectious pancreatic necrosis
IPNV	infectious pancreatic necrosis virus
IRA	import risk analysis
ISA	infectious salmon anaemia
ISAV	infectious salmon anaemia virus
JFAV	Japanese flounder ascites virus
JWG	Joint Working Group
MAF	Ministry of Agriculture and Forestry
NACA	Network of Aquaculture Centers in the Asia-Pacific Region
NaCl	sodium chloride
NMS	nervous mortality syndrome
NTF	National Task Force on Imported Fish and Fish Products
OIE	Office International des Epizooties (World Organisation for Animal Health)
OMV	*Oncorhychus masou* virus
PCR	polymerase chain reaction

PFU	plaque forming units
PKD	proliferative kidney disease
PKX	proliferative kidney disease agent
PL	plasmacytoid leukaemia
PSAV	Pacific salmon anaemia virus
QP	Quarantine proclamations
Quarantine Act	*Quarantine Act 1908*
RLO	rickettsia-like organism
RSIV	red sea bream iridovirus
RT–PCR	reverse transcriptase polymerase chain reaction
SD	standard deviation
SFIV	sheatfish iridovirus
SLV	salmon leukaemia virus
SPD	salmon pancreas disease
SPDV	salmon pancreas disease virus
SPS Agreement	WTO Agreement on the Application of Sanitary and Phytosanitary Measures
STP	sewerage treatment plant
TCID	tissue culture infectious dose
TEM	transmission electron microscpoy
TSGA	Tasmanian Salmonid Growers Association
TSV	taura syndrome virus
UDF	ulcer disease of flounder
VBNC	viable but non-culturable
VDV	viral deformity virus
VEN	viral erythrocytic necrosis
VER	viral encephalopathy and retinopathy
VERV	viral encephalopathy and retinopathy virus
VHS	viral haemorrhagic septicaemia
VHSV	viral haemorrhagic septicaemia virus
VNN	viral nervous necrosis
WAFIC	Western Australian Fishing Industry Council
WTO	World Trade Organization
YAV	yellowtail ascites virus

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