IMPORT HEALTH RISK ANALYSIS:
SALMONIDS FOR HUMAN CONSUMPTION

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1. EXECUTIVE SUMMARY

A risk analysis has been undertaken by the Ministry of Agriculture (MAF) Regulatory Authority (MAFRA) as a result of a USA request for New Zealand to consider market access for wild Pacific salmon from the USA. The decision to make the risk analysis more generic (to examine disease risks from wild and aquacultured salmonid products from a wide range of countries) was a New Zealand decision, and was taken so that the risk analysis might serve a broader purpose as a decision making tool for considering market access for salmonid products.

The product considered with the risk analysis, referred to hereafter as the *commodity,* has been further defined as product which has been derived from fish:

-        of species within the genera *Oncorhynchus, Salmo* and *Salvelinus*;

-        that were harvested from a population for which a documented health surveillance programme exists, which is administered by a competent government-authorised body and which has been assessed and approved by MAF;

-        that were not slaughtered as an official disease control measure as a result of an outbreak of disease;

-        that were processed in a premises under the control of a competent government-authorised regulatory body recognised by MAF as having equivalent food safety standards governing the processing of fish for export;

-        that have been headed, gilled and eviscerated during the above processing;

-        that have been individually inspected and graded during the above processing; and

-        that were sexually immature, or sexually maturing, but not sexually mature.

This definition is consistent with regulatory processes to provide animal health and food safety assurances, and industry practice to provide health guarantees and quality assurances.

Risk identification conclusions

1   The *commodity* has been defined in a manner consistent with industry practice for the production of salmonid products for human consumption. Certification from exporting regulatory authorities could be obtained to verify the definition.

2   There are no verified instances where importations of the *commodity* have led to disease entry and establishment. In many cases of disease agent introduction into a previously believed-free area the route of introduction was not able to be identified.

3   Pathogens and parasites of salmonids which are of international quarantine significance, of economic significance to salmonid aquaculture industries, which

adversely impact wild stocks of salmonids, and which are not known to be present in New Zealand exist in countries which might export the *commodity* to New Zealand if access were granted.

4   Introduced salmonids exist and are utilised by a variety of sectors in New Zealand, including the salmonid aquaculture industry, recreational fishers, Maori and the New Zealand public. The presence of salmonids contributes significantly to the economy in a variety of direct and indirect ways. Salmonids also have value other than purely economic.

5   Native salmoniform fish are present. Value is ascribed to these species by the fishery industries, Maori, conservation groups, and the New Zealand public. Obligations under the Biodiversity Convention need to be considered. If a salmonid disease were introduced into New Zealand, the impact on native salmoniform fish could range from epidemics with high morbidity and mortality, through to no discernable impact.

6   The consequence for salmonid-based industries of introduction and establishment of a pathogen or parasite of salmonids is difficult to predict, and would depend on many factors. The factors most likely to affect manifestation of disease are the actual organism introduced and its interaction with fish in the New Zealand environment. Impacts resulting from the introduction and establishment of some organisms are likely to be severe, and could cause long term disruption in the salmonid aquaculture industry and in recreational fishery enhancement programmes. Disruption would result from epidemics until management strategies for disease control are developed. For most diseases, management strategies have been developed overseas which are able to minimise disease impact and allow aquaculture and enhancement of wild stocks to continue. However, this invariably incurs costs associated with the initial research effort and ongoing disease management.

7   The combined economic and environmental impact of a salmonid disease introduction into New Zealand might be severely adverse. Overseas evidence has demonstrated that once introduced, eradication of aquatic animal diseases is unlikely.

Risk assessment conclusions

1   No disease introduction event has ever been reliably attributed as resulting from an importation of the *commodity* or similar type product. In many cases of disease agent introduction into a previously believed-free area the route of introduction was not able to be identified.

2   A qualitative assessment of the pathogens and parasites of salmonid fish has concluded that for the majority of organisms considered the risk of their introduction through importations of the *commodity* is negligible.

3   A qualitative assessment of the pathogens and parasites of salmonid fish has concluded that the risk of IPNV, IHNV, VHSV, EIBS, *Aeromonas salmonicida, Henneguya salminicola* and *Kudoa sp.* introduction through importations of the *commodity* is low.

4   A quantitative assessment used a Monte Carlo simulation model to conclude that *A. salmonicida* introduction through importations of the *commodity* is unlikely. In three scenarios modelled, the risk was estimated with 99% confidence to be less than 10^{-7} per tonne of *commodity* imported. The upper 95% confidence limits for the probability of *A. salmonicida* introduction per tonne of *commodity* imported were estimated as:

2.27×10^{-9} for commercially harvested wild Pacific salmon from North America;
6.24×10^{-8} for spawning wild Pacific salmon from North America;
5.48×10^{-8} for farmed Atlantic salmon from Norway;
1.15×10^{-7} for farmed rainbow trout from Denmark.

5   After making the conservative assumption that disease introduction through trade in eviscerated salmonids is possible, even though such introduction has never been demonstrated to occur, a series of quantitative risk assessments used actual historical data on international trade to conclude that the upper 95% confidence limit for the risk of such an event occurring is:

9.95×10^{-4} in the case of BKD, IHN and *A. salmonicida* introduction into New Zealand;
2.27×10^{-4} in the case of IHN and *Ceratomyxa shasta* introduction into Denmark;
3.16×10^{-4} in the case of IHN and *Ceratomyxa shasta* introduction into the United Kingdom;
1.02×10^{-4} in the case of ISA introduction into the United Kingdom;
5.44×10^{-5} in the case of *Ceratomyxa shasta* introduction into France;
2.53×10^{-5} in the case of *Ceratomyxa shasta* introduction into Japan;
4.81×10^{-5} in the case BKD, IHN and *A. salmonicida* introduction into Australia.

6   The annual volume of the *commodity* likely to be imported into New Zealand if market access is granted is approximately 100 to 500 tonnes. If New Zealand consumption of imported trout products attained United Kingdom levels, and demand was met wholly through importations, there is potential for this amount to increase to around 1,000 tonnes. Importations of over 1,500 tonnes are very unlikely given the present consumption patterns of salmon in New Zealand.

Risk management conclusions

1   Considering that the probability of an aquatic animal disease being introduced into New Zealand through imports of the *commodity* is likely to be negligible for most diseases and very low for others, continuing a prohibition on imports is inappropriate.

2   Considering that the probability of an aquatic animal disease being introduced into New Zealand through imports of wild Pacific salmon from the USA is not likely to be significantly different to that for the importation of wild Pacific salmon from Canada which are currently allowable, continuing a prohibition on imports of wild Pacific salmon from the USA is inappropriate.

3   Considering that the consequence of some aquatic animal diseases being introduced into New Zealand through imports of the *commodity* is likely to be severe for stakeholders in the salmonid fisheries of New Zealand and/or for native salmoniform fish, risk management measures over and above those recommended by the OIE Fish Diseases Commission are appropriate.

4   Risk management measures that could be applied include restricting exporting countries to those assessed by MAF as meeting food safety and aquatic animal health surveillance requirements, requiring the *commodity* definition to be verified within export certification, and/or requiring bulk product to be processed in New Zealand under MAF regulatory control.

5   Regulatory systems in Australia, Canada, USA, European Union and Norway would provide New Zealand with adequate food safety and aquatic animal health surveillance assurances if importations of the *commodity* were to be permitted.

6   An indication of how the risk management recommendations would be implemented by MAF can be gained through examination of the similar system set up to regulate imports of wild Pacific salmon from Canada, as documented within the *Import health standard for the importation into New Zealand of wild, ocean-caught Pacific salmon from Canada* (Appendix 2), *MAFRA Standard 154.02.16 Standard for premises processing imported salmon* (Appendix 3) and *MAFRA Standard 154.02.16.01 Standard for the supervision of premises processing imported salmon* (Appendix 4).

Risk communication

Very early on in the risk analysis process the Fishing Industry Board of New Zealand, the New Zealand Salmon Farmers' Association, the two largest private aquaculture companies, the New Zealand Fish and Game Council, and the Department of Conservation were invited to submit data to be considered within the risk analysis.

The Agricultural Security Consultative Committee (ASCC) and the ASCC Technical Subcommittee for Aquaculture have been briefed on progress throughout the risk analysis.

This document serves as the vehicle for public consultation on the issue of the importation of salmonid products for human consumption.

2.   INTRODUCTION

The Biosecurity Act 1993 requires that when considering importations into New Zealand of risk goods (that is, organisms, organic material or other things or substances which might pose a risk of exposure of organisms to damage or disease, or interfere with the diagnosis, management or treatment of pests or unwanted organisms) MAF should consider the likelihood of the risk goods bringing unwanted organisms into New Zealand, as well as the nature and possible effects on people, the environment and the economy of any unwanted organisms that the risk goods may bring into New Zealand.

Prior to 1983 New Zealand imported uncooked salmon for human consumption. In 1983 the Ministry of Agriculture (MAF) introduced a policy which required all importations of fresh water fish to be cooked. The policy reflected consideration that imports of uncooked fresh water fish might pose a risk of aquatic animal disease introduction.

New Zealand is a member of the World Trade Organisation (WTO), and is thus committed to fulfilling the obligations of the *Agreement on the application of sanitary and phytosanitary measures* (the SPS Agreement). These obligations include the need for sanitary measures to be maintained only while supported by scientific evidence, and based on international standards, guidelines and recommendations unless a risk analysis shows that a higher level of protection is justified. In the case of animal diseases, international standards, guidelines and recommendations are those developed under the auspices of the Office International des Epizooties (OIE) or world organisation for animal health.

After receiving a formal request to consider market access for wild Pacific salmon from Canada, in September 1994 MAF released a risk analysis examining the disease risks associated with imports of wild, ocean-caught, Pacific salmon into New Zealand from Canada. The risk analysis concluded that the disease risk was able to be managed, and an import health standard allowing imports was issued in June 1995.

In June 1995 the OIE Fish Diseases Commission (FDC) released the *International aquatic animal health code- 1995* (the Code). The Code describes a range of measures recommended during importation of a range of aquatic animal products. The Code does not recommend measures during trade in eviscerated fish products for human consumption, and this has been interpreted as an indication that the FDC considers such imports do not present a disease risk to the importing country.

In 1995 the USA requested MAF consider market access for wild, ocean-caught Pacific salmon from the USA. The development of an import health standard for Canadian product required considerable resource from within MAF and has led to very little actual trade, as subsequent data have shown (see section 4.7 of the Risk Assessment).

New Zealand has a longstanding request for Australia to allow market access for New Zealand produced salmon without requiring heat treatment. In 1996 Australia undertook to examine the request in a risk analysis. Australia has, in return, requested that New Zealand consider market access for Australian salmon and trout products.

MAF decided that a single risk analysis process generically examining the disease risks associated with imports of salmonid fish for human consumption would meet New Zealand's obligation to either align importation policy with the OIE's recommendations or scientifically justify any other importation policy, while ensuring consistency in determining the appropriate level of protection required for such trade. Such a risk analysis would be a more efficient use of limited available resources than a series of individual risk analyses examining a single country's market access request.

This document represents a generic risk analysis examining the disease risks to New Zealand associated with imports of salmonid fish for human consumption, and has been conducted in a manner MAF believes to be consistent with the OIE's recommendations for completing animal and animal product import health risk analysis.

The methodology followed within this risk analysis is considered by MAF to be consistent with the recommendations for conducting such analysis within the Code. The *commodity* considered within the risk analysis is clearly defined. The risks are identified by developing a list of potential diseases of concern (i.e. those that may be associated with importations of the *commodity*), and examining the consequences to New Zealand of their entry and establishment in New Zealand. The risks are assessed by applying three different methodologies for examining the likelihood of disease agent entry and establishment.

A qualitative assessment has been made following presentation of empirical data. The risk of pathogen introduction through importations is described as either "negligible" or "low". Qualitative risk assessment is not able to conclude that the risk does not exist. In this context, "negligible" can be taken to mean that the risk is so low that it need not be considered.

In the context of this risk analysis, an illustration of what is meant by a qualitative assessment of risk as being "low" is provided through quantitative assessment.

The risk estimations are presented as probabilities within both quantitative assessments. A useful way of interpreting these probabilities is to consider them as presenting the risk of a disease introduction per unit volume of imported product. In this respect, the expected annual volume of trade which may result from market access being granted is useful information, and this forms the subject of a separate assessment. It must be remembered that the risk will not vary from tonne to tonne or from year to year as a result of any earlier result i.e. no disease introduction during importation of 1,000 tonnes of product does not increase the probability of introduction with the next 1,000 tonnes imported.

During the quantitative assessment using the Monte Carlo simulation model actual survey data has been used to provide a conservative estimate of *Aeromonas salmonicida* infection prevalence in potential source populations. The values ascribed to prevalence are not intended to provide an actual estimate of prevalence in the source populations, and the data has been conservatively interpreted deliberately in order to present the worst case scenario.

Finally, risk management options for further reducing the likelihood of disease agent entry and establishment are presented.

3.   RISK IDENTIFICATION

3.1   DEFINITION OF *THE COMMODITY*

The product considered within this risk analysis is defined as headed, gilled and gutted salmonid flesh for human consumption (the *commodity*). The definition implies fish of a certain species, subjected to certain processes, and intended for a certain purpose. These factors can be further defined to form an absolutely clear definition of the *commodity* considered within the risk analysis which includes the regulatory controls and industry practices that typically influence production of the *commodity* in countries likely to export to New Zealand.

Family: Salmonidae

The family Salmonidae may be partially taxonomically classified as follows (from Stoskopf, 1993, and the National Centre for Biotechnology Taxonomy Homepage, at http://www.ncbi.nlm.nih.gov/Taxonomy/tax.html):

ORDER SALMONIFORMES
      FAMILY SALMONIDAE
            Subfamily Salmoninae
                Genus *Oncorhynchus*
                    Species: *O. clarki* (cutthroat trout)
                           O. gorbuscha (pink salmon)
                           O. keta (chum salmon)
                           O. kisutch (coho or silver salmon)
                           O. masou (masou or yamame salmon)
                           O. mykiss (rainbow or steelhead trout)
                           O. nerka (sockeye or kokanee salmon)
                           O. rhodurus (amago salmon)
                           O. tshawytscha (chinook, quinnat or king salmon)
                Genus *Salmo*
                    Species: *S. carpio*
                           S. fibreni
                           S. macrostigma
                           S. marmoratus
                           S. salar (Atlantic salmon)
                           S. trutta (brown trout)
                Genus *Salvelinus*
                    Species: *S. alpinus* (Arctic char)
                           S. confluentus (bull trout)
                           S. fontinalis (brook trout)
                           S. leucomaenis (whitespotted char)
                           S. malma (Dolly Varden)
                           S. namaycush (mackinaw or lake trout)
                           S. pluvius (Japanese char)
                Genus *Thymallus* (grayling)
                Genus *Coregonus* (Whitefish)

Genus *Prosopium* (Whitefish)
Genus *Stenodus*
Genus *Hucho* (Danube salmon and Taimens)
Genus *Brachymystax* (Lenok)

For the purposes of this risk analysis, the use of the term salmonid shall mean fish of a species within the genera *Oncorhynchus, Salmo* and *Salvelinus*. Species within these genera make up the largest proportion of the world's salmonid fish aquacultured or wild caught for human consumption. As such, fish from species within these genera are most likely to be imported into New Zealand as the *commodity*. Species within these genera also comprise a very large proportion of the literature reports on the aquatic animal diseases considered within this risk analysis. Market access requests for fish from other genera, sub-families or families may require additional analysis to determine whether the health risks are appreciably different.

The Conservation Act 1987 prevents the sale of wild salmon and trout in New Zealand as a conservation measure. MAF has obtained legal opinion that the prohibitions on sale relate only to wild salmon and trout taken in New Zealand, and that the importation and sale of salmon and trout products are therefore not prohibited.

Source population factors

Whereas a previous MAF risk analysis (MacDiarmid, 1994) examined the disease risks associated with importations of eviscerated salmon from specific source populations, the *commodity* considered within this risk analysis may come from aquacultured or wild-caught fish, from water of a variety of salinities, and from a number of countries. The eligibility of salmonid fish from any particular source to be imported as the *commodity* is restricted only by factors such as the requirements relating to the population from which the fish are harvested and the conditions under which the fish are processed.

As ocean-run salmonids approach and enter fresh water for spawning they sexually mature and may stop feeding. Both these mechanisms may act as physiological stressors increasing disease susceptibility. A previous MAF risk analysis (MacDiarmid, 1994) defined the product for importation as "wild ocean-caught Pacific salmon". The risk analysis noted that entry into fresh water and sexual maturation were factors that increased the probability of exposure and susceptibility to disease agents. Following the favourable consideration of the disease risk in that risk analysis, health certification was negotiated to accompany imported product which verified that the fish imported were not sexually mature and were caught prior to entry into fresh water. This health certification sought to reduce health risk.

After further investigation of the wild Pacific salmon harvesting and processing industries in the USA and Canada, it is apparent that no distinction is made on the basis of the locality of harvesting for any particular batch of fish. Conversely, sexual maturation is an important quality characteristic of wild salmon that is easily verified by processors, regulators, marketers and their clients. Stage of sexual maturation routinely forms an important grading criterion for wild salmon. Commercial salmonid aquaculture management seeks to avoid harvesting sexually maturing fish for a number of (mainly economic) reasons.

Verification of stage of sexual maturity within export certification is compatible with standard industry practice. Requiring fish to not be sexually mature would mean that, in all likelihood, the fish have not been exposed to the pathogen-contaminated water supplies of spawning grounds, nor been subjected to physiological stressors leading to increased disease susceptibility. Verification of ocean-caught status is less directly related to health risk, and is not compatible with practices within the wild salmon harvesting and processing industry, nor aquaculture industry.

A further requirement is that fish should be from a population for which a documented programme of aquatic animal health surveillance exists, sufficient to record the occurrence of the OIE listed diseases affecting salmonids, noting the category of occurrence (free, low sporadic, endemic, high). The health surveillance programme should be administered by a competent government authorised body. As the government authorised body with responsibility for aquatic animal health in New Zealand, MAFRA would also be responsible for assessing a potential exporting country's aquatic animal health surveillance programme and the competence of the administering authority.

The features and functions of surveillance and control programmes for marine fish diseases have been described (Håstein, 1996). The outcome of surveillance is data on disease occurrence which may be utilised for disease control purposes. Disease surveillance and control will normally be directed towards those diseases of most significance in limiting production or adversely affecting wild fish in a region. The type of control measures naturally vary from region to region, depending on disease distribution, epidemiology, the presence of local susceptible populations and their management.

Surveillance should include any wild salmonid populations in the locality if there is any degree of contact, including water sources, between the aquacultured and wild populations, as occurs with any fish reared in sea cages. However, this may be difficult to achieve. There are less opportunities for disease control in wild populations than in aquacultured populations. Health surveillance in wild populations is most often directed at broodstock because access to other life stages may be limited. Aquacultured fish will typically reflect the health status of wild fish they are in contact with.

Ideally, a requirement for fish to be derived from a population with health status monitoring through a disease surveillance programme would ensure that fish are not harvested as a disease control measure due to a disease outbreak. Commercial realities dictate that aquaculturalists seek to minimise their losses in the face of increasing disease incidence, and this situation probably cannot be avoided by regulation. It is, however, more realistic to expect fish slaughtered as an official disease control measure under the direction of the competent government health authority to be excluded from eligibility for export as the *commodity*.

Processing factors

The salmonid product most commonly traded internationally is fresh, head-on and gill-in, eviscerated fish. In recommending no health certification to accompany such fish, the OIE

reflects what is almost an international consensus[1] that this form of fish product poses no significant disease risk to an importing country (OIE, 1995a).

Several reasons make it extremely unlikely that a salmonid product in this form would be imported into New Zealand. These reasons are:

-   the New Zealand salmon aquaculture industry supplies local market needs for fresh salmon products, including high quality whole fish, at prices which are competitive internationally;

-   the distance from New Zealand of the major salmonid producing nations of the northern hemisphere would require airfreight of fresh product associated with high cost;

-   the small size of the domestic New Zealand market, meaning that fresh product would need to be imported in small lots so that it could be sold prior to deterioration. Air-freight importation of small quantities of product is unlikely to be a commercially viable proposal;

-   in the absence of locally produced trout products, the potential market in New Zealand for imported trout products is likely to be for the trout aquaculture industry's principal export product, a pan-sized head-on or headless, gilled and gutted smoked trout, or smoked trout fillets.

When international and domestic production and consumption of salmonid products are considered, it is entirely more likely that given access for salmonid products to the New Zealand market the following broad categories of product would be imported:

-   chilled consumer packs of value-added salmon products, such as smoked fillets;

-   frozen consumer ready packs of free-flow salmon fillets or steaks, or frozen semi-processed goods such as breaded or crumbed salmon flesh patties;

-   frozen headed, gilled and gutted salmon;

-   smoked, headed, gilled and gutted trout, vacuum packed in single portion consumer-ready retort pouches.

Each of the above categories is processed to a greater degree than the product the OIE considers to pose no significant health risk. The processing undertaken is likely to further lower the aquatic animal health risk associated with the imported product. However, the OIE has indicated that evisceration alone is an effective risk reduction measure, and that further processing is unwarranted as an animal health measure.

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[1]   Australia is alone in rejecting this position, and Australia's position is currently the subject of a WTO dispute process.

Heat treatments are currently required by *MAFRA Import health standard 152.10.08.101* for all fresh water fish products for human consumption imported into New Zealand, with the exception of wild, ocean-caught Pacific salmon from Canada which may be imported without heat treatment under a separate specific import health standard. A similar requirement for heat treatment of imported salmonid products exists in Australia. Salmonid producing nations have indicated a willingness to challenge the technical validity of this heat treatment requirement under international trading agreements, such as the SPS Agreement.

The OIE defines eviscerated fish as those from which the internal organs, excluding the brain and gills, have been removed (OIE, 1995a). Industry adopts a variety of dressing methods when whole salmonid fish are processed. In general, head and gills are left on the carcass by some dressing methods aimed at producing a fresh product for market. This is because the head and gills can be used as an indicator of the freshness of the product. Removal of the head and gills is likely to reduce the bioload of commensal and potentially pathogenic organisms in the product. This bioload reduction will result in reduced aquatic animal health risk. As it has already been established that fresh salmonid products are unlikely to be imported into New Zealand, requiring imported eviscerated salmonid products to have head and gills removed is a risk reduction measure consistent with the realities of any commercial trade that may result from this risk analysis.

Removal of skin would be a further means by which commensal and potentially pathogenic organism bioload could be reduced. However, a requirement for skin removal would restrict the range of products identified as a result of commercial considerations as being likely to be imported if market access is granted.

Typically all aquacultured salmonids are bled at harvest. Bleeding reduces blood spots, which will lead to down-grading, and is a means by which shelf-life may be extended as a result of bioload reduction. Bleeding does not routinely occur during harvest and processing of wild fish.

The product considered within this risk analysis is dead salmonids which have been headed, gilled and eviscerated, but not subjected to any temperature treatments. That is, they are chilled, headed, gilled and gutted fish. The fact that the salmonid products likely to be imported into New Zealand are likely to have been subjected to some additional form of treatment (freezing or smoking) provides a built-in conservatism to the risk analysis.

A further processing requirement relates to the regulatory environment under which the product is processed. Processing quality standards, although ostensibly to uphold public health and food safety standards, affect the aquatic animal health risk of the end product in much the same ways as public health risk is affected. These ways are:

-      by ensuring that fish that are overtly displaying physical characteristics of clinical disease are downgraded;

-      by minimising surface contamination through effective washing, leading to the removal of surface mucous, blood and debris on the skin and in the visceral cavity;

- by ensuring that evisceration is as complete as possible;

- by ensuring the end product is wholesome and fit for human consumption.

The physical characteristics of disease vary greatly. They range from no grossly detectable lesions, as in fish harvested while incubating disease or asymptomatic carriers of disease agent; to minor lesions grossly detectable on close individual examination, as in fish dying of peracute disease; to grossly obvious lesions, such as external ulceration or septicemic fish. Inspection of fish during processing functions as a quality measure. One outcome of inspection may be the detection of physical lesions such as noted above. Other outcomes may be the detection of physiological states that may affect quality, such as sexual maturity, or the detection of incomplete washing or evisceration. The outcome of inspection may be that fish exhibiting characteristics that affect quality are rejected, downgraded, and/or subjected to further processing. Downgrading may in turn result in fish either being filleted, or only used in manufacturing such as canning. Without pretending that individual examination of fish during processing is able to remove all diseased fish, it is obvious that restriction of product to fish which have been individually inspected will appreciably reduce the potential pathogen bioload of the *commodity*. This is the case despite the inspection of fish functioning primarily as a quality issue, rather than a disease issue, and despite any imposed restriction on the result of inspection i.e. restricting eligible product to that designated as certain grades as a result of inspection.

New Zealand processing standards equivalence

The *commodity* must have been processed in a premises under the control of a competent government-authorised regulatory body recognised by MAFRA as having equivalent food safety standards governing the processing of fish for export. As a result of such processing the fish must have been headed, gilled and eviscerated. During processing the fish must have been individually inspected and graded.

All fish exported from New Zealand is processed under MAF regulatory control, detailed within the following documents:

- *Regulatory Systems and Controls for the Export of Seafood*, an overview document produced by MAF Regulatory Authority (Seafood);

- The Fish Export Processing Regulations 1995;

- The Meat Act 1981;

- The New Zealand Fishing Industry Inspection and Certification Council Industry Agreed Standards.

The New Zealand standards for regulatory control over fish for export from New Zealand utilise a Hazard analysis at critical control points (HACCP) approach. Regulatory authorities are increasingly requiring HACCP systems to be used by food processors to implement food safety controls. The HACCP approach makes it easier for regulators to audit and to verify the

processor's compliance with regulatory requirements.

In order to facilitate trade (both import and export of product from New Zealand), MAFRA negotiates equivalence agreements with trading partners. In the case of fish, such equivalence agreements are negotiated on the basis of analysis of the exporting country's regulatory controls governing processing of fish for export, the compliance activities undertaken and the enforcement powers prescribed.

A minimum pre-requisite processing requirement of the *commodity* is that it must have been processed in a premises under the control of a competent government-authorised regulatory body recognised by MAFRA as having equivalent food safety standards governing the processing of fish for export. As a result of such processing the fish must have been headed, gilled and eviscerated. During processing the fish must have been individually inspected and graded.

Summary

In summary, the *commodity* may be further defined as product which has been derived from fish:

-        of species within the genera *Oncorhynchus, Salmo* and *Salvelinus*;

-        that were harvested from a population for which a documented health surveillance programme exists, which is administered by a competent government-authorised body and which has been assessed and approved by MAF;

-        that were not slaughtered as an official disease control measure as a result of an outbreak of disease;

-        that were processed in a premises under the control of a competent government-authorised regulatory body recognised by MAF as having equivalent food safety standards governing the processing of fish for export;

-        that have been headed, gilled and eviscerated during the above processing;

-        that have been individually inspected and graded during the above processing; and

-        that were sexually immature, or sexually maturing, but not sexually mature.

This definition is consistent with industry and regulatory authority practice to reduce animal and public health risk and provide quality guarantees during the production and processing of salmonid products for human consumption.

It should be noted that there are no verified instances of importations of *the commodity* leading to disease introduction and establishment in any country. In many cases of disease agent introduction into a previously believed-free area (for example *Aeromonas salmonicida* introduction into the United Kingdom, *Gyrodactylus salaris* introduction into Norway, IHNV

introduction into Norway, and *Myxobolus cerebralis* introduction into New Zealand) the route of introduction was not able to be identified.

3.2   DISEASES OF POTENTIAL CONCERN

Introduction

Risk identification must identify the pathogens and parasites of potential concern during importation of *the commodity*, for which the risk should be assessed. The initial objective of risk identification is to identify as complete a list as possible of the pathogens and parasites to which salmonid fish are susceptible utilising a wide range of appropriate sources. To obtain the list of diseases to be considered within the risk analysis from this initial list, two filters were then applied:

1   The pathogens and parasites of salmonids in New Zealand for which no significant international strain variation exists were filtered out of the initial list;

2   Organisms of the Order Metazoa were filtered out of the initial list.

The list of pathogens and parasites of salmonids resulting from this process is considered during the risk assessment.

OIE

The diseases listed by the Office International des Epizooties (OIE, 1995a; OIE, 1995b) for which salmonids have known susceptibility are the following:

Diseases notifiable to the OIE   Epizootic haemorrhagic necrosis (EHN)
                                        Infectious haematopoietic necrosis (IHN)
                                        Oncorhynchus masou virus disease (OMV)
                                        Viral haemorrhagic septicaemia (VHS)

Other significant diseases   Infectious pancreatic necrosis (IPN)
                                        Infectious salmon anaemia (ISA)
                                        Epizootic ulcerative syndrome
                                        Bacterial kidney disease (*Renibacterium*
                                        salmoninarum)
                                        Piscirickettsiosis (*Piscirickettsia salmonis*)

Previous MAF salmon risk analysis

Diseases considered within the previous MAF risk analysis (MacDiarmid, 1994) not listed above were the following:

Viral erythrocytic necrosis (VEN)
Erythrocyte inclusion body syndrome (EIBS)
Plasmacytoid leukaemia
Pancreas disease
Furunculosis *(Aeromonas salmonicida)*
Enteric redmouth (ERM)

Rosette agent
Loma salmonae
Enterocytozoon salmonis
Edwardsiellosis
Proliferative kidney disease (PKD)
Kudoa thyrsites
Ceratomyxa shasta
Herpesvirus salmonis
Vibriosis
Hitra disease
Henneguya salmonicola
Parvicapsular disease

AQIS salmon risk analysis

The list of diseases considered within the Australian *Salmon import risk analysis* (DPIE, 1996) included the following parasite, not listed above:

Whirling disease (*Myxobolus cerebralis*)

European Union

Salmonid diseases listed by the European Union in Annex A Lists I, II and III of Council Directive 91/67/EEC concerning the placing on the market of aquaculture animals and products are:

Infectious haematopoietic necrosis (IHN)
Viral haemorrhagic septicaemia (VHS)
Infectious pancreatic necrosis (IPN)
Bacterial kidney disease (*Renibacterium salmoninarum*)
Enteric redmouth (ERM)
Gyrodactylosis (*Gyrodactylus salaris*)
Whirling disease (*Myxobolus cerebralis*)

In addition, the EU took legislative measures in March 1993 in Commission Decision 93/144/EEC against infectious salmon anaemia (ISA), which prohibits the import of live fish and round fish (uneviscerated fish) from ISA infected regions of Norway. Importations of eviscerated fish from ISA infected regions of Norway have continued.

Australian aquatic animal quarantine review

Humphrey (1995) reviewed the diseases of aquatic animals for their significance to aquatic animal quarantine policy. Humphrey classified the pathogens of aquatic animals in a taxonomic manner and includes most of the pathogens listed above, and a great many others. Humphrey included a semi-quantitative assessment of relative quarantine importance, which was in effect a comparative qualitative assessment, with reference to the following characteristics of the pathogen or parasite: pathogenic significance, international spread, risk of entry into Australia

in absence of quarantine measures, risk of establishment in Australia, possible socio-economic consequences, possible ecological consequences, and difficulty of control or eradication. Although the scoring system employed by Humphrey gave a useful indication of the comparative significance of the pathogens and parasites discussed, the score cannot be assumed to have any direct relevance to the disease risk associated with the importation into New Zealand of the *commodity.*

Published review texts on aquatic animal pathogens and parasites

There are several published review texts on the parasites and pathogens of fish. The texts utilised during risk identification and risk assessment in this risk analysis are Wolf (1988), Stoskopf (1993), Austin and Austin (1993), Inglis et al (1993) and Woo (1995).

Pathogens and parasites of salmonids not considered within the risk assessment

1   Endemic pathogens and parasites

The pathogens and parasites of salmonids already present in New Zealand are reviewed in Section 3.5 of this risk analysis. The pathogens and parasites already present in New Zealand for which no significant strain variations are known to occur internationally are not considered within the risk assessment.

2   Metazoa

There are numerous metazoan parasites of salmonids (reviewed by Woo, 1995). Many are known to parasitise a wide variety of aquatic animals from families other than Salmonidae (e.g copepods). Others are parasites which have specifically adapted to parasitise salmonids (e.g *Gyrodactylus salaris*). Metazoan parasites tend to be large enough to be seen with the naked eye. Most parasitise either the intestinal tract or other visceral tissues (e.g. most nematodes), the gills (e.g. *Gyrodactylus salaris),* the tissues of the head (e.g. eye flukes) or the body surface (e.g. copepods).

Some metazoan parasites do infect the flesh of salmonids and other fish (e.g. *Anisakis* sp.). *Anisakis* sp. have been recorded in many species of fish in New Zealand (Hewitt and Hine, 1972).

The very small risk of introducing live parasites on a killed product is effectively negated for parasites associated with the tissues of the viscera, head, and gills by removal of these tissues prior to importation of the *commodity*. Parasites of the skin (e.g. copepods) or those that attach to the internal visceral cavity would be detected and removed during inspection of the product during processing.

Metazoan parasites tend to be obligatory parasites. Although some species have free-living stages, in general they would not be expected to survive long periods in a dead product out of the aquatic environment. Neither the previous MAF risk analysis (MacDiarmid, 1994) nor the AQIS risk analysis (DPIE, 1996) considered that there was any risk of introduction of any species of metazoan parasite in wild Pacific salmon for human consumption, despite many

species of metazoan parasites occurring in the source populations considered.

Although freezing and heat treatment such as smoking are not considered within the definition of the *commodity,* these processes are a further effective risk reduction measure for metazoan parasites.

Metazoan parasites are not considered further within the risk analysis.

Diseases and parasites of potential concern

The diseases and parasites of potential concern during importation of *the commodity* are:

Viral diseases
    Adenoviridae
        Strawberry disease virus of trout
    Birnaviridae
        Infectious pancreatic necrosis virus (IPN)
    Herpesviridae
        Herpesvirus salmonis
        Oncorhynchus masou virus (herpesvirus of Salmonidae Type II)
    Iridoviridae
        Epizootic haematopoietic necrosis virus (EHNV)
        Viral erythrocytic necrosis (VEN)
    Orthomyxoviridae
        Infectious salmon anaemia (ISA)
    Paramyxoviridae
        Chinook salmon paramyxovirus
    Picornaviridae
        Picorna-like virus of salmonids
    Reoviridae
        Chum salmon reovirus
        Coho salmon reovirus
        Taiwanese *Oncorhynchus masou* reovirus
        Taiwanese rainbow trout reovirus
    Retroviridae
        Atlantic salmon swimbladder sarcoma virus
        Plasmacytoid leukaemia
    Rhabdoviridae
        Infectious haematopoietic necrosis virus (IHNV)
        Viral haemorrhagic septicaemia (VHS)
    Togaviridae
        Pancreas disease
    Uncharacterised viruses
        Pacific salmon anaemia virus (Erythrocytic inclusion body syndrome)
        Rainbow trout intra-erythrocytic virus
        Intra-erythrocytic viral disease of coho salmon
        Atlantic salmon papillomatosis agent

Ulcerative dermal necrosis agent

A new virus of salmonids in Japan

Bacterial diseases

Aeromonadaceae representatives

Aeromonas salmonicida

Enterobacteriaceae

Citrobacter freundii

Edwardsiella tarda

Serratia liquefaciens

Serratia plymuthica

Yersinia ruckeri (Enteric redmouth disease)

Cytophagaceae

Cytophaga sp.

Flavobacterium sp.

Flexibacter sp.

Sporocytophaga sp.

Pseudomonadaceae

Pseudomonas sp.

Vibrionaceae

Plesiomonas shigelloides

Vibrio anguillarum

Vibrio salmonicida

Moraxellaceae

Acinetobacter sp.

Gram positive bacteria: the 'lactic acid' bacteria

Carnobacterium piscicola

Lactobacillus sp.

Lactococcus piscium

Streptococcus sp.

Vagococcus salmoninarum

Aerobic Gram positive rods and cocci (other than *Renibacterium salmoninarum*)

Coryneforms

Micrococcus luteus

Planococcus sp.

Rhodococcus sp.

Streptoverticillium salmonis

Renibacterium salmoninarum (Bacterial kidney disease)

Piscirickettsia salmonis (Piscirickettsiosis)

Other miscellaneous pathogens

Janthinobacterium lividum

Fungal diseases

Candida sake

Exophiala salmonis

Leptomitis lacteus

Ochroconis tshawytschae

Ochroconis humicola

Phoma herbarum

Saprolegnia sp
Schizotrytium-like sp.
Protozoan diseases
    Mastigophora (flagellates)
        Hexamita salmonis
        Ichthyobodo necatrix
        Trypanoplasma sp.
    Rhizopoda (amoeba)
        Thecamoeba hoffmani
        Vexillifera bacillipedes
    Apicomplexa
        Cryptosporidium sp.
    Microspora
        Enterocytozoan salmonis
        Loma salmonae
        Microsporidium takedai
        Pleistophora salmonae
    Myxozoa
        Ceratomyxa shasta
        Chloromyxum sp.
        Henneguya salminicola
        Kudoa sp.
        Myxidium truttae
        Parvicapsula sp.
        Proliferative kidney disease (PKX)
    Ciliophora (ciliates)
        Apiosoma sp.
        Carchesium sp.
        Tetrahymena rostrata
        Trichophrya sp.
    Unclassified
        Dermocystidium sp.
Diseases of uncertain aetiology
        Chinook salmon rosette agent
        Nervous mortality syndrome
        Haemorrhagic kidney syndrome

3.3   SALMONIDS IN NEW ZEALAND

Introduction

Salmonids are native to cool and cold waters of the Northern hemisphere, where they occur in fresh, brackish and salt waters. Salmonid species now occur on every continent, with the exception of Antarctica, as a result of human introductions (Bartley and Subasinghe, 1995). Some species must regularly spend a part of their life cycle in the sea. Others include some populations that go to sea for a time and some that do not, while yet other species may never enter the sea at all. Whether or not a part of their life is spent at sea, spawning in salmonids is invariably in fresh water.

Importation of salmonids into New Zealand

All species of salmonid fish present in New Zealand were introduced as a result of an intensive programme of importations aimed at the establishment of wild populations. In the period from 1870 through to 1930 (and on occasion in later years) many importations of fish eggs, and occasionally live fish, were made into New Zealand by a variety of agencies and private individuals. A wide range of salmonid and non-salmonid fish species from diverse locations were imported. The importations are documented in the archives of the various acclimatisation societies which were operating at that time, and have been summarised by McDowall (1990). Much of the information presented in this section is derived from the work of this author.

The importations of salmonids into Australia, similarly free of many salmonid pathogens of international significance, are documented in the AQIS risk analysis (DPIE, 1996). Numerous importations of salmonids into Australia occurred prior to 1966, and up until 1958 no quarantine or other sanitary measures were enforced. Chinook salmon eggs imported into Australia between 1963 and 1966 received formalin baths on arrival, and packing material was destroyed.

No formalised quarantine or other sanitary measures were enforced during early importations into New Zealand. Despite this, very few pathogens of international significance appear to have been introduced into New Zealand. Australia has been similarly fortunate. It seems likely that some important pathogens and parasites may have existed either in the source populations for these introductions or in the consignments themselves. For example, the bacterial pathogen of salmonids *Aeromonas salmonicida* was first described in Europe in 1894 and in North America in 1902. The first report from Great Britain of the organisms presence was in 1909, and by 1911 it was recognised to be present in a number of river systems in Great Britain (Austin and Austin, 1993). Other pathogens for which vertical or egg-associated transmission is more conclusively known to occur are *Renibacterium salmoninarum*, which was first recorded in Scotland in 1930 and in the USA in 1935 (Austin and Austin, 1993), and infectious haematopoietic necrosis virus (IHNV), which may have caused epidemics in salmonids on the Pacific coast of North America as early as 1926 (Wolf, 1988; McDaniel et al, 1994). Each of the initial isolations of these organisms were made from hatcheries. That is, disease in salmonids was clinically manifested when humans intervened to create artificial rearing systems with high stocking densities, artificial nutrition and other potential causes of stress in fish which are inherent in culturing systems. From an epidemiological viewpoint, it is

reasonable to assume that the organisms existed in wild populations of salmonids prior to their initial identification in salmonid hatcheries. That is, the salmonid pathogens probably evolved with the fish and hatchery rearing exacerbated them.

New Zealand sockeye salmon stocks are all derived from a single importation of 500,000 sockeye salmon ova from the Fraser River system, Canada, in 1901 (Graynoth and Hawke, 1985). Whether IHNV was already endemic in sockeye salmon populations of Canada at this time is unknown. Another not necessarily contradictory view is that human intervention into salmonid biology has influenced the evolution of salmonid pathogens. In either case, considering the number and diversity of salmonid introductions, it is likely that more complex considerations within the host-environment-pathogen interaction influenced the relative lack of pathogens or their evolutionary precursors in New Zealand and Australia than pure luck in the selection of source populations.

Many attempts at introduction failed as a result of egg or fry mortalities en route or in hatcheries in New Zealand. There appears to be no record of any investigation into the cause of these mortalities. Infectious disease must be considered a possibility.

Other importations partially succeeded, with fresh water populations established of fish species more usually known to be anadromous. Others succeeded in their aim of establishing self-sustaining wild populations of fish with biology in New Zealand comparable to that in their indigenous localities.

Salmonid species present in New Zealand

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 distribution and utilisation in New Zealand of the above salmonid species is briefly reviewed.

Salmo trutta

The brown trout was one of the first game fish to be introduced into New Zealand, and has become widely established throughout the country where habitat conditions have permitted. At present, the species is found in most river systems south of Coromandel. It is also occasionally found further north, although the rivers tend to be too warm for brown trout to thrive. On the East Coast of the North Island, from East Cape southwards to the Wairarapa, brown trout are not abundant, especially towards the coast. Brown trout are absent from the Chatham Islands, although they have been released there. They have not spread to Stewart

Island.

Brown trout populations in New Zealand are largely self-sustaining. They maintain numbers sufficiently dense to make full use of the available food supplies and territories. Some hatchery enhancement activity of brown trout populations is undertaken by the Department of Conservation (DoC). The brown trout is an important species within the fresh water recreational fishery of New Zealand. Current legislation controlled by DoC and within the Freshwater Fisheries Regulations 1983 classifies brown trout as a sport fish species, making it illegal for brown trout to be commercially farmed, or kept live for any other purpose, or to be sold live or dead.

Salmo salar

The Atlantic salmon was identified as a potential candidate for introduction very early in the settlement of New Zealand, and many of the earliest, and probably most, importations were attempts to establish a population in New Zealand. Today, Atlantic salmon are only found in Lakes Te Anau and Manapouri in Fiordland. The populations never established an anadromous habit, and were eventually fully landlocked by hydro-electric projects which dammed the rivers of the lower watershed cutting off access to the sea.

Atlantic salmon are the salmonid species most regularly used in salmon aquaculture worldwide, including in North America, Europe, and Australia. Some use of the species for commercial aquaculture occurs in fresh water ponds in the South Island and in sea-cages on Stewart Island. There appears to be growing interest in expanding the utilisation of Atlantic salmon within salmon aquaculture in New Zealand, including the importation of new genetic material. The genetic potential of current stocks in New Zealand is likely to be inferior if compared with strains available worldwide, as a result of the limited starting genetic base, years of in-breeding, no controlled selection for desirable traits, and fresh water adaptation. Strains of Atlantic salmon available worldwide include domesticated stocks with a long history of selection for desirable traits in aquaculture such as uniformity of size and conformation, high feed conversion, good reproductive traits in broodstocks, and placidity in captivity leading to less energy expended in metabolic functions other than growth.

Salvelinus fontinalis

The brook char is native to North America and the Canadian Arctic. It has been introduced to Europe, South America, Africa, Japan and Australia, as well as New Zealand.

The brook char is found in several upland lakes and small streams of the head waters of larger river systems. They are mainly in the centre of the South Island, and around Lake Taupo and the south of the Bay of Plenty in the North Island. Although classified as a sport fish and as such subject to the considerations within the Freshwater Fisheries Regulations 1983, the brook char is a species with lesser importance within the recreational fishery of New Zealand.

Salvelinus namaycush

This species in known in North America as lake trout or lake char. In New Zealand it has

become most widely known by its Indian name, mackinaw.

Mackinaw are only known to occur in a very limited region of the South Island, in Lake Pearson, near Arthur's Pass. Mackinaw hold little value in terms of utilisation by recreational fishers. However, it is possible with North American stocks in decline that the genetic value of the New Zealand fish has not been realised.

Oncorhynchus mykiss

Although well established in New Zealand, rainbow trout are more localised in distribution than brown trout. The rainbow trout in New Zealand is most notably a lake fish. Rainbow trout can be found around the thermal lake districts of the North Island, up to the Bay of Plenty and the Waikato. The largest population is found in the Volcanic Plateau of the central North Island. Rainbow trout are less predominant in the South Island and mostly confined to the central regions of the island.

The rainbow trout is an important species within the fresh water recreational fishery of New Zealand. Classification as a sport fish within the Freshwater Fisheries Regulations 1983 gives rainbow trout protection from commercial exploitation. Rainbow trout aquaculture is an important industry in many countries.

DoC undertakes an enhancement programme involving a number of small-scale hatcheries rearing eggs and juvenile classes from wild broodstock. Hatchery-reared trout are released into a number of waterways. These activities are the only aquaculture activities for trout species in New Zealand, and are tolerated, and indeed, protected and advocated by recreational fishers and conservationists. This is despite these groups having expressed in the past opposition to aquaculture of trout species. The major concern of opponents to trout aquaculture when this issue was examined by the Fishing Industry Commission in 1971-72 was potential disease and environmental impacts on wild fish populations from farming activities, including imports of new genetic stocks. The disease and environmental impacts resulting from recreational fishery enhancement activities do not appear to have been evaluated.

Oncorhynchus tshawytscha

Quinnat salmon (also called chinook and king salmon) have established anadromous stocks in the river systems of the east coast of the South Island. These stocks have suffered as a result of modifications to river systems, such as dams, particularly in the Waitaki watershed. Enhancement programmes maintain a modest population of wild stocks through hatchery spawning of anadromous broodstock, incubation of eggs, the release of juvenile classes, as well as some habitat restoration and maintenance of access to spawning grounds. These stocks make up an important seasonal recreational fishery in local areas of the South Island.

Quinnat salmon are the most important salmonid species in commercial aquaculture in New Zealand. Sea-cage rearing to harvest weights occurs at a number of locations in the South Island, and there are also a larger number of small fresh water pond rearing operations. Although classified as a sport fish within the Freshwater Fisheries Regulations 1983, over-riding classification within the Fisheries Act 1996 allows for quinnat, sockeye and Atlantic

salmon to be commercially exploited.

Oncorhynchus nerka

In New Zealand, virtually all sockeye salmon are found in Lakes Benmore and Ohau, with small numbers in Lakes Aviemore and Waitaki. Although early indications were that an anadromous population may have been established, hydro-electric projects have led to the populations becoming landlocked. The species has importance in local areas as a recreational fishery. Some small scale fresh water pond culture occurs in the South Island.

Indigenous salmoniform fishes

New Zealand has (or had, as at least one species is now extinct) 27 species of native fresh water fish. Fish from families within the Order Salmoniformes other than the introduced species of the family Salmonidae exist in New Zealand. The salmoniform fish species present in New Zealand other than salmonids are:

FAMILY RETROPINNIDAE
   Retropinna retropinna (common smelt)
   Stokellia anisodon (Stokell's smelt)

FAMILY PROTOTROCTIDAE
   Prototroctes oxyrhynchus (grayling) (extinct)

FAMILY GALAXIIDAE
   Galaxias argenteus (giant kokopu)
   G. fasciatus (banded kokopu)
   G. postvectis (shortjawed kokopu)
   G. brevipennis (koaro)
   G. vulgaris (common river galaxias)
   G. maculatus (inanga)
   G. gracilis (dwarf inanga)
   G. divergens (dwarf galaxias)
   G. paucispondylus (alpine galaxias)
   G. prognathus (longjawed galaxias)
   Neochanna burrowsius (Canterbury mudfish)
   N. apoda (brown mudfish)
   N. diversus (black mudfish)

The distribution and biology of these species was reviewed by McDowall (1990), who also reviewed the role of these species within the traditional Maori fisheries, and their role in modern commercial and recreational fisheries. The role of certain galaxiid species in the whitebait fishery is probably the most distinctive and valuable. Smelt are also commercially harvested on a small scale.

McDowall notes that there has been limited study of the relationships between the indigenous and exotic fish species in New Zealand. The impact of introduced salmonid species on native

fish occurs either through predation or by habitat displacement. At various times in New Zealand's recent history commentators have speculated on the role introduced salmonid species have played in the decline and eventual extinction of grayling. McDowall concludes that there can be little doubt that the decline in koaro in lakes in the central North Island is attributable to trout predation. McDowall notes that whitebait form an important food for lowland brown trout during the spring, but concludes that it is more likely that human-influenced habitat modification has influenced the decline of whitebait fisheries to a far greater extent. McDowall further notes that there has been a potentially terminal decline of dwarf inanga in rivers and lakes of Northland since trout stocking began in the 1960s.

3.4 UTILISATION AND VALUE OF SALMONIDS IN NEW ZEALAND

Identifying the stakeholders of the New Zealand salmonid fisheries

Salmonids were originally introduced into New Zealand for the purposes of recreational fishing. A substantial industry has built up around the recreational salmonid fishery. Enhancement programmes for wild salmonids are currently operated by the New Zealand Fish and Game Council (NZFGC) and the Department of Conservation (DoC) to support these activities.

Since their introduction, salmonids have become a part of the fauna within biological systems which conservation groups seek to preserve and enhance.

In the 1970s commercial salmonid aquaculture began in New Zealand, and in 1982 the first sea water net pen rearing of salmon was established. Since that time a significant export industry has been built up.

The presence of salmonids in the rivers and lakes of New Zealand and, in some cases, the displacement of native fish species has meant that salmonids have come to be regarded by Maori as a cultural resource of value. In some specific cases, Maori are recognised within legislation as stakeholders in salmonid fisheries.

There is wide participation by the general public within the recreational fishery of New Zealand. In general, the public is interested in conservation matters. Through these mechanisms, and as consumers of salmonid products, the New Zealand public is considered to be a stakeholder in the salmonid fisheries.

Thus, the stakeholders in the salmonid fisheries are identified as the following groups:

recreational fishing industry
conservation agencies and groups
salmonid aquaculture industry
Maori
the public

The utilisation of salmonids by, and the value of salmonids to, each of these stakeholder groups will be discussed so as to indicate the total value of the salmonid fishery of New Zealand.

Recreational fishing industry

The recreational fishing industry is less an industry as such, and more an activity with participation by local anglers, domestic tourist anglers, and international tourist anglers. A subset of recreational fishing is sports fishing, because sports fish are a subset of fresh water fish. Fresh water fish are defined by the Conservation Act as any species of fish, shellfish or Crustacea which spends all or part of its life cycle in fresh water. A fish species may be declared a sports fish by the Governor General, by Order in Council, pursuant to the

Conservation Act 1987. Once a fish species is declared a sports fish it is listed in the First Schedule of the Freshwater Fisheries Regulations 1983. The Freshwater Fisheries Regulations 1983 were originally made under the Fisheries Act 1983, but are now treated as being made under the Fisheries Act 1996. All species of salmonids present in New Zealand are listed as sports fish.

DoC and NZFGC are the statutory authorities charged by the government to manage sports fisheries pursuant to section 17 of the Conservation Act 1987. DoC is the government agency responsible for management of the Taupo sports fishery, and operates a hatchery enhancement programme for trout within this fishery (pers. comm. to Stuart MacDiarmid from John Holloway, Director, Estate Protection and Policy Division of Department of Conservation, December 1994). The Fish and Game Council of New Zealand is the national body to which each regional Fish and Game Council is affiliated. The Fish and Game Councils of each region prepare for approval by the Minister of Conservation such sports fish and game management plans as are necessary within their area of jurisdiction. There are three sport fishery hatcheries in the North Island, at Tongariro, Ngongotaha and Napier, which specialise in rearing rainbow trout for re-stocking recreational fresh water fisheries. The South Island has one sport fish hatchery at Wanaka.

In August 1996 MAF requested that DoC and the NZFGC provide an estimate of the value of the recreational salmonid fishery of New Zealand for the purposes of this risk assessment. No response was received from DoC, although submissions from DoC to MAF during a previous risk analysis were able to be used. The NZFGC response (pers. comm. Mike Britten, Assistant Director NZFGC, September 1996) noted that the NZFGC and its affiliated regional councils have not undertaken detailed studies to collect sociological information relating to the value of the fishery. The NZFGC provided details of five studies which give relevant information on this subject. In most cases, the expenditure of persons participating in recreational fishing has been estimated and this has been assumed to give an indirect indication of the value of the fishery. Whether or not this is a valid assumption has not been specifically addressed either within the publications themselves or by the NZFGC.

In most cases, it seems likely that if no recreational fishery existed those persons who were participating in the fishery would spend their money elsewhere in the domestic economy, and thus it would not be lost to the gross domestic product. Naturally, this is not true for international visitors who come here specifically to fish because of New Zealand's international reputation as a destination offering excellent recreational fishing. However, no estimate has been made of what percentage of total expenditure this group is responsible for.

The geographical distribution of recreational fishing leads to fishing related expenditure accounting for a high proportion of total expenditure in regions such as Rotorua, Taupo, and some South Island locations. Some of the studies estimated regional expenditure.

The estimations of value based on anglers' expenditure fail to recognise the investment made in the recreational fishing industries through enhancement activities, such as re-stocking programmes and habitat conservation and restoration projects. However, in the absence of other data to estimate the value of the New Zealand recreational fishery the documents referred to by the NZFGC are summarised. There has been no attempt to provide technical

criticism of the methodology employed in arriving at the estimates.

1   Scott (1987) examination of sport fisheries of New Zealand

In a Department of Scientific and Industrial Research (DSIR) bulletin examining the inland waters of New Zealand, Scott (1987) reviewed information on the fresh water recreational fishery. The number of individuals holding fishing licences was estimated at 6.2% of the population in the 1983-84 season. The mean expenditure by anglers in various regions was estimated through reference to previous studies which had employed diary schemes to gather data, after adjustment for inflation using the consumer price index. Travel, food and accommodation costs accounted for the largest share of expenditure. The total expenditure by anglers in 1983-84 was estimated at $65.7-74.2 million for all New Zealand, of which $10.8 million was spent in the Taupo region. The number of equivalent full-time jobs generated by angling was estimated as 1,484-1,676 for all New Zealand, and 244 for the Taupo district. The capital assets associated with expenditure in angling were estimated to be worth $683.7-772.2 million for all New Zealand, and $112.6 million for the Taupo district.

2   National Research Bureau phone survey, 1991

In 1991 the National Research Bureau (NRB) conducted a telephone survey of 3,495 persons over the age of 16 selected at random from telephone directories and supplemented with a group of Maori and Pacific Island persons (Jellyman, 1992). A total of 38% had fished once or more in the previous 12 months. Within this group, approximately one third had fished on 1-3 days, 4-14 days, and 15-60 days respectively. The survey design was not able to extract responses for fresh water fishing alone, although it was able to determine that 21% had fished in the sea from a boat, 21% had fished in the sea from land, 7% had fished in fresh water from a boat, and 10% had fished in fresh water from land. South Islanders were less likely than North Islanders to have fished in the previous 12 months, but more likely to have fished in fresh water. A total of 37% of all persons surveyed had fished for salmonids at some time, although only about 75% of these had purchased a licence. The average number of days fished annually by fresh water fishers was 4, and 12 for sea water fishers. From this survey a profile of the most likely fresh water angler was formulated as a South Island middle-aged European male with annual income over $40,000. The survey went on to ask a series of questions on expenditure on fishing, which included information on travel, accommodation, meals, boat running costs, clothing and equipment. The estimated total annual expenditure in New Zealand's recreational fishery was $745.2 million. Car travel (35.4%) and boat fuel and running costs (23.5%) accounted for most of total expenditure. The expenditure associated with fresh water fishing was estimated to be $150 million (20% of total). The survey did not itemise expenditure by region. However, as 42% of fresh water anglers had fished in the Taupo region, this region was estimated to account for expenditure of about $63 million. The total expenditure in recreational fishing was compared with the total seafood export earnings in the year to March 1992 ($1.1 billion). This comparison indicated that expenditure in recreational fisheries was about 75% the export value of the seafood industry.

3   Shaw (1990) evaluation of Rotorua fishing district during the 1986/87 season

A study in the Rotorua Licence District during the 1986/87 season (Shaw, 1990) estimated

that anglers in this district spent $17.05 million annually on their sport. Travel, accommodation and food accounted for over two thirds of this expenditure. Boating expenses accounted for an additional 17.8% of the total. The direct effect on the local economy of Rotorua was measured to be worth 102 jobs spread over 209 businesses. The estimated capital value of businesses directly related to anglers' fishing was estimated to be $11.2 million, and their total turnover $10.8 million.

4   Donnelly evidence at the Mataura River National Water Conservation Order Hearing

Phillip Donnelly, an economics consultant, produced evidence dated January 1990 during a Mataura River National Water Conservation Order Hearing which estimated the economic activity generated by trout fishing in the Mataura River. The estimates were made through reference to earlier surveys of angler activity for the Southland district and statistics on tourism from the New Zealand Tourist and Publicity Department. The final estimate of total expenditure was made through estimates of expenditure by three angler groups; local anglers, domestic visitor anglers and international visitor anglers. On the basis of estimated expenditure for the 1987-88 season the Mataura River trout resource was estimated to have a value between $17-35 million.

5   Trout Unlimited New Zealand extrapolation of USA data

Trout Unlimited New Zealand is an Auckland based company whose self-stated objective is to provide unlimited access for its members to fish for trout. In August 1989 the company's director, John Giacon, produced a document as "an appeal to the nation's decision makers, the media, and public not to pursue the current promotion of fresh water aquaculture, specifically trout farming". The document used a USA study titled *National Survey of Fishing, Hunting and Wildlife Associated Recreation* to provide base data from which extrapolations to the New Zealand situation were made. The estimate of total New Zealand expenditure in fresh water fisheries was $751 million, which included costs associated with travel, accommodation, meals, equipment, licence sales, magazines, fishing association membership fees, costs associated with accompanying non-participants (wife and children), and an estimation of expenditure by tourist anglers.

Since the above studies were conducted, there have been various attempts to update the estimates of expenditure through inflation adjustments. In a submission to the previous MAF salmon risk analysis (MacDiarmid, 1994), the Department of Conservation noted that Taupo anglers purchased 68,000 licences and that the economic worth of the Taupo fishery in 1994 dollars was nearly $97 million based on the NRB survey of 1991 (pers. comm. to Stuart MacDiarmid from John Holloway, DoC, December 1994). In another submission during the same risk analysis, the Taupo Fishery Advisory Committee similarly calculated the Taupo fishery to be worth $96.8 million based on the NRB report (pers. comm. to the Prime Minister from Graham Pyatt, Chairperson of the Taupo Fishery Advisory Council, December 1994).
Salmonid aquaculture industry

Fish farming in New Zealand is covered by the Conservation Act 1987 and the Fisheries Act 1996, and regulations made under both of these Acts. All salmonid species present in New Zealand fall within the definitions of fresh water fish and sports fish within the Conservation

Act.

The Conservation Act prevents the farming and sale of all sports fish, unless this is done pursuant to the Fisheries Act. The Freshwater Fish Farming Regulations defines the fish species which are covered by these regulations as those species specified by notice in the Gazette by the Chief Executive of the Ministry of Fisheries. The species of salmon listed by notice in the Gazette includes Atlantic salmon, quinnat/chinook/king salmon and sockeye salmon. Trout species are not on the list. The effect of this legislation is that salmon species are governed by the Fisheries Act and its Freshwater Fish Farming Regulations, which allows them to be farmed and sold, and trout species are governed by the Conservation Act, which bans them from being farmed or sold. Imported fish is not covered by these statutes.

The apparent dissonance between scientific logic and this legislation is obvious when the taxonomic position of the species involved is examined. The genera *Salmo* and *Oncorhynchus* are represented in both groups (salmon which may be farmed and trout which may not be). Neither is the designation based upon the biology of the species. Populations of sockeye and Atlantic salmon in New Zealand are completely landlocked in fresh water. Thus, species adapted in the New Zealand environment to fresh water are also represented in both groups.

The legislation covering aquaculture in New Zealand also includes the Resource Management Act 1991, which requires environmental considerations to take priority over other considerations when aquaculture proposals are considered.

The net effect of the legislation covering aquaculture in New Zealand is that any expansion of aquaculture activities, either designation of new farming sites or diversification of farming activities into new salmonid species, is likely to be difficult to achieve.

The New Zealand Salmon Farmers Association (NZSFA) is the national body representing the salmon aquaculture industry. The Fishing Industry Board of New Zealand (FIBNZ) is the national body representing the fisheries industries of New Zealand, and in that role also represents the salmon aquaculture industry. In August 1996 MAF requested that NZSFA and FIBNZ provide information allowing the value of the salmonid aquaculture industry to be assessed. Information has been received from FIBNZ, Mark Gillard (President of NZSFA), Paul Steere (King Salmon Company of New Zealand Ltd), and Kevin O'Sullivan (Big Glory Seafoods Ltd and NZSFA representative to the Agricultural Security Consultative Committee).

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. 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. 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). This figure represented less than 1% of the 575,000 tonnes total world production of farmed salmon in 1995 (statistics on international production from Food and Agriculture Organisation). Total world salmon production in 1995, including wild salmon harvests from regions such as Alaska

and British Columbia, was approximately 1,479,000 tonnes. In 1996 New Zealand produced 5,986.3 tonnes of salmon products worth $34.7 million. The total value of New Zealand's fisheries' exports was $676 million in 1995 and $643 million in 1996. Salmonid aquaculture is contributing significantly to the total export earnings as a commodity with relatively high value per unit volume.

Although the domestic production of farmed salmon remains a relatively low volume by international standards, annual production continues to increase at a considerable rate. The 1996 production represents a 43% increase over and above the previous year's production and a 200% increase over and above 1991 levels. These figures suggest an approximate 40% annual increase in production consistently over each of the last 6 years. This increased production is made all the more remarkable by the adversarial legislative climate in which the domestic salmon aquaculture industry exists and apparently flourishes. A 1992 report into the industry (Wildman, 1992) estimated that the New Zealand industry's production capacity was 3,000 tonnes, and that increases beyond that figure would be limited by the government's environmental policies.

Salmon aquaculture in New Zealand has been a privately owned and operated industry from its conception. Government support for the industry has been solely in the form of research conducted jointly with industry into smolt production and disease status. The most recent review of the New Zealand salmon aquaculture industry was prepared by the United States Department of Commerce National Marine Fisheries Service (NMFS) in 1992 (Wildman, 1992). There has been considerable change in the industry since that report.

To date all commercial salmon farming operations in New Zealand are in the South Island. After a recent merger of two of the three largest salmon aquaculture companies, in 1997 the industry comprises two companies which together produce over 90% of the total farmed salmon output and a number of very much smaller companies. The two largest companies are the King Salmon Company of New Zealand Ltd, based in the Marlborough Sounds and Nelson, and Big Glory Seafoods Ltd, based in Big Glory Bay in Stewart Island. There are a number of smaller operators in the South Island farming in sea water net pens around Banks Peninsula or in fresh water ponds.

Vertical integration is a feature of the structure of the two major companies within the New Zealand salmon aquaculture industry. This, their geographical separation, and the industry's separation from the enhancement activities of the recreational fishery, probably leads to a minimum of impacts resulting from each other's activities. Thus, a biosecurity strategy appears to have evolved, presumably by chance. The two major companies' supply of surplus hatchery stocks to smaller aquaculture enterprises is a potential source of disease dissemination. Effective hatchery disease surveillance minimises this risk.

1   King Salmon Company of New Zealand

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. King salmon is a species of salmon which, although of relatively high value in the market place, is not farmed in large volumes around the world. It is the largest species of Pacific salmon, and its high activity rate in net pens tends to lead to lower feed conversion ratios when compared with the more domesticated Atlantic salmon.

Canada also produces king salmon, and in 1993 British Columbia's total production of aquacultured king salmon was 7,345 tonnes, versus 14,468 tonnes of Atlantic salmon (pers. comm. Greg D'Avignon, Executive Director of the B.C. Salmon Farmers Association, October 1996).

The King Salmon Company of New Zealand Ltd company profile forecasts turnover for 1996 as $48 million, total assets (at peak of production cycle) of $47 million, and a staff of 353 at peak of harvest. The total peak stock inventory is 3.36 million fish in sea cages. The major markets by volume are Japan (64%), New Zealand (23%), Australia (5%), Asia (5%), and the USA (3%). The company profile notes that poor returns from the Japanese market in recent years are a result of the strong New Zealand dollar and weak Yen, as well as competition from other international producers. World salmon production is noted as increasing by 37% since 1990. The relatively weak domestic market for salmon has also been identified by the company as cause for concern, and a domestic marketing drive is planned. Access for uncooked salmon into Australia is noted as a priority issue which the company will vigorously pursue.

In 1996 the company was operating six sea cage sites in Marlborough, with a further two sites either leased and held for future expansion or undergoing planning approval procedures. The company also operates a fresh water farm site in Takaka, as well as three hatchery facilities producing 2.2 million smolt annually. All processing is undertaken at one of six company owned processing sites in Nelson.

2   Big Glory Seafoods Ltd

Big Glory Seafoods Ltd is wholly owned by the New Zealand fishing company Sanford Ltd. The company operates two farm sites, both in Big Glory Bay on Stewart Island. The company's salmon processing premises is in Bluff. Three hatcheries are owned and operated by the company, in Kaitanganta, Waitaki and Spring's Creek. In recent years the company has only farmed king salmon. The annual production of salmon by Big Glory Seafoods is approximately 1,100 tonnes, valued at around $8.2 million. The company employs 7 hatchery staff, 22 farm staff, and up to 90 factory staff on a seasonal basis (pers. comm. Kevin O'Sullivan, Big Glory Seafoods Ltd, March 1997).

Conservation groups

The role of DoC and NZFGC have already been discussed with reference to the recreational fishing industry. It is worth noting again that these agencies undertake a regulatory role and perform enhancement work in salmonid fisheries pursuant to the Conservation Act 1987. Specifically, in section 6 of the Conservation Act the roles of DoC are defined as to preserve so far as is practicable all indigenous fresh water fisheries and protect recreational fresh water

fisheries and fresh water fish habitats. The fact that salmonid fish species are prized by anglers and have become an important sport fishery is perhaps secondary to the principal reason for DoC's role in their conservation, which results from a recognition of the intrinsic value of particular species of New Zealand's indigenous and introduced fresh water fishes. Such recognition naturally defies attempts to quantify value. An analysis of the historical and continuing annual investment in conservation of fresh water fisheries presents one means by which their value might be quantified, but no such data have been presented by DoC.

New Zealand is party to the United Nations Environment Program Convention on Biological Diversity (the Biodiversity Convention). The overall objectives of the Biodiversity Convention are the conservation of biological diversity, the sustainable use of its components and the fair and equitable sharing of the benefits arising out of the utilisation of genetic resources. New Zealand's participation in the Biodiversity Convention is a statement of recognition of the intrinsic value of indigenous species, and conservation of indigenous fresh water fisheries is a responsibility arising out of this participation. Some conservation work for indigenous fresh water fish might indirectly benefit salmonids, such as through restoration of shared habitats and through increasing food supplies.

Salmonids are introduced species here, so their conservation and enhancement is not a responsibility arising out of the Biodiversity Convention, although whether approximately 70 years of isolation (the last introductions of salmonids into New Zealand were probably in the 1920s) may have led to a degree of genetic diversity in New Zealand salmonids compared with overseas stocks has not been assessed. Certainly the tendency for salmonids to return to the exact location of their spawning grounds is recognised as having led to specific genetic stocks spawning in distinct geographical locations in areas where salmonids are indigenous, such as the Pacific north west of America. Habitat modifications, or other mechanisms, in regions sustaining populations from which New Zealand salmonid stocks were derived may have lead to original stocks becoming endangered, another potential means by which the genetic diversity of our salmonids might have come to require protection. This hypothesis is similarly unassessed and beyond the scope of this analysis.

When the conclusions of McDowall (1990) are considered, that in some instances the presence of introduced salmonids has an adverse impact on indigenous fresh water fish species, it seems apparent that the conservation and enhancement of salmonids in this country must be carefully managed in order that it does not directly contravene the Biodiversity Convention.

Maori

The utilisation by Maori of fresh water fisheries resources prior to and subsequent to the introduction of salmonids was reviewed by McDowall (1990). Maori have traditionally utilised fresh water fisheries as a food source, and this utilisation extended to salmonids following their introduction. In more recent times Maori fishing rights embodied in the Treaty of Waitangi have been recognised as having precedence over the Crown's fisheries laws. This has lead to renewed recognition of the value of fresh water fisheries to Maori. In regions where the salmonid recreational fishery is an important economic resource Maori have become involved in fishery management. For example, the Tuwharetoa Maori Trust Board is represented on the Taupo Fishery Advisory Committee.

Traditional Maori utilisation of fresh water fisheries has long been recognised as being sustainable and based upon principles of conservation (McDowall, 1990, citing various authors). The value to Maori of the fresh water fishery is derived from both the actual and potential utilisation of the fishery as well as, and possibly more importantly, the intrinsic value of the fresh water fishery through it being part of the natural resources of New Zealand. Although value may be able to be quantified through estimating the actual and potential utilisation of fresh water fish by Maori, whether such a process would provide a good indication of the true value to Maori is questionable.

New Zealand public

The utilisation and value of the salmonid fishery to the New Zealand public is considered to be embodied within the discussions above.

3.5 HEALTH STATUS OF FISH IN NEW ZEALAND

The health status of fish in New Zealand has been determined through aquatic animal health surveillance. In 1977 MAF established a specialist fish disease diagnostic section at the Central Animal Health Laboratory (CAHL), Wallaceville, Lower Hutt. Since the early 1990s other organisations derived from MAF, including the Ministry of Fisheries and the National Institute of Water and Atmospheric Research, have also played a role in surveillance. Specific studies for research purposes have also contributed significantly to current knowledge of the pathogens and parasites present here.

In the period 1985 to 1994 CAHL conducted export testing of salmon farms to support exports of salmon to the USA. All lots (year, classes and species) on about 20 South Island salmon farming properties were tested annually for whirling disease and viruses. During this period 23,183 salmonids were sampled and tested by viral tissue culture examination, and 1,809 salmonids were sampled and tested for bacterial diseases (Anderson, 1996). A survey for infectious haematopoietic necrosis virus (IHNV) in wild and hatchery sockeye salmon concluded that New Zealand is free of detectable IHNV (Boustead et al, 1993). A survey for *Aeromonas salmonicida* concluded that New Zealand is free of this bacterial pathogen of salmonids (Anderson et al, 1994).

The following diseases and parasites of fish 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 (Hine, 1995). IPNV is recognised as a salmonid-pathogenic strain of a birnavirus that has many other serotypes (Wolf, 1988). Because strain differences exist internationally, IPNV must be considered during risk assessment.

Lymphocystis

The iridoviral disease lymphocystis is an infection of many fish species, and is particularly troublesome in ornamental fish. It is not known to affect salmonids. Lymphocystis has been diagnosed in New Zealand on two occasions in imported tropical aquarium fish which were still under quarantine (Durham and Anderson, 1981). That report notes that on one other occasion lymphocystis was diagnosed in a marine fish in New Zealand. The disease condition causes chronic hypertrophy of cells in the peripheral vasculature, often epithelial cells, with only a slight adverse impact on the host (Wolf, 1988).

Bacteria

Yersinia ruckeri

A strain of *Y. ruckeri*, causing enteric redmouth disease (ERM), has been isolated from salmonid farms and hatcheries here (reviewed by Hine, 1995). Numerous strains of *Y. ruckeri* are known to occur internationally (Austin and Austin, 1993). 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). Because of these strain differences, *Y. ruckeri* must be considered during risk assessment.

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). Because of these strain differences, *Vibrio anguillarum* must be considered during risk assessment.

Bacterial gill disease

Bacterial gill disease (BGD) is a condition primarily associated with yellow pigmented, filamentous, Gram-negative bacteria of the genera *Cytophaga, Flexibacter* and *Flavobacter* (reviewed by Turnbull, 1993). Bacterial gill disease occurs in New Zealand (Boustead, 1985). However, the bacteria associated with BGD infections in New Zealand have not been precisely defined, so BGD associated organisms will be examined during risk assessment.

Flexibacter columnaris

F. columnaris is the cause of columnaris disease, an infection of external tissues of many species of fresh water fish under environmental conditions favourable to the bacteria and stressful to the fish (reviewed by Wakabyashi, 1993). Many strains of the bacteria exist internationally, with a wide variation in virulence. *Flexibacter* sp. and *F. columnaris* occur in New Zealand (Boustead, 1982; Anderson, 1996). They have been recorded 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. Because of these uncertainties, *F. columnaris* will be considered during the risk assessment.

Aeromonas hydrophila

A. hydrophila is associated with a variety of disease conditions in aquatic animals, very often as a secondary invader of any ulcerated tissues in fresh water fish. *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). No significant strain variations are recorded for this bacterium (Austin and Austin, 1993).

Clostridium botulinum

Clostridium botulinum causes the normally fatal human ailment called botulism. Botulism associated with type C toxins occurs in New Zealand, although rarely (Hine, 1978). They are thought to originate from migrating sea birds.

Nocardiosis

Nocardia asteroides is considered to be an opportunistic pathogen of many fish species with a worldwide distribution (reviewed by Frerichs, 1993). The organism is abundant in soil. Losses of quinnat salmon in 1972 at one fresh water salmon farming location in New Zealand were attributed to nocardiosis (Boustead, 1985). No significant strain differences are known to occur.

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). In Tasmania *Paramoeba sp.* causes severe problems with amoebic gill disease in cultured Atlantic salmon (reviewed by Humphrey, 1995). Whether the difference in impact is the result of strain variations or environmental factors has not been studied. For this reason, *Paramoeba sp.* is considered during the risk assessment.

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). Strain differences do not occur. For this reason, *Myxobolus cerebralis* will not be considered during the risk assessment.

Ichthyophthirius multifiliis

Ichthyophthirius multifiliis is a protozoan parasite which 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. The possibility of strain variations exists, but these are likely to be strains physiologically adapted to temperature tolerance of host fishes rather than strains with a wide variation in pathogenicity (Dickerson and Dawe, 1995).

For a complete list of the other protozoan parasites recorded in New Zealand fishes, refer to Table 1.

Metazoa

Numerous species of metazoan parasites have been recorded in New Zealand fishes. A complete list is recorded at Table 1. Two review articles of aquatic animal parasites present in this country (Hewitt and Hine, 1972; Boustead, 1982) are the principal sources for the information presented in Table 1 for protozoan and metazoan parasites. The most comprehensive review article on parasites of New Zealand fishes (Hewitt and Hine, 1972) compiled data on the parasites of 140 fish host species, and noted that this represented only about one-quarter of the total fish species recorded from New Zealand waters. Despite compiling a list of 356 species of parasites, the authors concluded that the parasites of New Zealand's fish are poorly known.

3.6 THE CONSEQUENCES OF SALMONID DISEASE INTRODUCTION INTO NEW ZEALAND

Risk is a function of the probability of an adverse event and the consequences resulting from the event. The Biosecurity Act 1993 recognises this, and at section 21 requires any importation proposal to be assessed for its potential to introduce unwanted organisms into New Zealand, as well as the nature and possible effect on the people, the environment, and the economy on any unwanted organism introduction.

The SPS Agreement requires that when member countries of the WTO assess the risk to animal life or health and determine the sanitary measures to be applied to achieve the appropriate level of sanitary protection from such risk, they shall take into account as relevant economic factors the potential damage in terms of loss of production or sales in the event of the entry, establishment or spread of a pest or disease; the costs of control or eradication in their territory; and the relative cost-effectiveness of alternative approaches to limiting risk.

The OIE has defined the consequences of an aquatic animal disease incursion which should be considered as those adverse consequences affecting aquatic animal health, human health, aquatic ecology and ecosystems and the environment (OIEa, 1995).

In this study the potential consequences of a salmonid disease introduction are assessed through the following means:

1   By reference to the detailed Australian study on the impact of salmonid diseases within the *Salmon Import Risk Analysis* (DPIE, 1996);

2   Through an examination of the impact of the salmonid diseases already present in New Zealand;

3   Through an examination of the impact of salmonid diseases exotic to New Zealand in the overseas countries in which they occur;

4   In the absence of data on native salmoniform susceptibility to exotic salmonid diseases, through discussion of the range of potential consequences of disease introduction for native salmoniform fish.

Australian *Salmon import risk analysis*

The Australian *Salmon import risk analysis* (DPIE, 1996) included a section titled *Impact of salmonid diseases introduction into Australia* by the Australian Bureau of Agricultural and Resource Economics (ABARE). The document drew upon earlier reports utilising a stochastic investment model to estimate the long term impact on salmonid aquaculture and recreational fisheries in Australia likely to result from introduction and establishment of two diseases, furunculosis and infectious haematopoietic necrosis (IHN). The document also drew on information received in submissions during a period of public comment. The ABARE report suggests that the major salmonid industries would no longer be viable if important salmonid diseases were to become established in Australia. ABARE also indicated that there might be

social and economic impacts on the recreational fishing sector, and environmental effects including possible impacts on native fish and ecosystems. The major considerations in reaching these conclusions were the following:

1   Once established in a wild population, eradication of aquatic animal diseases is considered unlikely.

2   Within the salmonid aquaculture industries, impacts would be through lower survival rates of stock, costs of treatment of disease, and the loss of Australia's disease free status resulting in price effects for aquacultured Atlantic salmon exports to Japan.

3   The concentration of salmon farming in Australia within Tasmania, and the localised effects on the region resulting from disruption caused by a disease outbreak.

4   The difficulty in establishing the full impact of a disease outbreak in the tourism-based recreational fishing industry.

5   The uncertainty regarding susceptibility of native fish to salmonid diseases.

In deciding whether the ABARE report's conclusions are of relevance to the New Zealand situation, the similarities and dissimilarities between the New Zealand and Australian situations must be assessed.

Two important dissimilarities are immediately apparent: the environment and the salmonid species being farmed. Australian water temperatures are marginal for salmonids, and this was noted within the report as potentially increasing disease effects. The impact of *Paramoeba sp.* is certainly far greater in Tasmanian salmon aquaculture than in New Zealand despite that organisms presence here. The species of salmon which is most commonly farmed in each country differs, and this will certainly make a difference to the potential disease impact.

However, salmonid aquaculture industries in both countries operate in a relatively disease free environment with imported salmonid stocks derived from a limited genetic base. The Australian *Salmon import risk analysis* hypothesises that this situation will have led to Australian salmonid stocks having little inherited resistance to disease. This is an untested hypothesis, but it is used to conclude that there may be increased disease impact relative to impacts in countries where diseases are endemic.

Likewise, the same argument is used to hypothesise that tourism-based recreational fishing sectors in Australia are dependant on introduced salmonids surviving in the wild in environments to which they have had limited time to fully adapt. The result may be environmental stress, such as occurs from relatively higher water temperatures, leading to increased disease impacts. These impacts may limit the effectiveness of enhancement activities. If this were true, the same situation may apply in New Zealand.

In both countries there are populations of native salmoniform fish, and their susceptibility to salmonid diseases has not been assessed. The Australian *Salmon import risk analysis* notes

this absence of evidence, and takes a conservative position through an assumption that native salmoniform fish may be at least susceptible to disease as the most susceptible salmonid species. However in apparent contradiction to this assumption, the Australian *Salmon import risk analysis* further notes that there is no reason to consider that the risk to native salmoniforms would be significant for most pathogens which are, from experience overseas, characteristically specific in their host range.

Impact of the salmonid diseases already present in New Zealand

The manifestation of disease is a result of interaction between a host species, a disease agent and the environment. Salmonids are introduced species in New Zealand, and for this reason the impact the environment plays in disease manifestation could be expected to differ from that in other countries. The potential reasons for this difference include:

- The distribution of salmonids in New Zealand differs from other countries. Salmonid aquaculture facilities (including hatcheries focussing on wild stocks enhancement) tend to occur at relatively low density in New Zealand, with little geographic concentration. The concentrations that do occur, in Stewart Island and the Marlborough Sounds, are the result of a single company's activities in each case. Wild salmonids in New Zealand probably occur at densities lower than in regions where they are indigenous. This may mean a lower likelihood of diseases spreading epidemically or maintaining a high prevalence.

- New Zealand's meteorological and oceanographic conditions differ from other countries. This may affect host susceptibility, the clinical manifestation of disease, pathogen environmental survival, and the ability to transmit.

- New Zealand's native fauna and flora is unique, which may mean that species important in disease transmission as intermediate hosts do not occur, effectively blocking transmission, or other species may exist which amplify disease transmission or indirectly affect disease manifestation in unforeseen ways.

For the above reasons the impact of a disease incursion and establishment in New Zealand would not be expected to be completely predictable based on overseas experiences alone. An examination of the impact of those salmonid pathogens and parasites which already occur here may provide some indication of the impact of further disease introductions.

No major viral pathogens of international importance cause disease in New Zealand salmonids. An IPN-like birnavirus does occur, as demonstrated by isolations from returning salmon (Tisdall and Phipps, 1987). No clinical manifestations of disease have been attributed to the presence of this virus, and the impact on salmonids and native salmoniform fish of this IPN-like birnavirus is suspected to be negligible. However, because this virus is suspected to be a lowly pathogenic strain of IPNV, this example does not provide a representative indication of the potential impact of a major salmonid viral pathogen introduction.

The bacterial pathogens *Yersinia ruckeri*, *Vibrio anguillarum* and *V. ordalii* occur here (Boustead, 1982; Wards et al, 1991; Anderson et al 1994). In the case of *Y. ruckeri* and *V.*

anguillarum, strain differences in pathogenicity exist between New Zealand isolates and overseas isolates.

The first isolation of *Y. ruckeri* was made in 1982 from dying fingerlings on a Canterbury salmon farm. A second South Island salmon farm was found to be infected in 1990. Between 1991 and 1993 a survey was undertaken to determine the distribution of *Y. ruckeri* (Anderson et al, 1994). A total of 877 salmonids and indigenous fish, from 24 sites, were cultured. Isolations of *Y. ruckeri* were made from the two previously known infected salmon farms in the South Island, and from a North Island location. Subsequent investigation failed to confirm the presence of *Y. ruckeri* at the North Island site. *Y. ruckeri* is a notifiable organism, and since the 1994 survey there have been occasional isolations from South Island farmed quinnat salmon. In the known infected South Island sites, *Y. ruckeri* infections are clinically manifested as a relatively sudden onset of increased daily mortality (approximately 0.2% of the population per day) in juvenile quinnat salmon in fresh water during spring or early summer. Outbreaks are controlled by reducing stocking densities and feeding oxytetracycline. The impact of *Y. ruckeri* appears to be confined to the economic impacts on individual farms resulting from increased mortalities and the cost of control measures. There appear to have been no impacts on indigenous fish or on the recreational fishery as *Y. ruckeri* has not been isolated from fish other than farmed salmon and does not appear to be present in the North Island. This is despite the absence of regulatory intervention leading to major control and eradication efforts upon detection in salmon farms in recent years, and also despite salmon products from the South Island, presumably as well as recreational fishing equipment, being transferred regularly to the North Island. Highly pathogenic foreign strains of *Y. ruckeri* may not, however, follow the pattern established by the present New Zealand isolates.

Vibrio anguillarum appears to be prevalent in New Zealand, and has been isolated from sea water and fish from marine sites where salmon are farmed (Powell and Loutit, 1990). There have been no published reports of isolations from diseased fish here, although vibriosis-like disease in salmon farms is reported. Late access to these fish by laboratories has prevented aetiological identification. Powell and Loutit reported that the salmon farming industry was using vaccines produced overseas, despite the effectiveness of these vaccines against local isolates being questionable. New Zealand isolates are not reactive to antiserum to whole cell preparations of closely related foreign strains.

Anderson (1996) notes that disease outbreaks in salmon caused by *V. ordalii* have been rare since 1989 and antibiotic medication and vaccination are no longer necessary. *V. ordalii* was first identified here in 1984, and isolates are closely related to overseas strains (Wards et al, 1991). Disease due to *V. ordalii* was common during the warmer summer months in the early years of sea-cage rearing of salmon in New Zealand. There are no reports of vibriosis occurring in native salmoniform fish, although transmission studies have not been undertaken.

The available evidence suggests that *V. anguillarum* and *V. ordalii* in New Zealand have had minimal continuing impact on salmonid aquaculture, the recreational fishing industry and native salmoniforms, once appropriate management techniques to control disease outbreaks had been developed. The difference between New Zealand and foreign isolates of *V. anguillarum* may be significant.

Whirling disease was first identified in New Zealand in 1971 (Hewitt and Little, 1972), but was present for at least 5 years before it was identified (Boustead, 1993). Surveys to determine the extent of the parasite's distribution and the disease impacts have periodically been undertaken (Hewitt, 1972; Boustead 1993; Boustead, 1996). Whirling disease has been found in nine rivers or streams and seven hatcheries on the east coast of the South Island from the Waimakariri River to the Clutha River. In some cases isolation has prompted attempts at eradication through depopulation, but this has invariably been futile. A process of fish transfer authorization for controlling the spread of the disease is in place under the provisions of the Freshwater Fish Farming Regulations and through the requirements of DoC. Guidelines on restrictions on movements of fish from infected farms are used by Ministry of Fisheries when considering applications to transfer live fish from licensed fish farms or a sport fish hatchery. A control area is established on the east coast of the South Island and fish transfers beyond the control area are subject to more stringent test requirements. There are no controls on movement of dead fish and fishing equipment.

The presence of whirling disease has no direct adverse impact on the salmonid aquaculture industry. The most important aquaculture sites in New Zealand are outside the endemic area, and the salmonid species commercially aquacultured here are of low susceptibility to whirling disease. Boustead (1996) surveyed wild rainbow trout in the whirling disease endemic area, and concluded that the low incidence and intensity of infections and the absence of a decline in trout numbers probably indicate the parasite is not having a significant detrimental effect on rainbow trout populations. There are no reports of whirling disease in native salmoniform fish, although transmission studies have not been undertaken. The evidence suggests that the presence of *Myxobolus cerebralis* has had negligible direct impact on salmonid aquaculture, the recreational fishing industry and native salmoniforms in New Zealand. An important indirect effect of whirling disease presence is quarantine restriction placed on exports of salmon products to Australia.

New Zealand's relatively favourable aquatic animal health status makes an examination of impacts of endemic disease of limited relevance in predicting the potential impacts resulting from the introduction of serious exotic salmonid pathogens. Endemic diseases have had little impact on salmonid aquaculture, the recreational fishing industry and native salmoniforms. In some cases direct impacts, through stock losses and costs associated with management and treatment of disease, have resulted during the early period following pathogen recognition. Ongoing indirect impacts, such as the effect on market access, are important in specific cases.
Impact of salmonid diseases exotic to New Zealand in the countries in which they occur

Examining the experience of other countries may provide an indication of the potential impact on salmonid industries in New Zealand resulting from disease introduction. The potential impact of the New Zealand environment on disease manifestation will not be predictable in this examination.

Disease impacts may vary for the same disease between salmonid species, or in some cases salmonid stocks of the same species, and between regions, salmonid management systems, and salmonid industry sectors. The impact may also vary for endemic salmonid diseases in regions with indigenous salmonids, newly introduced diseases in regions with endemic salmonids, introduced salmonid species in regions with endemic salmonid diseases, and, of probably most

significance to New Zealand, newly introduced diseases in regions with introduced salmonids.

In the Pacific north west of North America IHNV is endemic. The strain present in Alaska is sockeye-adapted. IHNV has been suspected as the cause of epidemics in sockeye salmon as early as 1925. Epidemics have also occurred in chinook, chum and Atlantic salmon and in rainbow, steelhead and cutthroat trout. In Pacific salmon IHNV is a fresh water-associated disease, with spawning adults shedding high titres of horizontally transmitted virus in sexual fluids, and mortalities in alevins and pre-smolts in fresh water.

IHNV has hampered enhancement activities for sockeye salmon. In the period 1937 to 1975 no sockeye salmon releases were made in Alaska. Since 1975 sockeye enhancement programmes have been progressively less affected by IHNV and gradually more successful sockeye rearing to release has been possible as a result of greater understanding of the epidemiology of IHNV. The *Alaska Sockeye Salmon Culture Manual* (McDaniel et al, 1994) details the present Alaskan Department of Fish and Game recommendations for successful sockeye salmon culture. Iodine disinfection of eggs, virus-free water supplies and compartmentalisation in hatcheries have been successful in limiting outbreaks of IHNV.

The current impact of IHNV in sockeye salmon culture in Alaska results from occasional disease outbreaks in hatcheries, leading to stock losses and costs associated with control, the on-going research effort, and costs to hatcheries associated with implementation of specific management procedures.

When Atlantic salmon were introduced to the Pacific northwest of North America they probably had no previous exposure to the endemic IHNV strain. Atlantic salmon have been demonstrated to be more susceptible than sockeye and chinook salmon after exposure by immersion, injection and cohabitation (pers. comm. G S Traxler, October 1996). As a result, the disease has been manifested in a very different manner in cultured Atlantic salmon. While disease in Pacific salmon is largely confined to fresh water, epidemic mortalities have occurred in Atlantic salmon in sea water in growing fish up to harvest weights. The absence of effective preventive and therapeutic measures during grow-out phases has led to significant impacts in Atlantic salmon aquaculture in British Columbia.

The myxosporean parasite *Ceratomyxa shasta* is endemic in the Pacific north west of North America, and has been identified in salmonids in marine and fresh water environments from northern California to British Columbia (Bartholomew et al, 1989). High losses in susceptible strains of salmonids have occurred, in hatchery-reared and wild juveniles as well as in pre-spawning adults.

Although all salmonid species are probably susceptible, juveniles from waters containing the infective stage of the parasite are usually more resistant than strains from areas free of the parasite. The most successful approach to control is the introduction of resistant salmonids. Relocation of salmonids from areas where *C. shasta* is not present into areas where the parasite is endemic is likely to lead to serious disease in the relocated fish. Such introductions may also adversely affect the survival of resistant resident strains if interbreeding occurs.

The geographic isolation of the parasite despite large volume live fish and egg exports from

endemic areas is compatible with a hypothesis that an unknown factor local to the region is required for the completion of the life cycle. If so, New Zealand is probably not at risk of *C. shasta* introduction. However, the example illustrates that pathogens and parasites may impact upon salmonids in unforeseen ways, and that this impact may vary as a result of indirect influences.

Infectious salmon anaemia (ISA) provides an example of a previously unknown disease emerging and impacting in local salmonid aquaculture. Some aspects of disease epidemiology would be expected to already be known if an exotic disease agent is introduced into New Zealand, whereas nothing was known about ISA in 1984 when it first occurred in Norway. The Norwegian salmon aquaculture industry has suffered large direct costs associated with mortalities and disease control measures, as well as indirect costs associated with compliance with measures for product exports imposed by significant markets such as the EU. On top of this, there has been a huge research effort directed at elucidation of key elements of ISA epidemiology. The total impact of ISA is likely to be very large, but current research efforts remain directed at the nature and biology of the infectious agent and the epidemiology of the disease, and no report of the actual magnitude of impacts associated with ISA has been made to our knowledge. In an opening address to an ISA Workshop in September 1993, Bjørn Næss, Director of the Norwegian Central Veterinary Laboratory noted that losses due to fish disease problems in Norway amount to around US$100 million per year.

Norway's experience with the monogenean gill parasite *Gyrodactylus salaris* provides a further illustration of the consequences which may result from a disease or parasite introduction. The parasite is endemic in fresh waters draining into the Baltic Sea, and it is suspected that relatively recent introduction into Norwegian rivers occurred from that source. The parasite has become established in wild Atlantic salmon stocks in Norwegian rivers, causing mortalities. It is easily controlled in hatcheries through formalin treatment of water supplies. The parasite does not survive in full strength sea water.

Declines in wild stocks have been attributed to the parasite, and an eradication effort has been mounted. The eradication programme involves rotenone treatment of complete river systems followed by re-stocking with parasite free stock. This has been expensive and disruptive to ecosystems.

Chile has been imposing sanitary measures during importations of salmonid eggs, but despite these measures now recognises that BKD has been introduced and has become endemic. There is no effective treatment for BKD, although erythromycin injections of brood stock is effective in reducing, but not eliminating, vertical transmission. In aquaculture BKD is managed within hatcheries by techniques such as graded ELISA scoring identifying infected brood stock, the destruction of eggs from such fish, and through reducing stress in juvenile classes. The direct impacts of BKD are associated with increased mortality and the costs of disease control. Trade restrictions relating to BKD typically apply for live fish, eggs or uneviscerated products, whereas Chile's most valuable salmonid exports are for human consumption.

Australia is free of the major salmonid pathogens and parasites (DPIE, 1996). Despite enjoying this status, salmonid aquaculture in Australia suffers infectious disease problems. Amoebic gill disease (AGD) is the most important infectious disease problem facing

Tasmanian sea-cage salmonid culture (Munday, 1996). Control of AGD, achieved through gradual acclimatisation to sea water, fresh water bathing during outbreaks, and by farming the very susceptible sea trout only in brackish water, imposes management restrictions and costs.

AGD has also been recorded in British Columbia (Kent, 1992) and Ireland (Rodger and McArdle, 1996). The infectious agent associated with this disease, *Paramoeba sp.,* is a free-living opportunistic organism known to occur in New Zealand. Despite the presence of the organism, no serious disease manifestations occur here. This illustrates the importance of factors other than disease agent presence in predicting the consequence of disease agent entry.

Ascribing significance to the range of factors potentially contributing to the decline in wild salmonid populations in various European countries and on the Atlantic coast of North America is difficult. These factors include disease, habitat destruction, water quality and over-fishing. Hatchery enhancement programmes overseas, for example in the Pacific northwest of North America, have demonstrated that even in the presence of serious endemic diseases, salmonid populations devastated by habitat modifications (such as damming rivers for power projects) can be managed to retain a recreational fishing resource. Norwegian experience shows that even with emerging pathogens and introduced parasites a strong local aquaculture industry can exist. In both of these examples, significant financial investments have been made to ensure that impact on salmonids of disease and from environmental threats are managed to ensure fisheries are sustained.

Overseas experience indicates that once a disease is introduced into an aquatic animal population, eradication is unlikely. In some cases where introduction has been detected early and within a contained population such as a hatchery, stamping out has been effective (e.g. Norwegian stamping out of VHS in 1974).

The overseas examples, and the qualitative assessment of salmonid pathogens (section 4.1), illustrate that emergence of previously unknown disease conditions is a feature of salmonid aquaculture. Hitra disease in Norway and piscirickettsiosis in Chile are examples.

Native salmoniform fish

As noted previously, an Australian review attributed significance to the uncertainty of disease effects in native salmoniform fish (DPIE, 1996). This study assumed that native salmoniform fish may be at least as susceptible to disease as the most susceptible salmonid species. Such an assumption leads to a conservative view of the health risks for native salmoniform fish, which is that diseases could cause epidemics amongst native salmoniform fish.

Such a conservative view has not been substantiated in the case of endemic salmonid diseases. The fact that these diseases have not been recorded in native salmoniform fish in Australia or New Zealand suggests that the actual health risk to native salmoniform fish may be less than for salmonids. It is possible that the potential for native salmoniform fish to become infected is inversely proportional to the host specificity of a particular pathogen or parasite. That is, native salmoniform fish may be most at risk from the least host-specific pathogens or parasites. The probability of native salmoniform fish becoming infected would then progressively

decrease as susceptibility for any particular disease is confined to specific families within the Order Salmoniformes, to specific genera within the family Salmonidae, and to specific species within genera.

The risk of infection leading to an adverse impact such as clinical manifestation of disease or mortality, as opposed to subclinical infection, is more difficult to determine. Once again, one might conservatively assume clinical manifestation to be similar to that in the most severely affected salmonids, or assume manifestation similar to the most closely related susceptible species.

Conservative assumptions lead to conclusions of widespread epidemics and mortalities affecting native salmoniform fish. While recognising that native salmoniform fish may be more susceptible to infection and clinical manifestations may be more severe than in salmonid fish, the alternative assumptions described above should be examined so that a balanced view of the range of likely effects of a disease introduction is achieved.

IPNV has been known to infect representatives from at least 20 families, from primitive cyclostomes to advanced boney fishes (Wolf, 1988). As such, it might be assumed likely to infect native salmoniform fish. In many cases IPNV has been isolated from fish that appeared to be healthy. It is virulent for salmonids, and has also been shown to cause disease in non-salmonid fish such as anguillid eels. The manifestation of infection in native fish might be sub-clinical, as in many fish species, or clinical disease, such as in salmonids.

The natural hosts for IHNV are sockeye and chinook salmon, and rainbow and steelhead trout. Other susceptible species are Atlantic, chum and masou salmon. Cutthroat, brook and brown trout may be experimentally infected. Coho salmon are considered resistant, although adult carriers have been reported. Non-salmonid fishes (tubesnouts, shiner perch and Pacific herring) may be experimentally infected and suffer mortality (pers. comm. G S Traxler, October 1996). Thus, susceptibility within salmonid genera is variable, and a wider range of fishes are experimentally susceptible. For these reasons, the likelihood of New Zealand salmoniforms being naturally susceptible is low. However, if native salmoniform fish proved to be susceptible then the likelihood of a clinical disease manifestation is probably high.

The only known natural host of plasmacytoid leukaemia (PL) is chinook salmon. Sockeye and Atlantic salmon may be infected experimentally. The likelihood of native salmoniforms being naturally susceptible is probably negligible.

Although it would be possible to go through the list of salmonid diseases discussed and draw conclusions for each, these examples illustrate that a range of possible impacts exists, and that the pessimistic assumptions relating to Australian salmoniform fish made within the *Salmon import risk analysis* (DPIE, 1996) may not be valid for many of the diseases considered.

A further point worth noting is that as host specificity decreases and, under the alternative assumption presented above, the likelihood of native salmoniform fish being infected increases, the likelihood of disease introduction via pathways other than importation of the *commodity* probably also increases. Such pathways include the importation of ornamental fish, importation of marine fish for human consumption, natural migration and/or population

extensions, and ballast water and fouling of ships.

If this were true, then it would lead to the conclusion that for the organisms most likely to infect native salmoniform fish, importation of the *commodity* presents one pathway amongst many for introduction. When importation of the *commodity* presents the only pathway of introduction host specificity is presumably high, meaning that native salmoniform fish are unlikely to be naturally susceptible.

3.7   CONCLUSIONS

1   The *commodity* has been defined in a manner consistent with industry practice for the production of salmonid products for human consumption. Certification from exporting regulatory authorities could be obtained to verify the definition.

2   There are no verified instances where importations of the *commodity* have led to disease entry and establishment. In many cases of disease agent introduction into a previously believed-free area the route of introduction was not able to be identified.

3   Pathogens and parasites of salmonids which are of international quarantine significance, of economic significance to salmonid aquaculture industries, which adversely impact wild stocks of salmonids, and which are not known to be present in New Zealand exist in countries which might export the *commodity* to New Zealand if access were granted.

4   Introduced salmonids exist and are utilised by a variety of sectors in New Zealand, including the salmonid aquaculture industry, recreational fishers, Maori and the New Zealand public. The presence of salmonids contributes significantly to the economy in a variety of direct and indirect ways. Salmonids also have value other than purely economic.

5   Native salmoniform fish are present. In addition to the value ascribed to these species by the fishery industries, Maori, conservation groups, and the New Zealand public, obligations under the Biodiversity Convention need to be considered. If a salmonid disease were introduced into New Zealand, the impact on native salmoniform fish could range from epidemics with high morbidity and mortality, through to no discernable impact.

6   The consequence for salmonid-based industries of introduction and establishment of a pathogen or parasite of salmonids is difficult to predict, and would depend on many factors. The factors most likely to affect manifestation of disease are the actual organism introduced and its interaction with fish in the New Zealand environment. Impacts resulting from the introduction and establishment of some organisms are likely to be severe, and could cause long term disruption in the salmonid aquaculture industry and in recreational fishery enhancement programmes. Disruption would result from epidemics until management strategies for disease control are developed. For most diseases, management strategies have been developed overseas which are able to minimise disease impact and allow aquaculture and enhancement of wild stocks to continue. However, this invariably incurs costs during the initial research effort and ongoing disease management.

7   The combined economic and environmental impact of a salmonid disease introduction into New Zealand might be severely adverse. Overseas evidence has demonstrated that once introduced, eradication of aquatic animal diseases is unlikely.

Table 1. Pathogens and parasites of teleost fishes recorded in New Zealand.

Virus	Recorded details	Key references
IPNV-like birnavirus	Ocean-run quinnat salmon returning to the South Island	Tisdall and Phipps, 1987
Lymphocystis	Imported tropical fish undergoing quarantine	Durham and Anderson, 1981
Bacteria		
Yersinia ruckeri	Fresh water salmon hatcheries and wild locations of the South Island	Anderson et al, 1994
Flexibacter columnaris	farmed eels; also occasionally in cultured juvenile quinnat salmon and trout	Hine and Boustead, 1974 Boustead, 1982
Vibrio anguillarum	kidney of feral eel in Lake Ellesmere; survey of selected marine sites in New Zealand	Boustead, 1982 Powell and Loutit, 1990
Vibrio ordalii	salmon farms in South Island	Wards et al, 1991
Aeromonas hydrophila	various species of fresh water fish	Boustead, 1982
Mycobacterium marinum	imported tropical fish	Boustead 1982
Nocardia asteroides	fresh water quinnat salmon locations	Boustead, 1985
Hafnia alvei		Anderson, 1996
Clostridium botulinum		Hine, 1978
Fungi		
Saprolegnia sp.	Opportunistic infections	Boustead, 1982
Aspergillus sp.	Opportunistic infections	Boustead, 1982
Trichoderma sp.	Opportunistic infections	Boustead, 1982
Peyronellaea glomerata	Opportunistic infections	Boustead, 1982
Botrytis sp.	Opportunistic infections	Boustead, 1982
Fusarium merismoides	Opportunistic infections	Boustead, 1982
Algae		
Paramoeba sp.	mild disease in salmon farms of Stewart Island and Marlborough Sounds	Anderson, 1996
Protozoa		
Auberachia anomala	gall bladder of ling	Hewitt and Hine, 1972
Auerbachia monstrosa	gall bladder of javelin fish	Hewitt and Hine, 1972
Caliperia longipes	gills of *Ericentrus ruber, Trachelochismus melobesia*	Hewitt and Hine, 1972

Ceratomyxa aggregata	gall bladder of tarakihi	Hewitt and Hine, 1972
Ceratomyxa angusta	gallbladder of *Ellerkeldia semicincta*, sea perch	Hewitt and Hine, 1972
Ceratomyxa arcuata	gall bladder of *Anthias pulchellus*	Hewitt and Hine, 1972
Ceratomyxa castigata	gallbaldder of *Congiopodus leucopaecilus*	Hewitt and Hine, 1972
Ceratomyxa castigatoides	gall bladder of scarlet parrotfish	Hewitt and Hine, 1972
Ceratomyxa constricta	gall bladder of *Centriscops humerosus*	Hewitt and Hine, 1972
Ceratomyxa declivis	gall bladder of *Cyttus australis* , silver dory	Hewitt and Hine, 1972
Ceratomyxa elongata	gall bladder of *Lepidopus caudatus*	Hewitt and Hine, 1972
Ceratomyxa faba	gall bladder of *Arnoglossus scapha*	Hewitt and Hine, 1972
Ceratomyxa flexa	gall bladder of *Plagiogenion rubiginosus*	Hewitt and Hine, 1972
Ceratomyxa gemmaphora	gall bladder of red perch	Hewitt and Hine, 1972
Ceratomyxa gibba	gallbaldder of *Congiopodus leucopaecilus*	Hewitt and Hine, 1972
Ceratomyxa hama	gall bladder of *Arnoglossus scapha*	Hewitt and Hine, 1972
Ceratomyxa inconstans	gall bladder of trevally, sea perch, Japanese mackerel, horse mackerel	Hewitt and Hine, 1972
Ceratomyxa insolita	gall bladder of tarakihi	Hewitt and Hine, 1972
Ceratomyxa intexua	gall bladder of *Plagiogenion rubiginosus, hake*	Hewitt and Hine, 1972
Ceratomyxa inversa	gall bladder of ling	Hewitt and Hine, 1972
Ceratomyxa laxa	gall bladder of *Arnoglossus scapha*	Hewitt and Hine, 1972
Ceratomyxa minuta	gall bladder of hake, barracouta	Hewitt and Hine, 1972
Ceratomyxa moenei	gall bladder of bass	Hewitt and Hine, 1972
Ceratomyxa nitida	gall bladder of sole	Hewitt and Hine, 1972
Ceratomyxa polymorpha	gall bladder of red cod	Hewitt and Hine, 1972
Ceratomyxa recta	gall bladder of ling	Hewitt and Hine, 1972
Ceratomyxa renalis	urinary bladder of *Arnoglossus scapha*	Hewitt and Hine, 1972
Ceratomyxa subtilis	gall bladder of javelin fish	Hewitt and Hine, 1972
Ceratomyxa torquata	gall bladder of *Arnoglossus scapha*	Hewitt and Hine, 1972
Ceratomyxa uncinata	gall bladder of *Arnoglossus scapha*, lemon sole	Hewitt and Hine, 1972

Ceratomyxa vepallida	gall bladder of *Arnoglossus scapha*	Hewitt and Hine, 1972
Ceratomyxa sp.	gall bladder of many species	Hewitt and Hine, 1972
Chilodinella sp.	Gill infections in farmed eels	Boustead, 1982
Davisia diplocrepis	urinary bladder of *Diplocrepis puniceus*	Hewitt and Hine, 1972
Eimeria anguillae	Intestinal infections in farmed eels	Boustead, 1982
Endosphaera engelmania	hyperparasitic in *Trichodina multidentis*	Hewitt and Hine, 1972
Haemogregarina acanthoclini	Blood infection of rockfish	Hewitt and Hine, 1972
Haemogregarina bigemina	blood of many species	Hewitt and Hine, 1972
Haemogregarina coelorhynchi	blood of javelin fish	Hewitt and Hine, 1972
Haemogregarina hoplichthys	blood of flathead	Hewitt and Hine, 1972
Haemogregarina leptoscopi	blood of monk fish	Hewitt and Hine, 1972
Ichthyophthirius multifilis	Skin and gill infections in salmonids, eels and native galaxids	Boustead, 1982
Kudoa sp.	Muscle infection in red cod sample referred by Dept. of Health	Boustead, 1982
Leptotheca annulata	gall bladder of kahawai, hake, barracouta	Hewitt and Hine, 1972
Leptotheca minima	gall bladder of kahawai	Hewitt and Hine, 1972
Leptotheca pinguis	gall bladder of *Arnoglossus scapha*, sole	Hewitt and Hine, 1972
Leptotheca subelegans	bile of goby, *Diplocrepis puniceus*	Hewitt and Hine, 1972
Leptotheca sp.	gall bladder and urinary bladder of *Arnoglossus scapha*, *Congiopodus leucopaecilus*	Hewitt and Hine, 1972
Unidentified microsporidia	Muscle infections of butterfish; gill infections of black slickhead	Boustead, 1982
Myxidium incurvatum	gall bladder infection of many species	Hewitt and Hine, 1972
Myxidium sp.	Gill infections in farmed eels	Boustead, 1982
Myxobolus cerebralis	Fresh water hatcheries and rivers of South Island	Hewitt, 1972 Boustead, 1996
Myxobolus sp.	Skin and muscle infections in eels and native galaxids	Boustead, 1982
Myxosoma tripterygii	connective tissue of caudal peduncle of *Forsterygion varium*	Hewitt and Hine, 1972
Octosporea sp.	Infection of various tissues of jack mackerel	Boustead, 1982
Scyphidia acanthoclini	Gills infection of rockfish	Hewitt and Hine, 1972

Scyphidia sp.	Gills infections of farmed and feral eels	Boustead, 1982
Spaeromyxa tripterygii	gall bladder of *Forsterygion varium, Gobiomorphus*	Hewitt and Hine, 1972
Sphaerospora undulans	urnary bladder, ureters and oviducts of *Arnoglossus scapha, sole*	Hewitt and Hine, 1972
Sphaerospora sp.	Gall bladder of ling	Hewitt and Hine, 1972
Thelohanellus sp.	Skin infections of eels	Boustead, 1982
Trichodina parabranchiola	Gills and operculum of many species	Hewitt and Hine, 1972
Trichodina multidentis	Gills of many species	Hewitt and Hine, 1972
Trichodina sp.	Gill infections in eels	Boustead, 1982
Trypanosoma caulopsettae	blood of *Arnoglossus scapha*, sand flounder	Hewitt and Hine, 1972
Trypanosoma coelorhynchi	blood of javelin fish, red cod	Hewitt and Hine, 1972
Trypanosoma congiopodi	blood of *Congiopodus leucopaecilus*	Hewitt and Hine, 1972
Trypanosoma parapercis	blood of blue cod	Hewitt and Hine, 1972
Trypanosoma tripterygium	blood of many species	Hewitt and Hine, 1972
Zschokkella sp.	gall bladder of *Forsterygion varium*	Hewitt and Hine, 1972
Platyhelminthes		
Accacladocoelium alveolatum	sunfish	Hewitt and Hine, 1972
Allocotylophora polyprionum	gills of groper	Hewitt and Hine, 1972
Anahemiurus sp.	kahawai	Hewitt and Hine, 1972
Anchistrocephalus microcephalus	intestine of sunfish	Hewitt and Hine, 1972
Benthotrema richardsoni	intestine of lemon sole	Hewitt and Hine, 1972
Bivagina pagrosomi	Gills of snapper	Hewitt and Hine, 1972 Boustead, 1982
Bivesiculoides otagoensis	intestine of *Plagiogenion rubiginosus*	Hewitt and Hine, 1972
Bothriocephalus scorpii	intestine of red cod	Hewitt and Hine, 1972
Brevicreadium congeri	intestine of conger eel	Hewitt and Hine, 1972
Bucephalus longicornis	pyloric caeca and intestine of monk fish	Hewitt and Hine, 1972
Cardicola coridacis	gills and coelom (probably from blood) of butterfish	Hewitt and Hine, 1972
Cardicola whitteni	gills (probably from blood vessels) of tarakihi	Hewitt and Hine, 1972
Cemocotyle trachuri	gills of horse mackerel	Hewitt and Hine, 1972

Choanomyzus tasmaniae	intestine of *Notothenia macrocephala*	Hewitt and Hine, 1972
Clinostomum sp.	Flounder in Lake Ellesmere	Boustead, 1982
Coitocaecum anaspidis	intestine of many species, including brown trout, *Galaxias brevipinnis*, *Galaxias maculatus*	Hewitt and Hine, 1972
Coitocaecum tylogonium	intestine of *Centriscops humerosus*	Hewitt and Hine, 1972
Dactylogyrus sp.	Gills of grass carp in Rotorua	Boustead, 1982
Decemtestis pseudolabri	intestine of spotty	Hewitt and Hine, 1972
Deretrema minutum	intestine of *Galaxias maculatus*	Hewitt and Hine, 1972
Derogenes nototheniae	gills and stomach of *Notothenia macrocephala*	Hewitt and Hine, 1972
Derogenes varicus	stomach of many species, including brown trout	Hewitt and Hine, 1972
Diclidophora coelorhynchi	gills of javelin fish	Hewitt and Hine, 1972
Dihemistephanus lydiae	sunfish	Hewitt and Hine, 1972
Diplasiocotyle johnstoni	Gills of yellow eyed mullet	Hewitt and Hine, 1972
Diplopractodaeum plicitum	intestine of *Contusus richei*	Hewitt and Hine, 1972
Dolicoenterum longissimum	intestine of conger eel	Hewitt and Hine, 1972
Ectenurus lepidus	stomach of sea perch, horse mackerel	Hewitt and Hine, 1972
Encotyllable chironemi	gills of red moki	Hewitt and Hine, 1972
Eurysorchis australis	gills of kingfish	Hewitt and Hine, 1972
Genolinea anura	caeca of red moki	Hewitt and Hine, 1972
Genolinea dactylopagri	stomach of tarakihi, moki	Hewitt and Hine, 1972
Gonocerca phycidis	stomach of many species	Hewitt and Hine, 1972
Gonoplasius truttae	gills of kahawai	Hewitt and Hine, 1972
Gymnorhynchus horridus	liver of sunfish	Hewitt and Hine, 1972
Gymnorhynchus thyrsitae	muscle of barracouta	Hewitt and Hine, 1972 Boustead, 1982
Gymnorhynchus sp.	Cervical spine of butterfish	Hewitt and Hine, 1972
Gyrodactylus sp.	Gills of native galaxids in Lake Christabel	Boustead, 1982
Helicometra grandora	intestine of red gurnard, sea perch	Hewitt and Hine, 1972
Hepatoxylon trichuri	body cavity of many species	Hewitt and Hine, 1972
Heteraxinoides novaezealandiae	gills of horse mackerel	Hewitt and Hine, 1972

Heteraxinoides regis	gills of hake	Hewitt and Hine, 1972
Holorchis pulcher	intestine of moki	Hewitt and Hine, 1972
Hymenolepid cysticercoid	intestine of Rotorua smelt	Hewitt and Hine, 1972
Hypertrema ambovatum	intestine of parastic eel	Hewitt and Hine, 1972
Lacistorhynchus tenuis	body cavity of many species	Hewitt and Hine, 1972
Lecithochirium australis	stomach of barracouta	Hewitt and Hine, 1972
Lecithochirium conviva	stomach of conger eel	Hewitt and Hine, 1972
Lecithochirium flexum	stomach of conger eel	Hewitt and Hine, 1972
Lecithochirium genypteri	stomach of ling, horse mackerel	Hewitt and Hine, 1972
Lecithocladium excisum	intestine of red perch, stomach of blue cod	Hewitt and Hine, 1972
Lecithcladium magnocetabulum	stomach of brown trout, snapper	Hewitt and Hine, 1972
Lecithocladium seriolellae	stomach of brown trout, *Cyttus australis*, warehou	Hewitt and Hine, 1972
Lepidapedon australis	intestine of javelin fish	Hewitt and Hine, 1972
Lepidapedon congeri	intestine of conger eel	Hewitt and Hine, 1972
Megalocotyle australis	gills of sea perch	Hewitt and Hine, 1972
Megalocotyle johnstoni	gills of trumpeter	Hewitt and Hine, 1972
Megalocotyle latridis	gills of trumpeter	Hewitt and Hine, 1972
Microcotyle brevis	gills of *Forsterygii varium*	Hewitt and Hine, 1972
Microcotyle constricta	gills of blue cod	Hewitt and Hine, 1972
Microcotyle longirostri	gills of trevally	Hewitt and Hine, 1972
Microcotyle nemadactylus	gills of tarakihi	Hewitt and Hine, 1972
Microcotyle neozealandicus	gills of sea perch	Hewitt and Hine, 1972
Mitrostoma nototheniae	caeca and intestine of *Notothenia macrocephala*	Hewitt and Hine, 1972
Monogenea (3 species)	Gills of snapper in Wellington	Boustead, 1982
Myzoxenus crowcrofti	intestine of banded parrotfish	Hewitt and Hine, 1972
Neobivagina pelotretis	gills of lemon sole, sole	Hewitt and Hine, 1972
Neocreadium geniagni	Intestine, pyloric caeca and gall bladder of *Genyagnus monopterygius*	Hewitt and Hine, 1972
Neogruba seriolellae	gills of silver warehou	
Neolepidapedon cablei	intestine and caeca of southern hake	Hewitt and Hine, 1972
Neolepidapedon polyprioni	intestine of groper	Hewitt and Hine, 1972

Nybelina sp.	Stomach of kahawai, body cavity of barracouta, horse mackerel, John Dory	Hewitt and Hine, 1972
Odherium calyptrocotyle	intestine of sunfish	Hewitt and Hine, 1972
Opecoelus lotellae	intestine of southern hake	Hewitt and Hine, 1972
Opegaster caulopsettae	intestine of *Arnoglossus scapha*	Hewitt and Hine, 1972
Opegaster gobi	intestine of *Tripterygion* sp.	Hewitt and Hine, 1972
Pancreadium otagoensis	intestine of blue cod	Hewitt and Hine, 1972
Pelichnobothrium sp.	Pyloric caeca and rectum of brown trout	Hewitt and Hine, 1972
Phyllodistomomum anguillae	Urinary bladder of many species, including *Galaxias brevipinnis*	Hewitt and Hine, 1972 Boustead, 1982
Plagioporus dactylopagri	intestine of tarakihi	Hewitt and Hine, 1972
Plagioporus interruptus	intestine of scarlet parrotfish	Hewitt and Hine, 1972
Plagioporus pachysomus	intestine of *Cyttus australis*, blue cod	Hewitt and Hine, 1972
Plagioporus preporatus	intestine of red gurnard	Hewitt and Hine, 1972
Podocotyle caithnessi	intestine of conger eel	Hewitt and Hine, 1972
Proctoeces subtenue	posterior intestine of moki	Hewitt and Hine, 1972
Proenenterum ericotylum	intestine of *Notothenia macrocephala*	Hewitt and Hine, 1972
Proenenterum isocotylum	intestine of *Notothenia macrocephala*	Hewitt and Hine, 1972
Pseudaxine bivaginalis	gills of horse mackerel	Hewitt and Hine, 1972
Pseudobenedenia nototheniae	body surface of *Notothenia colbecki*, *Notothenia macrocephala*	Hewitt and Hine, 1972
Pseudocreadium monacanthi	intestine of leatherjacket	Hewitt and Hine, 1972
Pseudopecoeloides tenuis	gall bladder of horse mackerel	Hewitt and Hine, 1972
Pseudopecoelus hemilobatus	intestine of *Cyttus australis*	Hewitt and Hine, 1972
Pseudopecoelus japonicus	intestine of *Centriscops humerosus*	Hewitt and Hine, 1972
Pseudopecoelus vulgaris	intestine and caeca of sea perch	Hewitt and Hine, 1972
Rhynchopharynx paradoxica	sunfish	Hewitt and Hine, 1972
Stegodexamene anguillae	intestine of short finned eel, long finned eel muscle of *Galaxias brevipinnis,* and other species liver of native galaxids in Lake Chritasbel body cavity of upland bully in Eyre River	Hewitt and Hine, 1972 Boustead, 1982
Stephanostomum australis	intestine of red gurnard	Hewitt and Hine, 1972

Stephanostomum pristis	intestine of red cod	Hewitt and Hine, 1972
Steringotrema rotundum	gall bladder and stomach of blue cod	Hewitt and Hine, 1972
Sterrhurus lotellae	stomach of southern hake	Hewitt and Hine, 1972
Syncoelium thyrsitae	barracouta	Hewitt and Hine, 1972
Tagia gempylli	gills of hake	Hewitt and Hine, 1972
Telogaster opisthorchis	intestine of short finned eel, long finned eel muscle of many species including brown trout, *Galaxias brevipinnis*, *galaxias maculatus* body cavity of upland bully in Eyre River liver and gonad of brown mudfish in Lake Pounui	Hewitt and Hine, 1972 Boustead, 1982
Telorhynchus arripidis	intestine of kahawai	Hewitt and Hine, 1972
Tergestia agnostomi	intestine of yellow eyed mullet	Hewitt and Hine, 1972
Tricotyledonia genypteri	stomach of short finned eel, long finned eel, conger eel, ling	Hewitt and Hine, 1972
Trypanorhynch larvae	Ovary of quinnat salmon in Rangitata River	Boustead, 1982
Tubulovesicula angusticauda	stomach of many species	Hewitt and Hine, 1972
Udonella caligorum	gills of barracouta	Hewitt and Hine, 1972
Winkenthughesia australis	gills of *Lepidopus caudatus,* barracouta	Hewitt and Hine, 1972
Nematoda		
Ascarophis sp.	Stomach of many species	Hewitt and Hine, 1972 Boustead, 1982
Anguillicola australis	intestine of long finned eel Swim bladder of eel from eel farm on Rangitaiki River	Hewitt and Hine, 1972 Boustead, 1982
Anisakis sp.	Viscera, mesenteries and peritoneum of many species	Hewitt and Hine, 1972
Aphelenchus sp.	Stomach of *Galaxias maculatus*	Hewitt and Hine, 1972
Ascarophis sp.	Stomach of conger eel	Hewitt and Hine, 1972
Capillaria sp.	Stomach of many species	Hewitt and Hine, 1972
Contracaecum aduncum	intestine of many species, including brown trout	Hewitt and Hine, 1972
Contracaecum sp.	Stomach, intestine and body cavity of many species, including brown and rainbow trout Throat of rainbow trout in Lake Ida	Hewitt and Hine, 1972 Boustead, 1982

Cucullanellus cnidoglanis	stomach and intestine of snapper	Hewitt and Hine, 1972
Cucullanellus sheardi	intestine of moki	Hewitt and Hine, 1972
Cucullanellus sp.	Intestine of tarakihi	Hewitt and Hine, 1972
Cucullanus antipodeus	intestine of brown trout, *Rhombosolea* sp.	Hewitt and Hine, 1972
Cucullanus robustus	intestine of conger eel	Hewitt and Hine, 1972
Cucullanus sp.	Intestine of short finned eel, long finned eel	Hewitt and Hine, 1972
Dorylaimus sp.	Stomach of *Galaxias maculatus*	Hewitt and Hine, 1972
Eustrongylides sp.	Stomach wall of many species, including rainbow and brown trout body cavity of *Galaxias maculatus*	Hewitt and Hine, 1972 Boustead, 1982
Hedruris spinigera	stomach of many species, including brown and rainbow trout, *Galaxias maculatus*, quinnat salmon, and smelt	Hewitt and Hine, 1972
Paranisakiopsis australiensis	alimentary canal of javelin fish	Hewitt and Hine, 1972
Paranisakis sp.	Stomach wall of long finned eel	Hewitt and Hine, 1972
Paraquimperia sp.	Intestine of short finned eel, long finned eel	Hewitt and Hine, 1972
Philometra sp.	Body cavity of many species, nasal capsule of butterfish	Hewitt and Hine, 1972
Porrocaecum decipens	flesh of *Notothenia colbecki, N. macrocephala, N. microlepidota*, green back flounder	Hewitt and Hine, 1972
Porrocaecum sp.	Body cavity and muscle of barracouta	Hewitt and Hine, 1972
Spirocamallanus sp.	Intestine of long finned eel	Hewitt and Hine, 1972 Boustead, 1982
Stomachus marinus	stomach of barracouta	Hewitt and Hine, 1972
Acanthocephala		
Ancanthocephalus sp.	*Galaxias maculatus*	Hewitt and Hine, 1972
Corynosoma sp.	Intestine and pyloric caeca of brown trout	Hewitt and Hine, 1972
Heteracanthocephalus peltorhampi	sole	Hewitt and Hine, 1972
Micracanthorhynchus henirhamphi	stomach of *Reporhamphus ihi*	Hewitt and Hine, 1972
Neoechinorhynchus aldrichettae	Intestine of yellow eyed mullet in Lake Ellesmere	Boustead, 1982

Arthropoda		
Acanthochondria chilomycteri	mouth of porcupine fish	Hewitt and Hine, 1972
Aethon garricki	gills of tarakihi	Hewitt and Hine, 1972
Aethon morelandi	gills of moki	Hewitt and Hine, 1972
Aethon percis	gills of blue cod	Hewitt and Hine, 1972
Argulus japonicus	body surface of goldfish	Hewitt and Hine, 1972
Caligus aesopus	body surface of kingfish	
Caligus brevis	body surface of many species	Hewitt and Hine, 1972
Caligus buechlerae	body surface of *Tripterygion* sp.	Hewitt and Hine, 1972
Caligus longicaudatus	unknown host	Hewitt and Hine, 1972
Caligus pelamydis	operculum of barracouta	Hewitt and Hine, 1972
Caligus productus	operculum of bonito	Hewitt and Hine, 1972
Cecrops latreilli	gills of sunfish	Hewitt and Hine, 1972
Chondracanthus genypteri	gills of ling	Hewitt and Hine, 1972
Chondracanthus lotellae	gills and abdominal wall of red cod	Hewitt and Hine, 1972
Cirolana sp.	Mouth of quinnat salmon in Waitaki River	Boustead, 1982
Codonophilus imbricatus	mouth and throat of kahawai, sweep, horse mackerel	Hewitt and Hine, 1972
Codonophilus lineatus	throat of butterfish, mouth of *Reporhamphus ihi*	Hewitt and Hine, 1972
Congericola pallidus	gills of conger eel	Hewitt and Hine, 1972
Elytrophora brachyptera	operculum of long-finned albacore, blue-fin tuna	Hewitt and Hine, 1972
Gloiopotes huttoni	body of black marlin, striped marlin	Hewitt and Hine, 1972
Hatschekia crenata	gills of *Lepidopus caudatus*	Hewitt and Hine, 1972
Hatschekia quadrata	gills of porcupine fish	Hewitt and Hine, 1972
Irona melanosticta	gill arches and mouth of *Reporhamphus ihi*	Hewitt and Hine, 1972
Lepeophtheirus argentus	body surface of bream	Hewitt and Hine, 1972
Lepeophtheirus distinctus	ling	Hewitt and Hine, 1972
Lepeophtheirus erecsoni	body surface of moki	Hewitt and Hine, 1972
Lepeophtheirus hastatus	body surface of sunfish	Hewitt and Hine, 1972
Lepeophtheirus heegaardi	*Lepidopus caudatus*	Hewitt and Hine, 1972

Lepeophtheirus nordmanni	body surface of sunfish	Hewitt and Hine, 1972
Lepeophtheirus polyprioni	body surface of bass, groper	Hewitt and Hine, 1972
Lepeophthirius scutiger	body surface of spotty, scarlet parrotfish, banded parrotfish	Hewitt and Hine, 1972
Lernaea sp.	Surface of yellow eyed mullet in Manukau Harbour	Boustead, 1982
Lernanthropus microlamini	gills of warehou	
Livoneca raynaudi	gills of ling, *Notothenia colbecki*, *Rhombosolea* sp, stomach of red cod	Hewitt and Hine, 1972
Nerocila orbignyi	body of kahawai	Hewitt and Hine, 1972
Paeonodes nemaformis	gills, operculum and base of pectoral fins of brown trout and quinnat salmon	Hewitt and Hine, 1972
Paralernanthropus foliaceus	gills of hake, barracouta	Hewitt and Hine, 1972
Penella histiophori	body of black marlin	Hewitt and Hine, 1972
Philichthys xiphiae	skull of swordfish	Hewitt and Hine, 1972
Pseudocycnus appendiculatus	gills of long-finned albacore	Hewitt and Hine, 1972
Rebelula laticervix	head of *Coelorhynchus fasiatus*	Hewitt and Hine, 1972
Sphyrion laevigatum	gills and body surface of ling	Hewitt and Hine, 1972
Sphyrion sp.	Surface of tarakihi	Boustead, 1982
Trifur lotellae	gills of red cod	Hewitt and Hine, 1972
Annelida		
Makarabella manteri	branchial chamber of monk fish	Hewitt and Hine, 1972
Notobdella nototheniae	body surface of *Notothenia microlepidota*	Hewitt and Hine, 1972

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4.   RISK ASSESSMENT

4.1   QUALITATIVE ASSESSMENT OF THE VIRAL PATHOGENS OF SALMONIDS

4.1.1   Adenoviridae

All fish adenoviruses cause epithelial inflammatory or hypertrophic-type lesions in their hosts, and all are recorded as having only one natural host species. Wolf (1988) notes the occurrence of three adenovirus infections in fish: Atlantic cod adenovirus, dab adenovirus, and sturgeon adenovirus. None are known to be pathogenic for salmonids. McAllister (1993) and Humphrey (1995) have attributed strawberry disease of trout to an adenovirus.

Strawberry disease of trout is a non-fatal inflammatory skin condition of rainbow trout, first recognized about 30 years ago in hatcheries in the northwest of USA and Canada (McAllister, 1993). An adenovirus has been described as being associated with the epithelial lesions of strawberry disease (Fleury et al, 1985). No experimental challenge work has been carried out, so the aetiology of the disease remains uncertain and speculation has ranged from an infectious agent, designated strawberry disease virus, to local allergic reactions (Olsen et al, 1985). The disease has been observed in Oregon, Nevada, Idaho, Montana and California, with morbidity in some fish farms ranging up to 80% (McAllister, 1993). The disease has also occurred in France (Fleury et al, 1985; Olsen et al, 1985). The lesion is one of progressive inflammation with cellular infiltration of the dermis, hypodermis and underlying fascial planes, leading to focal necrosis and sloughing of the epidermis and scales. Affected fish normally recover in about two months. Diagnosis is based on clinical signs and histology. No treatment is known, although oxytetracycline reportedly shortens recovery time. The disease has only been transmitted by cohabitation (Olson et al, 1985).

Conclusions

1   Strawberry disease of trout may be caused by an infectious agent which is an adenovirus. Strawberry disease of trout is of economic significance for farmed and hatchery rainbow trout within the limited geographical range of the disease.

2   Fish clinically infected with strawberry disease lesions are unlikely to be processed for human consumption without suffering quality downgrading and/or trimming.

3   The titre of any infectious agent associated with sub-clinically infected fish, fish incubating strawberry disease or recovered fish is unknown. Whether infectious agent would survive on the *commodity* or in the environment is likewise unknown.

4   Transmission of strawberry disease of trout has only been demonstrated through cohabitation, suggesting direct pathways are necessary to transmit the disease.

5   The risk of strawberry disease of trout introduction through importations of the *commodity* is negligible.

4.1.2   Birnaviridae

The aquatic animal birnaviruses have been extensively studied. Infectious pancreatic necrosis virus (IPNV) is the prototype of the birnaviruses (Dobos et al, 1979; Brown, 1986). Up to ten serotypes of IPNV may exist (MacDonald and Gower, 1981; Okamoto et al, 1983; Hill and Way, 1983; Christie et al, 1988; Ross and Munroe, 1995). The relationship between serotypes of IPNV is the subject of continuing research. References to five other birnaviruses known to be pathogenic in fish species other than salmonids have been found in the literature (Humphrey, 1995).

IPNV

Introduction

Wolf (1988) provides a thorough review of the history of research into the long recognized disease entity now known to be caused by infection with IPNV. Although primarily recognised as a disease of fry and fingerling trout, the ability of IPNV to infect adult trout, other salmonid species and a wide range of non-salmonid aquatic animal species has been extensively researched.

Susceptibility and distribution

IPNV and IPNV-like viruses are considered to be ubiquitous in aquatic environments, in that they may be isolated from a wide range of aquatic animal species and they appear to have wide geographic distribution. Wolf (1988) lists 16 countries in Europe, Scandinavia, North and South America, and North Asia in which IPNV has been recorded. IPN-like viruses probably have even wider distribution than this list indicates.

IPNV is listed by the OIE as a significant pathogen of salmonids (OIE, 1995a). Brook and rainbow trout are particularly susceptible to lethal infection, while other salmonid species are less susceptible (McAllister and Owens, 1995). Wolf (1988) lists 16 species of salmonid fish and 19 families of fish other than Salmonidae from which IPNV has been isolated. A recent literature review (Humphrey, 1995) lists about 50 species of salmonid and non-salmonid fish from which IPNV has been isolated. IPNV has also been isolated from a wide variety of fish, molluscs and crustaceans in fresh water, estuarine and marine environments (Hill, 1982; Wolf 1988; McAllister 1993). The term "IPN-like viruses" has often been used to describe birnavirus isolates with many characteristics of IPNV but which appear non-pathogenic to salmonids.

McAllister and Owens (1995) assessed the virulence of 28 IPNV isolates by challenging brook trout. Isolates of IPNV from salmonid fishes were of mid-range to high virulence to brook trout, while isolates from non-salmonid fishes and from molluscs ranged from no to high virulence. IPNV has been shown to persist in scallops after inoculation and bath challenge, and although no replication occurred in scallops IPNV could be isolated from tissues for up to 11 months following inoculation (Mortensen et al, 1992). IPNV could be isolated from faeces of scallops following inoculation and bath challenge, and from prawns feeding on dead IPNV-contaminated scallops (Mortensen, 1993).

An IPN-like virus has been recorded in returning chinook salmon in New Zealand (Tisdall and Phipps, 1987). The New Zealand isolate appears to be avirulent in salmonids, as no disease entity has ever been associated with isolation of the New Zealand birnavirus (Hine, 1995).

In salmonid populations where IPNV occurs, prevalence and the impact of the disease can be expected to vary with the pathogenicity of the virus strain, water temperature, stocking densities and other stress-related factors. Under aquaculture conditions in Norway, IPNV disease has been demonstrated to result in a cumulative farm-level prevalence of 39.5% (Jarp et al, 1994). In Norway the risk of IPN is related to management practices such as mixing of smolts from many hatcheries in sea farms, as well as the age of the site and geographic location (Jarp et al, 1994). Another Norwegian study examining submissions of specimens from farmed salmon isolated IPNV from 63.7% of submissions, and concluded that all commercial Atlantic salmon sea water farms in Norway harbour IPNV carriers (Melby et al, 1991). Clinical IPNV infection is the main viral problem in both Norwegian farmed Atlantic salmon smolts following transfer to sea water and in many Norwegian hatcheries. IPNV has also been detected in rainbow trout and glass eel in Sweden (OIE, 1996).

In Denmark, cumulative data on laboratory submissions for the period 1990-1995 demonstrate that of 8,693 specimens (mainly rainbow trout) submitted for virological examination, 1,817 (20.9%) were positive for IPNV. A survey of 397 aquaculture facilities conducted in 1995 demonstrated that 87 (21.9%) were infected with IPNV. The majority (82 of 87) of IPN infected facilities were rainbow trout farms. Of the 611 pools of samples collected and submitted for diagnostic purposes during 1995, 69 (11.3%) were positive for IPNV (pers. comm. Niels Jørgen Olsen, Danish Veterinary Laboratory, 21 October 1996).

Although IPNV is known to occur in trout farms in the western states of the USA and has been suspected to occur in Alberta, Canada (Wolf, 1988), surveillance of wild salmon species in the Pacific northwest has not detected any fish infected with IPNV. In Alaska, surveillance data for the years 1980 to 1994 found no infected samples out of 11,004 Pacific salmon assayed (pers. comm. Linda Chaves, October 1995). In British Columbia, IPNV was not detected in any of the 2,331 wild, adult Pacific salmon submitted to the Pacific Biological Station between the period 1985 to 1994 (pers. comm. Trevor Evelyn, October 1996). In Washington, testing of salmon in the years 1991 to 1995 found no IPNV infected specimens from 51,947 samples (pers. comm. Linda Chaves, October 1995). Recently IPNV has been isolated from pen-reared coho salmon in Washington state and IPNV infected fish are frequently detected amongst returning fish within a stock of steelhead trout on the Columbia River (pers. comm. James Winton, North West Biological Science Centre, Washington, February 1997).

A survey in the northern part of Japan involving 11,095 females of six species and 155 males of two species of mature salmonids detected only two IPNV infected fish, although the study concluded that IPNV was probably widely distributed in the region (Yoshimizu et al, 1989). This conclusion is consistent with an earlier survey which isolated IPNV from trout in 24 of 27 hatcheries surveyed in Japan (Sano, 1972).

Pathology, epidemiology and control

McAllister (1993) and Wolf (1988) reviewed the clinical signs, pathology, transmission and epidemiology of IPNV infections. Briefly, acute infections normally occur in 1-4 month old salmonids. Older fish may undergo subclinical or unapparent infection. Older trout are known to be able to mount an immune response and clear the infection if infected as adults. Juvenile fish which survive infection tend to become carriers, with stress precipitated re-activation later in life leading to virus shedding and clinical disease. Clinical manifestation in fry and fingerlings is darkening, exophthalmia, abdominal distension, erratic swimming, and mucoid faecal pseudocasts. Mortality may approach 100%, but tends to be lower when water temperatures are low. Internally, pancreas, liver, kidney, spleen, and intestine exhibit gross pathology. Electron microscopy reveals virus particles in most internal organs. Internal organs, particularly caudal kidney, and sex products are the samples of choice for virus isolation. A variety of virus identification procedures is available to confirm a diagnosis of IPNV following virus isolation, with infectivity neutralization the standard method against which other methods are compared.

The infective dose of IPNV in salmonids has not been adequately defined. Frantsi and Savan (1971) were able to experimentally infect two month old brook trout fry held in 75 litre tubs at $10°C$ by feeding 10 ml of beef liver with 10^3 [tissue culture infectious doses$_{50}$ per gram of tissue (tcid$_{50}$/g)] virus/ml per 100 fish over a two day period.

Information on the titre of IPNV in tissues of infected fish can be extracted from a few key reports. Wolf (1988) reported titres in five adult carrier brook trout ranging from $10^{6.7}$ tissue culture infectious doses$_{50}$ per gram of tissue (tcid$_{50}$/g) in kidney to $10^{0.3}$ tcid$_{50}$/g in muscle. This information indicates that although muscle and skin may be infected, viral titres are likely to be several orders of magnitude (possibly 10^3 to 10^6) lower than visceral tissues.

Table 2. Average IPNV infectivity in selected tissues of five adult carrier brook trout (Wolf, 1988).

Tissue	Infectivity[a]
Kidneys	6.7
Stomach	4.6
Ovary	4.5
Liver	4.4
Spleen	3.3
Foregut	3.3
Hindgut	3.3
Muscle	0.3

[a] Infectivity expressed as exponent of $\log_{10}$ TCID$_{50}$/g.

In other studies with naturally infected brook trout, ovarian fluid was demonstrated to have the highest IPNV titre (10^8 plaque forming units (pfu)/ml), and titres in other tissues (skin and muscle were not included) ranged below this down to undetectable levels (McAllister et al, 1993). IPNV titres in kidney, spleen and pancreas of 2 year old rainbow trout up to $10^{3.5}$ virus/ml of homogenate were recorded in another study (Yamamoto, 1975). A further study of experimentally infected yearling brook trout (infected by both immersion and intra-peritoneal

injection) demonstrated mean IPNV viral titres in kidney, liver, spleen and pancreas ranging from 10^3 tcid$_{50}$/ gram of tissue to 10^4 tcid$_{50}$/ gram of tissue (Bootland et al, 1991).

A literature review (Humphrey, 1995) has summarised reports documenting the spread of IPNV resulting from the movement of infected live fish and eggs. There are no literature reports of IPNV having been spread via the movement of dead fish for human consumption.

Transmission of IPNV occurs horizontally via faeces, urine and sex products. A variety of animals can act as mechanical vectors, including chicken, heron, gulls and some crustaceans and molluscs. Herons fed 1g of IPNV infected trout fry shed IPNV in faeces for 7 days, and the faeces were able to transmit infection to rainbow trout fry (Peters and Neukirch, 1986). Vertical transmission occurs as a result of virus in the egg following virus associated with sperm penetrating the egg. IPN may be transmitted experimentally by injection, immersion and feeding. A carrier state plays an important role in the transmission of infection in hatchery conditions, with up to 90% of survivors of an epidemic becoming virus carriers. Carrier-mediated transmission may not be as important in nature. Prevalence of the carrier state probably also varies between salmonid species. Carrier prevalence may decrease with time. This may be a function of developing immunocompetence, as high antibody titres in carrier fish correlate with reduced viral titres in organs and faeces. Environmental conditions affect IPNV infection. Temperature may influence both host response and viral replication. Stress recrudescence of infections in carrier fish may occur with transport, overcrowding, increased temperature and low oxygen concentration.

No effective chemotherapeutic agents are available for treatment of IPN. Vaccines are in the early stages of development. There is no evidence of maternally transferred immunity. Resistance appears to be heritable and can be enhanced by selective breeding. Control is achieved by maintaining hatchery hygiene measures, screening of broodstock and minimising stress.

Survival and inactivation

Wolf (1988) reviewed the biophysical properties of IPNV. Humphrey (1995) also summarised the literature on the temperature and environmental stability of IPNV. IPNV has a relatively high thermal stability. Within pH 5.0-7.0, IPNV is stable in laboratory storage at 4°C for 4 months. Wolf (1988) also reports minimal decrease in IPNV titre of homogenized tissue samples stored in Hanks' balanced salt solution over 19 years, despite two power failures causing thawing of the stored samples.

In tissue culture, IPNV is completely inactivated within 16 hours at 60°C (Gosting and Gould, 1981). This study demonstrated that over 95% of the decrease in IPNV infectivity occurs rapidly, but that the inactivation rate then decreases abruptly. The initial rapid inactivation rate is temperature dependent. Humphrey et al (1991) reported negligible decrease in IPNV titre in tissue culture supernatant with serum at 40°C for 5.5 hours, and a 2-3 log decrease at 50°C for 3 hours, 60°C for 1 hour, and 70°C for 15 minutes.

The stability of IPNV in aquatic environments is the subject of several literature reports. Wolf (1988) reports persistence of infectivity in filter decontaminated water at 4°C for 5-6 months

and in municipal tap water at 10°C for more than 7 months. Wedemeyer et al (1978) reported IPNV stability for at least 8 weeks in either distilled, soft or hard lake waters; inactivation with chlorine at 0.2 mg/L in 10 minutes in soft water; inactivation with chlorine at 0.7 mg/L in 2 minutes in hard water; and inactivation with ozone at 90 mg/h/L in 10 minutes in hard water, and in 30 seconds in soft water.

Other studies have related IPNV stability to the presence of bacteria or natural microflora in water and demonstrated an inverse relationship, with infectivity persisting four times longer in filter decontaminated water or autoclaved estuarine water than in non-treated water (Toranzo and Hetrick, 1982; Toranzo et al, 1982). IPNV retains infectivity when frozen for long periods, and through freeze-thaw cycles (Wolf, 1988). Wolf (1988) summarised the stability of IPNV as shown in Tables 3 and 4.

Table 3. Stability of IPNV under various test conditions (Wolf, 1988)

Condition	Result
Municipal tap water at 10°C	<1 tcid$_{50}$/ml at 231 days
Non-treated canal water at 10°C	Not detectable after 14 days
Pond mud at 10°C	<1 tcid$_{50}$/ml at 70 days
Drying at 10°C	Persisted more than 4 weeks
10^6 rads gamma radiation	10% infectivity survived
2540 Å UV irradiation at 5 cm	100% inactivation at 60 min
2% NaOH solution (pH 11.9)	Total inactivation in 5 min
3% formalin solution	Total inactivation in 5 min
520 ppm chlorine	99% inactivation in <2 min

Table 4. Response of IPNV to selected disinfecting agents (Wolf, 1988)

Agent	Concentration (ppm)	Time (min)	Effect
Chlorine	40	30	Virucidal
Chlorine	16	30	Virucidal
Chlorine	0.7	2	Virucidal
Formalin	20,000	5	Virucidal
Formalin	2,000	60	Partly virucidal
Iodine	35	5	Virucidal
Iodine	16	5	Virucidal
Ozone	90	0.5-10	Virucidal
pH 2.5 (HCl)	-	60	Partly virucidal
pH 12.5 (NaOH)	-	10	Virucidal

The chlorine concentration of chlorinated water supplies in New Zealand is typically within the range 0.2- 0.5 mg/L, although concentrations up to 1 mg/L are used in some areas. Not all New Zealand water supplies are chlorinated (pers. comm. Wellington Regional Council Wellington City Water Management and Engineering Dept., April 1997).

Conclusions

1   IPNV is a serious, economically significant pathogen of salmonids and other fishes,

including marine fish species, and has a wide geographical distribution.

2   The isolation of an IPN-like birnavirus in chinook salmon returning to New Zealand is an indication that natural pathways for the introduction of IPNV into New Zealand probably exist.

3   Live fish, fish eggs, and untreated offal or fish farm wastes pose a high risk for spread of IPNV. There are no reports of dead fish for human consumption being the agent for transmission of IPNV.

4   The likelihood of fish clinically infected with IPNV being harvested and/or being processed for human consumption is low.

5   The likelihood of fish of carrier status for IPNV being harvested and/or being processed for human consumption is high.

6   The titre of IPNV in headed, gilled and gutted IPNV carrier salmonids processed for human consumption is likely to be relatively low.

7   Infectivity of any IPNV present in tissues of any fresh or frozen fish product could be maintained for long periods.

8   If imported in fish tissues and placed into the aquatic environment in New Zealand, some IPNV could remain infective for long periods.

9   The likelihood of IPNV remaining infective for long periods is reduced by widespread chlorination of New Zealand water supplies, although virus associated with organic material such as scraps is likely to avoid inactivation.

10   The risk of IPNV introduction through the importation of the *commodity* is low. The risk is likely to be lower than the risk of birnavirus introduction via natural pathways, which has already apparently occurred.

4.1.3 Herpesviridae

Introduction

Several viruses isolated from fish have been classified as herpesviruses (Wolf, 1988; Hedrick and Sano, 1989). Wolf (1988) listed 13 herpesviruses affecting fish, of which two affect salmonids: *Herpesvirus salmonis* and *Oncorhynchus masou* virus. McAllister (1993) listed three herpesviruses that affect salmonids: *Herpesvirus salmonis* (salmonid herpesvirus 1 or HSV)*,* Japanese salmonid herpesvirus and Lake trout herpesvirus. Both Wolf (1988) and McAllister (1993) further noted that several isolates of herpesviruses have been detected from Japanese salmonids, with the name Japanese salmonid herpesvirus used for all three. The three Japanese herpesviruses are *Nerka* virus (Sano, 1976), *Oncorhynchus masou* virus (salmonid herpesvirus 2 or OMV) (Kimura et al, 1981) and yamame tumour virus (YTV) (Sano et al, 1983; Hedrick et al, 1987). The salmonid herpesviruses from Japan and the USA are serologically distinct but share some common antigens (Hedrick et al, 1987). Recent reports from Japan indicate that up to 11 strains of herpesviruses affecting various salmonid species may be present in Japan (Yoshimizu, 1994; Yoshimizu et al, 1995). The Japanese salmonid herpesviruses are all closely related to OMV, and are clearly distinguished from salmonid herpesvirus 1. OMV is designated a notifiable disease by the OIE (OIE, 1995a).

Susceptibility and distribution

HSV was first isolated in 1971 from spawning rainbow trout at a single hatchery in Washington (Wolf, 1976). Its presence was confirmed in 1975 (Wolf, 1976), but it has not been isolated since (McAllister, 1993). Juvenile rainbow trout, chum salmon and chinook salmon have been shown to be susceptible to HSV (Wolf, 1988). Young Atlantic salmon, brook trout, brown trout, yearling rainbow trout and kokanee have been shown to be refractory to infection (Wolf, 1976; Wolf, 1988). HSV was controlled by destruction of the broodstock and disinfection of the hatchery. Follow-up surveys have failed to find HSV in any stations in the numerous countries that received eggs or fry from the hatchery (Wolf, 1988).

A closely related herpesvirus, designated steelhead herpesvirus, has been isolated from seven hatcheries and two lakes in California. In adult female steelhead trout returning to a Californian hatchery 5.8% of ovarian fluid samples were found to be positive for steelhead herpesvirus. Fingerling rainbow trout and chinook salmon are susceptible to infection, but brown trout and coho salmon are not (Eaton et al, 1989; Hedrick et al, 1987).

Nerka virus occurs naturally in kokanee fry in Japan. OMV was originally isolated from healthy fish, and subsequently demonstrated experimentally to be a pathogen of yamame fry and fingerlings (Sano, 1976). YTV has been isolated from a spontaneous tumour of yamame, and causes mortality in experimentally infected yamame (Sano et al, 1983). A large survey of hatcheries in Hokkaido indicated that OMV infection was widespread in northern Japan (Yoshimizu et al., 1989). Follow-up studies indicated that iodophor disinfection of eggs at the eyed stage had been an effective measure to control the spread and prevalence of OMV (Yoshimizu et al., 1989). Epidemic mortalities caused by OMV have been recorded in northern Japan in maricultured coho salmon in the 100-1,000g weight range. Mortalities ranged from 10-31% during the epidemic (Kumagai et al, 1994).

Lake trout herpesviruses cause sporadic incidents of epidemic mortality in hatcheries for lake trout in Lake Superior and Lake Michigan basins of the USA, dating from the mid-1980s. Brook trout, brown trout, rainbow trout, Atlantic salmon and chinook salmon are refractory. Natural epidemics occur in the spring, and may continue for several months (McAllister, 1993).

Pathology, epidemiology and control

Wolf (1988) and McAllister (1993) reviewed the clinical signs, pathology and epidemiology of the salmonid herpesviruses. Briefly, with the exception of YTV and coho salmon tumour virus which are oncogenic, herpesviruses of salmon affect fry, and in some cases fingerlings, causing inappetance, lethargy or hyperactivity, darkening and exophthalmos and high mortality. YTV causes spontaneous tumours in the perioral region of otherwise healthy fish leading to mortality. Both horizontal and vertical infection may play a role in the transmission of salmonid herpesviruses, with activation of latent infection at the time of spawning a possibility. The only documented natural sources of OMV are ovarian fluids and tumours of yamame. OMV has been transmitted experimentally to fry and fingerling chum salmon, kokanee and rainbow trout by immersion and by cohabitation (Kimura et al, 1981). Identification and destruction of infected stocks is the only effective means to control salmonid herpesvirus infections and is achieved through fish health surveillance programmes. Iodophor disinfection of eggs at the eyed stage is recommended as a control measure against OMV in Japanese hatcheries.

Survival and inactivation

Herpesviruses are ether, heat and acid labile (Wolf, 1988). As a group, herpesviruses are typically fragile in the environment. In 17 days OMV lost 99.9% infectivity at -20°C, and all infectivity at more than 15°C (Wolf, 1988). HSV is extremely temperature sensitive, requiring incubation in cell culture at 10°C rather than 15°C routinely used for virus isolation (Wolf, 1988). Holding eggs at temperatures higher than 16°C has been proposed as a possible control measure for HSV (Wolf, 1988)

Conclusions

1   The salmonid herpesviruses are significant pathogens of juvenile salmonids of certain species in specific geographical areas.

2   It is unlikely that salmonids clinically infected with a salmonid herpesvirus would be harvested and processed for human consumption, given the clinical aspects of the infection, the geographic distribution of the salmonid herpesviruses and the species and age susceptibility of salmonids.

3   Salmonids latently infected with a salmonid herpesvirus may be harvested and processed for human consumption. The viral titre of such fish is likely to be significantly reduced by evisceration.

4   It is unlikely that any salmonid herpesvirus present in headed, gilled and gutted

salmonids imported for human consumption would remain infective for any substantial period of time.

5 The risk of salmonid herpesvirus introduction through importations of the *commodity* is negligible.

4.1.4   Iridoviridae

Introduction

Wolf (1988) records seven iridoviruses pathogenic for fish. Another review of the literature (Humphrey, 1995) indicates that there are 13 iridoviruses affecting fish. Both sources record two iridoviruses known to be pathogens of salmonids: epizootic haematopoietic necrosis virus (EHNV) and viral erythrocytic necrosis (VEN). EHNV is designated a notifiable disease by the OIE (OIE, 1995a).

The iridoviral disease lymphocystis is an infection of many fish species, and is particularly troublesome in ornamental fish. It is not, however, known to affect salmonids.

Susceptibility and distribution

EHNV is confined to Victoria and New South Wales, Australia, and causes mortalities in wild redfin perch and farmed rainbow trout (Langdon et al, 1986; Langdon et al, 1988). Atlantic salmon are susceptible experimentally (Langdon, 1989). In a study of an outbreak in a rainbow trout farm in New South Wales, mortality of 0.033-0.2% per day was recorded in 125 mm forklength fingerlings, with total mortality for an outbreak in the 3-4% range. At the height of the outbreak, EHNV was demonstrated in 89% of clinically affected fish, 51% of dead fish and 4% of apparently healthy in-contact fish (Whittington et al, 1994).

VEN occurs in species from 23 genera of marine and anadromous fish on the Atlantic coast of the USA up to Greenland, in three genera in the Pacific north west of North America, and in four genera in Atlantic waters of Europe (Wolf, 1988; McAllister, 1993). Ten species of salmonids are known to be susceptible to VEN by intra-peritoneal injection of membrane filtered lysate, and two species have been infected by immersion challenge (MacMillan and Mulcahy, 1979).

Pathology, epidemiology and control

EHNV causes epidemics in redfin perch in the summer when water temperatures are relatively high (Langdon et al, 1986; Whittington et al 1994). Outbreaks in rainbow trout have occurred when the water temperature ranges from 11-17°C (Whittington et al, 1994).
The incubation period is affected by environmental temperature (Whittington and Reddacliff, 1995). Clinical signs in rainbow trout are non-specific: inappetance, abdominal distension, pallor of skin and fins, loss of equilibrium, flared opercula, and occasional ulceration of skin. Diagnosis must be confirmed by laboratory examination, with kidney, liver and spleen the preferred samples (Hyatt et al, 1991; OIE, 1995b). Outbreaks in trout farms may be related to epidemics in redfin perch in the watershed. The role that survivors of an outbreak play as carriers of infection has not been fully elucidated. There is no effective treatment for EHN. Control is by strict isolation and hygiene measures.

Wolf (1988) and McAllister (1993) reviewed the clinical signs, pathology and epidemiology of VEN. Clinical signs of VEN in severely affected fish are related to anaemia, and include darkening, pallor of the gills and internal organs, and low haematocrit. Mortality is generally

low, although epidemic mortality in herring due to VEN has been reported (Meyers et al, 1986). The virus causing VEN has not been isolated, although VEN can be experimentally transmitted. Marine reservoirs may be an important source of infection for salmonids. The virus may be vertically transmitted, as virus has been detected in yolk-sac fry and alevin of chum salmon (Rohovec and Amandi, 1981).

Survival and inactivation

EHNV persists for more than 2 years in tissues at -20°C; for more than 97 days in distilled water without decrease in titre; resists desiccation for more than 115 days at 15°C; is labile outside a narrow pH range either side of neutral; is inactivated at 60°C for 15 minutes, and 40°C for 24 hours; and is not completely inactivated by 400 mg/L hypochlorite in the presence of organic material (Langdon, 1989). Langdon concluded that the virus could survive for long periods in the aquatic environment and on fomites.

Wolf (1988) reported that VEN was completely inactivated at 60°C for 15 minutes. Efforts to isolate VEN in commonly used cell culture lines have all failed. Wolf (1988) found this unsurprising considering the cell tropism of VEN for cells of the blood and haematopoietic tissue. Failure to isolate the viral agent of VEN has hampered research into its biophysical properties.

Conclusions

1   EHNV is a serious pathogen of redfin perch and rainbow trout, with a geographical distribution limited to regions of Australia. It has not spread to any extent since epidemics of EHNV were first recorded 10 years ago.

2   Rainbow trout incubating or clinically infected with EHNV are unlikely to be harvested and processed for human consumption. However, a carrier status may exist in adult rainbow trout, and EHNV carrier fish could be harvested and processed for human consumption.

4   Heading, gilling and gutting is likely to remove the tissues containing the large majority of EHNV particles.

5   The risk of VEN introduction would be no greater, and probably less, in importations of the *commodity* than it would be for importations of dead uneviscerated marine fish, which are able to be imported for human consumption.

6   The risk of iridovirus introduction through importations of the *commodity* is negligible.

4.1.5   Orthomyxoviridae

ISA is a disease of farmed Norwegian Atlantic salmon. The disease was first recognised in 1984 (Thorud and Djupvik, 1988). Since then it has been detected from the south-west region of Norway along the coast to the far north (Thorud, 1991). The virus, termed infectious salmon anaemia virus (ISAV), remained unclassified until recently. ISAV has now been isolated and preliminarily characterised (Dannevig et al, 1995), and has morphological, functional and genomic properties consistent with those of an orthomyxovirus (pers. comm. Tore Håstein, August 1997).

Susceptibility and distribution

ISA has not been diagnosed in species other than Atlantic salmon, although experiments suggest that brown and rainbow trout could serve as carriers (Thorud, 1991; Nyland et al, 1995; OIE, 1996). Transmission experiments failed to produce clinical ISA in adult Arctic char, rainbow trout, wrasse and turbot (Hjeltnes, 1993). Natural outbreaks have occurred in all developmental stages of Atlantic salmon older than fry, providing that they have been exposed to sea water (Thorud, 1991).

ISA has not been recorded outside of Norway (OIE, 1996). This fact was utilised during an earlier MAF risk analysis to explain the concept of marker diseases (MacDiarmid, 1994). MacDiarmid (1994) reported a personal communication from Professor Tore Håstein, President of the OIE Fish Diseases Commission, that ISA's failure to spread outside of Norway, despite huge volumes of eviscerated salmon being exported indicated that the risk of spreading diseases by such exports is negligible.

Pathology, epidemiology and control

Clinical findings in affected fish are ascites, congestion, hepatic and splenic enlargement, pale gills and heart, haemorrhages in perivisceral fat, congestion of the foregut and severe haemolytic anaemia in the terminal stages. The principal histopathological change is haemorrhagic necrosis of the liver (Thorud, 1991). The infectivity of various internal tissues of experimentally infected Atlantic salmon at various times post-infection has been studied, and the infectivity of liver, kidney, spleen, plasma, head kidney leucocytes and red blood cells has been demonstrated (Dannevig et al, 1994).

Most ISA outbreaks occur in spring or early summer (Thorud, 1991). Epidemiological studies on ISA have demonstrated that poor biosecurity allowing passive transmission (proximity to other ISA affected sites and to salmon slaughterhouses) and active transmission (management practices which increase exposure to foreign biological material, such as the number of hatcheries from which smolts are obtained) are the major risk factors (Vågsholm et al, 1994; Jarp and Karlsen, accepted for publication 1996).

A reduction in ISA prevalence from 80 outbreaks during 1990 to fewer than ten confirmed during 1993 has been achieved by the Norwegian authorities through implementation of a series of control measures (Baalsrud, 1993).

Survival and inactivation

Transmission trials have demonstrated that ISAV is inactivated by heat (temperatures higher than 55°C for more than 1 minute), formaldehyde (0.5% for 16 hours), sodium hydroxide (pH 12 for 7 hours), formic acid (pH 4 for 24 hours), sodium hypochlorite (20mg/ml for 1 hour), ozone (8 mg/ml/l for 4 minutes) and UV irradiation (5 mJ/cm^2) (Torgensen, 1993). ISAV is thought to be unstable in the marine environment (Baalsrud, 1993).

Conclusions

1 ISA is a serious disease of Atlantic salmon thought to be caused by an orthomyxovirus. ISA is restricted to Norway.

2 Atlantic salmon are the only species known to be susceptible to ISA.

3 ISA infectivity has been demonstrated for viscera and blood. Bleeding and evisceration during processing of aquacultured Atlantic salmon is likely to significantly reduce infectivity.

4 The export of large quantities of salmon for human consumption from Norway without any restriction other than evisceration has not resulted in the spread of ISA to other countries.

5 The risk of ISA introduction through importations of the *commodity* is negligible.

4.1.6 Paramyxoviridae

Two paramyxoviruses affecting salmonids have been isolated.

Chinook salmon paramyxovirus (CSP) was isolated from spawning adult chinook salmon in Oregon (Winton et al, 1985). Wolf (1988) and Humphrey (1995) list CSP as being the only paramyxovirus affecting aquatic animals. The virus has been isolated in consecutive years from separate river systems in Oregon. No clinical or histopathologic signs of disease are present in infected fish. Virus has been isolated from the kidney and spleen of infected fish, but not from ovarian fluids or progeny of infected fish. The virus is inactivated by heating to 56°C for 6 hours or 37°C for 24 hours. It is stable through a freeze-thaw cycle at -20°C and -70°C (Wolf, 1988).

More recently, a paramyxovirus has been isolated from an Alaskan stock of adult chinook salmon (OIE, 1996). The relationship between the two paramyxoviruses appears not to have been definitively established.

Conclusions

1   Paramyxoviruses are of negligible significance as pathogens of salmonids.

2   Salmonids infected with a paramyxovirus are unlikely to be harvested and processed for human consumption, because of species susceptibility, the restricted geographic distribution of the viruses and the rarity of their occurrence in those areas where they have been reported.

3   The risk of paramyxovirus introduction through importations of the *commodity* is negligible.

4.1.7 Picornaviridae

Picorna-like viruses have been reported in barramundi, grass carp, redspotted grouper, salmonids, sea-bass, turbot and Japanese parrotfish (Wolf, 1988; Humphrey, 1995). The associated syndromes range from mass mortalities to no clinical signs.

Viral nervous necrosis (VNN) is a vacuolating encephalopathy primarily of Japanese parrotfish, but also of a number of other marine fish species. VNN has not been described in salmonids, although an Atlantic salmon demonstrating pathology including heart rupture has reacted positively to a serological test employing monoclonal antibodies against VNN virus (pers. comm. Dr Tore Håstein, August 1997).

There have been two reports in American Fisheries Society Fish Health Section newsletters of picorna-like viruses isolated from salmonids in the USA. A picorna-like virus was isolated from a population of Atlantic salmon fingerlings cultured in fresh water in Washington state which were experiencing low-level mortality over a long period (McDowell et al, 1989). No clinical signs of disease have been seen in experimentally challenged Atlantic salmon or rainbow trout, but the virus causes cytopathic effect in cell cultures. The virus has been isolated only once (McDowell et al, 1989). A picorna-like virus has also been isolated from adult brook trout, brown trout, cutthroat trout and rainbow trout at several locations in Northern California (Yun et al, 1989). No clinical signs or pathology were seen in adult or juvenile fish, although cytopathic effects were evident in cell culture. The virus could be transmitted horizontally by water-borne exposure. Isolation of virus from sex products suggested the possibility of vertical transmission but this has not been experimentally confirmed.

So, while picorna-like viruses have been reported in association with low-grade disease in salmonids their causative role has not yet been demonstrated.

Conclusions

1. Picornaviruses are important pathogens in aquatic animals. The picornaviruses known to infect salmonids do not appear to be of international quarantine or economic significance. Salmonids are probably of low susceptibility to VNN, if at all.

2. Salmonids infected with picornaviruses are unlikely to be harvested and processed for human consumption, because of species susceptibility, the restricted geographic distribution of the viruses and the rarity of their occurrence in salmonids.

3. The risk of picornavirus introduction through importations of the *commodity* is negligible.

4.1.8   Reoviridae

Wolf (1988) listed five reoviruses affecting fish. Another review of the literature (Humphrey, 1995) listed nine reoviruses of fish, of which five affect salmonids. The pathology associated with reovirus infections in salmonids is typically confined to the liver. The liver is also the sample of choice for virus isolation.

Bluegill hepatic necrosis virus was originally isolated from healthy oysters (*Crassostrea virginica*) in the USA. Experimental infection of rainbow trout has been demonstrated, and caused subclinical hepatic necrosis and occasional mortalities (Wolf, 1988). Chum salmon reovirus was originally isolated from healthy chum and masou salmon returning to Hokkaido (Winton et al, 1981). Chum, chinook, and kokanee salmon and rainbow trout can be experimentally infected. No clinical signs of disease are observed in either experimentally or naturally infected fish. Small foci of hepatic necrosis are seen histopathologically (McAllister, 1993). A reovirus of low pathogenic significance for naturally and experimentally infected salmonids has been isolated from coho and chinook salmon in Oregon (Winton et al, 1988). Two reoviruses have been reported in Taiwan. A reovirus affecting landlocked masou salmon has caused epidemic mortalities of fry and egg stages. The virus can be isolated from the liver and gills. (McAllister, 1993). A further reovirus in Taiwan affects rainbow trout (Humphrey 1995). Chum salmon reovirus has been the subject of survival studies in pond water which demonstrated sufficient retention of infectivity to sustain fish to fish transmission (Brady et al, 1993).

More recently, an aquareovirus was isolated from the ovarian fluids of adult chinook salmon in Alaska. Following DNA-hybridisation the isolate appeared different to the many other aquareoviruses isolated around the world (OIE, 1996).

Conclusions

1   Reoviruses of varying pathogenicity for salmonids exist within localised geographical distributions.

2   Salmonids infected with reoviruses are unlikely to be harvested and processed for human consumption because of species and age susceptibility and geographic distribution.

3   Heading, gilling and gutting of fish is likely to significantly reduce the number of infective particles in any reovirus infected salmonid harvested for export to New Zealand.

4   The risk of a salmonid reovirus introduction through importation of the *commodity* is negligible.

4.1.9 Retroviridae

Plasmacytoid leukemia (PL), or marine anaemia, may be caused by an oncogenic retrovirus. PL is a disease of chinook salmon in British Columbia (Kent et al, 1990). PL is not reported to occur anywhere else, although pathology similar to that of PL has been observed in chinook salmon reared in California and Washington state (Kent, 1992).

Although first recognised in farmed market-size chinook salmon between 2-4 kg (Kent et al, 1990), PL has also been shown to occur in wild-caught chinook salmon (Eaton et al, 1994). PL has been experimentally transmitted to chinook, sockeye and Atlantic salmon by injection, but cross-infections in sea water and fresh water proved difficult or impossible to establish, and the possibility of vertical transmission is being examined (Kent and Dawe, 1990; Newbound and Kent, 1991; Kent, 1992). Although the etiology of the disease has not been definitively determined, there is compelling evidence suggesting that PL is caused by an oncogenic retrovirus (Eaton and Kent, 1992; Kent and Dawe, 1993). The virus has been given the name salmon leukemia virus (Eaton and Kent, 1992).

The disease is characterised by pallor of the gills, anaemia, enlargement of the spleen and kidney, ascites and occasionally exophthalmia (Kent et al, 1990). Histologically there is massive proliferation of plasmacytoid cells in the kidney interstitium, spleen, intestinal lamina propria, pancreas, liver and heart, and in fish exhibiting exophthalmos also in the periorbital connective tissues, ocular muscles and choroid gland (Kent et al, 1990). The ubiquitous nature of PL in British Columbia and the lack of a documented spread of the disease suggest that PL is an endemic problem in farmed chinook salmon in British Columbia and not a spreading epidemic (Stephen and Ribble, 1995).

A swimbladder tumour has been observed in sea-caged Atlantic salmon in Scotland at a prevalence of about 5% (McKnight, 1978). Affected fish show no external lesions, but may be lethargic. Internally multiple nodular tumours 5-30mm in diameter are evident on the swimbladder and protruding into the abdominal cavity, with no evidence of invasion or metastasis (McAllister, 1993). Virus can be seen in electron microscopy of tumour tissue. The significance of the virus remains unknown. It has not yet been isolated in cell culture.

Humphrey (1995) has found reference to five other retroviruses affecting fish which are not known to affect salmonids.

Conclusions

1   Plasmacytoid leukemia is a disease of chinook salmon endemic to British Columbia, and is probably caused by an oncogenic retrovirus. The disease is of economic significance within its limited geographical distribution in farmed chinook salmon.

2   Despite on-going and large-scale exports of eviscerated chinook salmon out of British Columbia plasmacytoid leukemia does not appear to have been spread to any regions outside of British Columbia.

3   Salmonids with clinical plasmacytoid leukemia are unlikely to be harvested and

processed for human consumption

4   Atlantic salmon swimbladder sarcoma virus is suspected to be the cause of swimbladder sarcomas, a condition of farmed salmon of significance within the limited geographical distribution of the disease.

5   Salmonids infected with Atlantic salmon swim bladder virus are unlikely to be harvested and processed for human consumption, because of species susceptibility, geographic distribution and prevalence.

6   Heading, gilling and gutting of fish is likely to significantly reduce the number of infective particles of any Atlantic salmon swim bladder virus or PL infected salmonids harvested and processed for human consumption.

7   The risk of either plasmacytoid leukemia or Atlantic salmon swim bladder virus introduction through importation of the *commodity* is negligible.

4.1.10 Rhabdoviridae

Introduction

Two significant pathogens of salmonids are viruses in the family rhabdoviridae: infectious haematopoietic necrosis virus (IHNV) and viral haemorrhagic septicaemia virus (VHSV).

Two other rhabdoviruses are also known to affect salmonids, and can be dealt with briefly. A rhabdovirus causes hepatitis in fingerling, yearling and spawning rainbow trout in the Ukraine (McAllister, 1993). Wolf (1988) discusses the Ukrainian rhabdovirus as an isolate of VHSV. Rainbow trout can be experimentally infected with eel rhabdovirus, which has been isolated from eels and elvers experiencing haemorrhagic disease in North America, Europe and Japan. The significance of the rhabdovirus is unknown (Wolf, 1988). Wolf (1988) notes nine rhabdoviruses affecting fish, including the internationally important spring viraemia of carp (SVCV).

Strains of IHNV and VHSV are recognised in different geographical locations. Strains of both viruses found in North America and Europe differ. The genetic diversity of strains is greater for VHSV than for IHNV. This has led to the suggestion that strains of VHSV have become endemic to Europe and North America with little transmission between populations, while the relatively high degree of genetic uniformity between IHNV isolates suggests that transmission of virus is occurring between salmon populations (Oshima et al, 1993; Oshima et al, 1995). There is on-going research into the genetic uniformity of IHNV strains.

Both IHNV and VHSV are designated as notifiable diseases by the OIE (OIE, 1995a).

VHSV

Susceptibility and distribution

The species known to be susceptible to VHSV infection have been reviewed by Wolf (1988), McAllister (1993) and Humphrey (1995). In Europe, epidemics occur primarily in rainbow trout and brown trout. Northern pike, grayling, whitefish, pollan, lake trout and Atlantic cod are also susceptible to natural infection. In the USA, natural infections occur in chinook and coho salmon, steelhead trout, Pacific cod and Pacific herring in the Pacific northwest. Atlantic salmon, brook trout, golden trout, rainbow trout/coho salmon hybrids, giebel, sea bass and turbot have all been demonstrated to be susceptible to experimental infection. Recently, the North American strain of VHSV has been isolated from harvest size farmed Atlantic salmon in British Columbia from a sea water net pen site showing unexplained mortalities (Traxler et al, 1995).

The North American strain of VHSV is known to be endemic in the Northeastern Pacific Ocean among herring stocks, with cod, tubesnouts and shiner perch also serving as reservoirs (Meyers and Winton, 1995; Traxler et al, 1995). North American VHSV is moderately pathogenic for Pacific herring, causing occasional self-limiting epidemics, but has been shown to be relatively non-pathogenic for several species of salmonids. When eight species of salmonid fish were exposed by waterborne challenge to four North American isolates of

VHSV at 10^5pfu/ml for 1 hour, mortality ranged from 0-7% suggesting that North American isolates are substantially less virulent than European isolates (James Winton, pers. comm. October 1996). Salmonids and cod infected with VHSV in the Pacific northeast have probably become infected by preying on infected herring (Meyers and Winton, 1995). In Washington, viral testing of salmon in the years 1991 to 1995 detected 0.01% (1 pooled sample of fish out of 51,947 fish sampled) fish infected with VHSV (pers. comm. Linda Chaves, Director, USDC NMFS, October 1995)

There is circumstantial evidence that European isolates of VHSV are also endemic among marine fish in the Atlantic Ocean. North Sea cod are known to be a host for VHSV, and the virus is known to be closely associated with skin ulcers in this species (Smail, 1995). The high pathogenicity of the European strain of VHSV for salmonids may be the combined result of exposure of rainbow trout, which is an introduced species, in a stressful environment of intensive culture and the high rate of mutation inherent in all rhabdoviruses (Meyers and Winton, 1995).

Denmark, France, Germany, Italy, Austria and Luxemborg reported infection with VHSV in 1995 (OIE, 1995c). European Union legislation designates zones free of VHSV, and places restrictions on movement of live fish, gametes and uneviscerated fish of susceptible species from non-designated areas into zones designated free. Since their introduction in 1993, these measures appear to have limited the spread of VHSV in the EU. Free movement of eviscerated fish products within the EU has not been suggested as causing spread of VHSV.

In 1995 there were 489 fresh water trout farms in Denmark. Surveillance has demonstrated zones free of VHSV infection. New outbreaks are monitored and an eradication programme operates. Danish Veterinary Laboratory data for the period 1990-1995 demonstrate that of 8,693 specimens submitted for virological examination 205 (2.35%) were infected with VHSV. A survey of 397 aquaculture facilities conducted in 1995 demonstrated that 33 (8.3%) were infected with VHSV. All the infected facilities were rainbow trout farms. Of the 1,442 pools of samples collected during the 1995 survey, 15 (1.04%) were infected with VHSV (pers. comm. Niels Jørgen Olsen, Danish Veterinary Laboratory, 21 October 1996).

An outbreak of VHSV occurred in a turbot farm on the island of Gigha (off the west coast of Scotland) in 1995 (OIE, 1995c). An investigation concluded that the source of infection was either from North Sea Haddock used as unpasteurised food, or transmission from local marine fish via water or cohabitation (pers. comm. Alan Munro, August 1997). An eradication programme appears to have been successful, with continued monitoring of salmon farms confirming the absence of infection (OIE, 1996).

More recently, an outbreak of VHS occurred on a turbot farm at Cape Clear Island, County Cork, Ireland, in July 1997, and approved zone status of the island and its vicinity was suspended (pers. comm. John McArdle to Tore Håstein, 9 July 1997). The whole of mainland Great Britain remains an approved zone for VHSV.

In Bavaria, VHSV was detected in 16% of 1,413 cases submitted for diagnosis and neutralizing antibody was detected in fish on 60% of farms during a 5 year period (Wolf, 1988, citing Wizigmann et al, 1983). In Switzerland, examination of imported trout over a 5

year period detected a 1.5% prevalence of VHSV in 1,100 lots examined (Wolf, 1988, citing Meier, 1984).

VHSV has not been recorded in Asia, South America or Oceania.

Pathology, epidemiology and control

The clinical signs, pathology and epidemiology of VHSV have been reviewed by Wolf (1988) and McAllister (1993). These reviews refer primarily to European strains of VHSV.

North American strains of VHSV are opportunistic pathogens of herrings when stressed (Meyers and Winton, 1995). These strains are of low pathogenicity for salmonids, but the rapid mutation potential of rhabdoviruses may mean that the potential exists for them to become as virulent as European strains if introduced and allowed to persist in an intensive salmonid culture environment (Meyers and Winton, 1995).

Transmission of VHSV can occur to fish of all ages, although infections are more severe and mortalities higher in young fish. Natural infections occur by horizontal transmission of waterborne virus (Wolf, 1988). Vertical transmission has not been demonstrated and is thought not to occur (Wolf, 1988). Mechanical transmission on contaminated fomites may be important in the hatchery environment (McAllister, 1993).Water temperature is of paramount importance in the transmission and production of disease, with transmission and disease occurring in the range 1-12°C but not above 15°C (Wolf, 1988).

Herons may be able to act as mechanical vectors of VHSV (Peters and Neukirch, 1986). In contrast to IPNV, it has been demonstrated that VHSV loses its infectivity during passage through sea gulls, most likely as a result of the high acidity of the anterior digestive tract and the 40°C avian body temperature (Wolf, 1988).

Entry of the virus into susceptible fish is via the gills. The oral route has not been infective for salmonids, probably due to the acid lability of the virus. Pike have been infected by feeding infected trout (Wolf, 1988, citing de Kinkelin et al, 1979, and Ahne, 1980). Infection by immersion has been achieved by holding fish in 5 x 10^4 PFU/ml virus in 3 L of water for 3 hours (Wolf, 1988). Primary replication is in the gills and the vascular endothelium, principally of the kidney and spleen. Outbreaks of mortality occur at temperatures in the range 3-12°C, especially 3-5°C. Mortality and the proportion of virus carriers in survivors decreases at higher temperatures. Deaths at temperatures above 12°C are rare.

Acute clinical signs associated with epidemic mortality are lethargy, darkening, exophthalmia, anaemia, haemorrhages in the eyes, skin, gills and at the base of fins, and distended abdomen as a result of oedema of the kidneys, liver and spleen. Chronically infected fish are similarly affected but not as obviously haemorrhagic. Histological changes are confined to the kidney, liver and spleen. Virus concentrations are highest in the kidney and spleen, but also occur in sex products, urine, liver, heart and muscle. Little virus can be recovered in the faeces. Extravasation of blood can be found in the skeletal muscle of some infected fish (Wolf, 1988). A nervous form of VHS leads to high levels of virus in the brain, and presumably the spinal cord (pers. comm. Alan Munro, August 1997).

Control of VHSV is achieved through preventing contact between virus and host. Hatchery disinfection and restocking from pathogen free sources is the most effective means of control following epidemics in hatcheries. Survivors are resistant to reinfection (Wolf, 1988).

Table 5. Infectivity of different organs of rainbow trout that died during an outbreak of viral haemorrhagic septicaemia (determinations made after 8 months storage of specimens at -20°C) (Wolf, 1988)

Organ	Range of titres[a]
Kidneys	5.5-6.5
Spleen	4.5-6.8
Stomach	3.2-4.2
Muscle	2.5-4.5
Liver	2.2-3.2
Hindgut	1.8-4.2
Foregut	-2.2

[a] Infectivity expressed as exponent of $\log_{10}$ $TCID_{50}$/g.

Survival and inactivation

Wolf (1988) reviewed the survival and inactivation characteristics of VHSV. The virus is ether, heat and acid labile (at pH 3). It is stable at pH 5-10. The virus is stable through freeze-thaw cycles, but loses infectivity at temperatures above 20°C. VHSV is readily inactivated by a range of common disinfectants, but may persist in aquatic environments under the right environmental conditions for long periods.

Table 6. Effects of selected chemicals and environmental and physical factors on infectivity of Egtved virus (Wolf, 1988)

Agent or factor	Inactivation (%)	Period
Formalin 3%	100	5 min
NaOH 2%	100	5 min
Chlorine 500 ppm	99	<2 min
Actomar (I_2) 0.01%	100	5 min
Tap water at 10°C	90	14 days
Stream water at 10°C	90	14 days
Mud at 4°C, pH 7.4	>99	10 days
Drying at 15°C	>99	14 days
Gamma irradiation 1.7 x 10^5 rads in tap water	100	-
UV irradiation at 254 nm at 5cm	100	10 min

IHNV

Susceptibility and distribution

The species known to be susceptible to IHNV infection have been listed by Wolf (1988), McAllister (1993) and Humphrey (1995). Rainbow, amago, sea-run cut throat and kamloops trout and sockeye, Atlantic, chinook, chum and yamame salmon can be infected naturally. Northern pike can be experimentally infected. Coho salmon, brook, brown and cut throat trout are considered to be refractory to disease, although the virus has been isolated from clinically normal fish of these species.

The susceptibility of Atlantic, chinook and sockeye salmon to North American strains of IHNV has been evaluated by bath challenge in sea water, intra-peritoneal injection, and cohabitation with injected fish, in order to establish the potential risk of fish contracting IHN at netpen sites (pers. comm. G.S.Traxler, October 1996). In this study, Atlantic salmon were susceptible to IHN by each method of exposure, and produced higher viral titres than sockeye. Sockeye were susceptible to injection and cohabitation. Chinook were not found to be susceptible by any of the challenges.

Concern that a marine reservoir may exist or become established led to transmission trials being carried out involving marine fishes commonly found in and around net pens in British Columbia. Challenge experiments involving tubesnouts (*Aulorhynchus falvidus*), shiner perch (*Cymatogaster aggregata*), and Pacific herring (*Clupea pallasi harengus*) indicated that all three species were susceptible by intra-peritoneal injection with losses in excess of 50% in all species, and that herring were the most susceptible by immersion, with losses of 25% in exposed fish (pers. comm. G.S.Traxler, October 1996).

IHNV is endemic along the Pacific Coast of North America from northern California to Alaska. IHNV is suspected to be the cause of epidemic mortalities in sockeye salmon in Alaska as early as 1925 and again in 1946 (Wolf, 1988). In Alaska IHNV is endemic in tested stocks of anadromous sockeye salmon, and was a major cause of failure in culture of this species until the implementation of a strict statewide policy directed towards minimising losses in alevins (Meyers et al, 1990). IHNV is believed to have been introduced into Japan as a result of importations of sockeye salmon eggs from Alaska (Wolf, 1988). IHNV has also become established in regions of Europe, resulting in legislative measures to prevent the spread of disease. The virus has been disseminated to other areas of the USA and the world via the movement of live infected fish and contaminated fish eggs from the Pacific Northwest of North America (Wolf, 1988; McAllister, 1993). IHNV has also been shipped with eggs or fry to some locations in the USA where outbreaks have occurred in the original stock, but where the virus has apparently failed to become established (Wolf, 1988).

Some genetic variation amongst IHNV isolates occurs, but there may be a high degree of genetic uniformity relative to VHSV. Diversity is based upon geographic location rather than host species (Oshima et al, 1995). If viral populations were genetically isolated, more variation among isolates would be expected, given the high error rate of RNA polymerase (Oshima et al, 1995). This suggests continuing transmission of virus between salmon populations, although other factors may also explain the low level of genetic variation (Oshima et al, 1995).

Prevalence data for IHNV must be interpreted with caution, as important epidemiological aspects of infection must be taken into account when raw data on positive diagnoses in laboratory submissions are examined. In particular, the detectable presence of IHNV in Pacific

salmon is known to be associated with the stage of sexual maturation of fish sampled. IHNV is typically associated with spawning adults and clinically affected juveniles. On a single occasion IHNV has been isolated from adult sockeye salmon in sea water (Traxler and Roome, 1993). Sequential sampling studies of sockeye salmon returning from sea have demonstrated that IHNV is undetectable until during or after spawning (Mulcahy et al, 1984; Traxler, 1983). Atlantic salmon do not follow this pattern and infection with IHNV can occur at all stages of maturity (pers. comm. G.S.Traxler, October 1996).

Some data are available to indicate the IHNV prevalence expected in historically and newly infected regions. In Alaska, surveillance data for the years 1980 to 1994 indicate an IHNV prevalence of 21.6% (2,379 of 11,004) in Pacific salmon (pers. comm. Linda Chaves, October 1995). In British Columbia, of all the wild, adult Pacific salmon samples submitted to the Pacific Biological Station between the period 1985 to 1994, IHNV was detected in 2% (48 of 2,331) of cases (pers. comm. Trevor Evelyn, October 1996). In Washington, viral testing of salmon in the years 1991 to 1995 has shown 0.8% (399 of 51,947) to be infected (pers. comm. Linda Chaves, October 1995).

When the above data are examined on a species basis, the susceptibility of sockeye salmon to IHNV becomes obvious, with 34.3%(2,371 of 6,907), 21.1% (38 of 180) and 13.9% (239 of 1,710) infected in Alaska, British Columbia and Washington, respectively. In recent years the IHNV prevalence in Alaskan sockeye salmon production has declined since the introduction of hatchery measures such as egg disinfection with iodophor, use of virus-free water, and compartmentalization (Meyers et al, 1990).

Denmark, UK, Ireland and Norway have not recorded IHNV (OIE, 1995c). IHNV is present in France, and is particularly troublesome in rainbow trout aquaculture (Hattenberger-Baudouy et al, 1995). Parts of France are designated approved zones for IHNV (OIE, 1996). Quantitative data on IHNV prevalence for France and other European countries have not been obtained. Belgium, France, Germany, Austria and Italy reported infection with IHNV in 1995 (OIE, 1995c). European Union legislation designates IHNV free zones, and places restrictions on movement of live fish, gametes and uneviscerated fish of susceptible species from non-designated areas into designated free areas. These measures appear to be effective in limiting the spread of IHNV. Movement of eviscerated salmonid products for human consumption in the EU has not been associated with spread of IHNV.

Japanese surveillance data for the period 1976 to 1991 shows that of 15,432 mature females of five species of salmonid fish sampled at 47 locations in northern Japan, IHNV was detected from 11 fish (0.07%) (Yoshimizu et al, 1993).

Pathology, epidemiology and control

The clinical signs, pathology and epidemiology of IHNV have been reviewed (Wolf, 1988; McAllister, 1993). Most outbreaks occur in juvenile Pacific salmon during spring, in water temperatures of 12°C or less, although some outbreaks have been reported at temperatures of 15°C. Typically outbreaks affect fry or fingerlings, with mortality approaching 100%, while outbreaks in smolts normally result in lower mortalities. During epidemics there is massive

contamination of water and horizontal transmission of virus. Clinical signs in young fish include darkening, exophthalmia, pale gills, ascites, white faecal casts trailing from the vent, anaemia and erratic swimming (lethargy to whirling). Haemorrhages are evident at the base of pelvic and pectoral fins, at the vent, and occasionally in the skin musculature and mouth. Scoliosis and lordosis occur in 1% to 5% of survivors. Extensive pathology is present in the kidneys, spleen, liver, pancreas and in the granular cells of the alimentary tract. Infection in Atlantic salmon is not uncommon in older fish (up to harvest weights), and histological lesions may be more severe than in sockeye salmon (Traxler et al, 1993).

In nature, horizontal transmission is the most important means of spread of infection, with infected fish shedding virus in faeces, urine, and sex products causing contamination of eggs, fry and other fish. Virus may be present in ovarian fluids, and will cause superficial contamination of the egg, resulting in egg-associated transmission. During spawning there is contamination of water supplies and organic debris in river bed sediments with IHNV. Whether or not true vertical transmission occurs remains a subject of debate, but seems unlikely given the success of egg disinfection in Alaska.

Stressors within the hatchery environment (crowding, handling, nutrition, chemical exposure etc) may increase susceptibility in hatchery reared fish and contribute to epidemic disease outbreaks. Survivors of epidemics may become latent virus carriers, with physiological stresses associated with spawning causing reactivation of latent infection. The latent carrier state has not been demonstrated as IHNV is detectable only during or after spawning in sockeye salmon (Mulcahy et al, 1984), although infection in sockeye salmon in sea water has been reported on one occasion (Traxler and Roome, 1993). Infection with IHNV in sea water netpens is recognised as being a significant economic problem in culture of Atlantic salmon in British Columbia (pers. comm. Joanne Constantine, British Columbia MAFF, October 1996).

The virus can be mechanically transmitted by fomites and on birds, animals, and possibly insects.

The route of entry of IHNV may be from the gills into the circulatory system, or from the oral region into the gastrointestinal tract and then into the circulatory system. Traxler et al (1993) demonstrated that immersion for 3 hours in a concentration of 8.98×10^3 pfu/ml IHNV was sufficient to cause infection and death in Atlantic salmon, but not in sockeye or chinook salmon. The minimum gill titre required for infection to become systemic has been measured at 10^5 pfu/g (Mulcahy et al, 1983). Fish with gill titres lower than this showed only local gill IHNV infection. Once in the blood the virus is widely disseminated to all tissues (Drolet et al, 1994). The sequential progression of virus through tissues was determined by alkaline phosphatase immunohistochemistry (APIH) in steelhead fry, and it was shown that within five days of exposure (by immersion) virus is detectable in all tissues including muscle, skin and cartilage (Drolet et al, 1994).

The titre of IHNV in eight organs and two body fluids of 60 naturally infected sockeye salmon pre-spawning, at spawning, and post-spawning has been determined (Mulcahy et al, 1982). Despite virus being detected in every individual fish, it was not detectable in serum of prespawning females (of the four males in the population, stage of spawning was not specified). Visceral tissue titres were consistently high in pre-spawning females $(5.0 \times 10^1$ to

1.9 x 10^8 pfu/g), and in ripe and spent females (5.0 x 10^1 to 3.2 x 10^8 pfu/g). Viral titres in gonadal fluid were consistently highest, and the study concluded that gonadal fluid was the sample of choice for viral detection (Mulcahy et al, 1982).

A similar study reported IHNV titres in pre-spawning sockeye salmon females earlier in the spawning migration (Mulcahy et al, 1984). Pre-spawning fish were chosen at random from fish caught by a commercial purse seiner. Of 15 pre-spawning fish, 11 were positive for IHNV. Virus titres in various organs and fluids were measured (table 7).

Table 7. IHNV titres in tissues and fluids of 11 IHNV positive female pre-spawning sockeye salmon (of 15 pre-spawning fish sampled) (Adapted from Mulcahy et al, 1984)

Tissue/fluid	% positive	Geometric mean titre for positive fish[a]
Gill	91	3.58
Kidney	9	2.35
Spleen	27	2.52
Pyloric caeca	36	2.51
Brain	9	1.50
Eggs	9	2.40
Serum	0	-
Lower gut	0	-
Liver	0	-

[a] Mean viral titres are expressed as $\log_{10}$ pfu/g or pfu/ml for tissues or fluids respectively.

Mean viral titres in experimentally infected 20g sockeye salmon in sea water were 6.9 x 10^6 pfu/g in injected fish and 1.6 x 10^3 pfu/g in fish infected by cohabitation (pers. comm. Traxler, October 1996). Mean viral titres in experimentally infected 90g Atlantic salmon in sea water were 7.1 x 10^4 pfu/g in fish infected by immersion, 3.3 x 10^5 pfu/g in injected fish and 1.2 x 10^6 pfu/g in fish infected by cohabitation (pers. comm. Traxler, October 1996).

IHNV has also been detected in the mucus of naturally and experimentally infected juvenile and adult chinook and sockeye salmon and rainbow and steelhead trout. Virus titres ranged from 10^1 to 10^6 pfu/ml (La Patra et al, 1989).

Despite the above studies, the tissue residence site and concentration of IHNV during the period of latency remains unknown.

Combining the available data on tissue titres and infectious dose allows the likelihood of headed, gilled and gutted salmonids introducing IHNV into New Zealand to be illustrated using a calculation from the previous MAF salmon risk analysis (MacDiarmid, 1994). MacDiarmid assumed that 200 tonnes of product might be imported each year. This lead to a worst-case scenario estimate of 700 households consuming 0.87 kg of imported product on a single day.

If this consumption pattern occurred, and 17% of the salmon ended up as scraps in household waste water, MacDiarmid calculated that 51.76 kg of salmon scraps might enter the waste

water system, along with the 350,000 litres of waste water produced by this number of households on a daily basis.

We know that most populations of salmon harvested for human consumption will not have IHNV prevalence as high as spawning sockeye salmon. The data for spawning salmon indicate that prevalence of 14-35% may be expected. However, to ensure that the estimate of risk is conservative, we will assume that every fish was infected with IHNV.

We also know that clinically infected fish are unlikely to pass inspection and grading, so the infection of imported fish is likely to be the latent carrier state. However, to ensure further conservatism, we will also assume that the muscle, skin and bone contain IHNV titres equivalent to the geometric mean gill titre of IHNV positive pre-spawning sockeye salmon as determined by Mulcahy et al (1982) i.e. 5.8×10^3 pfu/g of tissue. In this case, the entire 51.76 kilos of scraps would contain a total of approximately 3×10^9 IHNV pfu.

When this viral load was suspended in 350,000 litres of waste water it would achieve a final dilution of 8.5 pfu/ml at the point of sewage discharge, if all 700 households were discharging their waste water into the same sewage system (in itself unlikely). This concentration is 3 orders of magnitude more dilute than the 8.98×10^3 pfu/ml for 3 hours Traxler et al (1993) demonstrated would infect Atlantic but not chinook or sockeye salmon.

The actual risk may be lower than even this calculation indicates, for a number of reasons. Salmon are unlikely to be found in the proximity of a sewage outfall (MacDiarmid, 1994). Atlantic salmon populations in New Zealand are land-locked, so this species is even less likely to contact sewage outfalls, and other salmon species require higher concentrations for transmission to occur. The viral concentration in the discharged waste water will decrease exponentially with distance from the outfall such that for each metre distance from the outfall dilution will increase by a cubic metre.

On the other hand, there are various factors not taken into account by this simplistic calculation which might increase the actual risk. These include the improbability that this viral load would be evenly distributed throughout the total daily waste water output; scraps may contain concentrations of virus and evade filtration processes; and susceptible marine fishes requiring lower infectious doses may be found in the proximity of a sewage outfall.

Nevertheless, the assumptions probably ensure that the calculation gives a conservative estimate of risk.

Survival and inactivation

IHNV is heat, acid and ether labile (Wolf, 1988). IHNV is readily inactivated by a variety of physical and chemical treatments. In particular, iodine and iodophor have been demonstrated to be 99.9% effective in inactivating the virus within 7.5 seconds of exposure to 0.1 mg/ml (Batts et al, 1991). The effect on IHNV of gamma irradiation, ultraviolet light, acetone, ether, formaldehyde, hypochlorite and ozone have been reviewed (Humphrey, 1995), demonstrating the lability of the virus to these disinfecting methods. IHNV is most stable within the pH range 6-8, and is inactivated 2-3 times faster outside this range (Pietsch et al, 1977). IHNV is

destroyed by exposure to 0.5 mg/ml chlorine in 5 minutes (Wedermeyer et al, 1978). IHNV is susceptible to thermal inactivation, with poor survival at temperatures of 32°C and above (Gosting and Gould, 1981). The presence of serum has a protective effect on IHNV, and enables the virus to resist freeze-thaw cycles. In the presence of serum IHNV could be expected to persist for several years at -20°C (Pietsch et al, 1977). In fish tissues, however, the virus is known to survive freezing poorly. Virus appears to only survive freezing well if suspending media are supplemented with high concentrations of dissolved protein, such as 10% serum, as a four log decrease, from 10^6 to 10^2, has been reported following a single freeze thaw cycle in fish tissues (pers. comm. Trevor Evelyn, August 1997).

Under the right environmental conditions, IHNV may persist for considerable periods outside the host. IHNV has been found to be stable at 10°C for 7 weeks in lake water, and at 15°C for 25 days in fresh water, for 14 days in sea water, and for 14 days in estuarine water respectively (Wedermeyer et al, 1978; Toranzo and Hetrick, 1982; Barja et al, 1982). Following widespread environmental contamination at spawning, IHNV viral titres may remain high in river sediments for months (Mulcahy et al, 1983).

Conclusions

1   VHSV and IHNV are serious pathogens of salmonid fish with well-defined geographical distributions.

2   Salmonid virus carriers are important in the epidemiology of both VHSV and IHNV, as may be marine fish species acting as reservoirs.

3   The relatively low genetic diversity of IHNV isolates from around the world, when compared to isolates of VHSV, may be the result of on-going transmission of IHNV among salmonid populations. This may reflect a greater risk of transmission of IHNV over VHSV through movements of fish and fish products.

4   Live fish, fish eggs, and untreated offal or fish farm wastes are the highest risk commodities for spread of VHSV and IHNV. Trade in eviscerated salmonids has not been suspected to have contributed to the spread of VHSV and IHNV.

5   European Union legislation allows eviscerated salmonid flesh for human consumption to be exported from VHSV and IHNV infected areas into approved free zones. This trade has not been suspected to have contributed to the spread of VHSV and IHNV.

6   The likelihood of a fish clinically infected with VHSV or IHNV being harvested and/or being processed for human consumption is low.

7   The likelihood of a fish latently infected with VHSV or IHNV being harvested and processed for human consumption is high.

8   The titre of VHSV or IHNV present in a headed, gilled and gutted carrier salmonid processed for human consumption is likely to be relatively low.

9   Infectivity of any VHSV or IHNV present could be maintained for long periods in tissues of any frozen fish product, but considerable inactivation occurs on thawing of the product, particularly with IHNV. Infectivity in fresh product is less likely to be maintained for long periods.

10   If imported in fish tissues and placed into the aquatic environment in New Zealand, some VHSV or IHNV could remain viable for extended periods.

11   The likelihood of environmental contamination of VHSV or IHNV in concentrations capable of causing infection of salmonids in New Zealand resulting from importations of the *commodity* is low.

12   The importation of uneviscerated marine fish species reported to act as reservoirs for IHNV or VHSV from endemic areas represents an alternative pathway for the introduction of these viruses into New Zealand.

13   The risk of VHSV or IHNV introduction through importations of the *commodity* is low.

4.1.11 Togaviridae

Although no togaviruses have been definitively accepted as causing diseases of salmonids, one disease is discussed here because of a recent report of suspected togaviral aetiology.

Pancreas disease of farmed Atlantic salmon has been recognised in Scotland, Ireland, Norway, the USA, France and Spain. The disease was first recognised in farmed Atlantic salmon in Scotland in 1976 (Munro et al, 1984). Natural infection is recognised in brown trout, and the disease has been experimentally transmitted to rainbow trout (Boucher et al, 1995).

Although associated primarily with post-smolts in sea water, the susceptibility of salmonid parr in fresh water has been demonstrated experimentally (McVicar and Munro, 1989; Houghton, 1995).

An infectious aetiology has been suspected for some time (McVicar, 1986), but it is only recently that a virus with physicochemical characteristics and morphology resembling the togaviruses has been isolated from infected fish and transmitted to salmon to cause the characteristic disease syndrome (Nelson et al, 1995).

The pathognomonic lesion in pancreas disease is vacuolation leading to generalised haemorrhagic necrosis of the acinar cells of the exocrine pancreas. Lesions in the cardiac and skeletal muscle, as well as in gills, eyes and gut, may occur as chronic sequelae to acute infection (McVicar and Munro, 1989). All fish in an affected population may suffer exocrine pancreas pathology, but a variable proportion go on to suffer atrophy of the exocrine tissue and lesions in other tissues. Recovery following infection occurs in a significant proportion of affected fish (McVicar and Munro, 1989). Infectivity of plasma, blood leucocytes, splenocytes and kidney homogenates has been demonstrated (Houghton, 1995). Infectivity was lost following exposure to pH 3 and to chloroform (Nelson et al, 1995).

Pancreas disease is one of the most economically important diseases in Scottish salmon farming (McVicar and Munro, 1989).

In France, isolation of a virus causing 'sleeping disease' of rainbow trout has been achieved in the RTG-2 (rainbow trout gonad-2) cell line, and the virus also appears to be a togavirus (OIE, 1996).

Conclusions

1   Pancreas disease is an economically significant disease primarily affecting juvenile Atlantic salmon in sea water in areas of Europe and the Western USA. Pancreas disease is caused by an infectious agent which may be a togavirus.

2   Little is known about the epidemiology of pancreas disease. However, sufficient is known to suggest that the likelihood of a fish clinically infected with pancreas disease being harvested and processed for human consumption is low.

3   The tissue distribution and titre of togavirus thought to cause pancreas disease is not

yet known, and neither is the means of natural transmission. The difficulty in microscopically detecting, isolating and transmitting the infective agent of pancreas disease may reflect low viral titres in target tissues and/or poor survival outside the host cell.

4   The risk of pancreas disease, or any other togavirus of salmonids, introduction through importations of the *commodity* is negligible.

4.1.12 Uncharacterised viruses

A number of diseases affecting salmonids are thought to be caused by viruses but the viral agent has not been positively identified. These diseases will be discussed individually.

Salmon anaemia virus/Erythrocytic inclusion body syndrome (EIBS)

Introduction

EIBS is a disease of salmonids characterised by a severe anaemia (Leek, 1985). The causative virus has not been isolated. The disease is associated with intracytoplasmic erythrocytic inclusions which, when studied by electron microscopy, reveal accumulations of 80 nm icosahedral virions (McAllister, 1993).

Susceptibility and distribution

Both juvenile and adult fish can be naturally infected with EIBS.

EIBS has been reported in chinook and coho salmon and cutthroat trout in the Western USA, in farmed Atlantic salmon in Norway, Ireland and Scotland, and in coho salmon in Japan (Piacentini et al, 1989; Bruslind et al, 1994; Lunder et al, 1990; Rodger et al, 1991; Takahashi et al, 1992; pers. comm. Alan Munro, August 1997).

EIBS is a major cause of mass mortality in coho salmon in saltwater net pens and fresh water culture in Japan (Takahashi et al, 1992). Rainbow trout can be experimentally infected (Piacentini et al, 1989). In Ireland EIBS was detected in all fresh water and marine farm sites examined, and prevalence in fish at sites ranged from 5-75% in fresh water and 23-95% in sea water (Rodger et al, 1991).

Pathology, epidemiology and control

In Pacific salmon EIBS is characterised by severe anaemia, leading to lethargy, dark or variable pigmentation, pallor of internal organs, and spleen enlargement (McAllister, 1993). EIBS in Atlantic salmon is less significant. Fish are apparently healthy and anaemia may not be present (pers. comm. Alan Munro, August 1997).

Diagnosis is confirmed by the presence of intracytoplasmic erythrocytic inclusions on examination of a blood film (Leek, 1987).

EIBS has been transmitted horizontally by waterborne exposure, intra-peritoneal injection of blood and kidney, and by oral intubation using infected tissues (Leek, 1985; Pelton, 1987; Piacentini et al, 1989; Arakawa et al, 1989;). The infectious dose is unknown. Whether vertical transmission or reservoirs of infection other than salmonids occur is also unknown. The sequelae to infection depend on the development of secondary infection; losses may be low, but will be higher if bacterial, fungal or parasitic secondary infections develop (Arakawa et al, 1989). Fish that recover are resistant to reinfection (Piacentini, 1989).

Disease outbreaks typically occur in cool water temperature months, and the appearance and persistence of viral inclusions is also temperature dependent (McAllister, 1993). Outbreaks may last for 5 months or more (Piacentini, 1989). No treatment or control methods are known, aside from management strategies to minimise stress (McAllister, 1993).

Survival and inactivation

No information is available on the survival and inactivation of the EIBS agent.

Conclusions

1   EIBS is an important pathogen of salmonids and is of economic significance in salmonid aquaculture in countries in which it occurs.

2   Pacific salmon clinically infected with EIBS are unlikely to be harvested and processed for human consumption without suffering quality downgrading.

3   Pacific salmon incubating EIBS, or possibly unapparent carriers of the EIBS agent, and infected Atlantic salmon may be harvested and processed for human consumption.

4   The amount of EIBS infectivity within any infected fish which is harvested is likely to be significantly reduced by bleeding and evisceration during processing.

5   The ability of the EIBS agent to survive in a product processed and imported for human consumption is unknown, as is the ability of the EIBS agent to persist in the environment and the dose required to infect salmonid species present in New Zealand.

6   The risk of EIBS introduction through importations of the *commodity* is probably low. The uncertainty reflects a lack of specific data concerning disease transmission.

Rainbow trout intra-erythrocytic virus

Wolf (1988) and McAllister (1993) describe a single instance in the USA of a fatal haemorrhagic disorder in a strain of rainbow trout. The instance was reported in 1977 and has not occurred since. The clinical signs affected adult females and consisted of exsanguinating haemorrhage, hypoxia, and sudden death. Erythrocytes of affected fish showed intracytoplasmic inclusions.

Conclusions

1   Rainbow trout intra-erythrocytic virus is of negligible economic or international quarantine significance.

2   The risk of rainbow trout intra-erythrocytic virus introduction through importation of the *commodity* is negligible.

Intra-erythrocytic viral disease of coho salmon

McAllister (1993) describes an intraerythrocytic virus detected in coho salmon in California and Washington causing severe anaemia. Pathological changes include diffuse degeneration of haematopoietic tissues of the kidney and spleen, accumulation of haemosiderin in macrophages, and the presence of numerous cytoplasmic inclusion bodies in circulating erythrocytes. Mechanism of transmission, and factors affecting prevalence and distribution are unknown.

Conclusions

1   Intra-erythrocytic viral disease of coho salmon is of negligible economic or international quarantine significance.

2   The risk of intra-erythrocytic viral disease of coho salmon introduction through importations of the *commodity* is negligible.

Atlantic salmon papillomatosis agent

Wolf (1988) and McAllister (1993) describe a benign epidermal papilloma occurring in Atlantic salmon in Great Britain, Norway, Sweden and the USA. The papillomas appear as single or multiple proliferations at any site on the body surface, which eventually slough and heal without evidence of invasion or metastasis. Mortality associated with secondary infection occurs in a low percentage of cases. Electron microscopic examination of papilloma tissue inconsistently reveals intracytoplasmic and extracellular retrovirus like particles, although attempts to isolate the virus have been unsuccessful.

Conclusion

1   Atlantic salmon papillomatosis virus is of minimal economic or international quarantine significance.

2   Fish clinically infected with Atlantic salmon papillomatosis virus are unlikely to be harvested and processed for human consumption without suffering quality downgrading.

3   The risk of Atlantic salmon papillomatosis virus introduction through importations of the *commodity* is negligible.

Ulcerative dermal necrosis agent

McAllister (1993) records ulcerative dermal necrosis as affecting adult Atlantic salmon and brown trout in Great Britain, Ireland, France and Sweden. The lesions occur as shallow ulcers which progress to deep ulcers, primarily on the head and adipose fin, but also on other body

surfaces. Secondary infections can lead to mortality. Electron microscopy of lesions has revealed what appear to be virus particles, but no virus has been isolated and ulcerative dermal necrosis has not been transmitted experimentally. The involvement of fungal organisms in the aetiology is suspected by some workers (pers. comm. Alan Munro, August 1997).

Conclusions

1   Ulcerative dermal necrosis is a disease of salmonids of limited international significance, for which an infectious aetiology has not been established.

2   Fish clinically infected with ulcerative dermal necrosis are unlikely to be harvested and processed for human consumption without suffering quality downgrading.

3   The risk of ulcerative dermal necrosis introduction through importations of the *commodity* is negligible.

A new virus of salmonids in Japan

An uncharacterised virus has been isolated from the brain of coho salmon, iwana, rainbow trout and ayu, and the ovarian fluid of masou salmon in the northern part of Japan (Oh M-J et al, 1995). The virus was isolated from juvenile and adult fish, and diseased fish exhibited abnormal swimming. The virus remains uncharacterised, although properties similar to a retrovirus have been noted.

The virus has been experimentally transmitted to coho salmon, masou salmon, iwana, steelhead trout and ito, and induced disease signs of spinning swimming and lethargic behaviour in infected fish, with cumulative mortalities up to 34% in coho salmon fry challenged by immersion, and 63% in coho salmon fry challenged by intramuscular injection. Viral antigen is detectable in kidney, brain and blood cells of infected fish.

Conclusions

1   An uncharacterised virus has emerged as a pathogen of salmonids in northern Japan. The economic significance of the virus is unknown. The geographical distribution of the virus appears to be very limited.

2   Fish clinically infected with the uncharacterised Japanese virus are unlikely to be harvested and processed for human consumption, considering the geographical distribution of the virus.

3   The ability of the uncharacterised Japanese virus to survive in a product processed and imported for human consumption is unknown, as is the ability of the uncharacterised Japanese virus to resist inactivation in the environment.

4   The risk of uncharacterised Japanese virus introduction associated with the importation of *the commodity* is negligible.

Other emerging viruses of salmonids

From time to time previously unknown viruses may emerge as pathogens of salmonids. Considering the range of viral pathogens examined in this risk analysis, the emergence of such pathogens is unlikely to affect the results of this risk analysis.

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4.2   QUALITATIVE ASSESSMENT OF THE BACTERIAL PATHOGENS OF SALMONIDS

4.2.1   *Aeromonas salmonicida*

Introduction

Aeromonas salmonicida is one of the most important fish pathogens because of its nearly worldwide distribution, diverse host range and economically significant impact on cultivated fish. More has been written about *A. salmonicida* than any other bacterial pathogen of fish. The review in this assessment will focus on aspects of the biology of the organism that potentially impact upon the risk of its introduction during the proposed importation of the *commodity*.

The traditional description of *A. salmonicida* is of non-motile, non-spore-forming, facultatively anaerobic, fermentative, Gram-negative rods, which produce a brown water-soluble pigment on tryptone-containing agar, which do not grow at 37°C, and which produce catalase and oxidase (Austin and Austin, 1993; Munro and Hastings, 1993).

Austin and Austin (1993) reviewed the literature relating to the strain variations that occur within the species, and summarised the debate on the taxonomical positioning of the species and various subspecies proposed. From their review, it appears that a consensus on these matters is some way off. For the purposes of this study it will suffice to note that strains of *A. salmonicida* have long been recognised which do not conform with the traditional description. These 'atypical' strains deviate from the classical description of the taxon over a number of biochemical and physiological properties, such as pigment production, biochemical profiles, motility and species predilection. Austin and Austin (1993) reviewed the numerous studies proposing a reviewed taxonomical classification of *A. salmonicida* on the basis of phenetic data, genotypic data, serological techniques, and bacteriophage typing. Probably the most enduring proposition is that of McCarthy and Roberts (1980). They proposed three subspecies of *A. salmonicida*, as follows:

Group 1: *A. salmonicida* subsp. *salmonicida*, comprising typical strains derived from salmonid fish;
Group 2: *A. salmonicida* subsp. *achromogenes*, comprising atypical strains derived from salmonid fish;
Group 3: *A. salmonicida* subsp. *nova*, comprising atypical strains derived from non-salmonid fish.

In an earlier MAF risk analysis (MacDiarmid, 1994), Dr Trevor Evelyn was reported as discussing the host range of *A. salmonicida* during the Canada- Australia Salmon Technical Meeting held in Nanaimo, British Columbia (BC), Canada, 1994. Evelyn notes that non-salmonids are nearly always infected with 'atypical' *A. salmonicida*. Non-salmonids may become infected with 'typical' *A. salmonicida* in stressful situations or when in close association with infected salmonids, but clinical manifestation seldom seems to result from these infections.

Susceptibility and distribution

A. salmonicida is the infectious cause of the disease furunculosis. Furunculosis occurs in nearly all species of salmonid fish, the one exception being rainbow trout which are more resistant to furunculosis than other species (Austin and Austin, 1993). Rainbow trout raised in sea water have been infected with 'atypical' *A. salmonicida* (Boomker et al, 1984). Furunculosis occurs in farmed salmonids in both sea water and fresh water. Outbreaks of furunculosis in Scotland diagnosed by the Marine Laboratory, Aberdeen, from 1979 to 1989 total 52 in fresh water sites and 127 in sea water sites (Munro and Hastings, 1993).

Atypical strains of *A. salmonicida* cause a variety of other disease conditions, both in salmonids and in non-salmonids. Ulcer disease and pasteurellosis in salmonids are now recognised as being caused by 'atypical' *A. salmonicida* (Austin and Austin, 1993; Munro and Hastings, 1993). 'Atypical' *A. salmonicida* infections in non-salmonids include carp erythrodermatitis in carp (Fijan, 1972), ulcer disease in goldfish (McCarthy and Roberts, 1980), and head ulcer disease in Japanese eels (Hikada et al, 1983). *A. salmonicida* has been isolated from Finnish flounders (Wiklund, 1995), turbot (Pérez et al, 1996), Atlantic cod (Cornick et al, 1984), sablefish (Evelyn, 1971), halibut and wrasse (Hjeltnes et al, 1995). Humphrey (1995) notes literature reports of 'atypical' *A. salmonicida* susceptibility in goldfish, silver bream, sablefish, minnows, carp, grayling, whitefish, chup, lampreys, plaice, bass and pike. It is likely that the species of marine and fresh water fish known to be susceptible to *A. salmonicida* will be expanded as further research is undertaken.

A. salmonicida has an essentially worldwide distribution, occurring in North America, South America, Europe, Asia, Australia and Africa (Austin and Austin, 1993; Munro and Hastings, 1993; Shotts and Nemetz, 1993; Humphrey, 1995). *A. salmonicida* is endemic in south eastern Australia, excluding Tasmania, having been introduced in goldfish imported from Japan in 1974 (Humphrey and Ashburner, 1993). The Australian strain of *A. salmonicida* is phenotypically distinct and conforms best with Group 3 strains, that is, 'atypical' strains associated with diseases in non-salmonid fish. Efforts are being maintained at the state level to limit the spread of the pathogen in Australia (Humphrey, 1995). *A. salmonicida* has not been reported in New Zealand, and survey data suggest that it is not present here (Anderson et al, 1994).

A disturbing recent development is the isolation of antibiotic resistant strains of *A. salmonicida.* Strains resistant to oxytetracyclines and potentiated sulphonamides have been isolated on the Atlantic coast of Canada (OIE, 1996).

Prevalence data for *A. salmonicida* must be interpreted with caution, as a number of factors will affect detected prevalence. These factors include the age, stage of sexual maturity and location (either fresh water or sea water) of the population surveyed, which may be inter-related. Numerous studies have demonstrated an increasing prevalence of detectable *A. salmonicida* infection as anadromous wild salmonids enter fresh water and mature sexually. Data from surveillance which targets fish exhibiting clinical signs of disease introduces a bias that will tend to indicate prevalence higher than would be expected in the general population. The diagnostic method employed also affects the detected prevalence. Increasingly sensitive diagnostic procedures are becoming available. Techniques such as ELISA are able to detect

the presence of *A. salmonicida* in asymptomatic fish when bacteriological culture failed to do so (Hiney et al, 1994). Aquaculturalists in many countries are able to diagnose furunculosis on the basis of clinical signs and treat using antibiotics. This may lead to under-reporting.

Data on the prevalence of *A. salmonicida* in wild Pacific salmon in the Pacific northwest of North America have been gained from a number of sources. An earlier MAF risk analysis (MacDiarmid, 1994) presented data from Pacific salmon submissions to the Fish Pathology Laboratory, Nanaimo, for the years 1972 to 1993 showing that of 21,493 sexually mature wild Pacific salmon cultured for *A. salmonicida*, 1,298 (6.0%) were found to be positive. Data from the same source for the period 1985-1994 show that 235 of 2,331 cases (10%) were positive for *A. salmonicida* (pers. comm. Dr Trevor Evelyn, October 1996). Data from Pacific salmon assayed for *A. salmonicida* by the Alaska Department of Fish and Game CFHD Division Fish Pathology Laboratories during the period 1980 to 1994 show that 87 of 3,615 cases (2.4%) were positive for *A. salmonicida* (pers. comm. Linda Chaves, Deputy Director, National Marine Fisheries Service, USDC, August 1995). As noted in the earlier MAF risk analysis (MacDiarmid, 1994), these figures reflect sampling biased towards sexually mature fish exhibiting clinical signs of disease at the time of egg collection, following stress associated with adaptation to fresh water, sexual maturation, and overcrowding in infected waterways.

A truer indication of the expected prevalence in wild Pacific salmon in the Pacific north west during the marine phase of their life cycle can be gained from specific surveys as fish enter fresh water at an early stage of sexual maturation. Two surveys of sockeye spawners performed in 1993 and 1995 in the Fraser River, B.C., detected 11 of 502 (2.2%) and 1 of 345 (0.3%) positive fish, respectively (pers. comm. Dr Trevor Evelyn, October 1996). An earlier MAF risk analysis (MacDiarmid, 1994) reported surveys in BC of wild sockeye and chum salmon prior to entry into fresh water which failed to detect any positive samples out of 600 fish tested (0%). The earlier MAF risk analysis concluded that infection with *A. salmonicida* in wild Pacific salmon prior to their entry into fresh water during the spawning migration was uncommon.

Numerous studies in Japan support the conclusion that prevalence of *A. salmonicida* in wild Pacific salmon harvested prior to entry into fresh water or sexual maturation is likely to be low (Nomura et al 1991, 1991a, 1992, 1992a, 1993). A total of 17,909 mature Pacific salmon of various species were sampled from 37 rivers in northern Japan from 1979-1991 (Nomura et al, 1993). No mature fish showed clinical signs of furunculosis. *A. salmonicida* could be cultured from the kidneys of 1,623 of 17,909 fish tested (9%), following holding without feeding for 1 month until sexual maturity. Individual populations in these studies had *A. salmonicida* infection rates of up to 31.1%. *A. salmonicida* could not be cultured from the kidneys of any of 480 (0%) immature harvest weight fish collected in set nets off Hokkaido. Neither could *A. salmonicida* be cultured from any of 2,010 juvenile masou and chum salmon fry collected from 11 hatcheries in Hokkaido. The conclusion of the authors of these studies was that *A. salmonicida* is widely distributed in the river systems studied, there is a seasonal pattern to annual prevalence with infections peaking in October, and fish appear to become infected upon return from sea water to fresh water during the spawning migration.

Furunculosis is recognized as being endemic in the main fish farming areas of Norway. Sanitary control measures, the increasing use of more effective vaccines, and low water

temperatures have significantly reduced the mortality of fish and the reliance on drugs such that furunculosis was not recognized to be a problem by the state veterinary services in Norway in 1995 (Næss and Håstein, 1995). In a study of 124 randomly selected Norwegian sea water farm sites housing post-smolts of Atlantic salmon the cumulative farm level incidence was 54.0% for furunculosis (Jarp et al, 1994). Furunculosis infection was significantly related to the location of the sea-site, presumably reflecting a reservoir of infection and the propensity for horizontal spread. Mortality associated with the furunculosis outbreaks was significant; 18.8% adjusted geometric mean mortality (95% confidence level) at sites experiencing furunculosis outbreaks compared with 10.8% mortality in non-furunculosis infected farms (Jarp et al, 1994). At the time of that survey no oil adjuvant based vaccination against furunculosis was used in Norway. Since the use of oil adjuvant based vaccines has become widespread, the farm level incidence of furunculosis has reduced every year since 1991, such that in 1996 there were 29 farms in all of Norway under restrictions for furunculosis according to Norwegian Fish Disease Legislation, and of these a diagnosis of furunculosis was made on only two during that year (pers. comm. Tore Håstein, Central Veterinary Laboratory, Oslo, Norway, March 1997).

The most common aquaculture species in Denmark is rainbow trout, and furunculosis causes few problems because of the resistance of this species (pers. comm. Niels Jørgen Olesen, Danish Veterinary Laboratory, 21 October, 1996). In 1995 of 54 pools of fish samples submitted for bacteriological testing, 15 were positive for *A. salmonicida* (giving a prevalence of 27.8%, in the unlikely event of all fish in every pooled sample being positive).

Pathology, epidemiology and control

The clinical signs, pathology and epizootiology of *A. salmonicida* infections have been reviewed by McCarthy and Roberts (1980), Austin and Austin (1993) and Munro and Hastings (1993).

Infection with *A. salmonicida* is by horizontal transmission. *A. salmonicida* maintains an epidemic cycle within host populations. Epidemics occur in juvenile or adult fish exposed to high levels of environmental contamination. Epidemics in hatchery and other fish farming conditions, which bring large populations of fish together under stressful conditions, have been an important feature of *A. salmonicida* infections during early salmonid aquaculture and enhancement programmes. There is some evidence to suggest that in the natural environment horizontal transmission does not occur as readily (McDermott and Berst, 1968). Carrier fish ensure that infection is transmitted between generations. Vertical transmission has not been demonstrated.

The possible routes of infection may be via gills, intestine, or skin damage. The production of a reliable model to infect salmonids with *A. salmonicida* has been the subject of research during development of vaccines, and has produced valuable data relating to the minimum infectious dose by various routes. The minimum infectious dose for Atlantic salmon in sea water by short duration (1-3 days) bath exposure has been measured at 10^4 cfu/ml, and for long duration immersion (3 weeks) at 10^2 cfu/ml (Rose et al, 1989). Immersion in concentrations of 10^2 cfu/ml for periods up to 1 week failed to cause infection. Intragastric intubation required doses of $>10^5$ cfu/fish to establish infection in Atlantic salmon.

An earlier MAF risk analysis (MacDiarmid, 1994) reported studies conducted at the Pacific Biological Centre, Nanaimo, in which immersion of stressed Pacific salmon in 10^3 cells/ml for 2-3 days failed to establish infection. A dose of 10^2 cells/kg administered by stomach tube was able to infect Pacific salmon, but not readily.

The susceptibility of 25g rainbow trout and 30g turbot to infection with *A. salmonicida* has been assessed (Pérez et al, 1996). Immersion in concentrations of 10^8 cfu/ml for periods of at least 12 hours was the minimum lethal dose required in rainbow trout, and was also the dose required to induce a carrier status in rainbow trout (i.e. a positive recovery of *A. salmonicida* on culture of kidney samples following stressing). Immersion in concentrations of 10^5 cfu/ml for periods of at least 12 hours was required to infect turbot, and a carrier status could not be demonstrated for turbot, with the exception of a single fish for which extenuating circumstances applied. Both species were refractory to challenge by intragastric intubation.

An experimental model for *A. salmonicida* infection of 25-50 g rainbow trout found LD_{50} to be between 1-5 x 10^3 cells by intramuscular injection and by 1 hour bath exposure at concentrations of 1 x 10^7 cells/ml (pers. comm. Christian Michel, August 1997).

A. salmonicida produces very powerful extracellular virulence factors which overcome the leucocytic defences of the host. *A. salmonicida* is then transported through the vasculature to localise in any of the organs.

Peracute, acute and subacute (chronic) forms of furunculosis are recognised in salmonids. As populations become older the disease often takes a more chronic course. A continuum of pathologies associated with peracute to subacute infection may be present in any infected population. Peracute furunculosis is confined to fingerling fish (McCarthy and Roberts, 1980). Infected fish die quickly with only slight external signs, possibly darkening, exophthalmia and haemorrhages at the base of the pectoral fins.

Acute infections are the most common form of the disease, and occur in young growing fish and adults. Acute disease is of sudden onset and can cause high mortality, with often few external signs. The septicaemia may be associated with darkening, lethargy, inappetance, pale gills and external haemorrhages. In sea-reared fish there may be extensive haemorrhage from the gills. Fish usually die within 2-3 days of infection. Bacteria may be cultured from most internal tissues. Detection of *A. salmonicida* in mucus has also been shown to be a non-lethal means by which infected fish can be detected (Cipriana et al, 1992). Internally there are petechiae on most serosal surfaces. Kidney, spleen, liver and swimbladder may be swollen. Whereas the liver and gills typically appear pale, as a result of low haematocrit, the spleen and kidneys are red and inflamed. The blood vessels of the intestine and pyloric caeca are inflamed, and the intestine is devoid of food and may contain an exudate of blood, mucus and cellular debris. There may be a bloody exudate in the peritoneal and pericardial cavities. Histologically, bacterial colonies in most organs and tissues are surrounded by necrotic debris, with very little visible host cellular response. This is a result of the bacterial production of proteolytic enzymes, which destroy host macrophages, monocytes, lymphocytes, and connective tissues.

In the subacute form of the disease there is a slower progression of infection, resulting in greater localisation in visceral organs, commonly kidney, spleen, blood vessel walls and

intestine, but also liver and gills. The lesion in the muscle tissues of the chronic form is that which has become known as a 'furuncle', although there is generally no pus accumulation. The muscle lesion is a swelling of necrotic tissue which is progressively walled off by scar tissue, and may or may not be ulcerated. The scarring associated with the muscle lesions renders the fish unmarketable.

Carrier or latent infections are important in the epidemiology of the disease. Survivors of the infection will harbour the organism in visceral tissues. It may be possible to isolate the organism from the kidney, gut, gill or skin. These covertly infected fish act as reservoirs of infection. Stress, such as handling stress in culture situations or stress associated with physiological re-adaptation to fresh water, cause recrudescence of the infection, which then initiates a renewed epidemic. Stress and trauma are important aspects of the epidemiology of *A. salmonicida* infections. Any trauma to the integument may act as a portal of infection. Lice damage skin, and may also transmit disease.

Data provided at the Canada-Australia Salmon Technical Meeting in Nanaimo BC, Canada (July, 1994) showed that in five chinook salmon dead from experimentally induced furunculosis viable counts of *A. salmonicida* ranged from $10^{7.2}$ to $10^{8.8}$ cells per gram of kidney tissue and from $10^{2.8}$ to $10^{5.3}$ cells per gram of flesh (average $10^{8.3}$ and $10^{4.7}$, respectively). Carrier chum and pink salmon have been determined to be loaded with $10^{3.7}$ viable *A. salmonicida* cells per gram of kidney tissue and 10^6 cfu/ml of coelomic fluid (Nomura et al, 1991; Nomura et al, 1992). These data suggest tissue titres several orders of magnitude (possibly 10^4) less in flesh than in kidney tissue, and several orders of magnitude less (possibly 10^5) in carrier fish than clinically infected fish.

From these data, MacDiarmid (1994) reasoned that tissue titres of *A. salmonicida* in the flesh of carrier salmon may be in the order of fewer than 10 viable cells per g of flesh. The conclusion was supported by a communication from Northern Ireland fish health experts which noted that *A. salmonicida* was unrecoverable from the flesh of healthy fish. MacDiarmid concluded that this level of *A. salmonicida* cells is not likely to provide an infectious oral dose or lead to concentrations capable of causing infection by immersion when the findings of Rose et al (1989) and the various other reported studies on infectious doses are considered.

In farming situations, management has been effective in reducing the impact of *A. salmonicida* infections. Siting farms away from sources of contamination, single-stock-class sites, reducing stress, screening of new fish introductions, and vaccination are all recommended to minimise the impact of furunculosis in endemic areas. Advances have been made in chemotherapy against furunculosis, and this is an area of continuing research. Some work is focussing on breeding disease-resistant fish strains.

Survival and inactivation

The survival and inactivation parameters of *A. salmonicida* have been extensively studied. The differing potential for *A. salmonicida* to survive in sterile versus non-sterile aquatic conditions was noted in the earlier MAF risk analysis, as was the fact that survival in non-sterile conditions mirroring those found in nature is of more direct relevance to a study of this type

(MacDiarmid, 1994). Effendi and Austin (1991) suggested that the survival of *A. salmonicida* in natural (non-sterile) conditions may be adversely impacted by the presence of potentially inhibitory organisms and their metabolites common in natural aquatic systems. Austin and Austin (1993) reviewed the experimental data concerning the survival of *A. salmonicida* in water, and also note that information gleaned from studies employing pre-sterilized water were not a good indication of the behaviour of the pathogen in the natural environment. Their findings of relevance (those concerned with survival in unsterilized conditions, as found in nature) are detailed below (table 8).

Table 8. Experimental data concerning the survival of *A. salmonicida* in unsterilized water (adapted from Austin and Austin, 1993).

Type of experimental system	Temp. (°C)	Survival time	Reference
Brackish water	-	16-25 days	Smith (1962)
	11-13	24 days	McCarthy (1980)
Distilled water	-	14 days	Horne (1928)
	-	4 days	Williamson (1929)
	-	7 days	Duncan (1932)
	20	9 days	Lund (1967)
Fresh water	-	24-30 hours	Duncan (1932)
	11-13	17 days	McCarthy (1980)
River water	-	2 days	Williamson (1929)
	-	2-17 days	Smith (1962)
	20	2 days	Lund (1967)
10^1-10^2 *A. salmonicida* cells/ml	15	*ca.* 3 days	Allen (1982)
10^5-10^6 *A. salmonicida* cells/ml	15	> 3 days	Allen (1982)
Sea water	-	2 days	Williamson (1929)
	-	24-30 hours	Duncan (1932)
	20	5-6 days	Lund (1967)
	11-13	8 days	McCarthy (1980)
	-	< 10 days	Rose et al. (1990b)
	20	6 days	Effendi and Austin (1991)
Tap water	-	3-4 days	Horne (1928)
	-	3 days	Williamson (1929)
	-	4 days	Duncan (1932)
	20	3 days	Lund (1967)

The data indicate the variability of experimental findings, but confirm that *A. salmonicida* may, under appropriate conditions, survive prolonged periods in fresh, brackish and sea water. Brackish water appears to be the medium resulting in the longest survival. In the majority of the above reports, an initial inoculum of 10^6-10^7 cells per ml was used. This is much larger than expected to be found naturally, except possibly during epidemics where moribund and dead fish have not been removed from the water source. The only experiments using lower initial inoculums reported a rapid reduction in numbers until 72 hours, at which time the organism became unrecoverable (Allen, 1982, cited by Austen and Austen, 1993).

The ability of mud to act as a reservoir for infection has also been examined (McCarthy, 1980). Once again, high initial inocula such as would only occur during epidemics were used, and it was demonstrated that under these conditions up to 10^5 viable cells per ml survived after 29 days. Other studies reported in Austen and Austen (1993) support the concept that following epidemics *A. salmonicida* is able to survive and even multiply in natural sediments, thereby providing a future source of infection. The possibility of fish farm equipment such as nets providing a reservoir of *A. salmonicida* has also been discussed (McCarthy, 1980), as has the ability of non-salmonid fish and other vertebrate and invertebrate animals to become infected and act as reservoirs of infection. Austen and Austen (1993) review the studies on other possible reservoirs, and conclude that *A. salmonicida* may be found in association with a variety of other aquatic animals, but that direct contact between infected salmonids is the primary means of disease transmission.

A. salmonicida is a facultative anaerobe, and therefore likely to survive on salmonid flesh or scraps. *A. salmonicida* remains viable in fish muscle tissue for 32 days (McCarthy, 1980). Other studies reviewed by Austin and Austin (1993) indicate survival in infected trout tissues for 28 days at 4°C, and 49 days at -10°C.

The ability of *A. salmonicida* to change its morphology and enter a dormant state during prolonged periods of survival outside of the host remains an area of active research. *A. salmonicida* cells have been detected in experimental micro-systems from which no colonies could be grown on routine bacteriological media. These cells have been labelled viable but non-culturable (VBNC). VBNC cells respond like culturable cells to indirect fluorescent antibody testing (iFAT), but can be distinguished morphologically (Effendi and Austin, 1995). The growth of *A. salmonicida* colonies on routine bacteriological media from aquatic micro-systems in which only VBNC cells could be detected by iFAT has been reported, however this apparent resuscitation probably resulted from the undetected presence of very low levels of truly viable bacteria within the system (Austin and Austin, 1993; Effendi and Austin, 1995). VBNC cells have been shown to be intact and contain DNA (Morgan et al, 1993). VBNC have been shown to demonstrate respiratory activity (Effendi and Austin, 1994, 1995). In concurrence with the earlier MAF risk analysis (MacDiarmid, 1994), Effendi and Austin (1995) conclude that *A. salmonicida* survives in the marine environment after plate counts have declined to zero, but that the surviving cells undergo major morphological changes, retaining some viability but losing pathogenicity.

Conclusions

1   *A. salmonicida* is an economically significant pathogen of salmonids and other fishes, including marine fish species, with a wide geographical distribution.

2   Live fish and untreated offal or fish farm wastes are the highest risk commodities for spread of *A. salmonicida*. There are no reports of dead eviscerated fish for human consumption being the agent for transmission of *A. salmonicida*, although the means of *A. salmonicida* introduction was not determined in many cases.

3   The likelihood of a fish clinically infected with *A. salmonicida* being harvested and/or being processed for human consumption is low.

4   The likelihood of a fish of carrier status for *A. salmonicida* being harvested and/or being processed for human consumption is high.

5   Heading, gilling and gutting of *A. salmonicida* carrier salmonids during processing is likely to significantly reduce the amount of infectivity present.

6   Any *A. salmonicida* present following processing may survive for sufficient time to be present in the *commodity* at the time of importation.

7   If imported in fish tissues and placed into the aquatic environment in New Zealand, *A. salmonicida* could retain viability for long periods but would probably retain pathogenicity for much shorter periods.

8   The likelihood of an infective dose of *A. salmonicida* for salmonids building up in the environment as a result of importations of the *commodity* is low.

9   The risk of *A. salmonicida* introduction into New Zealand through the importation of *the commodity* is low.

10   Of the pathogens qualitatively assessed as being of low risk, the risk of *A. salmonicida* introduction through importations of the *commodity* is potentially highest as a result of the possible occurrence of concentrations of pathogen in flesh. Quantitative risk analysis will provide an estimation of the level of risk posed by *A. salmonicida.*

4.2.2   Enterobacteriaceae

The enterobacteriaceae comprise some 100 species of facultatively anaerobic, non-spore-forming, Gram-negative rods. They are widely disseminated in soil, water, plants and animals. The genera *Edwardsiella* and *Yersinia* contain important pathogens of salmonids.

Three further enterobacteriaceae species have been recorded as pathogens of salmonids. *Citrobacter freundii* is common in eutrophic fresh water, and has been implicated in septicaemic disease in Atlantic salmon and rainbow trout in Spain, the USA and Scotland (Austin and Austin, 1993). Pathogenicity in salmonids has been experimentally confirmed by intra-peritoneal injection of rainbow trout inducing mortality. *Serratia liquefaciens* has been isolated from Atlantic salmon in marine cages in Scotland and has been implicated in disease conditions causing high mortality (Austin and Austin, 1993). Pathogenicity in Atlantic salmon has been experimentally confirmed by intramuscular injection which caused myoliquefaction and death. *Serratia plymuthica* has been associated with disease in rainbow trout fingerlings in Spain and Scotland (Austin and Austin, 1993). Investigations in each of the above cases noted probable contact with polluted water sources. Considering the known association of enterobacteriaceae with aquatic systems polluted with organic material, it is likely that the above disease instances represent opportunistic infections rather than serious emerging primary pathogens of salmonids. The infections were manifested as generalised septicaemia in juvenile classes of stock, so it is unlikely that infected fish would be harvested and processed for human consumption. Other enterobacteriaceae are recorded as pathogens of fishes other than salmonids. Pollution has been commonly implicated as an extenuating circumstance.

Edwardsiella spp.

Introduction

Two species of *Edwardsiella* are important pathogens of aquatic animals; *E. ictaluri* and *E. tarda*. The two organisms cause distinct disease syndromes of importance during fresh water fish culture. These syndromes differ greatly in their occurrence, clinical signs, pathogenesis and severity.

E. ictaluri causes enteric septicaemia in catfish. This is a highly contagious disease which seriously affects catfish culture in the southern USA. Mortality during epidemics can be as high as 50% (Plumb, 1993). Although *E. ictaluri* is reportedly specific for ictalurids (catfish and cyprinids), isolations from a wide range of other fish species are increasingly being reported, including experimental transmission to chinook salmon and rainbow trout (Baxa et al, 1990). This finding has implications for aquaculture and wild stocking of salmonids and ictalurids, but little direct relevance to this risk analysis. *E. ictaluri* is listed by the OIE as a significant disease of aquatic animals (OIE, 1995a).

E. tarda is the cause of septicaemia in warm water fish in the USA and Asia. *E. tarda* occurs widely in aquatic animals, ponds and mud, and is typically an opportunistic pathogen. *E. tarda* can cause gastroenteritis in man.

Susceptibility and distribution

Plumb (1993) records the following species to be naturally susceptible to *E. tarda;* channel catfish, chinook salmon, carp, crimson seabream, Japanese flounder, Japanese eel, largemouth bass, mullet, red seabream, striped bass, tilapia, and yellowtail. Rainbow trout must be added to this list, following isolation in this species in Australia (Reddacliff et al, 1996). *E. tarda* is known to be widely distributed through the USA, Asia and Africa, as well as in Australia. In Australia *E. tarda* has been isolated in ornamental fish, captive wild eels, saratoga and knifefish at Sydney's Taronga Zoo, and captive little penguins (Reddacliff et al, 1996). *E. tarda* infection of humans has also been reported.

Although *E. tarda* is considered to be ubiquitous in aquatic environments, fish pathogenic strains may have specific geographical distribution in Japan, Germany, Taiwan, Singapore and North America (Humphrey, 1995). Four serotypes of *E. tarda* are recognised (A, B, C and D), with serotype A the most virulent in aquatic animals (Austin and Austin, 1993).

E. tarda has twice been isolated in New Zealand, from a cetacean and a seal at Massey University (Stan Fenwick, unpublished data, pers. comm. of September 1997).

Pathology, epidemiology and control

Environmental parameters such as water temperature and organic load influence occurrence and severity of disease outbreaks. Most outbreaks occur at water temperatures above 30°C when high levels of organic material are in the water (Austin and Austin, 1993). Various aquatic invertebrate as well as vertebrate species (snakes, frogs, turtles, gulls and humans) may act as reservoirs of infection. *E. tarda* may also form a normal part of fish microflora, and for this reason it remains unclear whether *E. tarda* should be viewed as a primary or opportunistic pathogen in fish. The LD_{50} by intra-peritoneal injection has been determined for chinook salmon and steelhead trout as 4.1×10^6 and 5.6×10^6, respectively (Amandi et al, 1982). The LD_{50} by intra-peritoneal injection increased at lower temperatures. In the same report water-borne challenge of steelhead trout at concentrations up to 2.0×10^8 cells/ml inconsistently caused mortalities ranging from 0-15% in chinook salmon and steelhead trout. In another study the lowest LD_{50} by water-borne challenge was determined to be 3.1×10^7 cells/ml (Song et al, 1982). Amandi et al (1982) detected *E. tarda* in kidney and intestine of pre-spawning and post-spawning chinook salmon carcasses, but not freshly killed and spawned chinook salmon. They concluded that *E. tarda* is an opportunistic infection in chinook salmon. From their review of pathogenicity studies, Austin and Austin (1993) conclude that *E. tarda* is not particularly virulent.

Clinical signs of *E. tarda* infection in catfish are initially small cutaneous lesions, which progress to abscessation of muscles of the body and tail and eventually large gas-filled putrefactive lesions (Meyer and Bullock, 1973). Clinical signs in yearling and broodstock rainbow trout from an outbreak in Australia included mucopurulent cloacal discharges, bloated abdomens, and fish swimming upside down (Reddacliff et al, 1996). Gross pathology suggestive of septicaemia was observed in all fish in the Australian outbreak, including petechial haemorrhages on serosal surfaces of viscera, pericardium and gills, grossly abnormal spleens and livers and fibrinous exudates in peritoneum and pericardium.

Control may be achieved by chemotherapeutic means, with *E. tarda* reported to be susceptible

to a number of antibiotics commonly used in aquaculture, including oxytetracycline. Removal of predisposing factors is important in long term control, particularly the improvement of water quality. Research into control via vaccination and dietary supplementation is continuing and shows some promise.

Survival and inactivation

No relevant data on the survival and inactivation parameters of *E. tarda* have been found. Humphrey (1995) cites a report of *E. ictaluri* surviving in mud at 25°C for 95 days.

Conclusions

1   *Edwardsiella* spp. are important and economically significant pathogens of aquatic animals, with wide geographic distribution. Among salmonids, only chinook salmon and steelhead/rainbow trout have been shown to be naturally susceptible to *Edwardsiella* spp. In salmonids *Edwardsiella* spp. appear to be opportunistic pathogens, with outbreaks occurring in association with poor water quality.

2   Because of the clinical signs associated with septicaemia, salmonids clinically infected with *Edwardsiella* spp. are unlikely to be harvested and processed for human consumption.

3   The infective dose of *E. tarda* for salmonids is relatively high. The likelihood of an infective dose of *E. tarda* for salmonids building up in the environment as a result of importations of the *commodity* is low.

4   The isolation of *E. tarda* in a cetacean and a seal in New Zealand is an indication that natural pathways for the introduction of *E. tarda* probably exist.

5   The risk of *Edwardsiella* spp. introduction through the importation of the *commodity* is negligible.

Yersinia ruckeri

Introduction

Yersinia ruckeri causes enteric redmouth (ERM) in salmonids, particularly rainbow trout. *Y. ruckeri* comprises a group of Gram-negative rods, motile by means of seven or eight flagella. Five or six serotypes exist worldwide, recognised on the basis of whole-cell serology (Austin and Austin, 1993; Stevenson et al, 1993). *Y. ruckeri* occurs in New Zealand. Although the New Zealand isolate has not been definitively typed, isolates sent to the MAFF UK Fish Diseases Laboratory, Weymouth, reacted strongly to type I and weakly to type II antisera. Biochemical similarities to the Australian strain have been noted (pers. comm. Colin Anderson, MQM Animal Health Laboratory, 23 January 1997). Type I (Hagermann) was first isolated in rainbow trout culture in Idaho in the USA. The existence of types II (Idaho), III (Australian), V (Colorado) and VI (Ontario) has since been confirmed, but the organism

designated as type IV has been eliminated as a result of DNA hybridization (Stevenson et al, 1993). Stevenson et al (1993) discuss the findings of a survey which examined 163 isolates of *Y. ruckeri,* and determined serogroups on the basis of the lipopolysaccharide surface antigen (0-antigen). Strains belonging to serogroup 0:1 (which includes type I, the strain most often associated with disease) accounted for 110 (67%) of isolates examined. Strains belonging to serotype 0:2 (which includes some of serovar II, also associated with disease) accounted for a further 25 (15%) isolates.

Susceptibility and distribution

Stevenson et al (1993) reviewed the species known to be susceptible to *Y. ruckeri. Y. ruckeri* was first isolated in rainbow trout. However, its host range probably includes all salmonids. It has been isolated in rainbow, steelhead, brook and brown trout, and coho, chinook, sockeye and Atlantic salmon. *Y. ruckeri* has also been isolated from diseased emerald shiners, minnows, cisco, whitefish, sturgeon and turbot, as well as from apparently healthy goldfish, carp, eels, burbot, coalfish and Arctic char. *Y. ruckeri* has been experimentally transmitted to channel catfish, sole and gilthead. *Y. ruckeri* has also been isolated from muskrat, kestrels, turkey vultures, seagulls, crayfish, humans, sewage and river water. *Y. ruckeri* is known to cause disease in salmonids in North and South America, Great Britain, Denmark, Norway, Finland, Germany, France, Ireland, Australia and New Zealand.

Pathology, epidemiology and control

As noted above, ERM occurs most commonly in rainbow trout under intensive culture, usually in fingerling fish approximately 7.5 cm in length. Water temperature and stress are important exacerbating factors in disease occurrence and severity. Disease is most severe at water temperatures of 15-18°C, and least severe at 10°C or below. Stressing fish by placing them in water temperatures of 25°C or over will cause carrier fish to shed the organism. Handling fish and overcrowding (causing excess ammonia and metabolic waste and decreasing oxygen levels) are stressors which may precipitate outbreaks. The importance of *Y. ruckeri* infected asymptomatic carrier fish has long been recognised. Mortalities typically occur 5-20 days subsequent to stressing of a population in which there may be carrier fish.

The ability of birds to spread infection is suspected on the basis of recovery of the organism from the faeces of seagulls.

Numerous studies have been conducted to determine the LD_{50} of *Y. ruckeri* strains by injection and immersion, with variable results. Austin and Austin (1993) concluded that large numbers of organisms are required to initiate outbreaks, and that type I strains are most pathogenic to rainbow trout (LD_{50} of 3.0 x 10^5 cells per ml), then type II (LD_{50} of 1.0 x 10^7 cells per ml) followed by type III (LD_{50} as yet undetermined). Stevenson et al (1993) presented a summary of transmission studies, and also noted that LD_{50}, although a convenient means of quantifying virulence, does not take into account the ability of *Y. ruckeri* to cause low level mortalities and decreased growth rates in chronically infected fish over a considerable length of time. In this respect, the infectious dose of *Y. ruckeri* remains unknown and the data from studies of LD_{50} should be interpreted with a degree of caution, especially considering the wide host range.

Clinical signs associated with an outbreak are reddening of the mouth, darkening of the skin pigmentation and cutaneous petechiae. Disease may be manifested in peracute, acute, subacute and chronic forms. A generalised septicaemia occurs. In dead fish haemorrhages are visible in the intestine and muscle, and the spleen and kidneys may be swollen. The organism is recoverable from most internal organs, although anterior kidney is the diagnostic sample of choice. In asymptomatic carriers, *Y. ruckeri* is initially recoverable from the kidney, lower intestine, spleen and liver. Eventually the pathogen localizes in the lower intestine, where it is periodically shed in the faeces.

Oral, injectable and immersion vaccines for *Y. ruckeri* are available. Chemotherapeutics and management to minimise stress are also important aspects of control.

Survival and inactivation

The ability of *Y. ruckeri* to survive for long periods in the environment has been known for some time. There is, however, some conjecture over whether this ability is important in the epidemiology of disease in salmonids. *Y. ruckeri* can survive for more than 3 months in sterile river, lake and estuary sediments and water (Romalde et al, 1994). These authors also report states of non-culturability (dormancy) during which virulence is maintained, and an ability to resuscitate dormant states. The applicability of these findings in a natural (non-sterile) environment is uncertain.

Conclusions

1   *Y. ruckeri* is an important and economically significant pathogen of salmonids, particularly rainbow trout, with a wide geographic distribution including New Zealand. Strains varying in pathogenicity exist. Exactly where the New Zealand strain fits into the taxonomy of *Y. ruckeri* is presently uncertain.

2   Movement of live fish constitutes the most likely means of transmission of *Y. ruckeri*. Unrestricted movement of eviscerated fish is not suspected to have led to transmission of the strains of *Y. ruckeri* present in New Zealand, or been suspected to have led to transmission overseas.

3   Because of the age/size of fish likely to be clinically infected and the clinical signs associated with septicaemia, the likelihood of salmonids clinically infected with *Y. ruckeri* being harvested and processed for human consumption is low.

4   Asymptomatic carrier salmonids infected with *Y. ruckeri* may be harvested and processed for human consumption.

5   Heading, gilling and gutting is likely to significantly lower the number of infective particles remaining in a *Y. ruckeri* carrier fish.

6   *Y. ruckeri* would be likely to survive on the *commodity* and may survive for long periods if placed into aquatic environments in New Zealand.

7   The infective dose of *Y. ruckeri* for salmonids and other susceptible species is unknown. The LD$_{50}$ in rainbow trout appears to be relatively high. The likelihood of sufficient environmental contamination of *Y. ruckeri* to provide the LD$_{50}$ in salmonids occurring as a result of the importation of the *commodity* is low, although whether this can be interpreted as meaning the likelihood of sufficient environmental contamination to provide an infectious dose for fish occurring is similarly low cannot be said with any certainty at present.

8   The risk of exotic strains of *Y. ruckeri* introduction through importations of the *commodity* is negligible.

4.2.3 Cytophagaceae

The Cytophagaceae are a group of gram-negative rods or filamentous bacteria, which typically exhibit gliding motility and which may show varying morphology on differing growth media. Because of this varying morphology, members of the genera are difficult to identify and taxonomy is confused. The group includes the genera *Cytophaga, Flavobacterium, Flexibacter* and *Sporocytophaga.* Four disease conditions caused by members of the Cytophagaceae are of significance in salmonids; bacterial cold water disease (BCWD), columnaris disease, bacterial gill disease (BGD) and fin rot. The following is largely derived from reviews by Austin and Austin (1993), Holt et al (1993), Wakabayashi (1993) and Turnbull (1993a).

BCWD is caused by infection with *Flavibacterium psychrophilus* (syn *Cytophaga psychrophilus, Flexibacter psychrophilus*). BCWD is a serious septicaemic condition of hatchery-reared salmonids, in particular coho salmon in the Pacific Northwest. All salmonid species are probably susceptible, but epidemics in coho salmon in hatcheries of the north-western USA during early spring is the typical manifestation. The bacterium is also recognised in Europe and has been associated with disease in rainbow trout in France. Belgium and Ireland have also recently reported increasing problems in rainbow trout fry associated with *F. psychrophilus* infection, as has Chile (OIE, 1996). The disease occurs in the United Kingdom and Denmark.

Alevins or fry may succumb to BCWD, with high mortalities (up to 30-50%) being more common in alevins. Mortalities are highest at temperatures below 15°C. The classic lesion in fry is an ulceration of the skin and muscle of the peduncle. Lesions in other parts of the body such as the vent, anterior of the dorsal fin and lower jaw may also occur. Amongst survivors of an epidemic spinal deformities and/or a whirling disorder may occur several months later, and *F. psycrophilus* can be isolated from the brains of these fish. Yearling coho salmon may also develop BCWD, exhibiting typical ulcerated lesions or no external signs. There may also be anaemia and gill haemorrhage.

Adult carrier fish are important in the epidemiology of the disease and the organism can be isolated from spleen, kidney, ovarian fluid and milt. Vertical transmission occurs, probably via egg-associated transmission resulting from large numbers of the bacteria in ovarian fluids and milt. Horizontal transmission has only been demonstrated when the mucus and dermis of challenged fish have been experimentally broken.

F. psychrophilus has also been recovered from muscle lesions and kidneys of 100-500g (i.e. up to harvest-weight) rainbow trout in Ontario, Canada, with severe necrotising myositis (Lumsden et al, 1996). The report concludes that necrotising myositis is an unusual presentation of the common disease BCWD.

Columnaris disease is caused by *Flavibacterium columnaris* (syn *Flexibacter columnaris*), and is reported to occur in North America, Asia and Europe. All fresh water fish are probably susceptible in conditions favourable to the bacterium and stressful to the fish. Rainbow, steelhead and brook trout and coho and chinook salmon are susceptible. Outbreaks occur at water temperatures of 15°C or above. Early lesions appear as a white spot on the head, gills,

fins or body, surrounded by a zone of reddish tinge, or as pale, saddle-shaped lesions ventral to the dorsal fin and occasionally elsewhere. Under-running of tissues around lesions leads to extensive gill and skin ulcerations. As the organism is not found in the blood until extensive ulceration has occurred, it is thought that it gains entry to the blood via the lesions. Long term survival of the organism in water provides a reservoir of infection, as may reservoir hosts of species other than salmonids. Overcrowding and other factors which may lead to stress and/or integumentary damage are also important.

Bacterial gill disease and fin rot are infections of intensively reared salmonids and other fresh water fish caused by bacteria of the genera *Cytophaga, Flavobacterium,* and *Flexibacter.* These diseases have a worldwide distribution, and occur in New Zealand. The bacterial species involved in infections in New Zealand have not been typed. *Flexibacter psychrophilus* has been isolated in Tasmania from farmed Atlantic salmon experiencing moderate to severe erosions of the fins, with morbidity in excess of 80%. The phenotype of the Australian isolate was found to be similar to northern hemisphere isolates (Schmidtke and Carson, 1995). On the east coast of Canada, and possibly in other parts of the world, *Flavibacterium branchiophila* (syn *Flexibacter branchiophila*) is a significant cause of BGD in farmed salmonids. There is growing evidence that this species may act alone to cause disease (Ferguson et al, 1991; Speare et al, 1995). In BGD, bacterial colonisation of the lamellae of the gills results in inflammation, epithelial hyperplasia, lamellar fusion and eventual necrosis. This process results in clinical signs associated with respiratory distress, including gasping, swimming near water inlets or the surface, and mucous strands trailing from the gills. BGD has no specific temperature dependancies. Horizontal transmission occurs. The bacteria involved are possibly a normal part of the surface microflora of fish, and infection may relate to environmental conditions causing stress. The bacteria involved in BGD may also be involved in fin rot conditions of salmonids, which result from secondary opportunist invasion of wounds caused by biting, handling etc.

Conclusions

1   The Cytophagaceae are important and economically significant pathogens of salmonids with a probable near worldwide distribution. Cytophagaceae may be a normal component of the salmonid microflora, and clinical disease may reflect the stressful environmental conditions which result from intensive culture.

2   Diseases caused by Cytophagaceae occur in New Zealand. The similarity of New Zealand isolates to isolates from other parts of the world has not been adequately examined.

3   Because of the age class of fish likely to be clinically infected and the clinical symptoms of infection, salmonids clinically affected by Cytophagaceae are unlikely to be harvested and processed for human consumption.

4   Salmonid fish asymptomatically carrying Cytophagaceae are likely to be harvested and processed for human consumption.

5   Heading, gilling, evisceration and washing to remove mucus is likely to reduce the

number of infective particles of Cytophagaceae remaining on a salmonid processed for human consumption.

6   The risk Cytophagaceae introduction through importations of the *commodity* is negligible.

4.2.4 Pseudomonadaceae

Pseudomonadaceae are strictly aerobic, Gram-negative, straight or curved rods, motile by polar flagella. They are widely distributed in the aquatic environment. *Pseudomonas* species often form part of the normal surface or enteric microflora of many species of fish. They are common opportunistic or secondary invaders. In a few instances they have been recorded as primary pathogens of salmonids (Austin and Austin, 1993; Inglis and Hendrie, 1993).

Pseudomonas fluorescens is ubiquitous in fresh water ecosystems. In addition to being an opportunistic pathogen associated with fin and tail rot conditions, *P. fluorescens* causes pseudomonad septicaemia, a haemorrhagic condition of probably all species of cultured fresh water fish. Pseudomonad septicaemia is associated with stress or improper management, such as poor water quality and/or high stocking density. Rainbow trout are susceptible. Superficial ulcers caused by *P. fluorescens* have been recorded on IPNV chronically infected rainbow trout (Inglis and Hendrie, 1993). Ulcers and haemorrhages on the gills and fins have also been reported in trout (Austin and Austin, 1993). Control is readily achieved by correcting the underlying cause, in conjunction with antibiotic therapy.

Pseudomonas pseudoalcaligenes has been isolated in mixed cultures from extensive skin lesions in 100g rainbow trout in the UK (Austin and Austin, 1993). In this case, fish farm waters were contaminated by sewage. *Pseudomonas chlororaphis* has been reported in one instance to have caused heavy mortalities in farmed amago trout in Japan (Inglis and Hendrie, 1993).

Conclusions

1   Pseudomonadaceae are ubiquitous organisms in aquatic environments. *Pseudomonas* species are ubiquitous in fresh water environments, comprise a normal part of the microflora of many species of fresh water fish, and act as opportunistic pathogens of aquatic animals including salmonids. On rare occasions they have been demonstrated to be primary pathogens of aquatic animals.

2   Salmonids clinically infected with Pseudomonadaceae are unlikely to be harvested and processed for human consumption.

3   Because Pseudomonadaceae comprise a normal part of fish microflora, pathways exist which are more likely to lead to their introduction than the importation of the *commodity*.

4   The risk of Pseudomonadaceae introduction through importations of the *commodity* is negligible.

4.2.5 Vibrionaceae

Introduction

Vibrionaceae are facultatively anaerobic, Gram-negative, straight or curved rods. Vibrios are non-spore forming and motile by polar flagella. They are ubiquitous in marine and estuarine aquatic environments, particularly when static waters and soft benthos occur in combination with high organic loads, such as in estuarine sediments (Hjeltnes and Roberts, 1993). Vibrios cause acute bacterial septicaemia or chronic focal lesions in a wide range of fish species. Disease in fish is very often associated with some other stress, such as physical trauma, high stocking density, or poor water quality. Various species are pathogenic for fish, and infections in salmonids have been shown to be caused by *V. anguillarum, V. ordalii, V. salmonicida,* and *Plesiomonas shigelloides.* A variety of strains of the above species are known to exist and pathogenicity varies between strains. Vibrios are important pathogens of marine cultured aquatic animals due to their widespread occurrence in a variety of species and the economic impact of an outbreak of disease.

Vibriosis occurs in New Zealand salmonid aquaculture. *V. ordalii* has been isolated from salmon with clinical vibriosis and isolates have been shown to be closely related to the type strain (Wards et al, 1991). *V. anguillarum* has also been isolated from marine environmental samples in New Zealand, but these isolates have been shown to differ significantly from northern hemisphere pathogenic strains (Powell and Loutit, 1990).

V. anguillarum is serologically diverse. Austin and Austin (1993) reviewed the physical and biochemical characteristics, as well as the current status of strain and serotype classifications. Up to 10 serotypes have been proposed on the basis of cross-agglutination and cross-absorption tests with 'O' antigen.

V. salmonicida is the cause of 'Hitra disease', or 'cold water vibriosis'. Austin and Austin (1993) reviewed studies categorising strains on the basis of biochemistry, plasmid profiles, and serology.

Plesiomonas shigelloides has been recorded as causing bacterial septicaemia in 1-2 year old rainbow trout which experienced high mortality in northern Portugal, and suspected as having caused disease on other occasions (Austin and Austin, 1993).

Susceptibility and distribution

Egidius (1987) noted over 40 species of marine fish, including most of the salmonid species, in which *V. anguillarum* has been recorded as causing disease. Disease has also been recorded in fresh water culture, although it is primarily a marine phenomenon and disease in fresh water is probably caused by introduced marine vibrios (Egidius, 1987). Vibriosis due to *V. anguillarum* occurs worldwide.

V. salmonicida affects farmed Atlantic salmon in Norway, Scotland, the east coast of Canada and the USA, Iceland and the Faroe Islands. Rainbow trout may be infected experimentally, and *V. salmonicida* has also caused mortalities in cod. Since being first recorded in Norway in

1977, *V. salmonicida* has emerged as an economically important pathogen in Atlantic salmon culture in that country. The history and impacts of the disease in Norway have been reviewed (Håstein, 1993). Control of *V. salmonicida* through vaccination programmes has been relatively successful. From the peak in 1984 when 124 fish farms were affected by disease, in 1995 there were seven Norwegian fish farms affected by *V. salmonicida* outbreaks (Næss and Håstein, 1995). The use of vaccines has significantly reduced mortality in outbreaks in Canada and New England, USA (OIE, 1996).

Pathology, epidemiology and control

As *V. anguillarum* is ubiquitous, clinical disease is often associated with stressing mechanisms such as warm water temperatures, high stocking densities, high salinity or high organic load in the water. Parasitism or handling trauma may also precipitate disease. Transmission and pathogenicity studies have demonstrated that infection may be via the gills, the intestinal tract, or damaged integument. Following immersion of fish in *V. anguillarum* suspensions, the bacteria adhere to and colonise the gills, integument and the intestinal mucosa, and from there spread locally or via the blood to the liver, spleen, and muscle (Austin and Austin, 1993). The infective dose required by immersion and injection has not been precisely defined. The dose required to cause disease by injection may be relatively low. Evelyn (1971) infected chum and sockeye salmon by intra-peritoneal injection of 0.1 ml containing 10^7 viable cells of *V. anguillarum*. Sawyer et al (1979) established infection in Atlantic salmon by 1 hour immersion in 1×10^5 cells/ml. Neither of these experiments determined the minimum infectious dose.

Vibriosis caused by *V. anguillarum* occurs in peracute, acute, and subacute (chronic) forms. In juvenile fish, bacteraemia occurs early in the course of the disease, and bacteria are typically uniformly distributed throughout the tissues. Heavy mortalities are a common feature of disease outbreaks. Anaemia is a feature of the disease. Peracute infections in young cultured salmonids may cause few clinical signs other than periorbital and abdominal oedema. In older fish, acute and chronic forms occur. In the acute form, dark skin swellings and deep seated muscle lesions form, leading to tissue necrosis caused by large numbers of colonising bacteria. Chronically infected fish have deeper muscle lesions, characterised by granulation and scar tissue formation. Pale gills reflect severe anaemia. Fibrinous adhesions form in the abdomen. Histopathologically the muscle lesions show severe necrosis and ulceration of muscle and eventually the overlying epidermis. In chronic lesions there is concurrent reorganisation and necrosis of muscle lesions. Liver typically exhibits severe focal necrosis, and spleen and kidney show depletion and necrosis of haemopoietic elements.

Coldwater vibriosis or "Hitra disease, caused by *V. salmonicida*, normally occurs in late autumn through to early spring, when water temperatures are at their lowest, but it can occur year round. Clinical outbreaks are restricted to sea water or brackish water. All sizes and classes of Atlantic salmon may be affected, but disease in Norway most commonly occurs during the first winter period in sea water when fish are approximately 0.5-1.5 kg. The largest and fastest growing fish are often noted as the class most susceptible to disease (Håstein, 1993).
Disease is most commonly recorded in Atlantic salmon, and this species has been shown to be more susceptible than rainbow trout, with LD_{50} ranging from 4×10^6 to 1×10^8 cells per fish by injection (Austin and Austin, 1993). The disease is characterised by a generalized

haemorrhagic septicaemia. Fish may die peracutely with few clinical signs, or may be seen in sea cages with exophthalmos, abdominal distension and haemorrhages in the vent region. Internally there is pallor due to anaemia. Haemorrhages occur at the fin base and at the rectum, and ulcerations on the operculum or dorsum. Petechial haemorrhage over most serosal surfaces may be present. Pathology is typical of haemorrhagic septicaemia such as caused by *V. anguillarum*, including the muscle lesions.

Winter ulcer is a further disease described in Atlantic salmon with vibrios implicated in the pathogenesis (Lunder et al, 1995). Skin changes in this condition are characterised by ulcers of varying size, which may extend down into the underlying muscle. There is severe inflammation of the dermis and muscle tissue.

Effective vaccines against *V. anguillarum* and *V. salmonicida* have been developed. Vaccination by immersion, in food, or by injection is effective. Outbreaks may be controlled by the use of antibiotics, although antibiotic resistance is increasingly reported.

Survival and inactivation

V. anguillarum and *V. salmonicida* have been shown to survive long periods, up to 50 months and 14 months, respectively, in sea water with added nutrition (Hoff, 1989). In the same study, the long term starvation survival potential of *V. anguillarum* was demonstrated in high salinities, with no die off within 4 weeks at salinities of 10-35 parts per thousand. However, the fragility of *V. anguillarum* in fresh water environments was also demonstrated in this study, with 5 ppt salinity lethal after short exposure for *V. anguillarum* in the late exponential-growth phase, and salinities less than 10 ppt lethal after starvation.

V. anguillarum may not be as fragile in low salt environments as this study indicated, as it is able to be isolated on commercial tryptose soy agar containing less than 5 g/l NaCl (pers. comm. Christian Michel, August 1997).

The survival of vibrios in sediments is thought to be the means by which transmission cycles within fish farms are maintained from year to year. In Norway, *V. salmonicida* has been detected in sediment samples from diseased farms several months after an outbreak of disease (Enger et al, 1989).

Conclusions

1   *V. anguillarum* is a serious and economically significant pathogen of salmonids and other fishes, including marine fish species, with wide geographical distribution. New Zealand isolates of *V. anguillarum* are of low pathogenicity to salmonids.

2   *V. salmonicida* is a serious and economically significant pathogen mainly affecting farmed Atlantic salmon, with a geographical distribution encompassing Norway, Scotland, eastern Canada and USA, Iceland and the Faroe Islands.

3   The likelihood of fish clinically infected with *V. anguillarum* or *V. salmonicida* being harvested and/or being processed for human consumption is low.

4   Because Vibrionaceae are commonly part of the normal fish microflora and widespread in marine environments, the likelihood of a fish carrying either *V. anguillarum* or *V. salmonicida* being harvested and/or being processed for human consumption is high.

5   Heading, gilling and gutting is likely to significantly reduce the number of infective vibrio particles on salmonid fish.

6   If imported in fish tissues and placed directly into the marine environment in New Zealand, *V. anguillarum* or *V. salmonicida* could survive for long periods. However, if imported in fish tissues and placed into a fresh water environment (such as in processing premises, kitchens, and wastewater systems), *V. anguillarum* or *V. salmonicida* is less likely to survive for extended periods.

7   The likelihood of high concentrations of *V. anguillarum* or *V. salmonicida* building up in the environment as a result of importations of headed gilled and gutted salmonids for human consumption is low.

8   The risk of *V. anguillarum* or *V. salmonicida* introduction through importations of the *commodity* is negligible.

4.2.6   Moraxellaceae

Acinetobacter sp. are Gram-negative, facultatively anaerobic, non-motile rods. They are difficult to identify because they are biochemically unreactive. They are thought to be common inhabitants of fresh water and marine ecosystems, and populate the skin, gills and digestive tract of salmonids (Austin and Austin, 1993).

On a single occasion *Acinetobacter* sp. has been implicated as a primary pathogen in Atlantic salmon (Austin and Austin, 1993). The isolation was from 60 sexually mature Atlantic salmon wild broodstock, which were experiencing mortalities in conjunction with a haemorrhagic septicaemia-like condition.

Conclusions

1   *Acinetobacter* sp. comprise a normal part of the microflora of salmonids. On a single occasion *Acinetobacter* sp. has been implicated as causing disease in Atlantic salmon.

2   The risk of *Acinetobacter* sp. introduction through importations of the *commodity* is negligible.

4.2.7   The 'lactic acid bacteria'

Two disease conditions affecting salmonids must be discussed in the context of Austin and Austin's (1993) taxonomical classification of the "lactic-acid bacteria" i.e. anaerobic, fermentative, Gram-positive bacteria. The disease conditions are pseudo-kidney disease and streptococcosis. The taxonomy of the 'lactic acid bacteria' is subject to on-going change.

Austin and Austin (1993) reviewed the literature on the disease condition pseudo-kidney disease, and the bacteria that cause it. Pseudo-kidney disease is a septicaemia that is most commonly recognised in post-spawning rainbow trout. Typically, there is a mixed bacterial population isolated from infected fish, including lactic acid bacteria species such as *Carnobacterium piscicola, Lactobacillus* sp., *Lactococcus piscium* and *Vagococcus salmoninarum,* as well as *Aeromonas hydrophila, Pseudomonas fluorescens* and Enterobacteriaceae. Most species are common isolates in fresh water environments worldwide. The condition is characterised by ascites, internal haemorrhages, and pathology of the kidney, liver and spleen. Pseudo-kidney disease appears in most cases to be an opportunistic infection in stressed fish. The infection has been reproduced by injection of rainbow trout with lactic acid bacterial isolates.

Kitao (1993) reviewed the literature on the disease condition streptococcosis. Over twenty species of marine and fresh water fish have been affected by streptococcosis in Japan, the USA, South Africa, Singapore, Australia and Europe. Among the salmonids, rainbow trout and amago trout are susceptible. The disease syndrome is a septicaemia occurring both sporadically and epidemically, involving both alpha-haemolytic and beta-haemolytic strains of *Streptoccocus* species, as well as *Enterococcus* species. Isolates show a high degree of variation in biochemical properties, making species designation difficult in most cases. *S. iniae, S. agalactiae* (formerly *S. difficile*) and *Lactococcus garviae* (formerly *E. seriolicida*) are the more common isolates in fish infections.

Streptococcosis is characterised by ocular lesions (exophthalmia, corneal opacity, retrobulbar congestion, hyperaemia and haemorrhages), and haemorrhagic lesions on the dorsum, operculum, mouth, the caudal peduncle and the anal area. The lesions are generally more superficial than lesions of furunculosis or vibriosis, but will ulcerate and become necrotic gradually. Inflammation and haemorrhage from the gills may occur. Internally, spleen and liver are commonly affected, and kidney and heart less commonly. A serosanguinous exudate in the peritoneal cavity may lead to adhesions. *Streptococcus* species have been shown to survive for long periods in water and mud surrounding fish farms following outbreaks of disease. During an outbreak, environmental contamination may result from release of bacteria from dead or dying fish, and fish may become infected by horizontal transmission. Contaminated diets have also been demonstrated to be an important cause of disease in Japan. In rainbow trout, epidemics tend to occur when water temperatures are higher than 20°C.

Epidemics of streptococcosis occur in rainbow trout in Australia (Carson, 1990). Munday et al (1993) assessed the pathogenicity of Australian isolates of *Streptococcus* species for rainbow trout, Atlantic salmon and brown trout. Experiments by these authors confirm the occurrence of clinical streptococcosis is temperature dependant, and Atlantic salmon and brown trout are less susceptible to clinical streptococcosis.

Conclusions

1   Pseudo-kidney disease and streptococcosis are economically significant diseases of many fish species, including rainbow trout. The bacteria causally associated with these diseases appear to have wide geographical distribution, and some species are known to occur in New Zealand. The disease tends to occur in the presence of a stressor such as high water temperatures or in post-spawning fish.

2   Salmonids clinically affected with pseudo-kidney disease or streptococcosis are unlikely to be harvested and processed for human consumption.

3   Because these bacteria may be commonly isolated from the environment of fish farms, fish carrying these bacteria may be harvested and processed for human consumption.

4   The numbers of these bacteria present in headed, gilled and gutted salmonids processed for human consumption are likely to be low.

5   The likelihood of high concentrations of these bacteria building up in the environment as a result of importations of headed gilled and gutted salmonids for human consumption is low.

6   The risk of pseudo-kidney disease or streptococcosis introduction through importations of the *commodity* is negligible.

4.2.8   Other aerobic Gram positive rods and cocci

Austin and Austin (1993) reviewed the literature reports of aerobic Gram positive bacteria causing disease in salmonids. Within this group of organisms *Renibacterium salmoninarum* is of particular note as a pathogen of salmonids, and will be dealt with individually in the following chapter. A brief summary of Austin and Austin's (1993) findings regarding other aerobic Gram-positive bacteria follows.

Coryneform bacteria have been isolated from healthy rainbow trout. When injected intraperitoneally into juvenile rainbow trout they can induce mortality. Earlier reports of corynebacterium infections in salmonids were probably confusing coryneform bacteria with *Renibacterium salmoninarum,* which used to be classified as a coryneform but is now in a distinct genus. The status of coryneform bacteria as fish pathogens is presently uncertain.

Micrococcus luteus has been reported as causing disease in farmed rainbow trout in Argentina, and a micrococcus has been associated with diseased rainbow trout fry in the UK. The UK isolate was suspected to be involved in mortalities due to rainbow trout fry syndrome. Pathogenicity experiments involving intramuscular and intraperitoneal injection of the isolate into rainbow trout fry led to mortalities.

Mycobacteriosis is a common, widely distributed, chronic progressive disease which affects more than 150 marine and fresh water fish species, and probably involves a wide assortment of *Mycobacterium* species (Frerichs, 1993; Austin and Austin, 1993). The disease is a chronic progressive condition, characterised by inflammation and ulceration of the skin. The course of the disease may be several years. The bacteria involved are common in the aquatic environment. The disease is recorded in Australia (Humphrey, 1995). New Zealand imports many species of live ornamental fish of susceptible species, and it is unlikely that a chronic state of infection would be detected during quarantine.

Nocardiosis occurs in many species of fresh water and marine fish. *Nocardia asteroides*, a common soil and fresh water organism, is most commonly involved. Nocardiosis affects most age groups, and causes small white spots in dermis, muscle, gills and internal organs. The condition may progress in a manner similar to mycobacteriosis.

Planococcus sp. is increasingly being recognised in the UK as a cause of disease in farmed rainbow trout and Atlantic salmon. In Atlantic salmon the syndrome is of round, recessed white spots on the head. In 500g rainbow trout the condition may be ascites and kidney pathology. In rainbow trout fry, the condition may be manifested as rainbow trout fry syndrome, causing mortalities exhibiting anaemia, pale gills, liver and kidney and splenic pathology.

Rhodococcus sp. has been associated with disease in Canadian farmed chinook salmon and Atlantic salmon. In chinook salmon the condition manifests as ocular oedema leading to exophthalmia. In Atlantic salmon progressive granulomas in the kidney with low level mortalities have been recorded.

Conclusions

1   Various species of aerobic Gram positive bacteria are associated with disease conditions in salmonids. The disease conditions tend to have limited geographical distribution, although the bacteria associated with the condition probably have a much wider distribution. The disease conditions tend not to be of great economic or international quarantine significance.

2   The risk of aerobic Gram positive bacteria causally associated with disease conditions in salmonids being introduced through importations of the *commodity* is negligible.

4.2.9 *Renibacterium salmoninarum* (bacterial kidney disease)

Introduction

Renibacterium salmoninarum is the causative agent of bacterial kidney disease (BKD), and the disease has been reviewed on a number of occasions (Fryer and Saunders, 1981; Klontz, 1983; Munro and Bruno, 1988; Elliott et al, 1989; Evelyn, 1993; Fryer and Lannan, 1993). *R. salmoninarum* is of economic significance to the salmonid husbandry, in particular for Pacific salmon, because of its wide distribution in fresh water and marine environments, its chronic nature prior to development of clinical manifestations, its propensity for vertical transmission, and the inefficacy of the main therapeutic compounds used in treating fish (OIE, 1995b). *R. salmoninarum* is designated a significant pathogen of fish by the OIE (OIE, 1995a).

R. salmoninarum is the sole species within the genus *Renibacterium,* and is fastidious in its growth requirements. It is slow-growing, Gram-positive, non-acid-fast, non-spore-forming, and non-motile. Austin and Austin (1993) reviewed the ongoing investigation into the taxonomic positioning of *R. salmoninarum*. Forty-four strains of *R. salmoninarum* have so far been isolated, falling into two antigenic groups.

Susceptibility and distribution

R. salmoninarum causes disease in cultured and feral fish of the family Salmonidae. The genus *Oncorhynchus* (Pacific salmon, including rainbow trout) have experienced the greatest losses, and appear particularly susceptible. The genera *Salmo* and *Salvelinus* may also be infected, as may Danube salmon, *Hucho hucho,* and grayling, *Thymallus thymallus*. Other species have been experimentally infected (reviewed by Evelyn, 1993, and Lannan and Fryer, 1993), including Pacific herring, sablefish, shiner perch, common shiner, and flathead minnow. Evelyn (1993) notes an unpublished report of *R. salmoninarum* occurring in Pacific herring living in net-pens with BKD affected coho salmon. However, a survey of Pacific herring and other non-salmonid fishes living in and adjacent to net-pens failed to detect any naturally infected fish (Paclibare et al, 1988). Thus it is concluded that fish species other than salmonids are unlikely to be important in the epidemiology of *R. salmoninarum* infection, and conditions necessary to establish infection in these species are unlikely to occur under natural conditions. Blue mussels, *Mytilus edulis*, a common fouling organism on net-pens, have been shown to rapidly clear *R. salmoninarum* suspended cells in sea water and inactivate the bacteria (Paclibare et al, 1994). The authors concluded that the mussel was unlikely to act as a reservoir of infection, and more likely plays a beneficial role in reducing the frequency of horizontal infection.

R. salmoninarum occurs on the east and west coasts of North America, in Japan, Western Europe and Chile (OIE, 1995b). Neither the disease nor the bacteria have been recorded in Australia or New Zealand.

Data on the expected prevalence of *R. salmoninarum* infection must be interpreted with caution. Potential stressors such as the stage of sexual maturity and adaptation to either fresh or sea water (i.e. as returning adults or as smolts, respectively) lead to increased susceptibility. Sampling regimes for routine broodstock screening also introduce a potential bias through targeting fish showing overt signs of disease. The prevalence of *R. salmoninarum* infection is

known to vary widely within different stocks. The average prevalence of infection in a large population may not be a good indication of the prevalence within particular stocks.

There are significant difficulties in diagnosis of *R. salmoninarum* by culture or other means. *R. salmoninarum* is a slow growing organism with specific nutrient requirements. Culture on specialized media is required for long periods, up to 12 weeks (OIE, 1995b). Serological diagnostic methods (agglutination tests, immunofluorescent antibody tests, ELISA) are available, and are generally sensitive when *R. salmoninarum* tissue concentrations are high such as in advanced infections. As the ELISA technique has been developed and perfected, there have been conflicting reports on whether it or selective culture provided the greatest sensitivity of detection of low level infections (Pascho et al, 1987; Gudmundsdottir et al, 1993; Meyers et al, 1993; White et al, 1995). The ELISA is now established as the most sensitive diagnostic technique. IFAT is also able to diagnose a carrier state. Early work on the development of a standardised polymerase chain reaction (PCR) method to detect *R. salmoninarum* is promising, and the test may detect as few as 1 or 2 cells of the bacterium (Brown et al, 1994; Magnússon et al, 1994).

Data on the prevalence of *R. salmoninarum* in wild Pacific salmon in the Pacific northwest of North America for the period 1985-1994 show that 369 of 2,331 cases (15.8%) were positive for *R. salmoninarum* (pers. comm. Trevor Evelyn, October 1996). Data from the same source for the period 1972 to September 1993, presented in the earlier MAF risk analysis (MacDiarmid, 1994), indicate that prevalence was 4.6% during this period (1,011 positive of 25,984 fish). Also reported in the earlier MAF risk analysis were the results of testing of 600 salmon (300 sockeye and 300 chum) sampled at sea in British Columbia and tested for *R.. salmoninarum*. No kidney samples in these fish were positive by FAT and culture, but 3.3% of sockeye and 4.7% of the chum salmon were positive by ELISA. Data from Pacific salmon assayed by the Alaska Department of Fish and Game CFHD Division Fish Pathology Laboratories during the period 1980 to 1994 show that 3,206 of 51,895 fish (6.1%) were positive for *R. salmoninarum* (pers. comm. Linda Chaves, Deputy Director, National Marine Fisheries Service, USDC, August 1995).

An interesting perspective can be gained through examination of epidemiological data on infection with *R. salmoninarum* in pre-smolts, smolts and sea water phase fish in the Pacific northwest of the USA. A study which collected pre-smolt chinook and coho salmon and steelhead trout during downstream migration in the Columbia River detected an average 20% prevalence of *R. salmoninarum* in fish after holding them in fresh or sea water for 180 days (Sanders et al, 1992). Another study followed two groups of chinook salmon, one with high prevalence of BKD and the other with low prevalence, during downstream migration (Pascho et al, 1993). Survival of fish through the downstream migration was higher in the low-prevalence group. An earlier study (Banner et al, 1986) had collected salmonid fish in the coastal waters off Oregon and Washington. *R. salmoninarum* was detected in 11.0% of 878 chinook salmon (2.8% exhibiting overt signs) and 4.0% of 2,276 coho salmon (0.3% exhibiting overt signs).

These data suggest that, at least in the Pacific north west of America where *R. salmoninarum* is endemic, infection is detectable in all life stages of Pacific salmon, and that at any given time some members of an infected population will be dying from BKD. The data also suggest that

prevalence of infection in fish during fresh water stages (i.e. from fry to pre-smolts and again as spawning adults) is higher than during the marine phase. This may be due to a greater likelihood of horizontal transmission occurring at these times.

BKD was diagnosed for the first time in Norway in 1980 (Næss and Håstein, 1980). Since that time infection in wild and cultured fish has been recorded in seven river systems. In 1990, 76 salmon farms were under restrictions for BKD according to the Norwegian Fish Diseases Act (Næss and Håstein, 1990). In 1995 12 farms were under restriction (Næss and Håstein, 1995).

BKD was diagnosed in a rainbow trout farm in Denmark for the first time in March 1997, and has subsequently been demonstrated on a further six fish farms (pers. comm. Niels Jørgen Olesen and Ellen Lorenzen, Danish Veterinary Laboratory, September, 1997).

Pathology, epidemiology and control

Fryer and Lannan (1993) and Evelyn (1993) reviewed the clinical signs and pathology of BKD. Clinically infected fish show exophthalmia, abdominal distension, superficial blebs or blisters, haemorrhagic areas and deep abscesses on various parts of the body. Internally, the early stages of infection are characterised by small greyish-white lesions beneath the kidney capsule, with occasional similar lesions on liver and spleen. With time the lesions increase in size and number, the kidney becomes swollen, grayish-white and necrotic. In Atlantic salmon petechial haemorrhages are observed in the muscles lining the peritoneum. Histologically, advanced destruction of the haematopoietic tissue of the kidney is the principal feature.

An earlier MAF risk analysis (MacDiarmid, 1994) presented unpublished data on the viable *R. salmoninarum* count in the flesh and kidney of six chinook salmon dead from experimentally induced BKD. Kidney tissue averaged 4.6 x 10^9 viable cells per gram of tissue, and flesh 7.4 x 10^6 viable cells per gram of tissue. It was concluded that the flesh of fish dead of BKD contains approximately 10^3 times fewer *R. salmoninarum* cells than the kidney tissue. Examination of the data from testing of 600 salmon caught at sea led to the conclusion that, even in fish positive by ELISA, tissue concentrations must have been lower than 10^3 cells per gram of kidney. If the ratio of kidney to flesh bacterial titres noted above for fish dead of BKD holds true for carrier fish, the expected tissue titre for flesh of a carrier fish would be in the order of 1-10 cells per gram.

Transmission of *R. salmoninarum* occurs both horizontally and vertically. Vertical transmission through infected gametes plays an important role in maintenance of infection in susceptible populations. *R. salmoninarum* is located inside the ovum (Evelyn et al, 1984), and surface disinfection is ineffective in preventing transmission. The success of *R. salmoninarum* as a pathogen undoubtedly is a result of the ability to spread vertically. Natural horizontal transmission has been demonstrated to occur (Mitchum and Sherman, 1981), although the mechanism is not fully understood. Ingestion of faecal material may cause infection, as *R. salmoninarum* is shed in the faeces of infected fish. No data have been found from which the minimum infectious dose of *R. salmoninarum* can be deduced. Infection has been achieved by immersion of chinook salmon in a concentration of 10^4 cells per ml for 15 minutes (Murray et al, 1992).

Horizontal transmission is the only means by which importation of *the commodity* could lead to BKD establishing in New Zealand. If clinically infected fish are downgraded during inspection and processing, a conservative estimate of the *R. salmoninarum* titre in the *commodity* can be made by assuming this to be equal to the maximum possible titre in the kidney of ELISA-positive culture-negative carrier salmon (i.e. 1×10^3 viable cells per gram of tissue). Under the assumptions made in the discussion of IHNV (page 95) one can estimate that the concentration of *R. salmoninarum* at sewage outfall might be approximately 1.5×10^{-1} cells per ml. This is approximately 5 orders of magnitude less than Murray et al (1992) demonstrated would infect chinook salmon by immersion. While recognising that Murray's study did not provide an estimate of minimum infective dose, this calculation gives what is probably a conservative estimate of the risk of importations of the *commodity* leading to BKD establishment in New Zealand, although uncertainty remains with respect to the dose required to infect by the oral route.

Infections progress more rapidly at higher temperatures, although the greatest mortality may occur at temperatures below $12°C$. Diet and water chemistry may also affect incidence and severity of BKD, although this is an area requiring further study.

In reviewing methods of control of BKD, Fryer and Lannan (1993) note that antimicrobials have been effective in reducing disease levels, but unsuccessful in eliminating *R. salmoninarum* from fish host populations. Efforts to decrease vertical transmission include erythromycin treatments of brood stock during the period prior to egg-take. No effective and reliable vaccine against BKD is available. Brood stock segregation into heavily infected and lightly or uninfected groups is also used to control the prevalence of BKD in progeny. The selection of naturally resistant fish stocks for use in culture is another promising strategy.

Survival and inactivation

Austin and Austin (1993) reviewed the limited data on survival of *R. salmoninarum* in the environment. *R. salmoninarum* is an obligate intracellular organism, with limited survival outside the host in fresh or sea water. *R. salmoninarum* has been recovered from fish tank sediments for up to 21 days in the absence of any fish. In filter-sterilized river water, *R. salmoninarum* survived up to 28 days after which there was a rapid decline in numbers. *R. salmoninarum* is unable to compete with members of the normal aquatic microflora, and thus survives in the natural aquatic environment for limited periods only.

An earlier MAF risk analysis (MacDiarmid, 1994) reported the results of experiments conducted at the Pacific Biological Station, Nanaimo, in which the average number of *R. salmoninarum* cells in flesh was measured before and after one freeze thaw cycle to $-20°C$. In unfrozen fish average number of viable cells was 8.0×10^6 per gram of tissue. In frozen fish average number of viable cells was 1.8×10^6 per gram of tissue, a reduction of 77.5%. Although demonstrating the ability of the organism to survive freezing, this is nevertheless a significant reduction in terms of the numbers of viable cells likely to be present in carrier fish which are subjected to freezing and thawing during processing.

A recent study (Pascho et al, 1995) has demonstrated the sensitivity of *R. salmoninarum* to chlorine. At chlorine concentrations as low as 0.05 mg/L 99% inactivation occurred within 18

seconds at pH 7 and 15°C. The chlorine concentration of chlorinated water supplies in New Zealand is typically within the range 0.2- 0.5 mg/L, although concentrations up to 1 mg/L are used in some areas. Not all New Zealand water supplies are chlorinated (pers. comm. Wellington Regional Council City Water Management Dept., April 1997).

Conclusions

1   *R. salmoninarum* is an economically significant pathogen of salmonids with a wide geographical distribution.

2   Live fish and fish eggs are the highest risk commodities for spread of *R. salmoninarum*. There are no reports of dead eviscerated fish for human consumption being the agent for transmission of *R. salmoninarum,* although in many cases the means of introduction is not known.

3   The likelihood of a fish clinically infected with *R. salmoninarum* being harvested and processed for human consumption is low.

4   The likelihood of a *R. salmoninarum* subclinically infected or carrier fish being harvested and processed for human consumption is high.

5   The number of *R. salmoninarum* present in a headed, gilled and gutted subclinically infected or carrier fish processed for human consumption is likely to be low.

6   As a result of *R. salmoninarum* being highly sensitive to chlorine, the use of chlorinated water for washing and de-sliming fish during processing is likely to reduce the possibility of *R. salmoninarum* surviving on the outside of fish.

7   If imported in fish tissues and placed into the aquatic environment in New Zealand, *R. salmoninarum* would not be expected to remain viable for extended periods. Widespread chlorination of New Zealand water supplies, and the use of such supplies during any processing and preparation of imported salmon, is likely to reduce the possibility of *R. salmoninarum* remaining viable for extended periods.

8   The likelihood of *R. salmoninarum* titres in the New Zealand environment sufficient to initiate infections in chinook salmon by immersion resulting from importations of the *commodity* is low.

9   The risk of *R. salmoninarum* introduction through importations of the *commodity* is negligible.

4.2.10 *Piscirickettsia salmonis* (piscirickettsiosis)

Introduction

Piscirickettsiosis is a septicaemic condition of salmonids caused by infection with the rickettsial agent *Piscirickettsia salmonis,* a pleomorphic/coccoid, Gram negative, obligate intracellular parasite (Turnbull, 1993b). The syndrome emerged as an important cause of

disease in salmonids in Chile in 1981 (Bravo and Campos, 1989), the cause of which was later confirmed as *P. salmonis* (Cvitanich et al, 1991). *P. salmonis* is the first rickettsial agent to be implicated in the aetiology of a major fish disease. It is considered a significant disease by the OIE (OIE, 1995a).

Susceptibility and distribution

P. salmonis has been detected in coho, chinook, pink, Atlantic and Sakura salmon, and rainbow trout. The pathogen has been isolated in Canada, Chile, Ireland and Norway (OIE, 1995b).

In Chile the disease was first recognised in coho salmon. During 1989 approximately 1.5 million 200 g to market sized (2 kg) coho salmon died in epidemics (Cvitanich et al, 1991). Monthly mortalities on individual farms ranged from 1-40%, with cumulative totals of 70%. Typical mortalities in recent outbreaks have been 30% in coho salmon, 20% in rainbow trout and 10% in Atlantic salmon (OIE, 1996).

In British Columbia the disease syndrome has been recognised since 1970 in pink salmon held for experimental purposes (Kent, 1992). Since 1991 *P. salmonis* has caused a disease syndrome in farmed Atlantic and chinook salmon which is less dramatic than that seen in Chile (Kent, 1992; Brocklebank et al, 1993). *P. salmonis* has not been detected in wild salmonids in British Columbia.

Epidemic mortalities of the proportions seen in Chile have not been a feature of *P. salmonis* infections in Norway and Ireland. The disease syndrome reported in Atlantic salmon in these countries is a focal necrotising hepatitis (Rodger and Drinan, 1993; Næss and Håstein, 1993). In Norway, although *P. salmonis* was first isolated in 1992, the disease was probably occurring as early as 1988 (Næss and Håstein, 1993).

Pathology, epidemiology and control

The clinical signs and pathology associated with *P. salmonis* infection in salmonids in Chile were described by Cvitanich et al (1991). The most consistent signs were pale gills, swollen kidneys, enlarged spleens and a haemolytic anaemia. The most characteristic lesions, which are seen in fewer than 20% of heavily infected fish, are creamy to yellow sub-capsular nodules scattered diffusely through the liver. Histologically these lesions are described as a focal to diffuse necrotising hepatitis accompanied by necrotising vasculitis. Petechial haemorrhages are frequently observed on the stomach, intestines, pyloric caeca, swim bladder and visceral fat. Many fish show scale loss and raised areas of skin in the dorso-lateral regions, and patchy haemorrhagic areas on the ventral surfaces. Lesions in kidney, spleen, liver, heart, brain, intestine, ovary and gills are characterised as a vasculitis with associated necrosis. Rickettsial organisms are abundant in all tissues, typically intracellular within macrophages. The lesions in farmed Atlantic and chinook salmon in British Columbia are reported to be very similar (Kent, 1992), but may include exophthalmia, ulcerative stomatitis, raised erythematous masses in the branchial cavity, and solitary shallow ulcers on the flanks and cranio-dorsal to the tailfin (Brocklebank et al, 1993).

Initially infection with *P. salmonis* was thought to only occur in sea water. Since 1981 disease has occurred regularly in Chilean coho salmon smolts 6 to 12 weeks after entering sea water in the spring and autumn (Lannan and Fryer, 1994). *P. salmonis* has on occasion been isolated during the pre-smolt fresh water phase of rainbow trout and coho salmon (Bravo, 1994; Gaggero et al, 1995). However, the only report of *P. salmonis* occurring in diseased fish in fresh water noted concurrent infection with *Renibacterium salmoninarum* and a 2-3°C rise in water as possibly influencing the appearance of clinical signs (Gaggero et al, 1995).

How *P. salmonis* is transmitted requires further research. At present there is no evidence of vertical transmission (Turnbull, 1993), but there have been conflicting reports regarding horizontal transmission in fresh and sea water.

Cvitanich et al (1991) transmitted infection by injecting fish, and also observed infection in cohabitant fish in sea water and fresh water. The authors assumed horizontal transmission occurred via infectivity in fish faeces, although direct transmission of rickettsiae is known to be rare.

Lannan and Fryer (1994) noted that a pre-requisite feature of any organism capable of direct horizontal transmission is extracellular survival in the aquatic environment, and their experiments demonstrated infectivity remained in sea water after 2 weeks. In the same study, no infectivity was detectable after 28 days in spent culture medium. These results led to a conclusion that as *P. salmonis* could survive for periods of at least 2 weeks, direct transmission was a possibility in the marine environment. In fresh water, however, no infectivity could be demonstrated after re-suspension or at any time thereafter. The authors noted that this rapid inactivation would limit the opportunity for direct transmission in fresh water. This conclusion is supported by earlier studies which did not observe horizontal transmission in fresh water (Garcés et al 1991).

In Chile, the screening of fish tissues for *P. salmonis* by Gram-staining prior to selection as broodstock and iodophor treatment of fertilized eggs have been effective in restricting the impact of disease in fresh water hatcheries (Cvitanich et al,1991). There is no known method of restricting spread in sea water (Turnbull, 1993).

Survival and inactivation

P. salmonis is rapidly inactivated in fresh water, but survives for at least 2 weeks (but probably less than 28 days) in sea water (Lannan and Fryer, 1994).

Tissue homogenates from livers of infected chinook salmon lost the capability to induce disease in fish or cytopathic effect in cell culture after incubation at 37°C for 16 hours (Brocklebank et al, 1993). This suggests sensitivity to elevated temperature.

Conclusions

1   *Piscirickettsia salmonis* is an economically significant disease of salmonids with a known geographical distribution that includes major salmonid production areas of Chile, British Columbia, Ireland and Norway.

2   The likelihood of a fish clinically infected with *P. salmonis* being harvested and processed for human consumption is low.

3   Salmonids subclinically infected with *P. salmonis* could be harvested and processed for human consumption.

4   Heading, gilling and evisceration of any subclinically infected fish harvested and processed for human consumption is likely to significantly reduce *P. salmonis* infectivity.

5   As *P. salmonis* is an obligate intracellular organism, it would probably not survive for extended periods on the *commodity*.

6   If imported in fish tissues and placed directly into the marine environment in New Zealand, *P. salmonis* might survive for periods up to two weeks. However, if imported in fish tissues and placed into a fresh water environment (such as in processing premises, kitchens, and wastewater systems) any *P. salmonis* present in imported fish tissues is likely to be inactivated quickly.

7   The likelihood of high concentrations of *P. salmonis* building up in the environment as a result of importations of the *commodity* is low.

8   The risk of *P. salmonis* introduction through importations of the *commodity* is negligible.

4.2.11 Other miscellaneous pathogens

Epitheliocystis

Epitheliocystis is a common, widely distributed, readily transmitted chronic chlamydial infection of the skin and gills of certain marine and fresh water fish species (Austin and Austin, 1993; Turnbull, 1993b). The disease is known in fishes in a wide range of Northern and Southern Hemisphere countries, including Australia (Humphrey, 1995). Infections are known in tropical and cold water species and in marine and fresh waters. New Zealand imports many species of live ornamental fish of susceptible species, and it is unlikely that a chronic state of infection would be detected during quarantine. Epitheliocystis is not reported as causing problems in salmonid culture in the Northern Hemisphere.

Janthinobacterium lividum

Austin and Austin (1993) reviewed reports of *Janthinobacterium lividum*. In 1991 *J. lividum* was associated with two cases of rainbow trout fry syndrome, in Scotland and Northern Ireland. Typical clinical signs were exophthalmia, pale gills, darkening, swollen abdomen and skin lesions. In 1992 the bacteria was associated with skin lesions in 100-200g rainbow trout suffering from enteric red mouth. *J. lividum* is part of the normal microflora of fresh water and fish. These reports probably reflect an ability for *J. lividum* to infect salmonids as an opportunistic pathogen.

Conclusions

1   Disease problems in salmonids occasionally result from infections with non-host specific or opportunistic environmental bacteria.

2   Pathways of introduction for these bacteria exist which are far more likely than the importation of the *commodity*.

3   The risk of non-host specific or opportunistic environmental bacteria introduction through importations of the *commodity* is negligible.

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4.3   QUALITATIVE ASSESSMENT OF THE FUNGAL, ALGAL AND PROTOZOAN PARASITES OF SALMONIDS

4.3.1   Fungi

Although numerous species of fungi have been reported to be pathogens of salmonid fishes, few are recognised as being of economic significance and none are ascribed any degree of international quarantine significance. Most fungal parasites of salmonids cause disease in fresh water; the notable exception is *Ichthyophonus hoferi*. Most also are common environmental organisms and cause disease as opportunistic invaders of stressed fish.

Possibly the most common fungal disease of salmonids is **saprolegniasis**. Saprolegniasis is caused by a range of fungal species from the genera *Saprolegnia, Achyla, Aphanomyces* and *Leptolegnia*. The name saprolegniasis has come to incorporate almost any fungal infections of the skin of fish, despite such infections commonly being caused by species of fungi not within the order Saprolegniales (Pickering and Willoughby, 1982). Some species are recognised as having greater pathogenicity. All are common saprophytes. For diagnostic purposes species identification is unnecessary and rarely performed. The disease is worldwide in distribution, and affects a wide range of fish species including salmonids (Chacko, 1993). All salmonid species are susceptible.

Saprolegniasis is an integumentary or gill mycosis, usually secondary to stressing factors. A characteristic cotton wool appearance accompanies the proliferation of hyphal masses which adhere to and invade the skin or gills. Erosion and ulceration of the skin or gills causes osmo-regulatory failure. *Saprolegnia* species are also common hatchery saprophytes causing characteristic cotton wool appearance to degenerating eggs. Removal of dead eggs, and the use of formalin for dip treatment are common practices in salmonid hatcheries to control saprolegniasis. Saprolegniasis is common in aquarium fishes (Reddacliffe, 1985). New Zealand's import controls for aquarium fishes are unlikely to have prevented the introduction of a wide range of saprolegniasis species from being introduced.

Ichthyophonus hoferi is a fungal pathogen of mainly marine fish species that is of economic significance in salmonid netpen culture in North America. The disease also occurs in fresh water, and infections have been reported in rainbow trout which were being fed infected marine fish (Kent, 1992). Rainbow trout may be particularly susceptible to disease (Chacko, 1993). Many species of wild marine fish are infected, and in some species the prevalence is high, such as in Atlantic herring and plaice (Kent, 1992). The disease probably has a worldwide occurrence, although there are no literature reports of infection in New Zealand fishes. *I. hoferi* occurs in Australia (Reddacliffe, 1985; Humphrey, 1995; Munday, 1996). The fungus is spread from fish to fish by ingestion of spores or infected fish. Infections in Atlantic salmon smolts in their first summer in sea water have been observed in British Columbia, and it is unclear how and when the fish became infected (Kent, 1992). Infected fish are lethargic and emaciated, and may show curvature of the spine. White granulomatous lesions occur in the visceral organs, heart and muscle. The spores are visible in histological sections of lesions. When the host dies the spores produce a germination tube and branched hyphae. In plaice and haddock this occurs 15-30 minutes after death (Kent, 1992).

Exophiala salmonis causes **cerebral mycetoma**, occasionally in epidemics, in cutthroat trout, lake trout and Atlantic salmon in Canada (Chacko, 1993). The life cycle of the causative organism and predisposing conditions for the disease are unknown. A related species, *E. pisciphila*, causes a granulomatous inflammatory condition in a variety of fresh water and marine fish, including channel catfish (Chacko, 1993). Salmonids affected by cerebral mycetoma show ataxia, whirling swimming patterns, exophthalmus, and cranial cutaneous ulcers. Lesions are often confined to the brain, and are a chronic granulomatous reaction from which the fungus may be cultured. Attempts to reproduce the disease have failed.

***Branchiomyces* spp.** are parasites of gill tissues. Brown trout, Arctic char, and rainbow trout are susceptible (Chacko, 1993), as are a variety of non-salmonid fish species (reviewed by Humphrey, 1995). The geographic distribution includes USA, Europe, Africa, Israel, and Asia. The disease, termed branchiomycosis, is considered a major problem in eastern European fish culture (Humphrey, 1995).

Candida sake causes **candidiasis** in Amago salmon (reviewed by Chacko, 1993). The disease syndrome is characterised by stomach distension, caused by viscid fluid and gas bubbles.

Leptomitis lacteus has been reported to invade the eggs of rainbow trout, as well as a number of other fish species, in the UK (reviewed by Humphrey, 1995).

Ochroconis tshawytscha and *O. humicola* cause the disease **phaeohypomycosis**, which has been reported in rainbow trout and coho and chinook salmon in the USA (Chacko, 1993). Disease occurs as outbreaks with low morbidity. External signs are blister-like areas on the sides of fish, exophthalmus, and pale oedematous gills. Internally, there are enlarged kidneys, and a pale, haemorrhagic liver and spleen. Exposure of rainbow trout to suspensions of the fungus failed to induce disease, although intra-peritoneal injection succeeded. This suggests that horizontal transmission is unlikely in the absence of other stressing factors.

Paecilomyces farinosus is noted by Chacko (1993) as the cause of low-level mortalities among farmed Atlantic salmon in Scotland. Infected fish showed reddened vents and swollen abdomens. The fungus was found only in the wall of the swim bladder.

Phoma herbarum is a plant saprophyte that is infective for the fry and fingerlings of rainbow trout, coho and chinook salmon. The resulting disease, characterised by swollen swimbladder and eventual invasion of all other visceral organs, is recorded only in the USA and UK. The causative fungus is probably ubiquitous (Chacko, 1993, Humphrey, 1995).

Humphrey (1995) also notes a single report of a ***Schizotrytium*-like sp**. associated with epidermal lesions in farmed rainbow trout in the UK.

Conclusions

1   Two significant fungal diseases of salmonids, saprolegniasis and ichthyophoniasis, are caused by organisms which have suspected worldwide distribution. Insufficient data are available to definitively state whether the causative species are already present in New Zealand.

2   Numerous other fungal organisms have been reported to act as pathogens in salmonids. The disease syndromes typically are confined to discrete geographical regions, affect juvenile life stages, such as eggs or fry, and cause clinical signs that would lead to rejection or downgrading during processing. As such, the likelihood of a salmonid clinically infected with a fungal pathogen being harvested and processed for human consumption is low.

3   The fungal organisms which can be pathogenic in salmonids tend to be ubiquitous in aquatic environments, and may be commensals of healthy fish. For this reason, the likelihood of harvesting and processing a salmonid for human consumption which is subclinically infected with a fungal pathogen is high.

4   Heading, gilling, evisceration and washing during processing for human consumption is likely to significantly reduce infectivity of any subclinically infected fish.

5   Pathways for introduction of fungal pathogens of salmonids other than via the importation of the *commodity* exist. The low host specificity of many fungal pathogens means that importations of ornamental fish may have already introduced into New Zealand a range of fungal organisms potentially capable of opportunistically infecting fish.

6   The risk of fungal diseases of fish introduction through importations of the *commodity* is negligible.

4.3.2 Algae

Algae are a large and diverse group of organisms which are normally free-living in aquatic environments. Algae make up a significant component of phytoplankton, which form an important link in the carbon cycle in aquatic systems. Certain conditions may act as a trigger to cause algal population blooms, which may have adverse effects on many fish and invertebrate species. Algal blooms periodically cause fish kills in coastal waters throughout the world.

Kent (1992) reviewed the mechanisms by which algae may kill fish during an algal bloom. The mechanisms are through physical damage to gills by diatom spines, through asphyxiation by oxygen depletion, through gas-bubble trauma as a result of oxygen super-saturation from algal photosynthesis, and through direct chemical toxicity caused by icthyotoxins. Numerous algal species have been recorded as being involved in fish kills around the world, including in New Zealand. In Big Glory Bay, Stewart Island, *Heterosigma akashiwo* caused mortalities in cage reared chinook salmon in January 1989 (Chang et al, 1990). That report summarised the previously recorded phytoplankton blooms in New Zealand coastal waters.

International attention has recently focussed on the potential for harmful algae and other free-living aquatic organisms to be transferred in ships' ballast water.

Conclusions

1   Fish from water affected by an algal bloom are unlikely to be harvested and processed for human consumption.

2   Heading, gilling, evisceration and washing are likely to remove the majority of algae from any fish.

3   The likelihood of algae remaining viable on the *commodity* for extended periods is unknown, but is assumed to be low.

4   Pathways of algae introduction exist which are far more likely than the importation of the *commodity*.

5   The risk of potentially harmful algae introduction through importations of the *commodity* is negligible.

4.3.3 Mastigophora (flagellates)

The phylum Mastigophora (flagellates) comprises many orders of protozoa common in aquatic systems.

Five genera within the order Dinoflagellida (the dinoflagellates) are reported as parasites of fish. Many dinoflagellates are parasites of invertebrates. The principal mode of pathogenicity in fish is through the production of icthyotoxins, which may cause mass mortalities in wild or cultured fish (Noga and Levy, 1995). The icthyotoxin-producing dinoflagellates tend to parasitise the gills and skin of fishes in warm waters. Conditions conducive to dinoflagellate population blooms will lead to effects in local fish populations. Icthyotoxins are unlikely to be host specific.

There are numerous other organisms pathogenic for fish within the Mastigophora. Of these, the organisms which are known to be pathogenic in salmonids are *Hexamita salmonis, Icthyobodo (= Costia) necator*, and *Trypanoplasma (= Cryptobia) salmositica*.

Hexamita salmonis

Hexamita salmonis is a common flagellate parasite of the intestinal tract of salmonids. It is known to occur in Europe, North America and Asia. *Hexamita salmonis* is non-host specific, infecting a wide variety of fresh water and marine fishes. Rainbow trout and Atlantic salmon fingerlings, yearlings and smolts are most commonly infected. Infections are sporadic in aquaculture and rare in wild fish (Woo and Poynton, 1995).

Hexamita salmonis has a direct life cycle. Transmission occurs in fresh and sea water. Infection is by ingestion of cysts or trophozooites. Multiplication occurs in the intestine, and cysts and trophozooites are released in the faeces. Skin lesions may also be a source of infection. Aquaculture conditions facilitate transmission through crowding. *Hexamita salmonis* parasitises the intestine, causing chronic signs such as anorexia, emaciation, weakness, pale gills, abdominal distension, faecal pseudocasts, darkening, and exophthalmia. On necropsy there is anaemia, ascites, enteritis, and pale mucous intestinal contents. Systemic infections are also reported.

An organism closely resembling *Hexamita salmonis* has caused severe systemic infection with 50% mortality in chinook salmon at one net pen site in British Columbia (Kent, 1992). Systemic infections have also been reported in Atlantic salmon in Norway. These infections may represent a new, highly invasive form of *Hexamita salmonis* (Kent, 1992). Systemically infected Atlantic salmon may be in good condition but be significantly smaller than non-infected fish, and may behave abnormally, suffering increased morbidity and mortality. Systemically infected chinook salmon may appear normal or have distended abdomens. Gills are pale as a result of anaemia. The most obvious pathological change is an extremely hypertrophied liver (Kent, 1992). There may be serosanguinous ascitic fluid, enlarged spleen and kidneys, and petechiae occur throughout the skeletal muscles. Diagnosis is confirmed by identification of the organism in Giemsa stained wet mount preparations of blood or visceral organs.

Icthyobodo necator

Icthyobodo (= *Costia*) *necator* is an ectoparasite causing icthyobodiasis (costiasis) in freshwater and marine fish. It has been reported in North America, Europe and Japan (Woo and Poynton, 1995), but its true distribution is probably much greater than this. Humphrey (1995) records a diagnosis in Australia of costiasis in imported weather loach (a freshwater tropical fish common in home aquaria).

The life cycle involves a free-living ovoid to spherical form which is motile by flagella. The free-living form selects a site on a fish, either the skin or gills, and transforms to the parasitic form which attaches via a ventral disc. Both the free-living and parasitic forms of the organism are believed to be able to encyst under adverse environmental conditions, and cysts in the water become a source of infection and means of spread (Woo and Poynton, 1995).

The parasite is common in fresh water-reared fish, including salmonids, eels and goldfish. It has also been observed in Atlantic salmon in sea water. Further discoveries of the parasite in purely marine origin fish, such as flounder and haddock, have led to the view that a marine-adapted form may exist (Kent, 1992, Woo and Poynton, 1995). The organism is non-host specific; probably all species of fresh water fish are susceptible, including many species of ornamental fish commonly imported for home aquaria.

Malnourished and young fish are more severely affected than healthy adults. High mortality can be associated with heavy infections, as a result of gill damage. The organism multiplies rapidly by binary fission, and heavy infections can develop quickly if left untreated. Clinical signs include flashing, listlessness, anorexia, spots or films on fins and body surface, swollen gills and increased mucous secretions. The parasites can be observed in wet mounts of the gills or skin of affected fish. Control is achieved using external treatments of formalin or malachite green.

Trypanoplasma sp.

According to some classifications, *Trypanoplasma* sp. and *Crytobia* sp. may be members of a single genus of biflagellate organisms which inhabit fresh water. The taxonomic status of members of the genus requires clarification. Although used synonymously by some workers, the names *Trypanoplasma* sp. and *Crytobia* sp. have been proposed to also be used to distinguish organisms on the basis of life-cycles and morphology.

Trypanoplasma sp. are endoparasites of the blood system of fish and have indirect life cycles utilising leeches as intermediate hosts. *Crytobia* sp. are ectoparasites of the gills or body surface or endoparasites of the intestinal tract, and have direct life cycles (Woo and Poynton, 1995). Five species are known to cause disease in fish. One of these species, *T. salmositica* infects Pacific salmon species along the west coast of North America and in East Asia. *T. salmositica* is transmitted indirectly by a leech vector, but may also be transmitted directly. Salmon may be infected prior to smoltification, and one study (Woo and Poynton, 1995) reported prevalence in the range 3% to 21% in smolts. Mortalities probably continue after entry into sea water, and further infections occur upon return to fresh water. Prevalence in spawning salmon in fresh water is low in September, and about 100% in December. Infections are detectable from 5 days after entry into freshwater, and parasitaemias increase the longer the fish are in fresh water. *T. salmositica* can be grown in vitro, but requires serum

supplementation. *T. salmositica* is a parasite of cold water fish, and becomes sluggish and dies at temperatures over 21°C (Woo and Poynton, 1995). Clinical signs of infection in Pacific salmon species are exophthalmia, splenomegaly, anaemia and anorexia. The severity of the disease is related to the size of the inoculum, the water temperature, diet and genetics of the fish. In some cases of disease in coho and chinook salmon 100% mortality has been recorded. Sculpins (*Cottus* sp.) may be infected with *T. salmositica* without succumbing to disease and are thought to be a natural reservoir of infection.

Conclusions

1   *Hexamita salmonis, Ichthyobodo necatrix* and *Trypanoplasma salmositica* are common parasites of salmonids and other fishes, with wide geographical distributions. They occasionally cause economically significant disease syndromes in salmonids.

2   The likelihood of a fish clinically infected with one of these protozoan parasites being harvested and processed for human consumption is low, given the geographic distribution, the likely age of clinically infected fish, and the clinical signs.

3   Given the organisms' common occurrence and widespread distribution, the likelihood of a fish subclinically infected with one of these protozoan parasites being harvested and processed for human consumption is high.

4   Processing by heading, gilling, evisceration and washing is likely to remove the majority of these protozoan organisms in infected fish.

5   In the case of *Hexamita salmonis*, intestinally infected fish are very unlikely to lead to organisms carried in or on the *commodity*. Systemically infected fish are unlikely to be processed without suffering quality downgrading rendering them ineligible as the *commodity*.

6   Pathways of *Ichthyobodo necatrix* introduction exist which are far more likely than the importation of the *commodity*.

7   The risk of *Hexamita salmonis, Ichthyobodo necatrix* or *Trypanoplasma salmositica* introduction through importations of the *commodity* is negligible.

4.3.4   Rhizopoda (amoeba)

Amoeba are free-living organisms common in aquatic systems. Amoebic gill disease is caused by two species, *Paramoeba sp.* and *Thecamoeba hoffmani,* which parasitise the gills of salmonids. The trigger for the change from free-living state to parasite is unknown.

Amoebic gill disease is recorded in salmonids in Europe, North America and Australia. *Paramoeba sp.* is present in New Zealand (Anderson, 1996), although only mild disease has been recorded. *Paramoeba sp.* causes significant disease problems in Atlantic salmon in sea cage culture in Tasmania.

T. hoffmani causes amoebic gill disease in fingerling rainbow trout, coho salmon and chinook salmon in hatcheries in the USA (Woo and Poynton, 1995). In contrast to *Paramoeba sp.*, *T. hoffmani* is a fresh water organism. The amoeba was seen between gill lamellae, which were short, abnormally wide, and exhibiting cellular hyperplasia. Mortality resulted from respiratory impairment.

Both Woo and Poynton (1995) and Humphrey (1995) note literature reports of systemic amoebic infections in salmonids, in the kidneys and visceral tissues of rainbow trout and brook char. The infections were apparently associated with significant mortalities.

Conclusions

1   Amoebic gill disease is an economically significant disease of cultured salmonids. One of the causative organisms is present in New Zealand waters, although disease is not commonly recorded.

2   Heading, gilling, evisceration and washing is likely to significantly reduce infectivity in any infected salmonid harvested and processed for human consumption.

3   The risk of pathogenic amoeba introduction through importations of the *commodity* is negligible.

4.3.5   Apicomplexa

The Apicomplexans include the coccidia, which are primarily intestinal parasites with direct life cycles characterised by complex developmental stages.

Humphrey (1995) notes that a *Cryptosporidium* sp. is known to be pathogenic in fish, including salmonids, and that a *Cryptosporidium* sp. has been isolated from ornamental fish imported into Australia.

Molnár (1995) notes that the fish parasitic species *C. nasoris* has an extremely broad host range. The clinical syndrome in fish is likely to be digestive disturbance and malabsorption, and in heavy infections, mucus production and enteritis. There are no reports of this organism being a serious pathogen of salmonids, either in cultured or wild fish.

Conclusions

1   Fish clinically infected with *Cryptosporidium* sp., or other coccidia of fish, are unlikely to be harvested and processed for human consumption.

2   Heading, gilling, evisceration and washing are likely to remove the majority of coccidial organisms from any fish.

3   Pathways for introduction of coccidial organisms exist other than the importation of the *commodity*.

4   The risk of *Cryptosporidium* sp., or other coccidia of fish, introduction through importations of the *commodity* is negligible.

4.3.6 Microspora

The Microspora are obligate intracellular protozoan parasites, occurring in all animals but particularly invertebrates. Species affecting fish are widely distributed geographically in a variety of host species. Dyková (1995) reviewed the literature on microsporean parasites of fishes.

Microspora life cycles are typically direct, involving complex development in the host and spore formation. The spore is the infective stage, and natural infection is by ingestion. Once ingested, an elaborate spore-hatching apparatus enables the sporoplasm to become intracellular in the host, typically within macrophages, and the parasite is transported to the target tissues. Species differ in their requirements for particular host cell types. Cycles of merogony produce great numbers of parasite stages by binary or multiple fission, leading to heavy infections. Sporogony produces spores, which are either actively released or remain in tissues until after death. Spores retain infectivity in water at 40°C for at least 1 year. Development in the host is dependent on host physiological responses and environmental conditions, in particular temperature. Low temperatures affect host response mechanisms and parasite development. Host reaction to microsporidia is directed towards isolation and elimination of the parasite. Cells may undergo hypertrophy and nucleus fragmentation, leading to xenomas. Alternatively, infected cells may be isolated by fibroblasts and phagocytised by macrophages. Diagnosis of fish microspora infections is by microscopic examination.

Three species of microspora are recognised as significant parasites of salmonid fish: *Microsporidium takedai*, *Enterocytozoon salmonis* and *Loma salmonae*.

Microsporidium takedai

M. takedai infects eight species of salmonids in Japan. In rainbow trout the prevalence may be 100%. In other species, the prevalence may be 86% to 93%. Infection is temperature dependent, and is restricted to the warmer period of the year. The parasite grows well in hosts in water at 18°C, but stops growing at 8°C. The target tissues are heart and skeletal muscle. In skeletal muscle *M. takedai* produces 6 mm spindle-shaped 'cysts', and in heart muscle 2 mm globular-shaped 'cysts'. In acute disease there may be high mortality and enormous numbers of 'cysts' in the trunk musculature, up to 130 per gram of tissue. Heavily infected fish are typically in poor body condition. The host tissue response is inflammatory and phagocytic cellular infiltration, which in experimentally infected yearling salmonids started on day 11 post infection. Macrophages phagocytise spores and are eventually transported across the epidermis to outside the host.

Enterocytozoon salmonis

Enterocytozoon salmonis (Chilmonczyk et al, 1991) has been recorded in fresh water reared chinook and kokanee salmon and steelhead trout in Washington, California and Idaho, and in chinook salmon in sea water net pens in British Columbia (Kent, 1992). *E. salmonis* has also been reported in rainbow trout in France (Dyková, 1995) and Atlantic salmon in Chile (Bravo, 1996) following importations of salmonid eggs from North America. The parasite is extremely small, and is found intra-nuclearly in haemoblasts of haematopoietic tissues such as kidney and

spleen, and also in smaller numbers in leptomeninges, skeletal muscle, coelomic mesenteries, hepatic sinusoids, and periorbital connective tissues (Hedrick et al, 1991). Another intra-nuclear microsporidium, *Microsporidium rhabdophilia,* is also known to infect salmonid fish in California but is not associated with any disease syndrome (Modin, 1981). *E. salmonis* has been experimentally transmitted by intra-peritoneal injection of juvenile chinook salmon with kidney tissue of naturally infected fish (Hedrick et al, 1991).

E. salmonis infection in British Columbian chinook salmon is often associated with concurrent neoplastic disease known as plasmacytoid leukaemia (PL), although *E. salmonis* has been cleared of involvement in the aetiology of this disease. Clinical signs associated with infection include anaemia, with packed cell volumes as low as 5%, and pallor of the gills. Infections associated with PL are characterised by lymphoblastosis and leukaemia typical of that disease (Kent, 1992). The progression of the disease is water temperature dependent. Experimentally infected juvenile chinook salmon held at 15-18°C were severely affected, and suffered 90% mortality. Fish held at temperatures of 9-12°C suffered lower mortalities over a longer period (Antonio and Hedrick, 1995). Diagnosis of *E. salmonis* can be made by examination of Gram-stained kidney impressions, or by histological examination of kidney tissue sections. Treatment with Fumagillin DCH may protect fish from infection (Hedrick et al, 1991).

Loma salmonae

*Loma (*formerly *Pleistophora) salmonae* is widespread in wild and hatchery-reared rainbow and steelhead trout and kokanee, chinook, coho and masou salmon in the Pacific coast of North America and Japan. It has also been reported in coho salmon in France. Epidemic mortalities in these species have occurred in hatcheries as a result of *L. salmonae* infections (Dyková, 1995). Experimental challenge of Atlantic salmon, Pacific herring and shiner perch was unable to establish *L. salmonae* infection (Kent et al, 1995). Another species, *Loma fontinalis,* has been described from brook trout in British Columbia (Morrison and Sprague, 1983). The two species, *L. salmonae* and *L. fontinalis*, are very similar (Dyková, 1995) and are considered together in the context of this study.

Infections in coho salmon in fresh water may be mild, but following transfer to sea water may be associated with significant pathological changes and mortalities. Peak prevalence of 33-65% in coho salmon from infected net pen sites in Washington have been recorded during the summer (Kent et al, 1989). Infection is typically acquired in fresh water. However, transmission has been demonstrated to occur in sea water (Kent et al, 1995). Transmission is direct, as in other microsporidia, and the spore is the infective stage. The primary site of infection is in the gills. Spores are released following death and breakdown of the tissues. Removing dead fish from net pens without delay has been suggested as a means of controlling the spread of infection (Kent et al, 1995). Spores can be detected in wet mount preparations of the gills, and xenomas can be seen on histology. Xenomas in secondary lamellae of the gills cause relatively little damage, whereas those in primary lamellae cause severe inflammation (Kent, 1992). Parasites can also be detected in heart, spleen, kidney and pseudobranchs. Rupture of xenomas and release of spores into the surrounding tissue is believed to be a contributing factor to the inflammatory response. Preliminary studies indicate that Fumigillan DCH may be useful in the control of *L. salmonae* infections (Kent and Dawe, 1994).

Conclusions

1   Microspora parasites are serious and economically significant pathogens of salmonid fishes which tend to have defined geographical distributions and specific host ranges.

2   Salmonids infected with *Microsporidium takedai* are unlikely to be harvested and processed for export to New Zealand because importation from Japan is unlikely. Heavily infected fish are unlikely to pass inspection during processing.

3   The risk of *Microsporidium takedai* introduction through importations of the *commodity* is negligible.

4   The probability of a fish subclinically infected with *Loma salmonae* or *Entercytozoon salmonis* being harvested and processed for human consumption is high.

5   Heading, gilling and evisceration is likely to remove the large majority of Microspora parasites, with the exception of *Microsporidium takedai*, from any infected salmonids.

6   *Loma salmonae* and *Entercytozoon salmonis* are obligate intracellular parasites. However, their spores may survive for long periods in aquatic environments. The ability of the parasite to survive in the *commodity* is unknown.

7   Ongoing large volume exports of salmonid products for human consumption from infected areas of the USA and Canada have not been associated with extension of the range of *Loma salmonae* or *Entercytozoon salmonis*.

8   The risk of *Loma salmonae* or *Entercytozoon salmonis* introduction through importations of the *commodity* is negligible.

4.3.7 Myxosporea

The Myxosporea (Phylum: Myxozoa) are a group of more than 1,250 species, the majority of which occur in fish. Although multicellular, most researchers still include Myxosporea in the kingdom Protista (Whitaker et al, 1994). Myxosporea are known from practically all organs and tissues in fish. However, relatively few are known to cause serious or fatal infections. This probably reflects an equilibrium attained between myxosporeans and their hosts during their evolution. Host specificity varies between species, and geographical distribution depends on that of the host. Lom and Dyková (1995) reviewed the literature on myxosporean parasites of fishes.

The taxonomy of the genera is based on morphology, particularly of the spores. Characteristics of the vegetative life stages and life cycle are insufficiently known to be of use during classification. Intensive research into the life cycles of certain species, such as *Myxobolus cerebralis,* the cause of whirling disease (which is present in New Zealand), has led to the development of the actinosporean transformation theory (Wolf and Markiw, 1984). The basic premise of this theory is that all Myxosporea have life cycles similar to that of *M. cerebralis*, whereby development occurs in two hosts, which in the case of *M. cerebralis* are salmonid fish and an aquatic oligochaete worm. Spores released from fish hosts are not infective to fish, but must undergo development in the intermediate (or alternate) host. Development in the intermediate host leads to an actinosporean stage which is infective to fish. Such a life cycle is unparalleled among the protists. It has two types of asexual proliferation, two different sexual processes, two sporogenies resulting in two completely different types of infectious spores. This type of two host life-cycle has now been demonstrated to occur for 14 Myxosporean species, belonging to six genera in four families. If the actinosporean transformation theory is confirmed, it would naturally lead to suppression of the class Actinosporea, and Actinosporea species would be assigned to separate myxosporean species once their life cycles were properly identified (Kent et al, 1994). There is also a theory of direct transmission of myxosporean spores, but this seems unlikely on recent evidence (Lom and Dyková, 1995).

The two host life cycle of freshwater Myxosporeans is summarised by Whitaker et al (1994). The stage infective to fish is the sporoplasm that is released from a multicellular actinospore. Following penetration of the fish epithelium, the sporoplasm migrates to the site of infection. The sporoplasm becomes a multinucleate plasmodium, which in turn produces spores containing one or two sporoplasms. Myxosporean spores have no antigenicity to host fish, and elicit a low degree of humoral response. The Myxosporean trophozoites are classified as coelozoic or histozoic plasmodia depending on whether they target body cavities or tissues respectively. The extent of damage to tissues and organs is dependent on the location of the parasites, the intensity of infection and the host response. These vary from species to species. Cycles of merogony in the host can give rise to heavy infections resulting from small inocula. Hypertrophy and hyperplasia are the most common progressive changes in host tissues (Lom and Dyková, 1995). The stage infective to the oligochaete alternate host is the multicellular spore released from the fish after death or with body fluid discharge. Vegetative development

occurs in the gut of the worm, culminating in the formation of the actinospore. This life cycle has yet to be demonstrated for marine fish myxosporeans. Aquatic oligochaetes are common in the marine environment. Other planktonic invertebrate species might also serve as alternate hosts. An ability for direct fish to fish transmission may also exist for some species (Whitaker et al, 1994).

Ceratomyxa shasta

Ceratomyxa shasta is a fresh water Myxosporean species which is only known to occur in the west coast of North America. Bartholemew et al (1989) reviewed the literature on the parasite. Cutthroat, steelhead and rainbow trout and pink, chinook, coho and chum salmon are susceptible, and the parasite has caused epidemics in cultured and wild populations. Studies indicate prevalence ranging between 11% and 100% in stocks of sexually mature adult salmonids of various species sampled on the spawning grounds of the Fraser River, British Columbia (McDonald, 1982), between 1.5% and 3.3% in seaward migrating juvenile chinook salmon in the Fraser River (Margolis et al, 1992) , and between 8.9% and 14% in seaward migrating chinook salmon on the Columbia River (Fryer, 1985). Different stocks of salmonids may vary in their susceptibility to infection. Fish infected as fingerlings remain parasitised during the marine phase and may continue to die from ceratomyxosis, although clinical disease has not been reported from market size marine phase salmonids or in adults freshly returned to spawn. The latter show infections only in the intestinal tract, suggesting that infection is acquired upon their return to fresh water. As sexually mature adults they spread the parasite during migration in fresh water to spawning grounds.

The source of infection is unclear. Mature spores released from fish are not infective for fish. The infective stage may be an actinosporean developing in oligochaetes, but this has not been definitively determined at the present time. The development of infection and host response is temperature dependent. Development of the disease may be suppressed at temperatures lower than 6.7°C (Udey et al, 1975). The parasite penetrates the posterior intestine, and eventually pervades all layers of the entire digestive tract and spreads to most other organs. Mature spores appear 30 to 35 days post exposure, and deaths occur 38 to 42 days post exposure. Infected fish do not mount a detectable antibody response. In lesions all host tissues are replaced by parasites, and in surrounding tissues there is hyperplasia, leucocytic infiltration and necrosis. No treatments are known to be effective.

Henneguya salminicola

Henneguya salminicola is a common parasite of many species of salmonids in the Pacific north west of America (Lom and Dyková, 1995). A study of 10,414 sockeye salmon from 52 freshwater localities in British Columbia detected 1,464 infected fish (14%), although prevalence in certain localities at certain times of the year ranged up to 100%. *H. zschokkei* is a very similar, perhaps identical, parasite that occurs in salmonids and various other species of non-salmonid freshwater fishes and whitefish in Russia (Boyce et al, 1985). The two species (if they are in fact two different species) will be considered together.

H. salminicola produces cysts in the muscle of infected fish. The cysts vary from 4-15 mm in diameter, and are filled with masses of spores and debris of destroyed muscle (Boyce et al,

1985). The cyst wall is host connective tissue resulting from the host's immune system's attempts to isolate the parasite. When located in the superficial musculature the cyst may be detected externally as rounded swellings. In other cases the cysts cannot be seen externally and will not be detected until the infected flesh is exposed, such as during filleting. The cysts are white and so stand out in contrast to the pink flesh. In Pacific salmon species the majority of cysts are found in the musculature between the dorsal fin and the caudal peduncle (Boyce et al, 1985). Because of the presence of cysts, portions of the catches of salmon, particularly chum and coho, may be rendered unsuitable for marketing. On occasion, salmon infected with *H. salminicola* have been exported from Canada and the USA and cysts detected in the importing country. There are no reports of *H. salminicola* being spread in this way. However, the instances have raised commercial concerns.

Salmon are infected during their first 6 months in freshwater, and prevalence increases over the whole rearing period. *H. salminicola* distribution may be related to areas where carcasses of spawned adults accumulate and decompose. The life cycle of *H. salminicola* has not been determined, although the feeding of spores from fish probably does not cause infection in fish (Boyce et al, 1985). This probably reflects a requirement for development to occur in an intermediate host. Cyst size increases throughout life. Cysts contain proteases which probably function to breakdown host tissues for parasite metabolism. After death, these proteases are released into the flesh. Although post mortem myoliquifaction is not a consistent feature of *H. salminicola* infection, enzymatic proteolytic activity continues after death and can result in some localised degeneration or 'milky flesh'. Ultrasound examination is, at present, the only reliable means of detecting cysts in whole fish, but is not routinely used in the industry.

Kudoa sp.

Myxosporeans from the genus *Kudoa* have been reported only in fish. *Kudoa* has a wide geographical distribution, and has been found in many parts of the world. There are 34 named species of *Kudoa*, of which 22 infect musculature. Species identification is based on spore morphology (Whitaker et al, 1994). Some marine fisheries in certain areas experience heavy infections with *Kudoa* species. For example, in the early 1950s a stock of English sole suffered rejection rates of up to 21% due to *Kudoa*. The Pacific hake fishery of north west America has seen 100% of some catches infected with *Kudoa* (Whitaker et al, 1994). Although myoliquefaction (or 'milky flesh') is not reported in New Zealand Pacific hake catches, there has to date been no active surveillance for *Kudoa* in New Zealand waters, although a study is planned (pers. comm. Dr Mike Hine, NIWA, November 1995 and October, 1996).

The species known to infect salmonids is *Kudoa thyrsites,* which is found in the somatic musculature of Atlantic salmon (Harrell and Scott, 1985) and coho salmon, as well as the cardiac musculature of coho, chinook and pink salmon and steelhead trout (Whitaker et al, 1994). A recent report suggests that brown trout are also susceptible (Holliman, 1994). *K. thyrsites* is known to affect 27 species of fish in total, and is known to occur in Europe, the west coast of North America, Japan, Australia and South Africa. This broad host range and geographic distribution suggests that, in evolutionary terms, *K. thyrsites* is an old parasite, and it seems likely that *K. thyrsites* occurs throughout its susceptible hosts' geographical distribution. This would suggest that it is present in New Zealand waters.

In contrast, *K. paniformis* infects only one stock of one fish species (Pacific hake) on the west coast of North America, suggestive of a new parasite. A parasite will often have a greater impact in a relatively new host-parasite relationship than in situations in which the host and parasite have had a longer association (Whitaker et al, 1994). This generalisation holds true for *Kudoa*, as *K. paniformis* has had a devastating effect on the commercial Pacific hake industry in the north Pacific.

The infective stage and route for *Kudoa* infections of fish has not been established. The infection site is the muscle fibre, which usually is infected by one plasmodium containing numerous vegetative stages and spores, although multiple infections are occasionally seen. The parasite grows and produces proteolytic enzymes which breakdown muscle fibre filaments to provide nutrition for the parasite. While the parasite is within the muscle fibre it is undetected by the host immune system. In Pacific hake, when the parasite or enzymes reach the sarcolemma there is a swift host response and development of a fibroblast layer around the parasite and creation of a pseudocyst (Whitaker et al, 1994). In Atlantic salmon, however, there is very little host response. Muscle fibres continue to enlarge until they rupture, and at this stage limited response involving fibroplasia and inflammation is observed (Harrell and Scott, 1985). It has been suggested that infection of netpen reared Atlantic salmon in British Columbia is widespread, and that other pre-slaughter or post-slaughter factors may affect the occurrence of myoliquefaction (Ribble, 1994). Factors which may affect the distribution of *K. thyrsites* include higher sea temperatures and the presence of a wide range and high number of planktonic species such as occurs with current mixing. These factors were hypothesised as potential explanations for the occurrence of *K. thyrsites* in south-west Ireland, but not in west or north-west Ireland or Scotland and Norway (Palmer, 1994).

The spores have no direct access to the external environment, so are only released upon disintegration of the muscle tissue following death. The proteolytic activity of the enzymes leads to softening of the flesh (myoliquefaction) resulting in an unappealing appearance and texture and rejection by the consumer. The liquefaction or cysts are not detected until some days after harvest, or until after smoking. The cysts are generally fairly evenly dispersed throughout the flesh, and distinctive as white marks 5-15 mm in diameter (Palmer, 1994). If *Kudoa* is found during harvesting of fish destined for smoking, subsequent harvests from the same stock may be diverted to sale 'in the round' (Palmer, 1994). This situation reflects the international perception of *Kudoa* as a quality issue in the marketing of fish rather than a quarantine issue.

Proliferative kidney disease (PKX)

Proliferative kidney disease (PKD) is believed to be caused by a myxosporean parasite designated PKX, which is presumed but not proven to be a member of the genus *Sphaerospora*. PKD is one of the most economically important diseases among commercially reared rainbow trout in Europe and causes significant losses among Pacific salmon and rainbow trout populations in western North America (Hedrick et al, 1993). PKD affects cultured and wild fish, typically infecting fingerlings in spring, although adults may also be infected (Lom and Dyková, 1995). PKD has been the subject of periodic major reviews (Clifton-Hadley et al, 1984; Hedrick et al, 1993), from which the majority of information presented below is derived.

PKD occurs in Europe, Canada and several western states of the USA. Probably all *Oncorhynchus* and *Salmo* species are susceptible, as well as Arctic char, grayling and pike. A direct correlation between the disease and the agent has never been established. Two developmental phases of PKX have been identified in the salmonid host. The first phase, or extrasporogenic stage, occurs in the blood and kidney interstitium and provokes a chronic inflammatory response characteristic for the disease. The second developmental form is the sporogenic stage, seen in the lumina of kidney tubules 2-3 weeks after extrasporogenic stages are first observed. The sporogenic stages may be present for periods long after recovery from clinical disease. PKX is commonly detected in stained kidney imprints, tissue sections or fresh mounts using phase microscopy.

The clinical signs of PKD are variable and non-specific. In light infections there may be few external signs, and in severe outbreaks morbidity may be 100%. Darkening, exophthalmos, abdominal distension and pale gills may be seen. Secondary infections by bacteria, viruses, fungi or other protozoa may give rise to numerous other clinical signs. Internally, the most obvious lesion is renal swelling, typically accompanied by splenic enlargement and ascites. Anaemia is a common feature, but varies in severity. Histologically the kidney tissue exhibits initial mild haematopoietic hyperplasia. As the disease progresses, chronic inflammation leads to a severe granulomatous response in the kidney during the peak of disease. PKX spreads to all organs and tissues via the blood and in severe infections granulomatous changes can affect the spleen, intestine, gills, liver and muscles. Necrotising vasculitis may also be a feature in severe infections (Lom and Dyková, 1995). Sporogenic stages are present in the kidney tubules as early as 6 weeks post-exposure, and continue to be present for several months following recovery from clinical disease. Renal swelling subsides after the loss of extrasporogenic stages, and by 20 weeks post-exposure the kidney appears macro- and microscopically normal.

Infections are not directly transmissible between fish by natural means. Experimental transmission has resulted from injection of cell-free, bacteria-free filtrate from grossly infected organs of diseased fish. Hedrick et al (1992) transmitted the disease by exposure of rainbow trout to water filtered to 50μm, as well as by exposure to material captured on a 500μm screen. This finding is consistent with the hypothesis that a second host residing in sediments releases a smaller infective stage found in the water. The intensity of the immune response in salmonids is suggestive that salmonids are abnormal hosts. Survivors of PKD generally acquire immunity such that they will be resistant to further outbreaks latter in the summer or the following year. Lighter initial infections are in some cases insufficient to induce immunity. Treatments using Fumagillin DCH in feed are currently under investigation.

Other Myxosporean parasites of salmonids

Myxidium truttae infects brown trout and other salmonids throughout Eurasia, and may cause serious inflammation of bile ducts and suppurative liver necrosis in Atlantic salmon. *Myxidium salvelini* infects the urinary tissues of sockeye salmon and at least five other salmonid species in North Pacific drainages of Asia and North America (Lom and Dyková, 1995).

Chloromyxum truttae infects Atlantic salmon yearlings, and adult rainbow trout at spawning.

The site of infection is the liver. Clinical signs are anorexia, emaciation and yellow tinged skin. The gall bladder becomes hypertrophied and there is inflammation and necrosis of the liver and intestine (Lom and Dyková, 1995).

Sphaerospora truttae infects the renal tubules and Bowman's capsules of brown trout in central Europe (Lom and Dyková, 1995).

Myxobolus neurobius infects brown trout in Eurasia, accumulating in the brain and spinal cord and causing atrophy of these tissues (Lom and Dyková, 1995).

A *Parvicapsula* sp. has caused epidemics in net-pen reared coho salmon in Washington state, USA. The parasites infected renal urinary epithelium, and spores were discharged via the urinary tract. Proliferative nephritis and renal hypertrophy resulted (Lom and Dyková, 1995). Pacific cod may be a reservoir for *Parvicapsula* sp. The parasite has been detected in coho salmon at one net pen site and in wild sockeye in British Columbia (Kent, 1992).

Conclusions

1   Myxosporean parasites are economically significant parasites of wild and farmed salmonids. Geographical distribution varies between species and probably reflects the distribution of host species (both fish and oligochaete species).

2   Given their wide geographical distribution and the potential for subclinical infections, the likelihood of salmonids infected with Myxosporean parasites being harvested and processed for human consumption is high.

3   Heading gilling and gutting of salmonids during processing is likely to remove the majority of Myxosporean parasites associated with visceral tissues, including *C. shasta* and PKX.

4   *C. shasta* is only found in the Pacific north-west of America. There appears to have been no spread of infection associated with large volume exports of salmonid products for human consumption from the region.

5   In regions where PKX is endemic, fish are likely to be exposed as juveniles and survivors will have acquired immunity. This reduces the likelihood of adult fish of harvest weights being infected.

6   Heading gilling and gutting of salmonids during processing would not be expected to remove Myxosporean parasites associated with muscle tissues, including *Kudoa* sp. and *Henneguya salminicola.*

7   Filleting is a processing step which would increase the likelihood of fish heavily infected with Myxosporean parasites associated with muscle tissues being detected and downgraded during processing.

8   Myoliquefaction in fish heavily infected with *Kudoa* sp. is likely to be at an advanced

stage by the time the *commodity* is able to be placed on the market in New Zealand. This would result is disposal of the *commodity*. Inappropriate disposal of the *commodity* in New Zealand could increase the risk of parasite introduction, as well as increase the likelihood of other pathogens potentially present in the *commodity* contacting a susceptible species in New Zealand.

9   There is insufficient information available to draw conclusions on the length of time Myxosporean parasites are likely to remain viable in the *commodity.*

10   There is insufficient information available to draw conclusions on the likelihood of Myxosporean parasites infecting oligochaete alternate hosts if imported into New Zealand. Endemic infection with *Myxobolus cerebralis* indicates that species capable as acting as intermediate hosts for Myxosporean parasites exist in New Zealand.

11   Pathways for introduction other than importation of the *commodity* exist for *Kudoa* sp. The parasite infects marine fish species which are able to be imported into New Zealand for human consumption.

12   The likelihood of Myxosporean parasite introduction through importations of the *commodity* is negligible for species such as *C. shasta* and PKX.

13   The likelihood of Myxosporean parasite introduction through importations of the *commodity* is low for species such as *Kudoa* sp. and *Henneguya salminicola*. Measures to increase the likelihood of *commodity* heavily infected with these parasites being detected and disposed of in a non-risk manner prior to the *commodity* being marketed in New Zealand are likely to further reduce the risk.

4.3.8 Ciliophora (ciliates)

Lom (1995) and Dickerson and Dawe (1995) reviewed the literature on ciliates of fishes. Ciliates are the most common and widely distributed symbionts of fishes. They are transmitted directly, and are practically ubiquitous. *Ichthyophthirius multifiliis* parasitises the surface epithelium of all freshwater fishes, and occurs worldwide including New Zealand. *Cryptocaryon irritans* parasitises the surface epithelium of many marine fishes, and is also thought to occur worldwide although it has not been reported in New Zealand. Species of *Trichodina* and *Chilodonella* are known to occur in New Zealand in freshwater eels and occasionally in salmon (Boustead, 1985; MacDowall, 1990). Ciliates tend to cause disease in stressed fish, particularly in aquaculture or aquaria. Although quite a bit is known of the biology of some ciliate genera, often there is scant knowledge of species differences within a genus.

The phylum can be divided into ectoparasitic and endoparasitic groups. The ectoparasitic genera, such as *I. multifiliis, C. irritans, Chilodonella* sp., most *Trichodina* sp., *Trichodinella* sp., *Tetrahymena* sp., and *Carchesium* sp., tend to be widely distributed in aquatic environments, and non-host specific. They parasitise the gills, skin, fins and external orifices such as the nares. They are either obligatory parasites (*Chilodinella* sp., *Trichodina* sp., *Trichodinella* sp.), with no free-living stages, or facultative parasites (*Tetrahymena* sp., *Carchesium* sp.), which are free-living and opportunistically parasitise stressed fish. *I. multifiliis* and *C. irritans* cycle between an obligate fish-associated stage and a free-living reproductive stage.

I. multifiliis and *C. irritans* are obligate parasites that grow only by feeding on live fishes. They cause characteristic clinical signs, white spots on skin and gills, an inflammatory reaction and invoke an immune response.

Ectocommensal ciliates attach to fishes but derive their nutrients from the water. They tend to exert no adverse effects on the host. A heavy population of ciliates attached to the gills may cause poor oxygen exchange, but such heavy populations tend only to occur in fish stressed by some other factor. *Apiosoma* sp. and *Trichophrya* sp. are two ectocommensal ciliate species with wide geographic distribution and host range.

Endoparasitic ciliates come from three trichodinid genera, and parasitise the urinary tract of some species of marine and freshwater fishes. They initiate pathologic changes, typically hyperplasia and desquamation of the epithelium. As a rule they tend to be more host specific. Lom (1995) does not record any species regularly infecting salmonids.

Ciliates are important parasites of cultured and wild fish, including food fish and aquaria fish. Fish culture management uses a variety of management and therapeutic methods to control ciliate parasitism, in particular sourcing pathogen-free water, regular draining and sanitization of ponds, correct nutrition, oxygen balance and stocking density, regular examination of fishes for parasites, and treatments when necessary.

Conclusions

1   Ciliate parasites of fishes are economically significant parasites in the culture of fish, including salmonids.

2   Ciliate parasites are ubiquitous in aquatic environments. Ciliate parasites are known to occur in New Zealand.

3   The likelihood of salmonids parasitised by ciliate parasites being harvested and processed for human consumption is high.

4   Heading, gilling, gutting and washing during processing of salmonids is likely to significantly reduce the number of ciliate parasites associated with the *commodity*.

5   The likelihood of ciliate parasites remaining viable on the *commodity* for extended periods is low.

6   Pathways of introduction for ciliate parasites exist which are more likely than the importation of the *commodity*.

7   The risk of ciliate parasite introduction through importations of the *commodity* is negligible.

4.3.9 *Dermocystidium* **sp.**

Lom and Dyková (1992) consider *Dermocystidium* spp. to be a repository for protozoan organisms of uncertain taxonomic status. The genus contains several organisms pathogenic for salmonids, with typically widespread distribution in Europe and North America. Some species are localised in gills, such as *Dermocystidium branchialis* in the gills of brown trout in Europe, and *Dermosporidium truttae*, described once from the gills of brown trout. Other species cause systemic infection, such as *Dermocystidium* sp., which has caused mortalities in Atlantic salmon, brown and rainbow trout, and *Dermocystidium macrophagi,* which systemically invades rainbow trout and localises in blood cells.

Conclusions

1   *Dermocystidium* spp. are of relatively minor significance as pathogens of salmonids.

2   Salmonids systemically infected with *Dermocystidium* spp. are unlikely to be harvested and processed for human consumption.

3   Heading, gilling and gutting is likely to remove the majority of *Dermocystidium* spp. associated with the *commodity*.

4   The likelihood of *Dermocystidium* spp. parasites remaining viable on the *commodity* for extended periods is unknown.

5   The risk of *Dermocystidium* spp. introduction through importations of the *commodity* is negligible.

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4.4 QUALITATIVE ASSESSMENT OF THE DISEASES OF UNCERTAIN INFECTIOUS AETIOLOGY

4.4.1 Rosette agent

Rosette agent is the name given to an unclassified intracellular protistan parasite with affinities to colourless algae and fungi. It causes a severe infectious disease in chinook and Atlantic salmon in the west and east coasts of North America (Kent, 1992). The disease has occurred repeatedly at an experimental research station in Washington, USA, where it caused epidemics with mortality of up to 90% in 3 year old chinook salmon during their second summer in sea water (Harrell et al, 1986). The organism has been isolated in cell culture, and younger salmon have been experimentally infected (Elston et al, 1986). A similar organism has been reported to cause disease in fresh water-reared Atlantic salmon in California and in sea water-reared Atlantic salmon on the Atlantic coast of Canada. Infected fish are anaemic, and have enlarged spleen and kidney. Giemsa staining of spleen and kidney imprints are able to detect the organisms in clusters within macrophages. There are no known treatments.

Conclusions

1   Rosette agent is an economically significant pathogen of chinook and Atlantic salmon in Canada and the USA.

2   Because of the limited geographical distribution of the disease and the clinical signs of infection, fish clinically infected with rosette agent are unlikely to be harvested and processed for human consumption.

3   Despite widespread exports of salmonids for human consumption from regions where rosette agent is endemic, there has been no transfer of rosette agent associated with these exports.

4   Evisceration is likely to significantly reduce the amount of rosette agent in any infected fish.

5   The risk of rosette agent disease introduction through importations of the *commodity* is negligible.

4.4.2   Nervous mortality syndrome

A nervous disease of post-smolt Atlantic salmon was first observed in 1992 at certain locations in north western Ireland (Rodger et al, 1995; Scullion et al, 1996). Since it first appeared the disease has spread to a limited number of other farms in the local area. The disease is manifested in post-smolts 6-8 weeks after transfer to sea water. Initial clinical signs are lethargy, and nervous signs such as abnormal swimming patterns, loss of balance and apparent unconsciousness. Within two or three days of the initial signs mortalities start, and rise to up to 90% of post-smolt stock within 4 weeks of the onset of clinical signs. Survivors behave and grow normally, and appear not to be affected by outbreaks in subsequent years' post-smolt stocks. The pathological findings in diseased fish are confined to the brains and spinal cords. Small parasite-like organisms were observed within 4 weeks of the start of sampling, and there were multifocal areas of gliosis and microglial activation, and occasional areas of necrosis. The parasites appear similar to the extrasporogenic stages of a Myxosporidean. Oral antiprotozoan agents did not have any conclusive benefits.

Conclusions

1   Nervous mortality syndrome is an economically significant pathogen of Atlantic salmon in Ireland.

2   Because of the limited geographical distribution of the disease, the age of affected fish, and the clinical signs of infection, fish clinically infected with nervous mortality syndrome are unlikely to be harvested and processed for human consumption.

3   The risk of nervous mortality syndrome introduction through importations of the *commodity* is negligible.

4.4.3   Haemorrhagic kidney syndrome

Recently there have been reports of a new syndrome affecting farmed Atlantic salmon on the Canadian east coast that has resulted in increased morbidity and mortality in affected stocks (Byrne et al, accepted for publication 1997). The syndrome has become known as haemorrhagic kidney syndrome (HKS). The aetiology of HKS remains unresolved.

The syndrome has been manifested as increased moribund and dead 2.5-3.5 kg Atlantic salmon in sea cages on the eastern seaboard of Canada. The problem has spread from the sea cage site at which it was first detected to surrounding sites. Affected fish are lethargic and anorexic, with no external lesions. The majority of fish in affected pens appear clinically normal. Mortalities have continued at a rate of approximately 350 fish per affected pen per week. At necropsy, pathological changes were not prominent, but often included swelling and/or reddening within the kidneys, pale gills, exophthalmus and serosanguinous ascites. Anaemia is a consistent feature. Histology of the kidneys revealed pronounced interstitial haemorrhage and acute renal tubular necrosis with casting. Pathology was less consistently observed in gill, spleen, liver, peritoneum, intestine, stomach and skeletal muscle. Muscle occasionally presented with multiple random myolysis, sometimes associated with minimal haemorrhage or congestion.

Ongoing investigation is pursuing the possibility of infectious and non-infectious aetiology for HKS. No significant infectious agents have thus far been isolated. Virus isolation using standard procedures failed to isolate any viruses. Standard bacteriological evaluation of kidney tissue produced zero to low numbers of mixed colonies with no significant pathogens recovered.

BKD was present in the population as a whole, yet there was little overlap between BKD and HKS infections, and the evidence does not appear to support a theory that HKS is an unusual manifestation of BKD. Electron microscopy suggested the presence of a virus, possibly a rhabdovirus, in the renal tubular microvilli. Further virus-like particles were seen within the cytoplasm of a small number of erythrocytes. The role of these virus particles is unknown. Their size and structure resemble erythrocytic infections including erythrocytic inclusion body syndrome, although other signs are not consistent with EIBS. All tissue samples were negative for infectious salmon anaemia virus using IFAT.

Conclusions

1   Haemorrhagic kidney syndrome is a newly emerged disease of farmed Atlantic salmon in a specific region of the eastern seaboard of Canada. The disease is economically significant within its limited geographical distribution. The aetiology of HKS is unknown.

2   If an infectious agent such has been visualised in kidney tissue and erythrocytes is involved in the aetiology of HKS, bleeding, heading, gilling and gutting Atlantic salmon during processing would be likely to significantly reduce infectivity associated with the *commodity*.

3   The risk of HKS introduction through importations of the *commodity* is negligible.

4   From time to time previously unknown diseases may emerge and affect fish populations from which the *commodity* is derived. Considering the range of pathogens and diseases considered in this analysis, such an event is unlikely to affect the conclusions of this analysis.

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**4.5   QUANTITATIVE RISK ANALYSIS: MONTE CARLO SIMULATION
          MODEL**

1.   Introduction

A Monte Carlo simulation model was developed using Excel[2] and @Risk[3] to model the risk of introducing furunculosis in chilled, headless, eviscerated salmonid fish imported from a range of environments and production systems.

The model was based closely on that described by a previous MAF risk analysis (MacDiarmid, 1994), with modifications made in consultation with a professional risk analyst.

2.   Model structure

Figure 1 shows the summarised model structure. The model consists of four sections. In the first section the number of infected fish imported per tonne of imported product is calculated. The probability of introduction of disease is then modelled for three different routes of infection. The final output of the model is the sum of the probabilities of these three routes.

The routes of possible introduction of infection are:
1.   Scraps being discarded into waterways,
2.   Contaminated wrappers introducing infection into watercourses,
3.   Contaminated kitchen wastewater.

The detailed structure of the four sections of the model is shown in figures 2 - 5. Figure 2 shows the steps in the calculation of the probability that at least one infected fish will be imported into a region.

The three regions used in the model are :
1.   Upper half of the North Island
2.   Lower half of the North Island
3.   South Island

The modelling of risk on a regional basis is based on the assumption that the consumption of imported fish will be related to human population. This has an important effect on the risk of introduction of disease because the distribution of salmonid fisheries in New Zealand is almost a mirror image of the distribution of human population, and the geometry of waterways is such that water flow from North to South (i.e from higher to lower human population density) does not occur. Thus the major consumption of imported fish is expected to be in region 1, where there are almost no salmonid fisheries and therefore less risk of disease introduction.

This technique of modelling each region separately and then summing the probabilities for each region is used in each of the subsequent major areas of the model.

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[2] Microsoft

[3] Palisade

Figure 3 shows the steps for calculating the probability (risk) in each region that infection will be introduced into fish through scraps, figure 4 shows the calculations for contaminated wrapping, and figure 5 shows the steps for wastewater.

3. Changes in the model

3.1 Changes in distributions used

The major change to the model of MacDiarmid (1994) was the replacement of triangular distributions with betapert distributions, made possible by upgrades to the @RISK software since 1994. The reason for the change was that the triangular distribution usually overemphasises the tails of the distribution and underemphasises the shoulders of the distribution, resulting in more extreme values being generated thereby inflating confidence limits.

The betapert distribution is considered ideal for modelling expert opinion of the uncertainty in a variable (Vose, 1996) . It uses the same three input values as a triangular distribution (i.e. minimum, most likely, and maximum) but does not suffer to the same extent from systematic bias problems. The betapert distribution produces a systematically lower standard deviation than the triangular distribution, particularly where the distribution is highly skewed.

The LogPert distribution was used in place of the betapert for distributions which cover several orders of magnitude, based on the assumption that the expert is essentially thinking in log units.

To improve the logic of the model, binomial distributions were used in place of triangular or pert distributions in modelling the number of infected fish imported per tonne.

A separate submodel was used to model the distribution of fish weight, which obviously affects the number of fish in a tonne of imported product.

3.2 Reduction in the number of distributions

A further change was a great reduction in the number of distributions to better reflect the source of the uncertainty actually being modelled. The result of this change is that the following variables are now modelled once for all regions, rather than independently for each region as in the earlier version of the model.

Proportion of scraps and wrappers not incinerated.
(P2) rl = (P2) r2 = (P2)r3 = (P4)rl = (P4)r2 = (P4)r3

Proportion of scraps and wrappers not buried
(P3)rl = (P3)r2 = (P3)r3 = (P5)r1 = (P5)r2 = P(5)r3

Probability that scraps contaminate water
(R6)rl = (R6)r2 = (R6) r3 = (R8)rl = R(8)r2 = (R8)r3 = (R10)r1 = (R10)r2 = (R10)r3

Scraps infect susceptible host when present
This is modelled once for fresh and estuarine water in all regions, but is modelled separately for sea water.
(R7)rl = (R7)r2 = (R7)r3 = (R9)rl = (R9)r2 = (R9)r3
and
(R11)rl = (R11)r2 = (R11)r3

Wrappers contaminate water
(R13)rl = (R13)r2 = (R13)r3 = (R15)rl = (R15)r2 = (R15)r3 = (R17)rl = (R17)r2 = (R17)r3

Wrappers infect susceptible host when present
(R14)rl = (R14)r2 = (R14)r3 = (R16)rl=(R16)r2=(R16)r3 = (R18)rl = (R18)r2 = (R18)r3

Probability that wrapper is contaminated given that fish are infected
(R12)rl = (R12)r2 = (R12)r3

Probability that kitchen wastewater will be contaminated given that fish are infected
(R19)rl = (R19)r2 = (R19)r3

Wastewater infects susceptible host when present
This is modelled once for fresh and estuarine water in all regions, but is modelled separately for sea water.
(R22)rl = (R24)rl = (R22)r2 = (R24)r2 = (R22)r3 = (R24)r3
and
(R26)rl = (R26)r2 = (R26)r3

The effect of the above changes was to reduce the number of distributions by 52.

4.   Data used

The model was applied to three scenarios which broadly represent the range of possible importations:
- farmed Atlantic salmon from Norway
- wild Pacific salmon from the Pacific Northwest of America
- farmed rainbow trout from Denmark

Two data items were necessary to adapt the model to each scenario: prevalence of infection in harvested fish and fish weight. All other input variables remained the same as those used by MacDiarmid (1994), assuming:
a)   evisceration is as effective in reducing risk in Atlantic salmon and trout as it is in wild ocean-caught Pacific salmon
b)   inspection, grading and washing act to reduce risk in a similar manner as they were estimated to act for wild ocean-caught Pacific salmon from Canada

i)   prevalence of infection in harvested fish

Estimates of infection prevalence are based on surveillance data obtained from fish health

authorities in potential exporting countries. Such data are limited. Moreover, such data can be expected to over-estimate the infection prevalence in commercially harvested fish populations, as aquatic animal health surveillance is generally targeted at fish from which an infectious agent is most likely to be isolated, including spawning fish and fish exhibiting signs of infectious disease. Surveys of fish harvested for human consumption are rarely carried out.

In order to provide a conservative estimate of the disease risk, available data were interpreted so as to overstate rather than understate the infection prevalence in commercially harvested fish.

ii)   distribution of fish weight

Fish weight is an important variable in the model. The final output is disease risk per tonne of imported product, and that is determined to a large extent by the number of infected fish that are imported. Therefore, the smaller the average fish weight, the more individual fish would be present in a tonne of product, and the more infected fish at any given infection prevalence.

The average live weight of each population group at harvest was estimated using available data and expert opinion. In each scenario, the effect of heading and evisceration was assumed to be the same as that estimated by MacDiarmid (1994) for wild ocean-caught Pacific salmon. The only difference was that this time the effect was modelled using a betapert rather than a triangular distribution. The parameters for the distribution were:

> Minimum :   65%
> Most likely :   71%
> Maximum :   73%

Thus, the proportion of a fish remaining after heading and evisceration was modelled as :
> betapert (0.65, 0.71, 0.73)

4.1   Farmed Atlantic salmon from Norway

4.1.1   *A. salmonicida* prevalence

A 1991 study of 124 randomly selected sea water netpen sites in Norway found *A. salmonicida* on 67 (54%) of those farms (Jarp et al, 1994). Since that time, vaccination with oil based vaccines has significantly reduced the number of farms diagnosed as infected with *A. salmonicida*. In 1996 only two farms were diagnosed as infected with *A. salmonicida* out of 600 farms in Norway (T. Håstein, pers. comm. with M. Stone, March 1997).

As data on *A. salmonicida* prevalence is the result of either random surveys or routine active surveillance, where bacterial culture is the diagnostic criterion, these data give a reliable estimate of the presence of infection including the carrier state.

One area of uncertainty remaining is the prevalence of *A. salmonicida* on infected farms. Data were not available on this, and it was not possible to elicit expert opinion on it. Therefore a number of assumptions had to be made to get estimates of prevalence for the model.

Current prevalence of *A. salmonicida* is assumed to be:
a)   at least as high as in 1996, assuming 10% prevalence on infected farms
b)   most likely around the prevalence in 1996, assuming 100% prevalence on infected farms
c)   not higher than the farm level prevalence in 1991, assuming 100% prevalence on infected farms

To use the existing data in the model, two further assumptions were made:
a)   all farms are approximately the same size
b)   all farms are contributing approximately the same proportion of the total amount of salmon exported from Norway.

From the above raw data, the following estimates of *A. salmonicida* prevalence were made for use in a betapert distribution in the model:

Minimum P :   0.001
Most likely P : 0.01
Maximum P :   0.54

4.1.2   Fish weight

Weight of slaughtered fish from 1987 to 1995 according to information from the Kontali advisory company in Norway (T. Håstein, pers. comm. with M Stone, March 1997) are shown in table 1.

Table 1. Average weight of harvested Atlantic salmon from Norway, by year

Year	No slaughtered fish in (millions)	Average weight slaughtered fish (kg)
1987	29. 4	3. 13
1988	55. 3	3. 20
1989	49. 3	3. 16
1990	40. 7	3. 19
1991	33. 7	3. 48
1992	43. 3	4. 15
1993	53. 7	4. 27
1994	67. 7	4. 13
1995	81. 0	3. 75

There appears to have been a general increase in weights over the past 5 years, which probably reflects a production trend. The average of the average weights over the past 5 years is 3.95 kg. This was used as the most likely average weight.

The values chosen from the above table for the model were:

> Minimum K :   3.13
> Most likely K: 3.95
> Maximum K : 4.27

This was modelled as betapert (3.13, 3.95, 4.27), which gave a mean number of 368 fish per tonne (standard deviation = 1.106). It was converted to a binomial distribution for the main model:

> Binomial (n, p)
>> where n = 369
>>> and p = 0.907821

4.2   Wild Pacific salmon from the Pacific Northwest of America

4.2.1   *A. salmonicida* prevalence

Two scenarios for prevalence of *A. salmonicida* in wild Pacific salmon from the Pacific Northwest of America were modelled: one being a "worst case" scenario reflecting the prevalence of infection in spawning salmon which have returned to fresh water; and the second being a more realistic scenario reflecting the prevalence of infection in Pacific salmon which are harvested commercially.

MacDiarmid (1994) made the point that the prevalence of *A. salmonicida* in spawning fish is far higher than could be reasonably expected to be found in fish commercially harvested for the fresh/frozen trade. This is because spawning fish will have been subjected to physiological stress associated with adaptation to fresh water, sexual maturation, crowding in waterways, and exposure to the higher levels of infection in that environment.

Harvesting of wild Pacific salmon begins when the fish move into coastal waters prior to moving up rivers to spawning grounds. Most fish are caught by purse seining or trolling at sea near coasts or in inlets to bays, and some are caught by set nets from shore close to river months. Some are harvested as they arrive at hatcheries, although these fish are normally not processed for export trade. Most harvesting is done in June and July, and spawning takes place from September to November.

Spawning Pacific salmon

Between 1985 and 1994, 235 (10%) of 2,331 cases of sexually mature wild Pacific salmon examined by the Pacific Biological Station, Nanaimo, were positive for *A. salmonicida* (T. Evelyn, pers. comm. with M. Stone, October 1996). This compares to the *A. salmonicida* prevalence of 6% in spawning salmon (1,298 / 21,495) used by MacDiarmid (1994) as maximum infection prevalence to model Canadian salmon imports.

The survey data were used in a beta distribution to model prevalence in fish harvested at spawning:

> beta (α_1, α_2)
>> where α_1 = positives + 1
>> α_2 = samples - positives + 1

giving   beta (235 + 1, 2331 - 235 + 1)
or   beta (236, 2097)

A beta (236, 2097) distribution has the following characteristics:

Minimum : 8.6×10^{-2}
Mean :   0.1
Maximum : 0.12

The use of spawning salmon prevalence data to model the *A. salmonicida* prevalence in commercially harvested fish would considerably overstate the disease risk for reasons stated earlier.

Commercially harvested Pacific salmon

MacDiarmid (1994) cited a survey carried out by Canadian Department of Fisheries and Oceans researchers which sampled 600 wild salmon before returning to fresh water. None of these fish were found to be infected with *A. salmonicida*. As MacDiarmid suggested, these fish are more representative of wild ocean-caught Pacific salmon harvested for export than spawning salmon.

These data were modelled as a beta distribution in the model.

In this case $\alpha_1 = 0 + 1 = 1$
                $\alpha_2 = 600 - 0 + 1 = 601$

giving beta (1, 601)

A beta (1, 601) distribution has the following characteristics:

Minimum : 7.6×10^{-7}
Mean : 1.7×10^{-3}
Maximum : 1.2×10^{-2}

This distribution is considered the most appropriate for use in modelling the *A. salmonicida* risk from commercially harvested salmon, as it is based on specific survey data of that population of fish.

4.2.2   Fish weight

For Pacific salmon, fish weight was modelled using a betapert distribution using the same input data as MacDiarmid (1994) i.e.

Minimum :   1.5 kg
Most likely : 2.5 kg
Maximum :   5.0 kg

This gave a mean number of 517 fish per tonne (standard deviation 5.13) which was converted to a binomial for the main model :

Binomial (n, p)

where n = 545
and p = 0.9496

4.3 Farmed rainbow trout from Denmark

4.3.1 *A. salmonicida* prevalence

Rainbow trout have lower natural susceptibility to *A. salmonicida* than other salmonid species, and the pathogen causes fewer clinical and production problems during rainbow trout aquaculture. Rainbow trout can, however, become infected with *A. salmonicida*.

The only surveillance data on *A. salmonicida* infection in farmed rainbow trout that were obtained comes from a survey in Denmark during 1995 (N. Olesen, pers. comm. with M Stone, October 1996). The survey visited farms and sampled 54 fish that were observed displaying clinical signs of ill-health. These fish were subjected to microbiological examination, and 15 (27.8%) of them were found to be positive for *A. salmonicida*.

No formal sampling regime was employed during the survey, and information on where sampled fish originated cannot be obtained. However, we know that fish exhibiting clinical signs of ill-health were targeted. This undoubtedly increases the chances of diagnosing *A. salmonicida*, as even if the organism is not the primary cause of ill-health, ill-health will increase susceptibility to infection from any *A. salmonicida* in the environment of the farm.

To use these data in the model, the following assumptions were made:
a)   the 54 fish were collected from 54 different farms;
b)   15 out of 54 provides an estimate of the prevalence of *A. salmonicida* infected farms in Denmark;
c)   all farms are contributing approximately the same proportion of the total amount of trout exported from Denmark;
d)   prevalence of *A. salmonicida* infected fish on infected farms were at worst 100%, most likely 50% and at best 10%.

With these assumptions, the above raw data were used in the model as follows:
Minimum P :   0.028 (2.8%)
Most likely P : 0.139 (13.9%)
Maximum P :   0.278 (27.8%)

Thus, prevalence was modelled as betapert (0.028, 0.139, 0.278).

The values are based on the very conservative but unlikely assumptions that a survey targeting sick fish has provided an estimate of *A. salmonicida* prevalence in the general population, and that an infection rate of 100% might occur on infected farms. The values are higher than those used to model ocean-caught and spawning wild Pacific salmon and the minimum and most likely values for farmed Atlantic salmon. This is despite the already mentioned fact that rainbow trout are less susceptible to *A. salmonicida* infection than either of these species.

Therefore, this distribution may be considered to be a worst case scenario.

4.3.2 Trout weight

Very little data were available on trout weight. Rainbow trout are harvested at around 1 kg in Irish aquaculture (L. Gronberg, pers. comm. with M Stone, June 1995), while trout in the USA are harvested at weights as low as 250 g for the portion-sized market (Jansen and McLeary, 1996).

Using this information, trout weight was modelled in the separate submodel by a betapert distribution with the following parameters:

$$
\begin{aligned}
&\text{Minimum :} \quad 0.25 \text{ kg} \\
&\text{Most likely :} \quad 0.8 \text{ kg} \\
&\text{Maximum :} \quad 1.1 \text{ kg}
\end{aligned}
$$

This gave mean number of 1875 fish per tonne (standard deviation 9.2) which was converted to a binomial for the main model :

$$
\begin{aligned}
&\text{Binomial (n, p)} \\
&\text{where } n = 1964 \\
&\quad \text{and } p = 0.9549
\end{aligned}
$$

5.   Results

For each scenario, the results from 1000 iterations of the model were collected by the @Risk software, and moved to an Excel spreadsheet for analysis and charting.

5.1   Distribution of model output

The mean probability that infection would be introduced ranges from 5×10^{-10} per tonne for commercially harvested Pacific salmon from the Pacific coast of North America, to 5×10^{-8} per tonne for farmed trout from Denmark. The results indicate with 99% confidence that the risk of introduction of *A. salmonicida* infection in any of these commodities is less than 10^{-7} per tonne of commodity imported.

Table 2 shows the summary output statistics for each of the four commodity scenarios.

Figures 6-9 are cumulative frequency distributions of the model output for each of the four commodity scenarios.

Table 2. Model Output: probability of *A. salmonicida* introduction per tonne of commodity imported (in all cases chilled, headless, eviscerated fish).

	Commercially harvested Pacific salmon from North America	Spawning Pacific salmon from North America	Farmed Atlantic salmon from Norway	Farmed rainbow trout from Denmark
Minimum	6.82×10^{-14}	1.07×10^{-9}	6.27×10^{-11}	6.55×10^{-9}
Mean	5.31×10^{-10}	2.19×10^{-8}	1.56×10^{-8}	4.98×10^{-8}

Standard deviation	8.24×10^{-10}	1.49×10^{-8}	1.49×10^{-8}	2.76×10^{-8}
90th percentile	1.31×10^{-9}	4.09×10^{-8}	3.35×10^{-8}	8.56×10^{-8}
95th percentile	1.84×10^{-9}	5.12×10^{-8}	4.48×10^{-8}	1.0×10^{-7}
99th percentile	4.15×10^{-9}	7.65×10^{-8}	6.64×10^{-8}	1.43×10^{-7}
Upper 95% confidence limit	2.27×10^{-9}	6.24×10^{-8}	5.48×10^{-8}	1.15×10^{-7}

5.2   Sensitivity analysis

Figures 10-13 are sensitivity analyses for the four scenarios modelled. These figures show that the uncertainty in model output is most strongly influenced by either the prevalence or the wastewater sections of the model.

Prevalence has more effect in the commercially harvested Pacific salmon and farmed Atlantic salmon scenarios i.e where prevalence is low, changes in prevalence have the largest effect on the model outcome. On the other hand, where prevalence is known or assumed to be high, such as in the scenarios for farmed trout and for spawning Pacific salmon, further changes to prevalence have little effect on model output. In that case changes to variables in the wastewater section produce the greatest changes in output.

6.   Discussion

The results of the simulation model show that there is little risk of introducing *A. salmonicida* in any of the four commodities considered.

The lowest risk of *A. salmonicida* introduction is that posed by commercially harvested Pacific salmon from North America, a result which is based on realistic prevalence data from surveillance on that specific population of fish. However, the risk for spawning Pacific salmon, which is based on prevalence estimates considerably higher than could be expected in commercially harvested Pacific salmon, is only half an order of magnitude higher.

Even where very conservative assumptions were made to enable the use of scant or incomplete surveillance data for farmed Atlantic salmon from Norway and farmed rainbow trout from Denmark, the risk is less than 1×10^{-7}.

7.   Conclusion

Whereas qualitative risk assessment concluded that the risk of *A. salmonicida* introduction through importations of the *commodity* is low, quantification of the risk further demonstrates that such an event is unlikely to occur, particularly given the relatively low annual volumes which are likely to be imported.

Figure 1. Summary of Model Structure

D

1. PREVALENCE
Diseased fish
entering 3 regions

I_1

2. SCRAPS
Risk of introducing
disease by scraps in
each of 3 regions

I_2

3. WRAPPERS
Risk of introducing
disease by contaminated
wrappers in each of 3
regions

I_3

4. WASTEWATER
Risk of introducing disease
in contaminated
wastewater in each of 3
regions

MODEL OUTPUT
Risk of introducing
disease into NZ

$I_1 + I_2 + I_3$

Figure 2. Prevalence

Figure 3. Scraps

R_6

Probability that scraps contaminate fresh water #

$(D)_r$

Probability that at least one diseased fish will be imported into region

R_8

Probability that scraps contaminate estuarine water #

R_{10}

Probability that scraps contaminate sea water #

Sp

Proportion of NZ fisheries located in the region #

P_1

Propotion of imported fish disposed of as scraps #

R_7

Probability that scraps will infect a susceptible host in fresh water #

R_9

Probability that scraps will infect a susceptible host in estuarine water #

R_{11}

Probability that scraps will infect a susceptible host in sea water #

S_1

Probability that scraps introduce infection into freshwater $(D)r * P_1 * P_2 * P_3 * R6 * Sp * R7$

S_2

Probability that scraps introduce infection into estuarine water $(D)r * P_1 * P_2 * P_3 * R8 * R9$

S_3

Probability that scraps introduce infection into sea water $(D)r * P_1 * P_2 * P_3 * R10 * R11$

P_2

Proportion of scraps not incinerated #

P_3

Proportion of scraps not buried #

P_1 → P_2 → P_3 →

P_1 → P_2 → P_3 →

$(I_1)r$

Probability that infection is introduced into region by scraps $S_1 + S_2 + S_3$

(I)

Probability that infection is introduced into NZ fish by scraps $(I1)r1 + (I1)r2 + (I1)r3$

\# = Distribution defined from expert opinion

Figure 4. Wrappers

R$_{13}$

| Probability that wrappers contaminate fresh water # |

(D)r | Probability that at least one diseased fish will be imported into region |

R$_{15}$

| Probability that wrappers contaminate estuarine water # |

R$_{17}$

| Probability that wrappers contaminate sea water # |

Sp

| Proportion of NZ fisheries located in the region # |

R$_{12}$ | Propotion of imported wrappers contaminated # |

R$_{16}$

| Probability that wrappers will infect a susceptible host in estuarine water # |

R$_{18}$

| Probability that wrappers will infect a susceptible host in sea water # |

R$_{14}$

| Probability that wrappers will infect a susceptible host in fresh water # |

W$_1$

| Probability that wrappers introduce infection into freshwater (D)r * R12 * P$_2$ * P$_3$ * R13 * Sp * R14 |

(D)r
R$_{12}$
P$_2$
P$_3$

W$_2$

| Probability that wrappers introduce infection into estuarine water (D)r * R$_{12}$ * P$_3$ * P$_3$ * R15 * R16 |

(D)r
R$_{12}$
P$_2$
P$_3$

W$_3$

| Probability that wrappers introduce infection into sea water (D)r * R12 * P$_2$ * P$_3$ * R17 * R18 |

P$_2$ | Proportion of wrappers not incinerated # |

P$_3$ | Proportion of wrappers not buried # |

I$_2$r

| Probability that infection is introduced into region by wrappers W$_1$ + W$_2$ + W$_3$ |

I$_2$

| Probability that infection is introduced into NZ fish by wrappers (I1)r1 + (I1)r2 + (I1)r3 |

= Distribution defined from expert opinion

Figure 5. Wastewater

(D)r — Probability that at least one diseased fish will be imported into region

DF — Proportion of sewage discharged directly into freshwater

Sp — Proportion of NZ fisheries located in the region #

R22 — Probability that contaminated waste water will infect susceptible host in fresh water #

WW1 — Probability that wastewater introduces infection into freshwater (D)r * R19 * SW * DF * R22 * Sp

P2 — Probability that kitchen wastewater is contaminated given that fillets are infected #

P3 — Proportion of sewage not treated adequately #

DE — Proportion of sewage discharged directly into estuarine water #

R24 — Probability that contaminated wastewater will infect a susceptible host in estuarine water #

WW2 — Probability that wastewater introduces infection into estuarine water (D)r * R19 * SW * DE * R20

DS — Proportion of sewage discharged directly into sea water #

R26 — Probability that contaminated sewage will infect a susceptible host in sea water #

WW3 — Probability that contaminated wastwater introduces infection into sea water (D)r * R19 * SW * DS * R26

(D)r
R12
SW

(D)r
R12
SW

I3r — Probability that infection is introduced into region by wastewater WW1 + WW2 + WW3

I3 — Probability that infection is introduced into NZ fish by wastewater I3r1 + I3r2 + I31r3
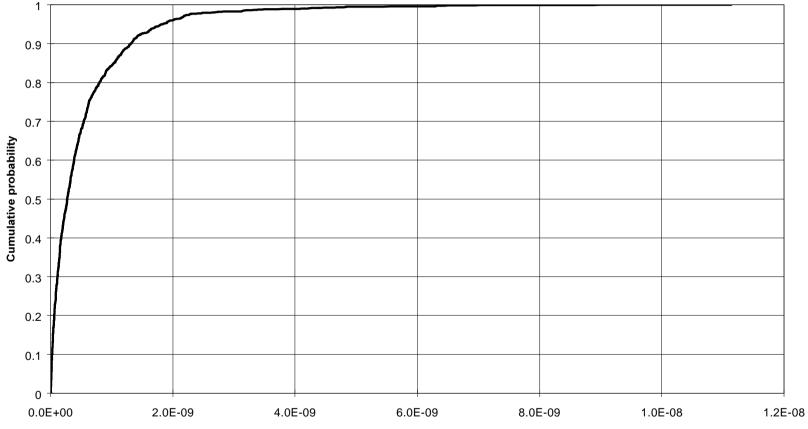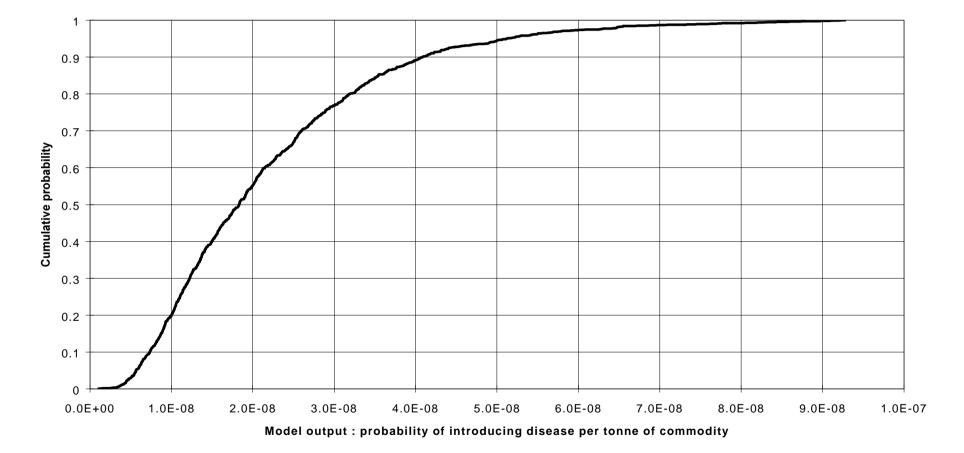
= Distribution defined from expert opinion

Figure 6. Commercially harvested wild Pacific salmon from North America : cumulative distribution of output probability
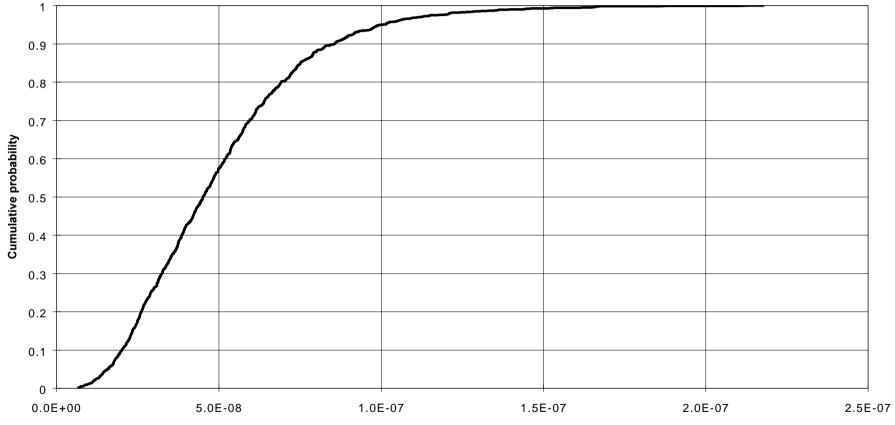
Cumulative probability

Model output : probability of introducing disease per tonne of commodity

Figure 7. Spawning wild Pacific salmon from North America : cumulative distribution of output probability

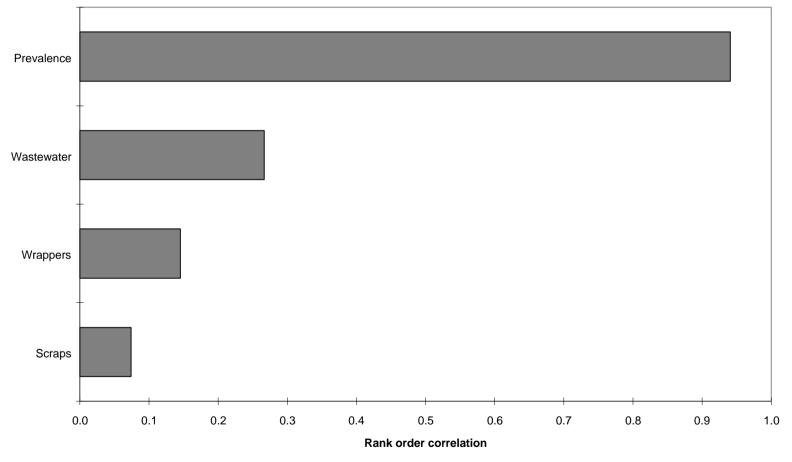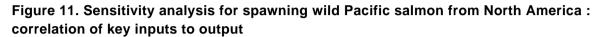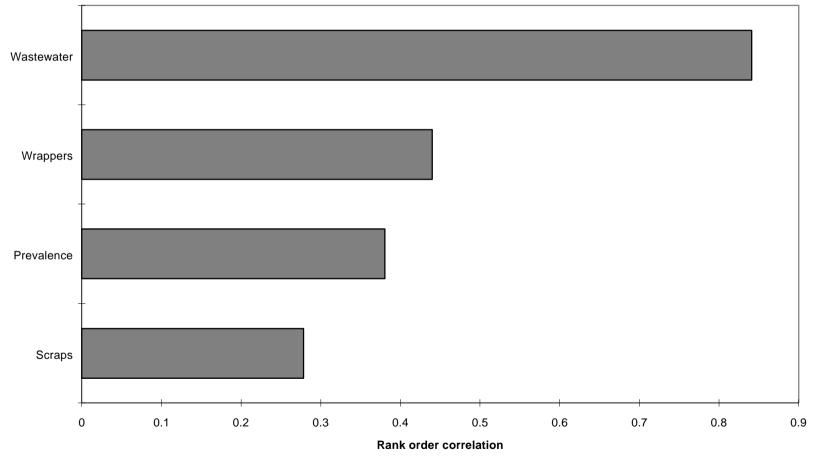**Figure 9. Farmed rainbow trout from Denmark : cumulative distribution of output probability**

Figure 10. Sensitivity analysis for commercially harvested wild Pacific salmon from North America : correlation of key inputs to output
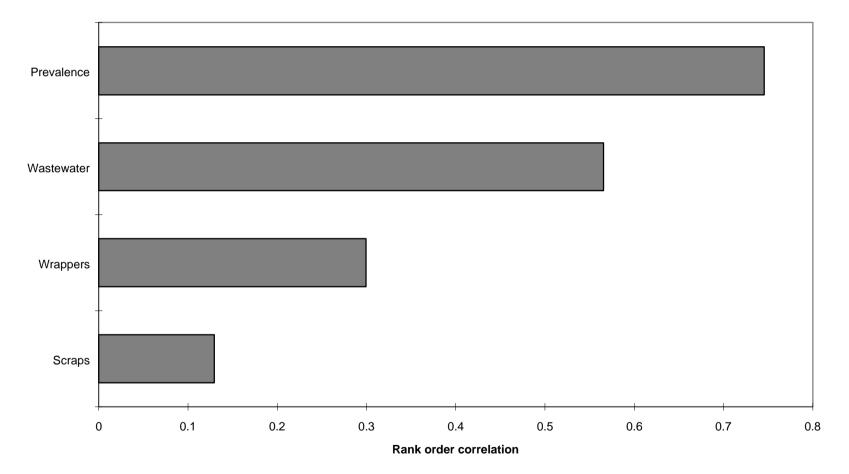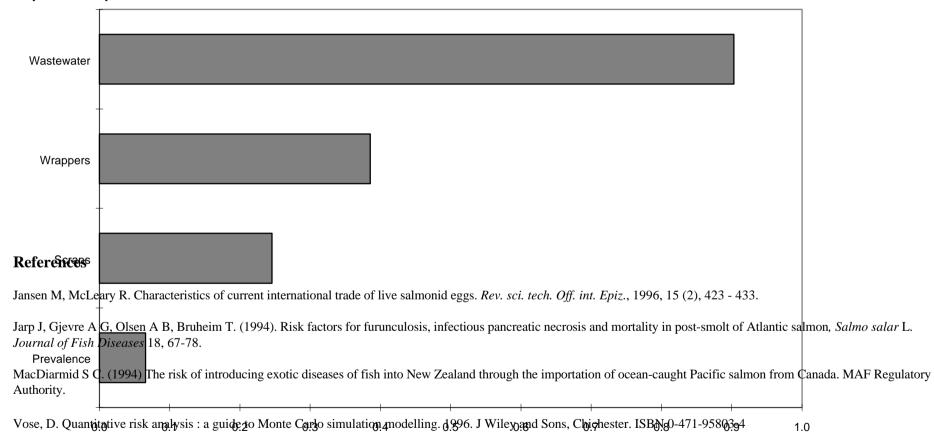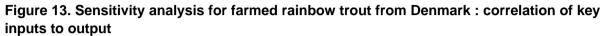
**Figure 11. Sensitivity analysis for spawning wild Pacific salmon from North America :
correlation of key inputs to output**

Figure 12. Sensitivity analysis for farmed Atlantic salmon from Norway : correlation of key inputs to output

Rank order correlation

Figure 13. Sensitivity analysis for farmed rainbow trout from Denmark : correlation of key inputs to output

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4.6   QUANTITATIVE RISK ASSESSMENT: ACTUAL HISTORICAL DATA

A discrete exposure process is characterised by the probability p of an event occurring at each trial. By examining historical data on salmon imports in situations where there have been no introductions of disease, it is possible to define the distribution of the probability of a disease introduction with each tonne imported. This produces a conservative estimate since it assumes that the possibility of a disease introduction does exist (Vose, 1997) and, even more conservatively, that prior to analysing the data, we would have considered that the probability of disease introduction could lie equally anywhere between 0 and 1.

The probability of a disease introduction occurring is modelled using the Beta distribution. This distribution returns the true probability p of an event (disease introduction in this case) happening, given that n trials gave been completed (that is, tonnes imported) and s "successes" (that is, disease introductions) observed.

$$p \quad = \text{Beta} (\propto_1, \propto_2)$$

Where
$$\propto_1 \quad = r + 1$$
$$\propto_2 \quad = n - r + 1$$
r   = number of disease introductions which have occurred.
n   = number of tonnes imported.

The approach used here makes an approximation in that it treats importation of tonnes of salmon as a discrete process when it is actually a continuous one. However, the approximation is an extremely close one where the units used mean we have a large number of trials, as in the examples cited here.

Surveillance and monitoring have confirmed that New Zealand salmonid populations are free from furunculosis, bacterial kidney disease (BKD) and infectious haematopoietic necrosis (IHN), all of which occur in British Columbia.

Between 1966 and 1983 just over 3,000 tonnes of Pacific salmon were imported into New Zealand from Canada (pers. comm. Iola Price, Director, Aquaculture and Oceans Science Branch, Department of Fisheries and Oceans, Canada). This period was largely prior to the establishment of salmon aquaculture in British Columbia, so this data is likely to represent importations of wild Pacific salmon. From these data it is possible to calculate the probability that a tonne of salmon could introduce one of these three diseases. By entering the data for disease introductions actually experienced (0) and tonnes of product actually imported (3,000) into the formula for a Beta distribution, using the software program @RISK, estimates for p are obtained.

For furunculosis, BKD and IHN, the risk of a disease introduction into New Zealand, based on actual historical data is less than;

Upper 95% confidence limit

New Zealand 1.23×10^{-3} per tonne

It should be borne in mind that this is a *pessimistic* risk estimate. Much larger volumes of table salmon have been exported from British Columbia into countries in which surveillance and monitoring have confirmed freedom from various significant diseases of salmon.

For example, between 1979 and 1991 the following tonnages of frozen, whole, dressed salmon (presumably including some farmed salmon) were exported to;

	Tonnes
Denmark	13,139
France	54,889
United Kingdom	9,454
Japan	117,936

(Data courtesy of Iola Price, Director, Aquaculture and Oceans Science Branch, Department of Fisheries and Oceans, Canada).

Surveillance has confirmed that IHN is not present in Denmark or the United Kingdom (OIE, 1995). From the data above, the probability of IHN being introduced per tonne of salmon imported can be calculated as less than;

	Upper 95% C.L.
Denmark	2.80×10^{-4}
United Kingdom	3.90×10^{-4}

The estimates for the United Kingdom are higher than the Danish ones because the volume of salmon imported into the United Kingdom was smaller.

All four countries are free from the disease ceratomyxosis. From the actual historical data estimates of the probability of introducing this disease can be calculated as less than;

	Upper 95% C.L.
Denmark	2.80×10^{-4}
France	6.71×10^{-5}
United Kingdom	3.90×10^{-4}
Japan	3.12×10^{-5}

Again, it must be recalled that these are all pessimistic risk estimates because they are based on the assumption that a disease introduction is possible. If data are pooled, for example by combining the tonnages exported to Denmark and the United Kingdom (22, 593 tonnes) calculation of the risk of introducing IHN per tonne gives the following estimate;

	Upper 95% C.L.
Denmark and the United Kingdom	1.63×10^{-4} per tonne

Infectious salmon anaemia (ISA) is a viral disease of Atlantic salmon which has never occurred outside Norwegian waters, despite large volumes of salmon having been exported from Norway. Between 1990 and 1995 Norway exported 1,014,976 tonnes of gutted Atlantic salmon and rainbow trout (pers. comm. Tore Håstein, Norwegian State Veterinary Laboratory). From these data one can calculate that the risk of spreading the viral disease ISA through trade in table salmon is less than;

	Upper 95% C.L.
All countries importing Norwegian salmon	3.60×10^{-6} per tonne

Of course, not all countries to which Norwegian salmon is exported have significant Atlantic salmon stocks, and ISA does not cause disease in any other species. Furthermore, not all importing countries have surveillance and disease monitoring programs. It is appropriate, therefore, to focus on countries with significant Atlantic salmon stocks and disease surveillance capability, such as the United Kingdom.

Since 1990 (the peak of the ISA epidemic in Norway) 29,142 tonnes of salmon were exported from Norway to the United Kingdom. From these historical data one can calculate that the risk of introducing ISA is less than;

	Upper 95% C.L.
United Kingdom	1.26×10^{-4}

Surveillance and monitoring programmes which are operated in Australia confirm that country's freedom from as many as 24 diseases of salmonids which are present in Canada (DPIE, 1996). Amongst these diseases are furunculosis, BKD and IHN. Between 1966 and 1975 Australia imported 76,655 tonnes of frozen, whole, dressed salmon from Canada (Data from Appendix 5 of the May 1995 draft *Import risk analysis,* Australian Quarantine and Inspection Service). This period was largely prior to the establishment of salmon aquaculture in British Columbia, so this data is likely to represent importations of wild Pacific salmon. From these historical data one can calculate that the risk of introducing furunculosis, BKD and IHN, and, indeed, a number of other diseases of salmonids is less than;

	Upper 95% C.L.
Australia	4.81×10^{-5} per tonne

It should be borne in mind that these are *pessimistic* estimates and the actual risk could be orders of magnitude smaller. However, as these risks have been calculated from actual historical data, the risks are unlikely to be greater. Indeed, if a logical examination of biological factors and fish processing systems had led one, prior to analysing the available data, to consider that the probability of disease introduction was likely to be small (that is, *not* equally likely to be anywhere between 0 and 1), one could have employed other techniques which would give upper confidence limits smaller than those shown by the Beta distribution.

The data presented above and the calculations made from them support the contention that there is little risk that significant diseases of salmonids can be spread through the trade in gutted salmon.

References

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4.7   RISK ASSESSMENT: VOLUME OF TRADE

The volume of the *commodity* likely to be imported if granted market access into New Zealand forms an important part of the risk assessment. This is because quantitative risk assessment methods express risk in terms of disease introductions per unit of product imported. The risk of a disease introduction is directly proportional to the volume of the *commodity* imported.

The volume of the *commodity* likely to be imported if market access is granted may be assessed through the following means:

1   Examining data on imports of salmon products prior to the prohibition in 1983;

2   Examining data on imports of wild, ocean-caught, Pacific salmon products from Canada since market access was granted in June 1995;

3   Examining data on consumption of salmon products in New Zealand;

4   Estimating the demand for products other than salmon products included in the definition of the *commodity*.

Importations of salmon products prior to the prohibition of imports in 1983

The Department of Fisheries and Oceans of Canada has provided data on exports to New Zealand of salmon products, other than canned, prior to the prohibition of imports in 1983. These data appear as Table 1. No other countries are known to have exported significant quantities of un-canned salmonid products to New Zealand. The Canadian data are considered to provide an indication of the demand for imported salmon products in this period. The amount of product annually imported from Canada between 1966 and 1983 ranged between 0 and 1070 tonnes. The average volume imported annually in this period was 167 tonnes, and the median for the period was 9 tonnes. No salmon was imported in 9 of the 18 years in this period.

The period these data cover is prior to the establishment of a salmon aquaculture industry in New Zealand. Thus, the demand for uncooked salmon products in New Zealand was, at that time, being met through importations. Imported salmon products today compete against locally produced salmon products, and this almost certainly decreases demand for imports. Conversely, in the period examined the New Zealand public were probably not conditioned towards salmon products being a readily available food commodity.

Importations of wild, ocean-caught, Pacific salmon products from Canada since market access was granted in June 1995

In June 1995 MAF issued an import health standard allowing the importation of fresh or frozen wild, ocean-caught, Pacific salmon from Canada. The import health standard classifies product according to whether or not it is commercially packaged for direct retail sale. Product

commercially packaged for direct retail sale may be issued biosecurity clearance at the border allowing importers to distribute it without MAF's regulatory intervention. Product not commercially packaged for direct retail sale, typically boxes of frozen, headed and gutted fish, must be processed in premises under MAF regulatory control prior to being distributed in New Zealand (see section 5 Risk Management).

Since June 1995 MAF has issued five import health permits (IHP) under this import health standard. Three IHPs have been issued for very small quantities of product commercially packaged for direct retail sale imported as samples by retailers. Two IHPs for product not commercially packaged for direct retail sale, totalling 40 tonnes, have been issued. These IHPs were both issued to the owner of the single MAF registered premises for processing imported salmon. All permits were issued in the 9 month period following the import health standard becoming available. There have been no permits issued since February 1996. Further, no retail company that imported samples of product went on to apply for permits for commercial quantities of product. This indicates that the imported product did not compete well against domestically produced salmon products.

As the wholesale price for wild salmon is less than aquacultured salmon in markets such as North America where both are freely available, it seems unlikely that imported aquacultured salmon products would retail for less than imported wild salmon products. The success of aquacultured New Zealand salmon in international markets indicates it is of comparable quality to overseas aquacultured salmon. The failure of imported wild salmon to compete and displace New Zealand aquacultured salmon indicates that imported aquacultured salmon would similarly be unlikely to compete and displace New Zealand aquacultured salmon to any great extent.

Consumption of salmon products in New Zealand

The previous MAF salmon risk analysis estimated that between 289 and 459 tonnes of locally produced salmon products were consumed in New Zealand annually (MacDiarmid, 1994). This estimate was made by assuming that between 10% and 15% of locally produced product was consumed within New Zealand. The amount of product exported in 1992 was 2,600 tonnes.

The New Zealand King Salmon Co. Ltd forecasts production of 5,300 tonnes (gilled and gutted weight) in the 1996/97 season, comprising 80% of total New Zealand production. The New Zealand King Salmon Co. Ltd estimates that sales in New Zealand will total 23% of production (New Zealand King Salmon Co. Ltd, 1996). If other salmon producing companies have similar marketing strategies (i.e. similar proportion of total production marketed domestically) the forecast annual New Zealand consumption of locally produced salmon products would be 1523.75 tonnes. This value is significantly higher than estimated by MacDiarmid as a result of the large increase in domestic salmon production, and a higher estimate of the proportion sold in New Zealand.

The 40 tonnes of wild, ocean-caught, Pacific salmon from Canada imported in the last 23 months represents a current market share of approximately 1.3% for imported salmon products

of the total amount of salmon consumed in New Zealand annually [estimated locally produced and consumed salmon + imported salmon].

In the United Kingdom farmed Atlantic salmon is consumed at a rate 1 tonne/1000 people/year and is increasing at 10-15% annually (pers. comm. Alan Munro, July 1997). If this level of consumption was achieved in New Zealand, over 3,000 tonnes of salmon would be required to support it. This is twice the present level of domestic consumption. The figure remains well within the New Zealand salmon aquaculture industry's production capacity, but suggests that the domestic market has room for some expansion.

The New Zealand King Salmon Co. Ltd intends major development of the domestic market in the coming year (New Zealand King Salmon Co. Ltd, 1996). Some New Zealand primary production industries acknowledge that imported products complement New Zealand product in the local market by helping maintain consistent supply. For example, the Pork Industry Board notes that competition from imports stimulates efficiencies and a drive to improve quality, which in turn stimulate demand (Ministry of Foreign Affairs and Trade, 1997). The New Zealand King Salmon Co. Ltd is able to supply the domestic market with fresh salmon products year round (New Zealand King Salmon Co. Ltd, 1996). The company accepts that market access for overseas produced salmonid products should be granted if disease risks are able to be managed, but is keen to expand the domestic market without requiring domestic production to be complemented by imports (pers. comm. Paul Steere, Chief Executive, New Zealand King Salmon Co. Ltd, October 1996). This seems achievable considering that 77% of production is presently exported.

Demand for products other than salmon products included in the definition of *the commodity*

None of the above estimations can be used to estimate the demand in New Zealand for salmonid products other than salmon, such as trout. There is currently no commercially available domestic supply of trout for human consumption because the Conservation Act 1987 prohibits the farming and sale of New Zealand trout. As such it is difficult to predict the domestic demand for trout products if they were to be imported.

In the United Kingdom there is a ready supply of trout for consumption as a result of the domestic trout aquaculture industry and imports from other European countries. Trout consumption in the United Kingdom is stable at about 250kg/1000 people/year (pers. comm. Alan Munro, July 1997). Approximately 800 tonnes of trout would be needed to meet a comparable level of consumption in New Zealand. However there may be differences between the United Kingdom and New Zealand which limit the accuracy of such an estimate, such as the eating habits of the respective populations, and the retail price of trout relative to other fish products.

Imports would likely be in a form commonly traded internationally, such as individually retort-pouch-packaged, smoked, headed, gilled and gutted fish or fillets. Such a product would need to be air-freighted to New Zealand, meaning that the retail price could be more than countries

which have domestic production or imports from neighbouring countries. Trout might initially attract attention as a novelty. In the longer term, it might compete with salmon (both domestically produced and imported) or create a new niche within the domestic fish, or even game products, markets. Overseas experience indicates that trout would be likely to attract the attention of restaurateurs and chefs. In particular, the ability to offer trout on the menus of restaurants in regions of New Zealand which specifically attract tourist visitors as a result of local trout fisheries may paradoxically create demand for imported product. As with salmon, it seems likely that a domestic trout aquaculture industry would reduce demand for imports.

Table 1. **Importation of Pacific salmon into New Zealand from Canada in the period 1966 to 1983 (Department of Fisheries and Oceans of Canada statistics)**

IMPORTS OF PACIFIC SALMON FROM CANADA (TONNES)					
Year	Chum	Coho	Sockeye	Spring	Other	Total
1966	54	0	0	0	0	**54**
1967	0	0	0	0	0	**0**
1968	0	0	0	0	0	**0**
1969	0	0	0	9	0	**9**
1970	0	0	0	0	0	**0**
1971	0	0	0	95	0	**95**
1972	0	0	0	0	0	**0**
1973	64	0	0	0	0	**64**
1974	0	0	0	0	0	**0**
1975	0	0	0	0	0	**0**
1976	0	0	0	209	0	**209**
1977	0	0	0	0	0	**0**
1978	0	0	0	0	0	**0**
1979	322	132	0	227	0	**681**
1980	0	0	32	0	68	**100**
1981	0	0	240	0	830	**1070**
1982	0	0	0	0	0	**0**
1983	476	0	0	254	0	**730**
TOTAL	**916**	**132**	**272**	**794**	**898**	**3012**

Conclusions

1   The domestic demand for salmon products has increased since salmon was able to be imported prior to 1983. This increase reflects the establishment of a domestic salmon aquaculture industry. If domestic demand continues to increase it will probably be able to be met by the domestic salmon aquaculture industry, which currently exports approximately 77% of production.

2   Data for wild, ocean-caught, Pacific salmon products imported since market access was granted in June 1995 suggest that the establishment of a domestic salmon aquaculture industry supplying the domestic market has significantly reduced the demand for imported salmon products reflected in data for imports prior to 1983.

3   The annual consumption of salmon products in New Zealand (excluding imported canned salmon products) is currently approximately 1,500 tonnes. Data for imports of wild, ocean-caught, Pacific salmon products indicate a market share of no more than 1.3%, and probably currently less, of this total.

4   Steadily increasing annual production and export volumes indicate that salmon from the domestic salmon aquaculture industry competes well in international markets against overseas aquacultured salmon. The low volume of product imported indicates that salmon from the domestic industry is competing well in domestic markets against imported wild, ocean-caught Pacific salmon from Canada.

5   Given the ability of domestically produced salmon to compete against both overseas aquacultured and wild salmon, it seems unlikely that the market share for imported salmon products would significantly increase if market access for a wider range of salmon products was granted.

6   As trout products for human consumption are not currently commercially available in New Zealand, there may be significant interest in importation of these products if market access was granted. Data on trout consumption in the United Kingdom suggests importations of approximately 800 tonnes of trout would be needed to meet a comparable level of consumption in New Zealand.

7   It is highly unlikely that the volume of the *commodity* imported into New Zealand annually will exceed 1,500 tonnes, the current annual domestic consumption of salmon products (excluding imported canned salmon products). A more realistic estimate in the near future may be between 100 to 500 tonnes, increasing up to around 1,000 tonnes as the market becomes established.

8   As with salmon, the establishment of a domestic trout aquaculture industry would be likely to reduce demand for importations.

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4.8   SUMMARY OF CONCLUSIONS OF THE RISK ASSESSMENT

1   No disease introduction event has ever been reliably attributed as resulting from an importation of the *commodity* or similar type product. In many cases of disease agent introduction into a previously believed-free area the route of introduction was not able to be identified.

2   A qualitative assessment of the pathogens and parasites of salmonid fish has concluded that for the majority of organisms considered the risk of their introduction through importations of the *commodity* is negligible.

3   A qualitative assessment of the pathogens and parasites of salmonid fish has concluded that the risk of IPNV, IHNV, VHSV, EIBS, *Aeromonas salmonicida, Henneguya salminicola* and *Kudoa sp.* introduction through importations of the *commodity* is low.

4   A quantitative assessment used a Monte Carlo simulation model to conclude *A. salmonicida* introduction through importations of the *commodity* is unlikely. In three scenarios modelled, the risk was estimated with 99% confidence to be less than 10^{-7} per tonne of *commodity* imported. The upper 95% confidence limits for the probability of *A. salmonicida* introduction per tonne of *commodity* imported were estimated as:

        2.27×10^{-9} for commercially harvested wild Pacific salmon from North America;
        6.24×10^{-8} for spawning wild Pacific salmon from North America;
        5.48×10^{-8} for farmed Atlantic salmon from Norway;
        1.15×10^{-7} for farmed rainbow trout from Denmark.

5   After making the conservative assumption that disease introduction through trade in eviscerated salmonids is possible, even though such introduction has never been demonstrated to occur, a series of quantitative risk assessments used actual historical data on international trade to conclude that the upper 95% confidence limit for the risk of such an event occurring is:

        9.95×10^{-4} in the case of BKD, IHN and *A. salmonicida* introduction into New Zealand;
        2.27×10^{-4} in the case of IHN and *Ceratomyxa shasta* introduction into Denmark;
        3.16×10^{-4} in the case of IHN and *Ceratomyxa shasta* introduction into the United Kingdom;
        1.02×10^{-4} in the case of ISA introduction into the United Kingdom;
        5.44×10^{-5} in the case of *Ceratomyxa shasta* introduction into France;
        2.53×10^{-5} in the case of *Ceratomyxa shasta* introduction into Japan;
        4.81×10^{-5} in the case BKD, IHN and *A. salmonicida* introduction into Australia.

6   The annual volume of the *commodity* likely to be imported into New Zealand if market access is granted is approximately 100 to 500 tonnes. If New Zealand consumption of imported trout products attained United Kingdom levels, and demand was met wholly

through importations, there is potential for this amount to increase to around 1,000 tonnes. Importations of over 1,500 tonnes are very unlikely given the present consumption patterns of salmon in New Zealand.

5.   RISK MANAGEMENT

There are the following options for managing the disease risk to New Zealand associated with imports of salmonid products for human consumption:

1   Continue the current prohibition of imports of uncooked products;

2   Continue the current prohibition on imports of aquacultured salmonid products, while allowing market access for wild, ocean-caught Pacific salmon from the Pacific coast of North America. Since June 1995 Canadian product has had market access to New Zealand. This risk analysis has demonstrated that the disease risk associated with importations of USA product is probably not significantly different to the risk associated with Canadian product.

3   Approve imports of aquacultured and wild salmonid products conforming with the definition of the *commodity* from a restricted range of countries assessed as having met appropriate regulatory requirements for aquatic animal disease surveillance and food safety. In this case, the definition of *the commodity* would be verified within export certification by an appropriate government regulatory authority in the exporting country.

4   Retain a level of regulatory control over product once imported to prevent accumulations of waste potentially contaminated with low titres of exotic pathogens contacting aquatic ecosystems in New Zealand, in recognition of the importance placed on dilution effects within the risk assessment.

These risk management options are discussed, with reference to the risk assessment, in order that what is considered an appropriate level of protection from risk may be determined.

Continuing the current prohibition

Currently all importations into New Zealand of salmonid products for human consumption must meet the requirements of *MAFRA Standard 152.10.08.101 Fresh water fish for human consumption* (Appendix 1), which requires all such product to be cooked. The only exception is wild, ocean-caught, Pacific salmon from Canada, which is able to be imported fresh or frozen under the requirements of a specific import health standard (Appendix 2).

In not making any recommendations for health requirements for countries importing eviscerated fish products for human consumption, the OIE considers such a product may be imported without risk of aquatic animal disease introduction (OIE, 1995). An obligation arising from New Zealand's membership in the World Trade Organisation and as a signatory to the SPS Agreement is alignment, where appropriate, with the recommendations for import health requirements for animals and animal products made by the OIE. When considering importations of any animal or plant product every member has the right to conduct risk analysis

to determine the level of protection appropriate for their circumstances. Such risk analysis must be scientifically based.

The risk assessment within this analysis has concluded that the risk of introducing exotic diseases during importation of the *commodity* is negligible for most diseases, and low for others. The risk assessment has quantified the risk for some diseases in some circumstances, and estimated the likely volume of trade. When the dual conclusions of the risk assessment are considered, that there is a low probability of disease introduction per unit volume imported and the total annual volume of imported product is likely to be relatively low, continuing the prohibition of imports is inappropriate.

The risk identification has concluded that the consequence of disease introduction could be severe for stakeholders in the salmonid fisheries of New Zealand, as well as for native salmoniform fish. Under these circumstances an appropriate level of protection may not be provided through alignment with the OIE's recommendations. Additional measures to manage the disease risk, such as those discussed below, are probably appropriate.

Wild Pacific salmon from North America

This risk analysis has been conducted as a result of a USA request for New Zealand to consider market access for wild Pacific salmon from the USA. The decision to make the risk analysis more generic (to examine disease risks from wild and aquacultured salmonid products from a wide range of countries) was a New Zealand decision, and was taken so that the risk analysis might serve a broader purpose as a decision making tool for the increasingly contentious issue of market access for salmonid products.

This risk analysis has concluded that:

1   Wild Pacific salmon from the USA and Canada are harvested at sea from populations which during the ocean going phase of their life-cycle are likely to be contiguous;

2   The health status of wild Pacific salmon from the USA and Canada is not so significantly different as to affect the disease risk associated with importations of headed, gilled and gutted salmon for human consumption;

3   Health surveillance in wild Pacific salmon populations conducted by both countries has been assessed by MAF through visits to the region, and is considered to provide an excellent reflection of the actual health status of the populations from which fish will be harvested;

4   Government regulatory control over food safety issues during harvesting and processing within both the USA and Canada has been assessed by MAF as being equivalent to New Zealand's own control, as determined by food safety agreements and through visits to the region.

The SPS Agreement requires WTO members to ensure that their sanitary measures do not arbitrarily or unjustifiably discriminate between members where identical or similar health conditions prevail, and that they are not applied in a manner which would constitute a disguised restriction on international trade. Any difference in sanitary measures applied by New Zealand to wild Pacific salmon from Canada, and to similar product from any other country, must be justified by a risk analysis. Information presented in this risk analysis demonstrates that importation of wild Pacific salmon product from USA would be associated with a level of disease risk not significantly different to that for Canadian salmon.

Countries assessed as having equivalent regulatory requirements

Importations of the *commodity* could be restricted to countries assessed by MAF as having appropriate aquatic animal health surveillance and food safety legislation and practices. MAF is considered to be the appropriate New Zealand government agency to perform this assessment, as domestic aquatic animal health surveillance and food safety for exported foods falls under its jurisdiction.

Salmonid producing nations from which MAF has received applications to allow importation of salmonid products for human consumption are discussed below.

1   United States of America

In December 1995 a Cooperative Arrangement between the Ministries of Agriculture and of Health, New Zealand, and the Department of Health and Human Services, Food and Drug Administration, United States of America was signed. The intention of the arrangement is to provide assurances about the safety, wholesomeness and truthful labelling of seafood products traded between the two countries. The arrangement specifically excludes fresh and frozen molluscan bivalve shellfish as these are covered by a separate earlier MOU signed in 1980. In December 1995, the FDA introduced regulations requiring all seafood processors to adopt the HACCP system. This requirement is not included in the current arrangement, although the provisions relating to HACCP are intended to be included in 1997. In essence, the Cooperative Arrangement is an agreement that the legislation relating to food safety and wholesomeness in both countries is equivalent and that product meeting those requirements can be traded between the two countries. MAF provides FDA with a list of premises that can export to the US and each consignment is accompanied by a MAF export certificate. FDA provides MAF with a list of premises found to require official US government regulatory action. FDA does not provide certification for each consignment.

The federal legislation for regulating aquatic animal health in the USA is Title 50 CFR part 16 The Salmonid Importation Regulations, administered by the US Fish and Wildlife Service, Department of the Interior. This legislation details health testing prior to any importation or inter-state movement of salmonids.

Alaska has the largest stocks of wild salmonids in the USA. The Alaskan Department of Fish and Game Division of Commercial Fisheries Management and Development (DCFMD)

administers an aquatic animal health surveillance and diagnostic programme. In salmonids the programme focuses on routine diagnostic screening of brood stock, and mortality investigations of hatchery classes of juvenile salmon. Two DCFMD fish health laboratories serve the state, each with a single fish pathologist, and they are located in Anchorage and Juneau. Routine brood stock screening is undertaken for exotic diseases of international importance (IPN, *Oncorhynchus masou* virus and VHS) as well as for endemic diseases (IHN, furunculosis, BKD and ERM), according to federal and state regulations. Specific surveillance is periodically undertaken to gauge the health status of salmon stocks for diseases of lesser importance, and some data is attained by way of investigation into specific health problems either at hatcheries or in wild stock.

Other states with salmonid fisheries, wild or aquacultured, also administer surveillance programmes. In Washington health surveillance is conducted by the co-managers of the wild salmon fishery, including the Washington Department of Fish and Wildlife; the Native-American tribes of Washington, who, since a federal ruling of 1970, have been allocated a 50% share of the fishery resources of the state; and federal authorities. The fish health policy has listed the following diseases as compulsorily notifiable: IHN, VHS, OMV, IPN, BKD, antibiotic resistant strains of *Aeromonas salmonicida* and *Yersinia ruckeri,* and whirling disease.

The National Marine Fisheries Service, US Department of Commerce, acts as resource managers and trade promoters for the marine fisheries of the USA, including the wild salmon fisheries of Alaska and Washington. The NMFS administers a voluntary export verification and certification service for the export of seafood products to trading partners needing assurances over and above those required for product to enter the domestic US market.

The aquaculture section within Animal and Plant Health Inspection Service (APHIS) of the US Department of Agriculture (USDA) is becoming more active within the aquaculture industries of the USA. The traditional role of overseeing federal regulations for imports is being expanded to cover other aspects of support for health surveillance programmes and export certification of live product.

A Joint Subcommittee on Aquaculture has been formed between APHIS, the NMFS and the Fish and Wildlife Service to delineate agency responsibilities and meet the aquaculture industry's animal health needs.

In October 1996 a MAF Veterinary Officer travelled to the USA to assess food safety processing practices and aquatic animal health surveillance in the wild and farmed salmonid fisheries of the Pacific Northwest of America, including Alaska and Washington. The visit included meetings and discussions with all agencies mentioned above.

2   Canada

In April 1996 an Equivalency Arrangement on Control Measures for the Safety and Quality of Fish and Fishery Products was signed between the Ministries of Agriculture and of Health,

New Zealand, and the Department of Fisheries and Oceans (DFO), Canada. The intention of the arrangement is to provide assurances about the safety, wholesomeness and truthful labelling of seafood products traded between the two countries. The arrangement covers all types of seafood. NZ provides Canada with a list of premises which can export to Canada and each consignment is accompanied by a MAF export certificate. Product from Canada is accompanied by export certification.

The Department of Fisheries and Oceans' (DFO) Inspection Branch is the federal agency responsible for providing export health certification for salmon exported from Canada. In 1997 a single food inspection agency was created to oversee food safety and inspection across many food processing sectors including seafood. The inspection of salmon products for export is conducted in accordance with the Fish Inspection Regulations, requiring all export processors to be registered with the DFO and to be operating a Quality Management Programme under HACCP principles. The DFO's Ocean Sciences Branch is responsible for the Fish Health Protection and Inspection Regulations, which is the legislation for aquatic animal health surveillance in Canada. The DFO administers a programme of health surveillance in the wild salmon fishery.

The British Columbia Ministry of Agriculture Fisheries and Food (MAFF) has responsibility for health surveillance within the province's aquaculture industries, as do other provincial government agencies in other provinces. Aquatic animal health surveillance in the aquaculture industry of British Columbia is passive, and relies upon the investigation of suspected disease problems by private or industry veterinarians, utilising the laboratory services of MAFF when required. There is no compulsory reporting of endemic diseases (including IHN, BKD, furunculosis). The Fish Health Protection Regulations requires any stocks moved within the province (or exported from the province) to be tested for the scheduled diseases.

In June 1994 a MAF Veterinary Officer travelled to British Columbia to assess food safety during processing and aquatic animal health surveillance in the wild salmonid fisheries of that state. In October 1996 a MAF Veterinary Officer travelled to British Columbia and Ottawa to audit certification of wild, ocean-caught Pacific salmon from Canada and to assess the salmonid aquaculture industry. Meetings and discussions with all the above agencies were held.

3   Australia

In December 1995 a treaty was signed between Australia and New Zealand providing for uniform food standards between the two countries. In general, foods which meet the requirements of one country can be traded in the other, amounting to mutual recognition of food safety equivalence. There are some exceptions to this. AQIS are continuing to monitor high risk foods (a category which contains a number of seafood products). The Trans-Tasman Mutual Recognition Agreement (TTMRA) will extend recognition of food safety equivalence.

The Australia New Zealand Food Authority (ANZFA) has responsibility for developing and maintaining food standards, although these responsibilities relate more to composition and labelling rather than food safety. ANZFA authority does extend into some areas of food safety.

Aquatic animal health surveillance in Australia is administered by state governments. The *Salmon import risk analysis* (DPIE, 1996a) summarised these activities. The Australian Quarantine and Inspection Service (AQIS), Department of Primary Industries and Energy, regulates imports of animals and animal products. Two recent studies have summarised Australia's current import policies for aquatic animals and products and set the direction for future policy (Humphrey, 1995; DPIE, 1996b)

4   European Union

In December 1996 the Ministry of Agriculture, New Zealand, and the European Community signed a Veterinary Agreement, the text of which is detailed in Council Decision 97/131/EC of 17 December 1996. Among many other issues of veterinary and public health equivalence covered by the Veterinary Agreement, the standards and systems for seafood production in New Zealand and European Union members are considered to be equivalent, with the exception of bivalve molluscan shellfish which are not grown above the seafloor. All seafood product exported from New Zealand to the EU must come from EU listed premises and be accompanied by export certification. All imports of seafood from the EU to New Zealand must be accompanied by export certification.

The European Community legislation on aquatic animal health is detailed within a variety of directives. The most important directives are 91/67/EEC of 28 January 1991 (and subsequent amendments) concerning the animal health conditions governing the placing on the market of aquaculture animals and products, which lists diseases of concern in trade of aquatic animals and products and provides certification requirements, and 93/53/EEC of 24 June 1993 (and subsequent amendments) which introduced minimum Community measures for the control of certain fish diseases. A wide array of other directives relate to disease status of zones within the EU for listed diseases (particularly IHN and VHS in salmonids), and approval of sampling plans and diagnostic methods for disease zoning.

5   Norway

New Zealand has no food safety equivalence agreements with Norway. Norway exports aquacultured salmon products to the EU. The Norwegian Fish Inspection and Quality Control Service, Directorate of Fisheries, provides verification and certification of the relevant EU requirements.

The Veterinary Service, Royal Ministry of Agriculture, is the government agency with regulatory control of aquatic animal health. New Zealand imports a variety of animal and animal products from Norway under certification provided by the Veterinary Service. Aquatic animal health legislation is detailed within the Interim Fish Diseases Act and Other Regulations Concerning Diseases in Aquatic Organisms, of August 1996, revised in June 1997, controlled by the Veterinary Services.

Verification of the *commodity* within export certification

Import health standards for animal and animal products imported into New Zealand commonly require verification of animal health requirements within export certification to be signed by government approved regulatory agencies in the exporting country. The export certification used is negotiated between MAF and the overseas government authority. When consignments arrive at the New Zealand border biosecurity clearance is dependent upon a documentary check of export certification to ensure it is correctly completed, and a physical check of the consignment to ensure it meets the conditions of eligibility within the import health standard.

Consignments of the *commodity* imported into New Zealand could be required to be accompanied by export certification which would confirm the consignment meets the defining criteria of the *commodity,* detailed on page 16. Such certification would be similar to that which is currently used to certify importations of wild, ocean-caught Pacific salmon from Canada. This appears within the Zoosanitary Certificate of the *Import health standard for the importation into New Zealand of wild, ocean-caught Pacific salmon from Canada* (Appendix 2).

In the context of this risk analysis, the *commodity* would be required to be accompanied by official health certification attesting that:

-        Imported product is derived from fish of species within the genera *Oncorhynchus, Salmo* and *Salvelinus*;

-        Imported product is derived from fish that were harvested from a population for which a documented health surveillance programme exists, which is administered by a competent government-authorised body assessed and approved by MAF;

-        Imported product is not derived from fish that were slaughtered as an official disease control measure as a result of an outbreak of disease;

-        Imported product is derived from fish that were processed in premises under the control of a competent government-authorised regulatory body recognised by MAF as having equivalent food safety standards governing the processing of fish for export;

-        Imported product is derived from fish that during processing have been headed, gilled and eviscerated, individually inspected and graded and that were sexually immature, or sexually maturing, but not sexually mature.

Regulatory control over the *commodity* once imported

The risk assessment has indicated that aquatic animal pathogens may contaminate the *commodity* in low concentrations. The likelihood of environmental contamination with these pathogens sufficient to comprise an infectious dose building up in an aquatic ecosystem in New Zealand as a result of importations of the *commodity* forms an important component of the overall assessment of risk. The risk assessment has concluded that this likelihood is low as a

result of the low initial concentration of pathogens on the *commodity* and the dilution effects which occur along the pathway from its intended use in New Zealand to contact with a susceptible species. These dilution effects are most effective when consignments of the *commodity* are divided into small quantities and widely distributed, and least effective when large consignments are processed in one location. This situation may occur if large volume consignments are imported in bulk form, such as frozen, headed, gutted fish, to be subsequently unpacked and processed into fillets or steaks. This would create a concentration of potentially contaminated packaging, scraps and waste water at the processing site which if allowed to discharge into an aquatic ecosystem would increase the likelihood of an infectious dose contacting a susceptible species.

The risk analysis process for wild, ocean-caught, Pacific salmon from Canada recognised this, and created a system of regulatory control over processing premises to control waste management. Product is classified by the import health standard as either commercially packaged for direct retail sale, and thus requiring no further processing in New Zealand, or not so. Product in the former category is eligible for biosecurity clearance at the border. Product in the latter category is not eligible for biosecurity clearance at the border but must be consigned to a transitional facility. Transitional facilities in this case are premises registered to *MAFRA Standard 154.02.16 Standard for premises processing imported salmon* and supervised according to *MAFRA Standard 154.02.16.01 Standard for the supervision of premises processing imported salmon.* These standards appear as Appendix 3 and 4 respectively.

As a result of an audit by during 1997 of the only registered premises, these standards have recently been updated and draft reviewed standards will in the near future be subjected to consultation. The technical requirements, which remain largely unchanged in the draft reviewed standards, include a quality system for the premises approved by MAF, approved methods of waste disposal, discharge of waste water into an urban sewage treatment system, fortnightly visits by a MAF Travelling Meat Inspector (TMI) throughout the processing period to verify continuing compliance with standards, and periodic audit of the operator and supplier of supervision by MAF Regulatory Authority.

Importations of the *commodity* could be subject to similar post-arrival regulatory control as has been put into place for wild Pacific salmon from Canada.

5.1   Conclusions

1   Considering that the probability of an aquatic animal disease being introduced into New Zealand through imports of the *commodity* is likely to be negligible for most diseases and very low for others, continuing a prohibition on imports is inappropriate.

2   Considering that the probability of an aquatic animal disease being introduced into New Zealand through imports of wild Pacific salmon from the USA is not likely to be significantly different to that for the importation of wild Pacific salmon from Canada which are currently allowable, continuing a prohibition on imports of wild Pacific salmon from the USA is inappropriate.

3   Considering that the consequence of some aquatic animal diseases being introduced into New Zealand through imports of the *commodity* is likely to be severe for stakeholders in the salmonid fisheries of New Zealand and/or for native salmoniform fish, risk management measures over and above those recommended by the OIE Fish Diseases Commission are appropriate.

4   Risk management measures that could be applied include restricting exporting countries to those assessed by MAF as meeting food safety and aquatic animal health surveillance requirements, requiring the *commodity* definition to be verified within export certification, and/or requiring bulk product to be processed in New Zealand under MAF regulatory control.

5   Regulatory systems in Australia, Canada, USA, European Union and Norway would provide New Zealand with adequate food safety and aquatic animal health surveillance assurances if importations of the *commodity* were to be permitted.

6   An indication of how the risk management recommendations would be implemented by MAF can be gained through examination of the similar system set up to regulate imports of wild Pacific salmon from Canada, as documented within the *Import health standard for the importation into New Zealand of wild, ocean-caught Pacific salmon from Canada* (Appendix 2), *MAFRA Standard 154.02.16 Standard for premises processing imported salmon* (Appendix 3) and *MAFRA Standard 154.02.16.01 Standard for the supervision of premises processing imported salmon* (Appendix 4).

References

Department of Primary Industries and Energy (DPIE). (1996a). Salmon import risk analysis: An assessment by the Australian Government of quarantine controls on uncooked, wild, adult, ocean-caught Pacific salmonid product sourced from the United States of America and Canada. Australian Commonwealth Government.

Department of Primary Industries and Energy (DPIE). (1996b). Report of the National Task Force on Imported Fish and Fish Products: A report into the implications arising from aquatic animal imports. Australian Commonwealth Government.

OIE. (1995). OIE International Aquatic Animal Health Code, Office International des Epizooties, 1995, first edition.

6. ACKNOWLEDGEMENTS

IMPORT HEALTH STANDARD FOR THE IMPORTATION INTO NEW ZEALAND OF SALMONIDS FOR HUMAN CONSUMPTION FROM SPECIFIED COUNTRIES (SHC)

USER GUIDE

The information in MAF animal and animal product import health standards is presented in numerically ordered sections with descriptive titles. Sections are grouped into one of four parts, designated alphabetically.

Part A. GENERAL INFORMATION contains sections of general interest, including those relating to the legal basis for MAF import health standards and the general responsibilities of every importer of animals and animal products.

Part B. IMPORTATION PROCEDURE contains sections which outline the requirements to be met prior to and during importation. Whether a permit to import is required to be obtained prior to importation is noted, as are conditions of eligibility, transport and general conditions relating to documentation accompanying the consignment.

Part C. CLEARANCE PROCEDURE contains sections describing the requirements to be met at the New Zealand border and, if necessary, in a transitional facility in New Zealand prior to any consignment being given biosecurity clearance.

Part D. ZOOSANITARY CERTIFICATION contains model health certification which must be completed by the appropriate personnel as indicated in the certification and accompany the consignment to New Zealand. When MAF has accepted health certification produced by a government authority in the exporting country as meeting the requirements of the model health certification this is noted. When no health certification is required to accompany consignments Part D. will note "none required".

PART A. GENERAL INFORMATION

1   IMPORT HEALTH STANDARD

1.1   Pursuant to section 22 of the Biosecurity Act 1993, this document is the import health standard for the importation into New Zealand of salmonids for human consumption from specified countries (SHC).

1.2   Obtaining biosecurity clearance for each consignment of salmonids for human consumption imported into New Zealand from specified countries (SHC) is dependant upon the consignment meeting the requirements of this import health standard.

1.3   This import health standard may be reviewed, amended or revoked if there are changes in New Zealand's import policy or the animal health status of the originating country, or for any other lawful reason, at the discretion of the CVO.

2   IMPORTER'S RESPONSIBILITIES

2.1   The costs of MAF in performing functions relating to the importation of salmonids for human consumption shall be recovered in accordance with the Biosecurity Act and any regulations made under that Act.

2.2   All costs involved with documentation, transport, storage and obtaining a biosecurity direction and/or biosecurity clearance shall be borne by the importer or agent.

3   DEFINITION OF TERMS

biosecurity clearance
As defined by the Biosecurity Act 1993.

biosecurity direction
Direction or authorisation given by an Inspector for uncleared goods to proceed to a transitional facility.

bulk form
Means product which is intended to be further processed and/or packaged in New Zealand prior to retail sale or use in the institutional trade. e.g. containers of more than one fish.

commercial consignment
Means a consignment comprising product intended for distribution and/or sale in New Zealand.

commercially packaged for direct retail sale
Means product not requiring further packaging and/or processing prior to retail sale or use in the institutional trade in New Zealand. e.g. retort pouch packaged single fish, hermetically sealed packages of fish portions.

CVO
The Chief Veterinary Officer, New Zealand Ministry of Agriculture and Forestry, or any person who for the time being may lawfully exercise and perform the power and functions of the Chief Veterinary Officer.

equivalence
Acceptance by the CVO that the circumstances relating to the importation of a consignment are such that the health status of the consignment is equivalent to the health status of a consignment that complies with the requirements of the import health standard.

headed, gilled and gutted

Means removal of all tissues comprising the head, the gills and the internal organs.

Inspector
As defined by the Biosecurity Act 1993.

MAF
The New Zealand Ministry of Agriculture and Forestry.

permit to import
A permit issued by the Director General of MAF pursuant to section 22 1(A) of the Biosecurity Act 1993 upon an importer's demonstration that certain requirements of the import health standard have been met in advance of an importation being made, such that a transitional facility is available to accept the consignment/s and a method and route of transport from the port of arrival to the transitional facility has been approved. The procedure for application and the information required for a permit to import are detailed within the import health standard.

private consignment
Means a consignment comprising product imported in an airline passenger's personal effects or by mail and not intended for sale in New Zealand.

salmonids
For the purposes of this import health standard, salmonids means fish in the genera *Oncorhynchus, Salmo* and *Salvelinus*.

specified countries (SHC)
Means countries with MAF-approved fish health and fish processing regulatory programmes from which salmonids for human consumption may be imported into New Zealand, as listed under ELIGIBILITY. The SHC is a unique code required by the Plant and Animal Quarantine Information System (PAQIS).

transitional facility
As defined by the Biosecurity Act 1993.

4   EQUIVALENCE

4.1   It is expected that the animal product will meet the conditions of this import health standard in every respect. If the products do not comply with the requirements, an application for equivalence may be submitted to MAF for consideration. Detailed information supporting the application for equivalence must be forwarded to MAF for a decision.

PART B. IMPORTATION PROCEDURE

5   PERMIT TO IMPORT

5.1   In the case of private consignments of product and product which is commercially packaged for direct retail sale:

5.1.1   Importations of salmonids for human consumption into New Zealand from specified countries (SHC) which meet the requirements of this import health standard may, subject to sections 27 and 28 of the Biosecurity Act, be given biosecurity clearance and do not require a biosecurity direction to a transitional facility. As such, they do not require a permit to import.

5.2   In the case of product in bulk form:

5.2.1   An application for a permit to import shall provide the following information:
(i) name and address of importer; and
(ii) name and address of exporter; and
(iii) description and quantity of the salmonids for human consumption to be imported; and
(iv) date of the proposed importation; and
(v) name and address of the TRANSITIONAL FACILITY to which the consignment is to proceed following importation; and
(vi) a letter from the authorised supervisor of the transitional facility stating that the facility is registered and is available for the dates proposed and has the capacity to accommodate the consignment proposed to be imported; and
(vii) the transport method and route during importation into New Zealand, which will be in accordance with all requirements for TRANSPORT TO NEW ZEALAND noted in this import health standard, and evidence of transit authority from countries on the transport route; and
(viii) the transport method and route during transfer from the port of arrival in New Zealand to the transitional facility.

5.2.2   A permit to import will be granted for a single consignment only.

5.2.3   A permit to import is required for all consignments of salmonids for human consumption imported into New Zealand from specified countries (SHC). Application for a permit to import shall be made at least 30 days prior to the proposed date of importation in writing to The Chief Veterinary Officer, Ministry of Agriculture and Forestry, PO Box 2526, Wellington, New Zealand.

6   ELIGIBILITY

6.1   The specified countries (SHC) from which importation under this import health standard is permitted are the following:
Australia, Canada, European Union Member Countries, Norway, the United States of America.

6.2   In the case of product imported as a private consignment:
(i) no permit to import is required;
(ii) export certification meeting the requirements of PART D ZOOSANITARY CERTIFICATION is not required;

(iii) the consignment must comprise headed, gilled and gutted fish commercially packaged for direct retail sale (see DEFINITION OF TERMS);
(iv) the consignment must not comprise more than 10 kg net weight of fish;
(v) the consignment is eligible for biosecurity clearance at the New Zealand border.

6.3   In the case of commercial consignments of product commercially packaged for direct retail sale:
(i) no permit to import is required;
(ii) the consignment must be accompanied by export certification meeting the requirements of PART D ZOOSANITARY CERTIFICATION;
(iii) the consignment is eligible for biosecurity clearance at the New Zealand border.

6.4   In the case of product in bulk form:
(i) the consignment must be accompanied by a valid permit to import;
(ii) the consignment must be accompanied by export certification which meets the requirements of PART D ZOOSANITARY CERTIFICATION;
(iii) the consignment is not eligible for biosecurity clearance at the New Zealand border, but must be directed to proceed to a transitional facility as described in Section 8 BIOSECURITY DIRECTION and Section 9 TRANSITIONAL FACILITY.

6.5   The product must comply with the requirements of the Food Regulations (1984) of New Zealand administered by the Ministry of Health and each consignment may be subject to inspection by an inspector appointed under these regulations.

6.6   The following information relates to a non-biosecurity eligibility restriction:
The Customs Import Prohibition (Trout) Order 1998, administered by the New Zealand Customs Service, prohibits the importation of trout and trout products in quantities exceeding 10 kilograms, or in quantities of less than 10 kilograms if the goods are intended for sale, between 7 January 1999 and 7 July 2000, except with the consent of, and subject to such conditions as may be imposed by the Minister of Conservation. Any consignment may be subject to inspection by an authorised person under the Customs and Excise Act 1996.
(**N.B.** The schedule of the above conditions notes the following as prohibited imports:
1. All species of trout, alive or dead, including but not limited to-
a. Brown trout (*Salmo trutta*);
b. Rainbow trout (*Oncorhynchus mykiss*);
c. American brook trout of char (*Salvelinus fontinalis*);
d. Lake trout or char (*Salvelinus namayusch*);
e. Cutthroat trout (*Oncorhynchus clarki*);
f. Goldern trout (*Oncorhynchus aguabonita*);
g. Gila trout (*Oncorhynchus gilae*);
h. Apache trout (*Oncorhynchus apache*);
i. Mexican trout (*Oncorhynchus chrysogaster*);
j. Any hybrid of a fish listed in paragraphs (a) to (i) of this item.
2. Any part of a fish listed in item 1 of this schedule.
3. Any fish product which is or is derived from a fish listed in item 1 of this schedule.)

7 DOCUMENTATION ACCOMPANYING THE CONSIGNMENT

7.1   The consignment shall be accompanied by appropriately completed health certification which meets the requirements of PART D. ZOOSANITARY CERTIFICATION.

7.2   Documentation shall be in English, but may be bilingual (language of exporting country/English).

7.3   It is the importer's responsibility to ensure that any documentation presented in accordance with the requirements of this import health standard is original (unless otherwise specified) and clearly legible. Failure to do so may result in delays in obtaining biosecurity direction and/or clearance or rejection of consignments.

PART C. CLEARANCE PROCEDURE

8 BIOSECURITY DIRECTION

8.1   In the case of product in bulk form:

8.1.1   Upon arrival in New Zealand the documentation accompanying the consignment shall be inspected by an Inspector at the port of arrival. The Inspector may also inspect the consignment, or a sample of the consignment.

8.1.2   A biosecurity direction may be given by an Inspector under section 25 of the Biosecurity Act 1993 authorising the consignment to move to the transitional facility named in the permit to import, providing that the documentation meets all requirements noted under PART D. ZOOSANITARY CERTIFICATION and the consignment meets the conditions of ELIGIBILITY.

9 TRANSITIONAL FACILITY

9.1   In the case of product in bulk form:

9.1.1   Imported product must be consigned to a transitional facility registered to *MAF Regulatory Authority Standard 154.02.16 Transitional facilities for processing imported salmonids*.

9.1.2   While in the transitional facility the imported product shall be subjected to the following conditions:
(i) such processing and/or packaging to render the product commercially packaged for direct retail sale;
(ii) management of any solid and liquid wastes produced during processing and/or packaging according to a MAF approved waste management plan.

10 BIOSECURITY CLEARANCE

10.1   In the case of product in bulk form:

10.1.1   On successful completion of the terms detailed under TRANSITIONAL FACILITY the consignment may, subject to sections 27 and 28 of the Biosecurity Act 1993, be given a biosecurity clearance pursuant to section 26 of the Biosecurity Act 1993.

10.2   In the case of commercial consignments of product commercially packaged for direct retail sale:

10.2.1   Upon arrival in New Zealand the documentation accompanying the consignment shall be inspected by an Inspector at the port of arrival. The Inspector may also inspect the consignment, or a sample of the consignment.

10.2.2   Providing that the documentation meets all requirements noted under PART D. ZOOSANITARY CERTIFICATION and the consignment meets the conditions of ELIGIBILITY, the consignment may, subject to sections 27 and 28 of the Biosecurity Act 1993, be given a biosecurity clearance pursuant to section 26 of the Biosecurity Act 1993.

10.3   In the case of private consignments of product commercially packaged for direct retail sale:

10.3.1   Upon arrival in New Zealand the consignment shall be inspected by an Inspector at the port of arrival.

10.3.2   Providing that the consignment meets the conditions of ELIGIBILITY, the consignment may, subject to sections 27 and 28 of the Biosecurity Act 1993, be given a biosecurity clearance pursuant to section 26 of the Biosecurity Act 1993.

PART D. ZOOSANITARY CERTIFICATION

11   NEGOTIATED EXPORT CERTIFICATION

11.1   The following documents are approved by MAF as being equivalent to the requirements of PART D. ZOOSANITARY CERTIFICATION, and are approved as export certificate/s to accompany imports of salmonids for human consumption into New Zealand when appropriately completed by an authorised representative of the exporting country's competent government agency for salmonid health and/or salmonid processing for human consumption:

11.1.1   Canadian Food Inspection Agency Fish Health Certificate for wild Pacific salmon products originating in Canada and intended for export to New Zealand [Ref: CFIA / ACIA 5024 (97/12) Newz 11/96].

11.1.2   U.S. Department of Commerce National Oceanic and Atmospheric Administration Health Certificate to New Zealand: Import conditions to issue a zoosanitary certificate for U.S. processed salmon intended for import into New Zealand;

11.1.3   Agriculture Fisheries and Forests Australia (formerly the Department of Primary Industries and Energy) Export Certificate EX46 "Certificate as to Condition", with additional certification for salmonids for human consumption exported to New Zealand as follows:
The products are:
1) derived from fish grown and harvested in Tasmania, or Victoria, or NSW;
2) derived from fish within the genera *Oncorhynchus, Salmo* and *Salvelinus;*
3) not slaughtered as an official disease control measure as a result of an outbreak of disease;
4) (i) headed, gilled and gutted;
(ii) individually inspected and graded, ensuring the product for export is free from visible lesions associated with infectious disease and fit for human consumption; and
(iii) found to be sexually immature, or sexually maturing, but not sexually mature.

12   MODEL ZOOSANITARY CERTIFICATION

COMMODITY: Salmonids for human consumption

CERTIFYING AUTHORITY:
Agency:
Department:
Country:

I. ORIGIN OF THE PRODUCT

(i). Name and address of producer:

(ii). Name and address of processor:

(iii). Processing premises registration number:

II. PRODUCT DESCRIPTION

(i). The product contained in this consignment is:

EITHER: commercially packaged for direct retail sale;

OR: in bulk form.

(Delete one of the above.)

(ii). Amount (in kgs) of product:

III. DESTINATION OF FISH

(i). Name and address of New Zealand importer:

(ii). Name and address of premises approved and registered under *MAF RA Standard 154.02.16* (in the case of product in bulk form):

IV. ZOOSANITARY INFORMATION

12.1   The product for export is derived from fish within the genera *Oncorhynchus, Salmo* and *Salvelinus.*

12.2   The product for export is derived from fish that were harvested from a population for which a documented health surveillance programme exists which is administered by a competent government-authorised agency.

12.3   The product for export is derived from fish that were not slaughtered as an official disease control measure as a result of an outbreak of disease.

12.4   The product for export is derived from fish that were processed in a premises under the supervision of a competent government-authorised regulatory agency with responsibility for food safety standards during processing of fish for export. During processing the fish were:
(i) headed, gilled and gutted;
(ii) individually inspected and graded, ensuring the product for export is free from visible lesions associated with infectious disease and fit for human consumption; and
(iii) found to be sexually immature, or sexually maturing, but not sexually mature.

Signature of authorised representative of competent government agency controlling salmonid health and/or salmonid processing for human consumption:

Official stamp and date:

Name and address of office:

N.B. Official stamp must be applied to all pages.

MAF REGULATORY AUTHORITY
Animal Health and Welfare

STANDARD 154.02.16

Standard

for Premises Processing

Imported Salmonids

Ministry of Agriculture
MAF Regulatory Authority
P O Box 2526
Wellington
New Zealand

CONTENTS

REVIEW

This MAFRA Standard is subject to periodic review. The next review date is 30 March 1997. Amendments will be issued to ensure the Standard continues to meet current needs.

ENDORSEMENT

This Standard is hereby approved pursuant to sections 39 and 40 of the Biosecurity Act 1993.

The forms on pages 11, 13 and 14 are hereby approved pursuant to sections 39 and 40 of the Biosecurity Act 1993.

_____
B D O'Neil
Chief Veterinary Officer
(acting pursuant to delegated authority)

Date:

AMENDMENT RECORD

Amendments to this Standard will be given a consecutive number and will be dated.

Please ensure that all amendments are inserted, obsolete pages removed and the record below is completed.

Amendment No:	Entered by:	Date:
1		
2		
3		
4		
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1.   INTRODUCTION

1.1   SCOPE

The primary purpose for the quarantine of imported salmonids during processing is to minimise any risks associated with the concentration of by-product potentially contaminated with disease biota. This is achieved by preventing by-product coming into contact with susceptible New Zealand fish species.

This MAFRA Standard specifies the registration, structural, and operating standards for premises processing imported salmonids in New Zealand and prescribes requirements with which the operators of such processing facilities shall comply.

It should be noted that in meeting the requirements of this Standard the operator of a processing premises is in no way absolved of responsibilities under the requirements of other relevant legislation; for example the Meat Act 1981, the Food Act 1991, the Food Hygiene Regulations 1974, and the Fish Export Processing Regulations 1995.

It should also be noted that a processing premises for imported salmonids registered to this Standard must not also be licensed as a fish export packing house under the Meat Act 1981.

1.2   REFERENCES

This Standard is an approved standard in terms of sections 39 and 40 of the Biosecurity Act 1993 and Regulations.

The following publications are referred to in this MAFRA Standard:

AS\NZS 9002 (1994): Quality Systems - Model for quality assurance in production, installation and servicing. Published by Standards New Zealand.

Import Health Standard for the Importation of Wild, Ocean-Caught Pacific Salmon into New Zealand from Canada.

1.3   DEFINITIONS

For the purposes of this MAFRA Standard the following definitions apply:

Approval

Approved in writing by the Chief Veterinary Officer or his/her delegated representative.

Biota

Any stage of any organism including fungus, bacterium, plant, virus, rickettsia, mycoplasma, protozoan, metazoan.

Contaminated by-product

Is all product, other than the end product, resulting from the processing or packaging of imported salmonids undertaken by the processing premises. Examples of contaminated by-product are trimmings of fish, imported packaging materials and water contaminated by contact with imported salmonids.

Chief Veterinary Officer

Is the chief technical officer (as defined in section 101 of the Biosecurity Act 1993) of MAF with animal health responsibilities.

End product

Any parts of the imported salmonids that are sold in New Zealand or re-exported.

Import Health Permit

A numbered document, issued under section 20 of the Biosecurity Act 1993, approving the importation of risk goods.

Import Health Standard

Any current schedule of conditions for the importation of salmonids into New Zealand issued in terms of section 22 of the Biosecurity Act 1993. This import health standard forms part of the import health permit.

Imported Salmonids

Any uncooked salmonid flesh product imported under a current import health standard.

ISO 9002

Is the Australian and New Zealand Standard (1994) Quality Systems- Model for quality assurance in development, production, installation and servicing.

MAF Regulatory Authority

The body within the Ministry of Agriculture responsible for regulatory functions.

Operator

The body corporate or person who has overall responsibility for the facility, its maintenance and operation in terms of section 40 of the Biosecurity Act 1993.

Person

   As defined in section 2 of the Biosecurity Act 1993.

Procedure

A document that specifies, as applicable, the purpose and scope of an activity; what shall be done and by whom; when, where, and how it shall be done; what materials, equipment and documentation shall be used; and how it shall be controlled.

Processing Premises

A premise, registered by the Director General MAF in terms of section 39 of the Biosecurity Act, that undertakes any packaging or processing of salmonids imported under the requirements of an import health standard, such as the Import Health Standard for the importation of wild ocean caught Pacific salmon into New Zealand from Canada, and prior to that product being placed for sale.

Quarantine Facility

A facility for organisms or organic material that may be harbouring pests or unwanted organisms.

Registered

Means registered by the Director General MAF as a quarantine facility under sections 39 and 40 of the Biosecurity Act 1993.

Salmonids

For the purposes of this standard, salmonids shall mean fish within the genera *Oncorhynchus, Salmo* and *Salvelinus.*

Supplier

The party contracted by MAFRA to supply supervisory services with respect to the compliance of these standards e.g MAF Quality Management.

1.4   SERVICE OUTLINE

Critical structural and operating requirements for premises for the processing of imported salmonids within this Standard are:

The processing premises shall be constructed and operated in a manner to ensure that all potentially contaminated by-products resulting from the re-packaging or processing of imported salmonids by the processing premises are disposed of in a manner approved by the Chief Veterinary Officer.

The over-riding principle shall be that no contaminated by-product shall be allowed to enter a marine or freshwater environment, be fed or come into contact with fish (of any species) prior to sufficient treatment to render it a non-risk good. It is

accepted that wastewater discharged to an urban sewage system will by dilution bring about the same effect.

Approval to operate a premises for the processing of imported salmonids is subject to registration in terms of the sections 39 and 40 of the Biosecurity Act 1993 and continued compliance with this Standard, the Biosecurity Act and Regulations.

Imported salmonids which meets the conditions of the import health standard and has been processed in accordance with this Standard shall be deemed to given biosecurity clearance.

2.   SERVICE REQUIREMENTS

2.1   GENERAL REQUIREMENTS

The processing premises shall be operated in accordance with this Standard, the conditions of the import health permit, import health standard and any other relevant legislation, such as the Food Act 1981, the Food Hygiene Regulations 1974, the Meat Act 1981 and the Fish Export Processing Regulations 1995.

The processing premises must not a be fish export packing house licensed under the Meat Act 1981.

The operator shall develop a quality assurance programme and procedures based on ISO 9002. These procedures shall address the requirements for the service outline indicated in section 1.4 above, and the technical requirements and procedures of section 3.4 below. The quality assurance programme, these procedures and any amendments to them shall be approved by the Chief Veterinary Officer.

The operator shall be registered (section 3.3). The operator may nominate another person, a manager, who shall have defined authority and responsibility for ensuring that the facility and operations comply with the procedures detailed in the quality assurance programme.

The operator shall provide the inspector, or any other representative of the Chief Veterinary Officer, access to the facility, records and documents for the purposes of inspection and/or audit.

Procedures and records of any contracted disposal company and any cartage contractors may also be audited at the discretion of the Chief Veterinary Officer.

3.　TECHNICAL REQUIREMENTS

3.1　APPROVAL OF THE PROCESSING PREMISES SITE

Where it is proposed to establish new processing premises, upgrade, or to increase the area of an existing processing premises, an application for site approval shall be submitted to the Chief Veterinary Officer. The application shall show, where applicable:

Details of the proposal, including the importation programme and the quantities of imported salmonids to be processed.

A site plan of the property which shows the location of the processing premises and the entrance/exit to the site. These shall also show a plan of the proposed waste disposal system. Drain pipes, the natural underground drainage of the site and location of streams form an important part of this plan.

Detailed plans of the proposed processing premises with notes as appropriate which show how the facility has been designed and will be operated to ensure the containment and treatment of contaminated by-products of imported salmonids.

Evidence shall be presented from the relevant regional council and district council that the proposed operation satisfies planning requirements under the Resource Management Act, Building Act or any other relevant legislation under which these Councils have jurisdiction. The Director General shall also be satisfied that the Local Authority has been properly informed about the project and assured of the risks involved.

Evidence shall be presented to show that the immediate neighbours of the property have been properly informed of the proposed project and that they have no unmanageable objections to the project.

Evidence shall be presented to show that a notice signed by the Director General has been placed in the local paper by the veterinary officer after approval of the proposal has been received from the local authority and acknowledgement received from the neighbours. The intent of this notice is to give the local community an opportunity to make representations concerning the proposal within a three week period.

The prospective operator shall apply for registration as an operator and for Police clearance at this time.

The Chief Veterinary Officer will advise the supplier if approval has been given.

3.2   REGISTRATION OF THE PROCESSING PREMISES

An application for registration of a processing premises shall be made by the operator to the Director General as required under Section 39 of the Biosecurity Act 1993. This shall be made through the supplier using the form on page 11.

When the facility and operational procedures meet the requirements of this Standard the supplier shall recommend to the Chief Veterinary Officer that the facility be registered as a quarantine facility.

A letter of registration shall be sent to the operator when the facility has been registered.

3.2.1 DE-REGISTRATION OF THE PROCESSING PREMISES

The Director General may cancel the registration of a processing premises if no longer satisfied that the facilities provided are suitable for the purpose or are not being maintained to this Standard.

APPLICATION FOR REGISTRATION OF A PROCESSING PREMISES FOR THE PROCESSING OF IMPORTED Salmonids AS A QUARANTINE FACILITY

PURSUANT TO SECTION 39 OF THE BIOSECURITY ACT, 1993.

Name of the Processing Premises: . . . .

Location address: . . . .

. . . .

Processing Premises operator's name: . . . .

Organisation: . . . .

Postal Address: . . . .

Telephone No: . . . .   Facsimile: . . . .

I, ... , being the applicant, declaring that the above facility meets the requirements of MAFRA Standard 154.02.16: Standard for Premises Processing Imported Salmonids, apply to have it registered by the Director General as a Quarantine Facility.

. . . .
      Signature of Applicant   Date

Form approved by the Chief Veterinary Officer pursuant to Section 39 of the Biosecurity Act, 1993.

3.3 REGISTRATION OF THE OPERATOR

The operator is responsible for the operation of the processing premises and ensuring that mechanisms are in place for resourcing the facility. Any person wishing to be an operator, who agrees to comply with this Standard, shall apply to the Director General, through the Chief Veterinary Officer, for registration as a quarantine operator, pursuant to Section 40 of the Biosecurity Act 1993. A form is provided on the following page.

If the facility is leased, the lessee, responsible for the operation of the processing premises, shall apply to be the operator and the contract with the owner shall clearly identify who is responsible for the maintenance of the premises and the resourcing of the operation. No part of the lease contract shall override the requirements of this standard in the operation of quarantine. This contract shall be made available to the Chief Veterinary Officer.

A letter of registration shall be sent to the operator by the Chief Veterinary Officer when the operator is registered.

3.3.1 COLLECTION OF PERSONAL INFORMATION ON INDIVIDUALS

In regard to any information being collected on the application for registration of a quarantine operator, this is personal information (being information identifying or being capable of identifying an individual person). Notification is hereby provided, in accordance with Principle 3 of the Privacy Act 1993, to individuals of the following matters:

1. This information is being collected for the purposes relating to the registration of quarantine facilities as per section 40 of the Biosecurity Act, 1993.

2. The recipient of this information, which is also the agency that will collect and hold the information is the Ministry of Agriculture, PO Box 2526, Wellington.

3. You are reminded that under Principles 6 and 7 of the Privacy Act, 1993, you have the right of access to, and correction of, any person information which has been provided.

3.3.2 DE-REGISTRATION OF THE PROCESSING PREMISES OPERATOR

The Director General may cancel the registration of an operator if no longer satisfied that the processing premises is being operated according to this Standard. Notice of cancellation shall be given in writing to the operator.

APPLICATION FOR REGISTRATION OF AN OPERATOR OF PREMISES FOR THE PROCESSING OF IMPORTED Salmonids (PROCESSING PREMISES)
PURSUANT TO SECTION 40 OF THE BIOSECURITY ACT, 1993.

The application for registration of the operator shall be made to the Director General by the person responsible for the processing premises and its operation. Qualifications and experience of the operator (and the manager, if a manager is to be appointed in lieu of the operator) to be detailed on a separate page.

Applicant's name: . . . .

Designation: . . . .

Organisation: . . . .

Postal address: . . . .

Telephone No: . . . .   Facsimile: . . . .

Name of Processing Premises: . . . .

I . . . . , being the person (the proposed operator) responsible for the processing premises named above, declare that:

I have the technical and financial resourcing mechanisms in place to maintain that facility.

I have read and understand MAFRA Standard 154.02.16. I will ensure that the operation of the processing premises is in accordance with MAFRA Standard 154.02.16.

I hereby apply for registration as an operator.

. . . .
      Signature of Applicant   Date

Form approved by the Chief Veterinary Officer pursuant to Section 40 of the Biosecurity Act, 1993.

Officer in Charge
Intelligence & Criminal History Section
Police National Headquarters
P O Box 3017
WELLINGTON

CONSENT TO DISCLOSURE OF CONVICTIONS

I, ...
       (Surname)   (Fore Names)

. . . .
       (maiden or any other names used)

Sex . . . . . (M/F)   Date and Place of birth . . . .

Nationality . . . . Address . . . .

. . . . ,

hereby consent to the disclosure by the New Zealand Police of any convictions I may have pursuant to this application to the **DIRECTOR GENERAL OF AGRICULTURE, NEW ZEALAND,** to be registered as a quarantine or containment operator.

(Note: Such a disclosure may **NOT** include information relating to any discharge under Section 19 of the Criminal Justice Act 1985, or Section 347 of the Crimes Act 1961, or Section 282 of the Children and Young Persons and their Families Act 1989.)

Signed . . . . Date . . . .

COMMENTS OF NEW ZEALAND POLICE

Send completed disclosure to:

    Personal: In Confidence
         Elizabeth Stoddart
         MAF Regulatory Authority
         P O Box 2526
         WELLINGTON

Form approved by the Chief Veterinary Officer pursuant to section 40 of the Biosecurity Act 1993

3.4 REQUIREMENTS OF A PREMISES FOR THE PROCESSING OF IMPORTED Salmonids (PROCESSING PREMISES)

The operator shall develop and implement a quality assurance programme and procedures based on ISO 9002. The quality assurance programme, these procedures and any amendments to them shall be approved by the Chief Veterinary Officer. The quality assurance programme should account for the following requirements:

3.4.1 Waste Management Plan

The operator shall develop a quality assurance programme and procedures to deal with all aspects of waste management of contaminated by-product produced as a result of the processing or packaging of imported salmonids undertaken at the processing premises.

These procedures shall minimise any risks associated with the concentration of by-product, potentially contaminated with disease biota, coming into contact with susceptible New Zealand fish species.

The procedures will be updated by the operator with the approval of the Chief Veterinary Officer so that they are at all times an accurate and detailed reflection of the waste management practises.

These procedures shall address the treatment or disposal of the following:

1. Disposal of imported packaging, by incineration or other approved method. (A contracted waste management company may be used.)

2. Disposal of non-liquid contaminated by-products by either complete incineration or rendering at a minimum of 85°C for at least 15 minutes. Such disposal may be on-site, or after transportation to a contracted company such as a waste management contractor or petfood/fertiliser manufacturer.

3. Disposal of liquid wastes potentially contaminated by imported salmonids by discharge into an urban sewage treatment system.

3.4.2 Audit by the Supplier

For audits and visits by the supplier the operator shall ensure the following:

All relevant records and procedures are made available to the supplier including records from any relevant contracted waste management services.

The operator's management representative is available for the purposes of assisting the inspector.

Systems audit at commencement of processing:
If the processing of imported salmonids is discontinued for more than six weeks the operator shall give the supplier at least 2 weeks notice of the impending commencement of processing of imported salmonids. This notice should be sufficient to ensure that the supplier is able to perform a systems audit against this Standard and the approved waste management plan before approval is given for the commencement of processing of imported salmonids.

Continuing fortnightly visits:
The operator shall advise the supplier when processing of imported salmonids is to take place so that the supplier can visit the premises each fortnight throughout the period when imported salmonids are being processed. During these visits the Inspector shall check waste management records and physically check waste management methods. During these visits the inspector may focus on specific randomly chosen aspects of the operation or a full audit against this Standard and the associated approved waste management plan.

3.4.3 Records:

The operator is required to demonstrate compliance with the requirements of this Standard by keeping continuous records of waste management and salmonids processing, together with evidence of active internal auditing procedures. The records should be available to the inspector during each visit to the processing premises.

Such records should be kept for a minimum of 7 years, and include as a minimum:

1. Records of registration applications and approval.

2. Records of dates, weights, import health permits, import health standards, processing details, end-product destination and invoices for every consignment of imported salmonids.

3. Records of the quantity of by-product treatment performed on-site related to the quantity of imported salmonids.

4. Records of the quantity of by-product treatment performed off-site related to the quantity of imported salmonids.

5. Records of every visit by the supplier, including audits, areas looked at during the visit, and the follow-up work resulting from such visits to ensure continued compliance.