Supplementary Import risk analysis: Head - on, gill - in Australian salmonids for human consumption.

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1 Introduction

1.1 Existing conditions resulting from previous MAF risk analysis

In August 1997 MAF released a risk analysis examining the biosecurity risks associated with importation into New Zealand of headed, gilled and gutted salmonids for human consumption (Stone et al, 1997). Following consultation, in September 1998 an import health standard was issued (MAF, 1998).

The measures within the import health standard include:

- restricting imports to specific source countries with regulation of fish production and processing industries by competent authorities approved by MAF;

- requiring certification of imports (except private non-commercial consignments) by the exporting country's competent authority, that the product is derived from fish:
  - 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 agency;
  - 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 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.

- restricting packaging and processing after arrival in New Zealand of imported product in bulk form. Any such processing prior to distribution to consumers must be performed in premises with appropriate waste management systems registered and supervised by MAF. (There are no post-arrival restrictions on product imported into New Zealand in a form allowing direct distribution to consumers).

1.2 Trout

The Customs Import Prohibition (Trout) Order 1998 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. This prohibition is not for biosecurity purposes.

The New Zealand parliament is currently considering a bill that would permanently prohibit importation and sale of trout in New Zealand.

1.3 Current conditions for imports of salmonids from Australia

In January 1999 MAF accepted certification proposed by the Australian Quarantine and Inspection Service (AQIS) to accompany exports. In summary, Australia was approved under the existing import health standard because:

- the Australian and New Zealand Food Safety Authority ensures uniform food standards between Australia and New Zealand;

- MAF accepts that AQIS is a competent authority to regulate food safety and zoosanitary assurances for foods exported from Australia to New Zealand; and

- MAF accepts salmonid health surveillance systems administered by the state veterinary authorities in Tasmania, Victoria, and New South Wales provide an acceptable level of assurance regarding population health status.

1.4 New market access request from Australia

During the annual Closer Economic Relations ministerial meeting in Canberra on 12 August 1999, New Zealand agreed to consider imports of eviscerated (gutted) salmonids for human consumption from Australia i.e. head-on, gill-in fish. Concerns had been voiced by Australia at the meeting that the AQIS 1999 risk analysis (AQIS, 1999) had recommended that salmonids from New Zealand (other than trout) could be imported [1] under measures that did not require removal of head and gills or post-arrival processing restrictions.

1.5 Commodity definition

The commodity considered in this risk analysis is eviscerated salmonids from Australia, produced and processed under the already approved regulatory systems which currently provide for market access into New Zealand for headed, gilled and gutted salmonids. The 1999 AQIS risk analysis reviewed the distribution and significance of salmonids in Australia, and the fish health surveillance programmes within individual states and territories.

This risk analysis will consider the biosecurity risks associated with imports of trout products, although non-biosecurity eligibility restrictions may affect eventual market access conditions for trout products.

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1 Although the 1999 AQIS risk analysis recommended to allow imports of uncooked salmonids for human consumption, the recommendations have not, to date, been implemented. Australia's zoosanitary measures for imports of salmonids for human consumption remain those subject to dispute between Canada and Australia. The World Trade Organisation Dispute Settlement Body Panel Report of 12 June 1998 summarised the Australian measures as:

- Quarantine Proclamation 86A of 19 February 1975 which prohibits importation of fresh, chilled or frozen salmon;

- The AQIS 1988 *Conditions for the importation of salmonid meat and roe into Australia* , which allow salmonids to be imported if treated according to the following time temperature parameters (temperatures relate to oven temperatures):

  35ºC for 7 hours
  40ºC for 5 ½ hours
  50ºC for 3 hours
  60ºC for 1 hour
  70ºC for 15 minutes
  120ºC for 12 minutes

The WTO Report concluded that the Australian measures were not based on a risk assessment, created arbitrary or unjustifiable distinctions in the level of sanitary protection considered to be appropriate in different situations, and were more trade-restrictive than required to achieve its appropriate level of sanitary protection.

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AQIS's import risk analysis on non-viable salmonids and non-salmonid marine finfish. http://www.aqis.gov.au/docs/qdu/salmontoc.htm

2 Hazard identification

2.1 Health status of Australian salmonids

There is evidence that the following disease agents/diseases may be present in salmonids in Australia (AQIS, 1999):

- *Aeromonas salmonicida* (some atypical strains)
- *Edwardsiella tarda*
- *Yersinia ruckeri* (some strains)
- *Vibrio anguillarum* (some strains)
- *Vibrio ordalii* (not in association with disease)
- *Kudoa thyrsites*
- Epizootic haematopoietic necrosis virus (EHNV)
- Lymphosarcomas in Atlantic salmon in Tasmania
- Aquabirnavirus (not in association with disease)
- *Paramoeba sp.*
- Streptococcosis

2.2 Health status of New Zealand salmonids

The health status of New Zealand salmonids has been reviewed during the previous MAF and AQIS risk analyses (MAF, 1997; MAF, 1999).

2.3 Diseases of potential concern

The diseases of potential concern during imports of salmonid products are those which occur in Australia but not in New Zealand, and include:

- EHNV
- *Aeromonas salmonicida* (atypical strains)
- Lymphosarcomas in Atlantic salmon in Tasmania
- Streptococcosis

Although there may be minor strain differences in several other agents that occur in both countries, they are excluded from consideration based on the absence of clinical evidence suggesting significant differences in pathogenicity and virulence.

References

AQIS's import risk analysis on non-viable salmonids and non-salmonid marine finfish. http://www.aqis.gov.au/docs/qdu/salmontoc.htm

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3 Risk Assessment

3.1 Epizootic heamatopoietic necrosis virus

3.1.1 Review of epidemiologically important disease information

3.1.1.1 Health status of Australia

EHN is a disease of redfin perch (*Perca fluviatilis*) and rainbow trout (*Oncorhynchus mykiss*) caused by an iridovirus. The disease is designated as notifiable by the OIE (OIE, 1997).

Epidemics cause serious mortalities in redfin perch in Victoria and New South Wales, (Langdon et al, 1986; Langdon et al, 1988). Disease has also been reported in South Australia. Atlantic salmon are experimentally susceptible (Langdon, 1989).

3.1.1.2 Prevalence in Australia

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). Although most disease is seen in growing rainbow trout, virus is recovered from older fish during outbreaks (Whittington et al, 1999).

Epidemics occur in the summer when water temperatures are relatively high, generally between 11-17°C (Langdon et al, 1986; Whittington et al 1994). The incubation period is also affected by environmental temperature (Whittington and Reddacliff, 1995).

EHNV may have contributed to declines in native fish populations, including galaxids, in Australia in recent decades (Langdon, 1989).

3.1.1.3 Tissue distribution and infective dose

Infection in rainbow trout may occur after cross-over from wild redfin perch or through movement of infected rainbow trout fingerlings (Whittington et al, 1999).

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 (Hyatt et al, 1991). The role that survivors of an outbreak play as carriers of infection has not been fully elucidated. There is no information on tissue distribution in clinically affected or carrier fish, but the virus has been isolated from pooled samples of liver, kidney, spleen, brain, gonad, ova, milt and gut in fish from an infected rainbow trout farm. Failure to detect EHNV in surviving fish 2 and 4 months after an outbreak suggests a low rate of carriage in rainbow trout (Whittington et al, 1994).

Redfin perch, which appear to be more susceptible to infection than rainbow trout, were infected and developed fatal disease within 28 days of bath exposure to 0.08 $TCID_{50}$/ml. Rainbow trout were resistant to infection by bath exposure to $10^{2.2}$ $TCID_{50}$/ml. (Whittington and Reddacliff, 1995). In another study, 1 out of 7 rainbow trout became infected following 1 hour immersion in 10^3 $TCID_{50}$/ml. (Langdon et al, 1988).

3.1.1.4 Survival and inactivation

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

3.2 Release assessment

The prevalence of EHNV infection may be very high in rainbow trout during epidemics of disease. Epidemics tend to occur in mid to late summer, though not annually, and have a restricted geographical distribution in NSW and Victoria. The prevalence of subclinically infected fish during epidemics may be around 4%. The prevalence in healthy fish in-between epidemics is unknown, but is probably very low.

During epidemics of EHN, fish smaller than harvest size are probably most severely affected. Subclinically-infected harvest-size fish, or those exhibiting very mild clinical signs, could be processed and pass inspection and grading. Those with externally visible lesions are likely to be trimmed and down-graded. The tissue distribution of EHNV in clinically healthy fish is unknown.

3.3 Exposure assessment

Redfin perch are more susceptible than rainbow trout, and probably represent the most likely exposure pathway for rainbow trout in this country. Redfin perch are common and widespread throughout New Zealand, inhabiting freshwater lakes, lagoons, ponds and sluggish rivers, although not in the Central Plateau of the North Island (McDowall, 1990).

Rainbow trout require high doses to become infected, but redfin perch are more susceptible. There is no information on infectious dose for galaxids. Although important information is lacking, in all likelihood the only means by which an infectious dose of EHNV could naturally arise would be if large amounts of rainbow trout from the endemic area were imported and furthered processed here, with direct discharge of wastes into the aquatic environment. EHNV probably survives well in the environment and on fomites, so may have the potential to accumulate under such circumstances.

3.4 Consequence assessment

Epidemics occur at temperatures of 11-17 °C. Such temperatures are probably common in many New Zealand waterways during the summer months.

The temperatures of Lake Taupo and its tributaries, where the major rainbow trout stocks of New Zealand are found, would not be limiting for EHNV. The ground water in-flow is an even 10°C ∓ 1°C all year round, and this temperature is also the bottom temperature of the lake all year round. Temperatures at 5-20 metres depth are between 20-22°C in summer. The Tongariro River has summer maximum of 13-17°C. The ranges reflect the diurnal shift. (Max Gibbs, National Institute of Water and Atmospheric Research, Hamilton. Personal communication with M. Stone, 7 September 1999.)

EHNV may also pose a threat to native galaxids, such as kokopu (whitebait).

3.5 Risk estimation

The likelihood of fish being infected with EHNV is probably moderate to high for rainbow trout harvested during summer in NSW, Victoria and South Australia; low for rainbow trout in this area at other times of the year; and negligible for other species and other locations in Australia. EHNV may be present in the viscera and brain in clinically infected fish. Whether a carrier state in adult rainbow trout exists is unknown. The possibility that virus may be present in eviscerated rainbow trout products from the endemic area must be considered.

EHNV appears to be relatively resistant to environmental inactivation, and so virus survival on imported product or in the environment would probably not be a limiting factor. Infection of redfin perch represents the most likely scenario for EHNV establishment in New Zealand, as rainbow trout are relatively resistant to infection. However, rainbow trout may be affected if establishment in redfin perch lead to epidemic mortalities here. The likelihood of an infectious dose for redfin perch building up in the environment is probably negligible if the dilution factors along the expected exposure pathways are not artificially circumvented. The only means by which this could possibly occur would be if large-scale processing of rainbow trout imported from the endemic area lead to accumulated scraps being discarded into an aquatic environment.

Heat treatment of imported product to 60°C for 15 minutes would ensure inactivation of EHNV.

If outbreaks of EHN were to occur in New Zealand, this would be a serious threat to fresh water fisheries resources, particularly redfin perch and rainbow trout populations. The potential for adverse impacts to native galaxids is a further serious concern.

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3.2 *Aeromonas salmonicida*

3.2.1 Review of epidemiologically important disease information

3.2.1.1 Health status of Australia

Typical *A. salmonicida* is not reported in salmonids in Australia.

Goldfish ulcer disease was reported on a Victorian farm in 1974 (Trust et al, 1980). Atypical *A. salmonicida* was first isolated in 1980. Transmission studies showed the organism caused septicaemia in Atlantic salmon, rainbow, brown and brook trout when inoculated intraperitoneally, and infection could be transmitted through cohabitation (Whittington and Cullis, 1988).

Atypical *A. salmonicida* was isolated from juvenile hatchery reared and wild-caught greenback flounder in Tasmania. No clinical disease was reported. (Whittington et al, 1995).

Aside from the above, atypical *A. salmonicida* has also been reported various species of fish in NSW, Western Australia, South Australia and Queensland (AQIS, 1999).

3.2.1.2 Prevalence in Australia

Clinical infection with *A. salmonicida* has not been reported in commercial salmonids in Australia, although strains of *A. salmonicida* present in that country are pathogenic for salmonids under experimental conditions (Peter Beers, Head of Aquatic Animal Section, Animal Quarantine Policy Branch, AQIS. Personal communication with M. Stone, 24 September 1999.) The prevalence of subclinical infections (covert carriers) in most parts of Australia is unknown. Intensive health surveillance and an absence of isolations from salmonids, as is the case in Tasmania, provide a strong assurance of a zero prevalence.

In general, where *A. salmonicida* is accepted as being endemic, hatchery water supply will have the greatest influence on the prevalence of covert infections in aquacultured salmonid populations. If hatchery water is obtained from wells or springs, it is likely to lead to a lower prevalence of covert carriers than if water is obtained from sources inhabited by wild fish. The effect on prevalence of covert carriers through movement to salt water is unknown. (Hiney et al, 1997.)

Data on prevalence of *A. salmonicida* in salmonid populations in other countries, and the factors influencing detected prevalence, have been previously reviewed (AQIS 1996 and 1999; Stone et al, 1997). This information related mainly to typical *A. salmonicida*.

3.2.1.3 Tissue distribution and infective dose

The clinical signs of furunculosis are those of systemic bacteraemia. These are likely to result in affected fish being downgraded.

Atypical *A. salmonicida* is probably less invasive, and clinical infections may manifest as locally erosive skin lesions without systemic invasion. Such lesions are also likely to result in trimming and downgrading.

The location of *A. salmonicida* in covertly infected fish remains uncertain. A model based on the known epidemiology has been proposed which suggests the sites of carriage in covert infections are those areas external to that defended by the immune system i.e. the mucus of the skin and the intestinal lumen. (Hiney et al. 1997).

The titre of typical *A. salmonicida* in mucus of apparently healthy salmonid fish may be 10^3 to 10^6 cfu/g (Hiney and Olivier, 1999; Cipriano et al, 1992).

The infectious dose for various salmonid species by a variety of routes has been previously reviewed (AQIS 1996 and 1999; Stone et al,1997). Some estimates of infectious doses that lead to transmission within pathways expected in nature (cohabitation, immersion and gastric intubation, reflecting ingestion) include long duration (3 weeks) immersion of Atlantic salmon in seawater with concentrations of 10^2 cfu/ml bacteria; intragastric intubation of Atlantic salmon with a dose of $> 10^5$ cfu/fish; immersion of 25 g rainbow trout in freshwater containing 10^8 cfu/ml for 12 hours; and intragastric intubation of Pacific salmon with 10^2 cells/kg.

However, this data exclusively relates to infectious dose of typical *A. salmonicida.* Atypical *A. salmonicida* is a less invasive organism, so infectious dose may be higher than for typical strains, all other influences being equal.

3.2.1.4 Survival and inactivation

These aspects have been previously reviewed, although once again the information relates only to typical strains (AQIS 1996 and 1999; Stone et al, 1997). *A. salmonicida* may survive for long periods in favourable environments outside the host, particularly following a high-dose initial inoculation and in the absence of competing microorganisms. Survival potential following low dose inoculation into a contaminated environment is probably much lower, although *A. salmonicida* may have the ability to enter a dormant state during prolonged periods of survival outside the host. Survival for long periods on salmonid flesh has been demonstrated at various temperatures (for 28 days at 4°C, and 49 days at -10°C).

3.2.2 Release assessment

The prevalence of atypical *A. salmonicida* infections in salmonids in Australia is unknown, although in Tasmania it is probably zero. Naturally occurring clinical disease in Australian salmonids has not been reported.

Inspection during processing ensures fish with clinical infections are subject to trimming of skin lesions or downgrading if showing signs of bacteraemia. Subclinically infected fish will pass inspection without trimming or downgrading.

The sites of carriage of atypical *A. salmonicida* in subclinically infected fish are probably the gastrointestinal tract and the mucus layer of the skin, whereby the organism escapes the host's immunological defences. Although it is known that relatively high titres of typical *A. salmonicida* can be carried in the mucus of healthy fish, it is not known how this data relates to atypical *A. salmonicida*. Thorough washing to remove mucus and evisceration is likely to significantly reduce the amount of infectivity.

3.2.3 Exposure assessment

Typical *A. salmonicida* may survive for relatively long periods on salmonid flesh and in favourable environments outside the host, although low doses deposited into contaminated environments would probably not retain the ability to infect fish for long. Very large amounts of typical *A. salmonicida* are necessary to produce concentrations that will provide an infectious dose for salmonids by immersion. Much lower doses are required to produce infection by ingestion in Atlantic and Pacific salmon, although trout may be refractory to infection by this route. It is not known how this information relates to atypical *A. salmonicida*.

3.2.4 Consequence assessment

Strains of atypical *A. salmonicida* present in Australia have been experimentally demonstrated to be pathogenic for salmonids present in New Zealand. Atypical *A. salmonicida* has a wide host range, and so the potential for infections in other fish must also be considered.

The impact of the organism's presence in Australia has been very low.

3.2.5 Risk estimation

The likelihood of significant quantities of atypical *A. salmonicida* being present on product derived from Australian salmonids that were clinically healthy at slaughter and that have been eviscerated and washed clean of mucus is very low. In the case of product from Tasmania, the likelihood is negligible. The likelihood of quantities of atypical *A. salmonicida* sufficient to provide an infectious dose for fish in New Zealand building up in the environment as a result of imports of such a product is negligible. The likelihood of significant adverse consequences from introduction of the strains of atypical *A. salmonicida* that are present in Australia is also low, certainly much lower than the expected impact for wild and aquacultured salmonids from introduction of typical strains. The overall risk is negligible.

References

AQIS's import risk analysis on non-viable salmonids and non-salmonid marine finfish. http://www.aqis.gov.au/docs/qdu/salmontoc.htm

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3.3 Lymphosarcomas in Tasmanian Atlantic salmon

3.3.1 Review of epidemiologically important disease information

3.3.1.1 Health status of Australia

The only published reference to lymphosarcomas in Tasmanian Atlantic salmon is the AQIS risk analysis, which referred to the disease as a plasmacytoid leukaemia-like condition (AQIS, 1999). The aetiology of the condition in Australia is unknown. The manifestation as lymphosarcomas suggests a retrovirus may be involved, although there is no direct evidence to support this. This would suggest a similarity to plasmacytoid leukemia of chinook salmon in British Columbia (Kent et al, 1990) which is caused by an oncogenic retrovirus given the name salmon leukemia virus (Eaton and Kent, 1992). Pathology similar to that of plasmacytoid leukaemia has also been observed in chinook salmon reared in California and Washington state (Kent, 1992).

3.3.1.2 Prevalence in Australia

No reliable estimate of prevalence of the condition in Australia has been made. One series of estimates made from industry records of gross kidney lesions at harvest found a variation between populations of between 0.05% to 0.27% in market size fish from the same original source (AQIS, 1999). Given the gross method to make the diagnosis, it may represent an under-estimate of the prevalence in that particular population.

3.3.1.3 Tissue distribution and infective dose

No aetiological agent has been isolated for lymphsarcomas in Tasmania, and there is no information on tissue distribution and infective dose. Transmission studies have not been undertaken (AQIS, 1999).

Mortality is rare, with most diagnoses made by post-mortem inspection at harvest. The pathology is typically solid focal tumours in the kidneys, occasionally involving the whole kidney. Lesions may also occur in the liver and muscle. Lesions in the choroid of the eye have been seen at least once, and a leukaemic pattern in the blood has also been seen, though rarely. Neoplastic infiltratrion in the gut, pancreas and spleen, as found with plasmacytoid leukaemia in Canadian chinook salmon, have not been seen in Tasmanian Atlantic salmon (AQIS, 1999).

In Canada, plasmacytoid leukaemia occurs in farmed and wild-caught chinook salmon, typically one year after entering sea water (Eaton et al, 1994). 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. In fish exhibiting exophthalmia the proliferation is also apparent in the periorbital connective tissues, ocular muscles and choroid gland (Kent et al, 1990).

3.3.1.4 Survival and inactivation

As no aetiological agent has been isolated for lymphosarcomas of Atlantic salmon in Tasmania, there is no information on survival and inactivation.

3.3.2 Release assessment

In Australia the condition has only been reported in Atlantic salmon from Tasmania. Gross post-mortem lesions at slaughter suggest 0.27% of fish may be affected.

Based upon the distribution of pathology, evisceration would be expected to remove the majority of infectivity (assuming an infectious aetiology). There could be some residual infectivity associated with kidney tissue left in the carcass after evisceration, lesions in muscle, and in rare cases associated with blood or tissues of the eye.

3.3.3 Exposure assessment

Transmission studies have not been completed. There is no specific information on which to base an exposure assessment.

3.3.4 Consequence assessment

The condition has apparently not had a significant impact in the Atlantic salmon aquaculture industry in Tasmania. There have been no studies to investigate effects of the condition on productivity. The disease appears to be regarded as an incidental finding during slaughter.

Certainly the disease is more of a concern in chinook salmon raised in net-pens in British Columbia. If the conditions in Canada and Australia are related, the impact in New Zealand could be more significant than that seen in Australia because the industry here is based on net-pen culture of chinook salmon.

3.3.5 Risk estimation

The risk of lymphosarcomas must be considered in the context of imports of Atlantic salmon from Tasmania. The prevalence appears to be relatively high. An infectious aetiology has not been confirmed, but an unidentified retrovirus may be involved. Because no infectious agent has been identified, critical information on tissue distribution, infectious dose and survival is lacking. The distribution of pathology, including kidney, muscle, blood and eye, suggests that residual infectivity may remain in eviscerated Atlantic salmon. As a group, retroviruses survive poorly outside the living host.

There is little information available on lymphosarcomas in Tasmanian Atlantic salmon, so an accurate estimate of the likelihood of introduction and establishment is no possible. A reasonable assumption might be that, given the apparent lack of impact and spread in Australia despite the low key approach taken to the disease's presence, it is probably less likely to be spread by eviscerated fish products than other more infectious diseases previously considered. On that basis the likelihood of introduction is very low.

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3.4 Streptococcosis

3.4.1 Review of epidemiologically important disease information

3.4.1.1 Health status of Australia

Streptococcosis is a septicaemia that occurs both sporadically and as epidemics, caused by both alpha-haemolytic and beta-haemolytic strains of *Streptoccoccus* spp., as well as *Enterococcus* spp. 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. The infections occur in many species of marine and fresh water fish, and in many countries (Kitao, 1993).

Epidemics of streptococcosis occur in rainbow trout in Australia (Carson, 1990). Isolations of related organisms from various species of fish reported in Australia (AQIS, 1999) include: *Enterococcus seriolicida* in Victoria; *Lactobacillus piscicola* and *Streptococcus* sp. in NSW; *Vagococcus salmoninarum* and *Lactococcus garviae* (not seen since 1992) in Tasmania; *Enterobacter* sp. in South Australia; and *Streptococcus iniae* from barramundi in Queensland.

3.4.1.2 Prevalence in Australia

There is no information on prevalence in Australian salmonids, although it would seem that in recent years the impact of this disease in aquaculture has been successfully managed through attention to various aspects of husbandry. Atlantic salmon and brown trout are less susceptible to clinical streptococcosis than rainbow trout, and changing species in circumstances where the disease has made rainbow trout culture unprofitable appears to have been a successful strategy in Tasmania (Munday et al, 1993).

The epidemiology appears to be that of an opportunistic infection by a group of related bacteria with fish-pathogenic properties. Disease tends to manifest when young fish are stressed, particularly by high temperatures. Studies involving Australian isolates demonstrated that manifestation of disease was temperature dependent (Munday et al, 1993). The source of infection may be the environment (bacteria released from diseased fish, which then survive in sea water or mud), carrier fish, or contaminated diets (Kitao, 1993).

3.4.1.3 Tissue distribution and infectious dose

The clinical signs of streptococcosis are those of bacteraemia, with widespread haemorrhage and inflammation. Bacteria are widely distributed in such fish, and can be seen on smears from kidneys and other organs. The brains are a good site for detection of the causative bacteria (Kitao, 1993), presumably reflecting widespread dissemination in bacteraemic fish and late post-mortem invasion by other contaminants at this site.

A wide variety of studies have examined various horizontal routes of transmission, by inoculation, immersion and ingestion. Transmission occurs readily when fish are stressed.

3.4.1.4 Inactivation and survival

The causative organisms of streptococcosis have been shown to survive for long periods in water and mud surrounding fish farms following outbreaks of disease (Kitao, 1993).

3.4.2 Release assessment

The prevalence of streptococcosis in market size fish is likely to be very low. Diseased fish would not pass inspection and grading because of the obvious clinical signs. Infectivity associated with carrier fish would be low, and would be further reduced by evisceration.

3.4.3 Exposure assessment

The organisms involved in streptococcosis survive well in the environment. The infectious dose in young fish stressed by high temperatures may be low. However, the low level of infectivity associated with the imported commodity and the various dilution effects along the exposure pathway would most likely ensure that the risk of an infectious dose arising as a result of imports is low.

3.4.4 Consequence assessment

The nature of the group of organisms involved in streptococcosis suggests that the range of organisms already present in New Zealand probably interact with fish to produce non-specific opportunistic infections under similar stressful conditions associated with outbreaks of streptococcosis. For instance, the bacteria *Vibrio ordalii, Aeromonas hydrophila, Hafnia alveii*¸and *Nocardia asteroides* all occur in New Zealand and cause infections in stressed fish. It seems probable that organisms associated with streptococcosis in other countries also occur here already. Adverse consequences for fish health, over and above the clinical manifestations already seen, seem unlikely.

3.4.5 Risk estimation

The level of infectivity in imported eviscerated fish is likely to be low. The organisms survive well and may infect stressed fish when present at low levels. However, opportunistic pathogens that interact with fish in similar ways are already present here, so additional or unexpected adverse consequences are unlikely. The risk posed by organisms that cause streptococcosis is negligible.

References

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4 Exposure pathways

A number of criteria would have to be met if eviscerated fish for human consumption are to be the vehicle for introduction of fish diseases (MacDiarmid, 1994). The risk assessment has discussed the likelihood of imported product being contaminated with exotic pathogens, and the consequence of introduction. A discussion on exposure pathways is warranted as it may help to put the risks into a more general perspective.

4.1 Waste management: scraps, packaging and wastewater

The form of the imported product would influence the amount of waste generated. In general, the amount of uncooked product likely to be discarded would be related to the amount of processing prior to import. For instance, processed products such as fillets, steaks and other consumer-ready portions would result in fewer scraps. Such products would also require less preparation in New Zealand kitchens, thereby reducing the potential for contamination of kitchen wastewater. On the other hand, these products would tend to generate higher levels of packaging. In general, in each scenario the reverse is true for imports of whole eviscerated fish.

Imported fish will be a relatively expensive commodity, and this will affect consumer behaviour with regard to discarding scraps. Heads, skins, fins and frames might be discarded uncooked, although often the whole fish would be cooked, thereby inactivating any associated exotic pathogens. Of the uncooked scraps that are discarded, most would be either composted, incinerated or buried at landfills. Similarly, most packaging would also end up either incinerated or buried in landfills. Only a very small proportion of imported product is likely to contact an aquatic environment through disposal of uncooked scraps or packaging.

Kitchen wastewater may become contaminated during processing of raw imported product. Most kitchen wastewater generated in New Zealand enters metropolitan sewerage schemes, where any contamination would be subject to massive dilution effects. The aquatic environment within such schemes (high levels of faecal coliforms, household detergents etc) would adversely affect the survival of most exotic pathogens of fish.

4.2 Distribution of imported product and susceptible species

The distribution of imported product would most likely follow population distributions, with the majority being consumed in metropolitan areas. The high levels of liquid and solid waste generated in such centres would ensure large dilution effects.

In general, the major population centres are remote from aquatic animal habitats where species susceptible to the diseases of concern would be found. The tourist destinations of Rotorua and Taupo are an exception. Ironically, the ban on sale of fish taken in New Zealand, which receives strong support from the recreational fishing sectors of these communities, might create a demand in restaurants in these areas for imported product.

Several reasons make the prospect of picnickers discarding imported product directly into a salmonid habitat, thereby by-passing all the expected risk reduction mechanisms in the normal exposure pathway, an unlikely scenario for disease introduction. Only a small proportion of imported product will be consumed by picnickers in an outdoor setting. That which is consumed in such settings would be likely to have been pre-prepared, and so

subjected to heat treatment. Imported product will be expensive, and therefore unlikely to be discarded in significant quantities. Although head, frames and skin would be discarded, uncooked whole fish are an unlikely picnic food. Finally, the risk assessment has concluded that if exotic pathogens are present on the product, they will be in very low concentrations. These factors combine to make the likelihood of a susceptible fish receiving an infectious dose of an exotic pathogen as a result of discarded food scraps negligible.

4.3 Potential accumulation

If imported product is contaminated with low levels of exotic pathogens, the low proportion of product discarded as uncooked scraps and the large dilution effects for scraps, packaging and wastewater would ensure the risk of an infectious dose arising in the aquatic environment of a susceptible species is negligible.

However, this might not be the case if a single premises were to process or re-package large amounts of imported product, and wastes associated with these operations were discharged directly into a waterway. The risk of an infectious dose arising in the waterway may not be negligible. Although the likelihood of this scenario actually happening is probably small, the potentially severe adverse consequences demand that it should be actively avoided.

4.4 Control over post-arrival processing

Earlier MAF risk analyses (MacDiarmid, 1994; Stone et al, 1997) acknowledged the potential risks in respect of accumulations of scraps, packaging and wastewater in association with processing or re-packaging bulk imported product. MAF has developed standards to manage this risk by requiring any processing or re-packaging of bulk product after arrival in New Zealand to occur in registered premises. Registered premises must have approved waste management practices, including wastewater discharge into a metropolitan sewerage scheme.

References

MacDiarmid S C. The risk of introducing exotic diseases of fish into New Zealand through the importation of ocean-caught Pacific salmon from Canada. MAF Regulatory Authority. 1994.

Stone M A B, MacDiarmid S C, Pharo H J. Import health risk analysis: salmonids for human consumption. Ministry of Agriculture Regulatory Authority, New Zealand. 269 pages. 1997.

MAF. Import health standard for the importation into New Zealand of salmonids for human consumption from specified countries (SHC).

MAF. MAF Regulatory Authority Standard 154.02.16 Transitional facilities for processing imported salmonids.

5 Expected Volume of Trade

5.1 Current volume of imports

Since approval of the Australian Quarantine and Inspection Service export certificate under the MAF *Import health standard for the importation into New Zealand of salmonids for human consumption from specified countries (SHC),* there have been no importations of headed, gilled and gutted salmonids from Australia (Source: R. Weston, New Zealand Customs Service, personal communication with M. Talbot, Ministry of Foreign Affairs and Trade, 14 September 1999).

5.2 Price comparison for salmon

The form of salmon product most commonly traded internationally is head-on, gilled and gutted fish.

The distribution price in Sydney of Royal Tasmanian Salmon is currently AUD$16.90 per kg of head-on, gilled and gutted product. The price varies between AUD$15.90 and AUD$17.90 at different times of the year. (Source: Peters, Sydney Fish Market, personal communication with M. Stone, 15 September 1999.)

The distribution price in Auckland of New Zealand King Salmon is currently NZD$11.20 per kg of head-on, gilled and gutted product. The price does not significantly vary during the year. (Source: Paul Steere, Chief Executive, New Zealand King Salmon Company, personal communication with M. Stone, 15 September 1999.)

5.3 Trout

It is currently illegal to sell trout taken in New Zealand. If New Zealand consumers are given the choice to purchase imported trout, the novelty factor and the lack of local competition might ensure some level of market uptake for imported product. Following the announcement in August 1998 of the decision to allow imports of salmon, trout and char, MAF received interest from the commercial sector for imports of trout from Australia for distribution to restraurants.

The 1998 MAF risk analysis considered consumption patterns of trout in the United Kingdom, where there is a ready supply from local and imported aquaculture sources. Similar levels of consumption here as in the United Kingdom would result in imports of approximately 800 tonnes. This estimate does not take into account eating habits of the respective populations, or the retail price of trout relative to the other fish products with which it would compete.

Australia currently produces 2,118 tonnes of rainbow trout, of which 3-4% is exported and the rest sold on the domestic market.

At the present time, imports of trout into this country are restricted to private consignments of less than 10kg per person, unless by dispensation from the Minister of Conservation. The current parliamentary process examining the import and the sale of trout might have a significant impact on the eventual volume of imports.

5.4 Estimate of volume

Given the present commercial incentives, it seems unlikely that any significant quantities of salmon will be imported from Australia into New Zealand.

On the other hand, there may be significant interest in trout, but the actual level of imports may eventually be determined by factors other than the commercial ones. If trout were allowed to be sold in New Zealand, the likely volume of imports could be somewhere between 200-500 tonnes annually.

6 Risk assessment conclusions

6.1 The risk of atypical *Aeromonas salmonicida* being introduced into New Zealand from Australia is negligible so long as:

- fish are harvested from populations which are not experiencing unexpectedly high prevalence of infections such as would occur during an epizootic of disease;
- processing ensures any fish with lesions or other clinical signs of infection are trimmed and/or downgraded;
- all fish are eviscerated and thoroughly washed of mucus during processing.

6.2 The risk of lymphosarcomas of Tasmanian Atlantic salmon being introduced into New Zealand if eviscerated fish were imported is very low.

6.3 Importation of eviscerated rainbow trout from the EHNV endemic area of Australia might lead to product harbouring the pathogen being imported. If imported product were directly distributed to consumers, dilution effects along the exposure pathway would make the likelihood of an infectious dose arising in the aquatic environment negligible. However, further processing or packaging of imported product could result in accumulation of potentially contaminated scraps, packaging and wastewater. If waste from a facility undertaking further processing or packaging of large quantities of imported product were discharged directly into a waterway habitat, the likelihood of an infectious dose arising might not be negligible. The consequences of introduction could potentially be severely adverse, and warrant a conservative approach to risk management.

6.4 Heat treatment (60ºC for 15 minutes) of eviscerated trout products from EHNV endemic areas would also reduce the risk to negligible levels.

6.5 The risk posed by organisms associated with streptococcosis is negligible. The probability of introduction is low, but of more relevance is the probable absence of any significant consequence associated with introduction. This disease is caused by opportunistic pathogens infecting stressed fish, and numerous other bacteria present in the environment already fulfil a similar role.

7 Recommendations

7.1 Importation of eviscerated salmonids from Australia be permitted subject to certification by AQIS (except private non-commercial consignments) that the product is derived from fish:

- belonging to 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 agency;
- that were not slaughtered as an official disease control measure as a result of an outbreak of disease;
- that were processed in 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) gutted; and
  (ii) individually inspected and graded, ensuring the product for export is free from visible lesions associated with infectious disease and fit for human consumption.

7.2 In addition to the above measure, rainbow trout[2] from the EHNV endemic area must be:

EITHER commercially packaged for direct retail sale;

OR subject to post-arrival quarantine restrictions such that re-packaging and processing prior to distribution to consumers must be performed in premises with appropriate waste management systems registered and supervised by MAF;

OR subject to heat treatment to raise the internal core temperature to 60ºC for 15 minutes.

[2] The measures stated here are only those considered appropriate to manage the biosecurity concerns. As stated earlier in the risk analysis, imports of trout products into New Zealand currently face unrelated restrictions.