Rev. sci. tech. Off. int. Epiz., 16 (1), 135-145

Contamination of fish products: risks and prevention

P.M. Hine [1] & S.C. MacDiarmid [2]

(1) National Institute of Water and Atmospheric Research, P.O. Box 14-901, Kilbirnie, Wellington, New Zealand
(2) Ministry of Agriculture and Fisheries Regulatory Authority, Ministry of Agriculture, P.O. Box 2526, Wellington, New Zealand

Summary
The risks of contamination of finfish products with active pathogens largely depend on the type of product concerned and disposal methods of the importing country. Frozen fish used as bait or to feed high-value species present the greatest risk as vehicles of contamination because they are unprocessed. Freezing preserves viral – and some bacterial – pathogens, thus the use of such fish as bait can introduce those pathogens into natural waters. Conversely, processed fish, particularly fillets, which have been heat-treated or cooked, present the lowest risk. If fish are processed after importation, care must be taken to ensure effective waste disposal, with particular attention to the prevention of scavenging by avian vectors and drainage from landfills into natural waters. Liquid waste should be disinfected and disposed of well away from natural waters.

Keywords
Disinfection – Disposal – Finfish – Pathogens – Processing – Risk analysis.

Introduction

The principles of epizootiology applied to diseases of mammals and birds can, in many respects, be applied to fish. However, pathogens in aquatic species, unlike those of terrestrial species which face the barriers of desiccation during transmission between hosts, exist in a medium which supports them and can actively transport them. Trade in finfish differs from trade in molluscs and crustaceans because it involves freshwater species, particularly salmonids, carp and catfish. Pathogens in freshwater products may pose a higher risk than pathogens in marine products, because of the close association of humans with fresh water. As most of the information on which risk can be assessed relates to temperate freshwater fishes, particularly salmonids, these will be dealt with in detail. When considering importation of marine or tropical species, authorities in importing countries must act conservatively in potentially high risk situations.

Importation decisions should be based on the outcome of risk analyses, which calculate the cumulative probabilities of survival of a potential pathogen, from the origin of the product in the exporting country to the disposal of waste in the importing country. Estimation of probabilities involves not only the survival of the potential pathogen through processing, its introduction into waters with susceptible hosts and the establishment of infection, but also the likelihood of detection and prevention of introduction. The factors determining the latter, such as evaluation of the competent authority in the exporting country, are common to all trade in animal products. The Office International des Epizooties (OIE) listed diseases and code of practice are given in the OIE *International Aquatic Animal Health Code*, and methods of diagnosis are detailed in the OIE *Diagnostic Manual for Aquatic Animal Diseases*. This paper considers the survival of potential pathogens through processing, and the likelihood of their introduction into, and establishment in, the importing country, how such risk may be minimised, and how risk analysis may be applied to such situations.

Survival of pathogens during processing

Fish are traded either live, chilled, frozen, pickled, smoked, cooked or canned. The survival of pathogens under different conditions is illustrated in the Appendix. Freezing preserves the infectivity of viruses, and the lower the temperature, the greater the preservation. Some bacterial pathogens

(*Aeromonas salmonicida, A. hydrophila, Pseudomonas fluorescens, Edwardsiella ictaluri, E. tarda*) also survive freezing down to between –10°C and –20°C for 20 to 60 days, but whether long-term survival occurs at lower temperatures is unclear. Freezing and thawing may also inactivate some bacterial pathogens, such as *A. salmonicida* and *Renibacterium salmoninarum*, sometimes reducing numbers by orders of magnitude (42). *Streptococcus* sp. may be transmitted in frozen fish fed to yellowtail (*Seriola quinqueradiata*), in which case it can survive freezing for up to six months (52). The spores of *Myxobolus cerebralis*, the cause of salmonid whirling disease, survive at –20°C for more than three months (17).

Heating rapidly inactivates infectious haematopoietic necrosis virus (IHNV) and spring viraemia of carp virus (SVCV), but takes longer to inactivate viral haemorrhagic septicaemia virus (VHSV), whilst infectious pancreatic necrosis virus (IPNV) requires pasteurisation temperatures (90). *A. salmonicida* is readily killed above 50°C, but *R. salmoninarum* remains viable at temperatures approaching pasteurisation (91). Hot smoking kills the spores of *M. cerebralis* (96).

The effect of pickling in brine on viral and bacterial pathogens is unclear, but it is likely that pathogens often found in the marine environment, such as IPNV and *Vibrio* spp., will survive better than those of freshwater fishes, such as *E. ictaluri*. Pickling in brine does not destroy halophilic bacteria (7), but the effect this method has on fish pathogens is unclear. *A. salmonicida* appears to be sensitive to low pH: IHNV and *R. salmoninarum* less sensitive; and IPNV and mycobacteria relatively insensitive (91). It appears unlikely that pathogens would survive in pickled or brined products for long periods.

Unprocessed whole fish pose the highest risk of contamination because the gut and viscera are the sites of greatest infection with viruses, bacteria and metazoan parasites. Whole frozen fish are traded for human consumption after processing in the importing country, or as bait, for fish or pet food, or to make fish meal. There has been very little research into diseases of baitfish, most of which are clupeiforms, although herrings (*Clupea harengus*) have been subject to several studies. The parasites and pathogens of herrings and baitfish include VHSV (48, 49, 50), viral erythrocytic necrosis (VEN) (47), *Vibrio anguillarum* (9), pasteurellosis (23), *Pseudomonas anguilliseptica* infection (41), *Streptococcus* sp. (98), *Ichthyophonus* (73), and the myxosporean *Kudoa thyrsites* (38). VHSV, *V. anguillarum*, pasteurellosis, *P. anguilliseptica* and *Ichthyophonus* all cause epizootics, and *K. thyrsites* causes myoliquefaction, destroying product quality (92).

The risk of transmission of gastrointestinal parasites and pathogens can be reduced by gutting and heading fish, and by extracting the kidney, liver and spleen, which are major sites of viral and bacterial infection. Care must be taken to remove the entire kidney, as it lies directly below the spine and is not removed at evisceration. Heading removes the risk from infections of the nervous system, such as viral encephalopathy and retinopathy (55), and of the cranium, such as *M. cerebralis*. Removal of the spine and ribs further reduces the risk from *M. cerebralis*, and removal of skin and fins reduces the foci of infection in haemorrhagic septicaemias. However, viral (14, 56, 57, 58, 78) and bacterial (4, 54, 88) pathogens may remain in fillets.

Introduction and establishment in waters of the importing country: risks and prevention

Imported processed fish may be sold, unmodified, for human consumption, while whole or partially processed fish may be further processed (see below). Whole frozen fish for bait or fish food may be introduced directly into natural waters. Such a practice involves a high risk, and fish fed to other fish may transmit diseases, such as vibriosis (9), streptococcosis (52, 81, 98) and ichthyophoniasis (11). VHSV may be common in marine fishes (48, 49, 50, 71, 74): isolates of the virus survive a wide range of salinities (59), and therefore VHS may be transmitted and become established by the use of unprocessed marine fish as feed for other fish species. In 1995, frozen pilchards (*Sardinops sagax neopilchardus*) imported from California, Chile, Peru and Japan were fed to sea cage tuna (*Thunnus maccoyii*) in Australia, resulting in an epizootic among pilchards, starting off the coast of South Australia and spreading along approximately 6,000 km of coastline of Australia and New Zealand. Although a herpesvirus associated with these mortalities (30, 93) has yet to be demonstrated to be the cause of the epizootics, and despite the fact that Evans' postulates of disease causation (83) have not yet been fulfilled, scientists investigating the mortalities believe that the introduction of whole frozen pilchards into waters with resident pilchard populations poses a high risk of introducing such a pathogen.

Post-importation processing carries a high risk if waste products are introduced into natural waters in which potential pathogens may survive. Ideally, solid wastes should be incinerated. If this is not possible, they should be buried on land where there is no drainage into nearby natural waters. While disposal on the surface of landfills may lead to dispersal of pathogens by avian vectors (17, 22, 42, 61), the reduction in pH which accompanies putrefaction leads to inactivation of many pathogens. *Renibacterium salmoninarum*, *A. salmonicida*, *Mycobacterium chelonei* and IHNV are inactivated within hours at pH 3.8-4.3 (91). However, IPNV is resistant to low pH and may survive for many days under the same conditions.

Water used in processing should be treated prior to disposal (2, 5, 13, 31, 33, 39, 40, 60, 79, 80, 87), but the use of toxic

chemicals should be avoided, particularly if waste is discharged directly into waterways or the sea. Ultraviolet light, ozone and chlorine do not pollute natural waters, but gases from the treated water must be allowed to evaporate off in well ventilated surroundings before the water is discharged. If freshwater is ponded before discharge, care must be taken to keep eels (*Anguilla* spp.) out, as they can tolerate mildly toxic water, can become infected by many serious pathogens (IHNV, VHSV, IPNV, eel vesiculoviruses, eel herpesvirus, *A. salmonicida, A. hydrophila, A. jandaei, P. anguilliseptica, E. tarda, V. anguillarum, V. furnissii, V. vulnificus*), and can migrate overland between waterways. Working surfaces and instruments used in fish processing plants should be regularly disinfected, as should the aquaculture establishments themselves (87).

Cooking food is likely to destroy fish pathogens in contaminated fish, although lightly cooked or raw fish may still contain viable organisms. Although some fish pathogenic viruses (22, 42, 61, 77) and also whirling disease spores (17) can survive passage through homeotherms, the low pH of the stomach makes it unlikely that they will survive passage through humans. Uncooked fish also poses a greater risk if disposed of at sites where birds may scavenge.

Should viable viral or bacterial pathogens enter natural waters, they are unlikely to be killed by temperature or pH, but salinity-intolerant species (IHNV, *A. salmonicida, Edwardsiella* spp.) may not survive for long in sea water, and halophilic species (IPNV, *Vibrio* spp.) may be killed by freshwater (Appendix). Many pathogens survive for several days or months in estuarine waters and in sediment. Channel catfish virus disease (CCVD) rapidly loses infectivity in mud, IPNV loses infectivity more slowly, possibly because of sediment microbial flora, and *R. salmoninarum* is not found in sediment near fish farms, but can survive in sediment in experimental tanks (Appendix). *M. cerebralis* spores are viable after more than five months in mud at 13°C (17).

Even if susceptible hosts exist in waters contaminated by potential pathogens, they still have to be exposed to an infective dose of the organism for infection to become established. Studies on disease induction by immersion in pathogens (6, 26, 46, 67, 69, 89) cannot be readily related to the infective dose in natural waters because factors relating to the environment (temperature, pH, salinity, hardness, microbial flora, water flow rates), the host (species, age, immunocompetence, stock density) and pathogen (strain, virulence, titres) all determine the infective dose. When carrying out a risk assessment it must be concluded that the greater the amount of potential pathogen introduced into the proximity of susceptible hosts, the higher the risk that infection will become established.

Carrying out a risk analysis

An analysis of the likelihood of introducing exotic diseases into New Zealand through the importation of Canadian table salmon (44) provides an example of how a risk assessment may be carried out. For many years Canadian trade officials had been seeking access to New Zealand for a number of commodities processed from wild, ocean-caught Pacific salmon (*Oncorhynchus tshawytscha*). On account of their isolation from northern hemisphere salmon fisheries, the fish stocks of New Zealand are considered to be free from a number of diseases present on the west coast of North America. For this reason, New Zealand authorities had adopted a 'zero risk' stance with respect to imports of uncooked Canadian salmon products and refused access to the New Zealand market. The arguments against granting access had been based solely on a perceived microbiological threat. That is, if a disease could be identified as present in Canadian fish stocks, that was considered sufficient reason to deny access. No attempt was made actually to assess whether the introduction of disease was likely. It was sufficient to postulate the possibility. However, in 1992 the New Zealand Ministry of Agriculture agreed to carry out an analysis of the risks of introducing exotic fish diseases through the importation of headless, eviscerated, wild, ocean-caught Pacific salmon from Canada.

An examination of the literature and consideration of submissions from local salmon farmers, recreational fishers and conservation groups led to a decision to include in the risk analysis some 23 diseases which are present in North American fish stocks. The diseases included:

- infectious pancreatic necrosis
- infectious haemorrhagic necrosis
- viral haemorrhagic septicaemia
- viral erythrocytic necrosis
- erythrocyte inclusion body syndrome
- pancreas disease
- plasmacytoid leukaemia
- *Aeromonas salmonicida*
- bacterial kidney disease
- enteric redmouth
- salmonid rickettsial septicaemia
- rosette agent
- *Loma salmonae*
- *Enterocytozoon salmonis*
- proliferative kidney disease
- edwardsiellosis
- *Kudoa thyrsites*
- *Ceratomyxa shasta*
- *Herpesvirus salmonis*
- vibriosis
- Hitra disease (*Vibrio salmonicida*)
- *Henneguya salmonicola*
- parvicapsular disease.

It was recognised at the outset that not every disease posed an equal threat. Each disease differs in the likelihood of being introduced in the commodity and in the adverse impact such introduction would have on local fish stocks, farmed and wild. A non-quantitative assessment of the risk of

introducing disease through the importation of Pacific salmon was carried out for all 23 diseases. These non-quantitative risk assessments recognised that for table fish to serve as a vehicle for the introduction of fish disease, a number of criteria must be met:

- the disease must be present in the waters of origin
- the disease must be present in the particular fish caught (or the flesh must have become contaminated during processing)
- the pathogen must be present in the imported tissues
- the diseased flesh must pass inspection and grading procedures
- the pathogen in the flesh must survive storage and processing and be present at an infectious dose
- the pathogen must be able to establish infection by the oral route or by being present in the water in which the fish swims
- scraps of the flesh product must find their way into a susceptible fish host in New Zealand or an infectious dose of pathogen must come into contact with a susceptible fish host by some other means.

The likelihood of each of these criteria being met will be different for each disease. However, taking them all into consideration, the non-quantitative assessments led to the conclusion that of all the diseases present in North American salmonids, furunculosis, caused by the bacterium *A. salmonicida*, is the one which would be most likely to be carried in the type of commodity under consideration. This conclusion was based in part on the fact that, of all the diseases considered, none result in greater numbers of pathogens being present in the flesh of infected fish.

The next stage was to conduct a quantitative risk assessment which focused on *A. salmonicida*, taking into account what was known of the prevalence of *A. salmonicida* in wild, ocean-caught Pacific salmon, the distribution and numbers of *A. salmonicida* found in infected Pacific salmon, the effect of processing on the numbers of the pathogen remaining in the tissues of infected fish, the survival of *A. salmonicida* in the environment, the dose required to infect susceptible fish (of any species), and waste management practices in New Zealand.

Much of the data on incidence of furunculosis in ocean-caught Pacific salmon, distribution and numbers of *A. salmonicida* in infected fish and the effect of freezing and thawing on survival of the pathogen was previously unpublished.

Quantitative risk assessment is a decision-making tool which employs science but is not itself a pure science. It is a blend of inductive and deductive logic which is used to examine the components of risk in a structured way. Risk assessment must deal with situations as they arise and must tolerate the mathematical limitations of the disease prevalence estimates or other data. By breaking down the overall risk into various components (the 'input variables'), a risk assessment is designed to focus attention on the specific criteria which must be satisfied before a disease introduction occurs. Until relatively recently, quantitative risk assessments have assumed and combined a series of average, conservative and worst-case values to derive a point estimate of risk that is seen to be conservative. However, such an approach has major limitations, making it very difficult to put estimates into perspective and often, through their bias, focusing on scenarios which will rarely, if ever, happen. Rather than focusing solely on 'worst-case' scenarios, risk analyses based on Monte Carlo simulation models, using variables which are defined by a range of values, give decision makers a much better picture of the risk and the uncertainty surrounding it.

To assess the risk of introducing *A. salmonicida* into the fish stocks of New Zealand through imports of Pacific salmon, the essential steps in the pathway of possible disease introduction were identified, broken down into their components, and estimates of risks applied to each component (44). For each input variable examined, minimum, most likely and maximum estimates (that is, triangular distributions) were obtained by examination of published material and consultation with experts. The final risk estimates were derived from a simulation model which made calculations iteratively using the PC software program @RISK©. Results were displayed as probability distributions.

The quantitative assessment concluded that the risk of introducing *A. salmonicida* into the farmed, recreational or native fish stocks of New Zealand through the importation of chilled, headless, eviscerated salmon is extremely remote. The model estimated that there is a 95% probability that the risk of disease introduction is less than 1×10^{7} per tonne of commodity imported. To put this into perspective, the analysis pointed out that the entire annual production of wild, ocean-caught Pacific salmon in Canada is no more than 100,000 tonnes.

The analysis recognised that the risks associated with other diseases would be cumulative to those posed by *A. salmonicida* and that any risk posed by the other diseases must be added to that posed by furunculosis. However, the analysis also outlined reasons for considering that no disease is more likely to be introduced than *A. salmonicida*, and that the cumulative risk of disease introduction is unlikely to be significantly greater than the range of risk estimates described for *A. salmonicida*.

Concluding remarks

Risk analysis takes into account the prevalence of pathogens in the population from which the finfish products are derived, the probability of their surviving in the product during the

process of importation, the probability of the pathogen coming into contact with local fish stocks after importation and the repercussions of such contact. There is a significant body of information on the survival of fish pathogens under a range of environmental conditions, as discussed in this paper, but there are still major gaps in current knowledge.

Risk assessment is a complex discipline and risk assessments often do not resist an adversarial climate, such as sometimes surrounds proposals to import products which compete with those produced locally. Opposition to import proposals tends to focus on one or two aspects of the uncertainty surrounding the proposal. However, uncertainty and subjectivity do not imply chaos and, despite the uncertainties, one may have confidence that the 'true risk' is unlikely to exceed the estimate resulting from a conservative risk assessment process. Members of the World Trade Organisation are obliged to be consistent in their decision-making with respect to imports. The adoption of risk analysis is a means of ensuring such consistency and the data presented here are intended to assist those wishing to apply risk analysis to trade in finfish products. ■

Risques et prévention de la contamination des produits à base de poisson

P.M. Hine & S.C. MacDiarmid

Résumé

Les risques de contamination des produits à base de poisson par des agents pathogènes non inactivés dépendent, dans une large mesure, du type de produit en cause et des méthodes d'élimination des déchets pratiquées par le pays importateur. Les poissons congelés utilisés comme appât ou pour nourrir des espèces aquatiques de plus grande valeur sont les premiers vecteurs potentiels de contamination, dans la mesure où ils n'ont reçu aucun traitement. En effet, la congélation ne tue ni les virus ni la plupart des bactéries et l'utilisation de ce poisson comme appât peut introduire des agents pathogènes dans les eaux naturelles. En revanche, le poisson transformé, particulièrement les filets cuits ou traités à haute température, présentent un risque négligeable. Si le poisson est transformé après l'importation, l'usine de transformation doit éliminer complètement ses déchets en évitant notamment que ceux-ci puissent être accessibles aux oiseaux ; il convient également d'empêcher tout ruissellement des décharges vers les eaux naturelles. Les eaux usées doivent être désinfectées et évacuées loin des eaux naturelles.

Mots-clés

Agents pathogènes – Analyse des risques – Désinfection – Élimination des eaux usées – Poisson – Transformation.

■

Riesgos y prevención de la contaminación de productos a base de pescado

P.M. Hine & S.C. MacDiarmid

Resumen

El riesgo de contaminación de productos a base de pescado por patógenos activos guarda una estrecha relación con el tipo de producto del que se trate y los métodos de eliminación de desechos empleados en el país de importación.

Debido a la ausencia de procesamiento previo, el pescado congelado que se utiliza como cebo o para alimentar a especies acuáticas de mayor valor presenta un riesgo más alto de contener agentes infecciosos. Debido a que la congelación no destruye los virus –ni algunas bacterias–, el uso de pescado congelado como cebo puede introducir dichos patógenos en las aguas naturales. El pescado procesado, por el contrario, y en especial los filetes (que han sido tratados por calor o precocinados), presentan el nivel más bajo de riesgo. Cuando el tratamiento del pescado es posterior a su importación, es necesario cerciorarse de la eliminación eficaz de los desechos, procurando especialmente evitar su consumo por parte de pájaros vectores y su posible filtración desde los vertederos hasta aguas naturales. Es preciso desinfectar los desechos líquidos y procurar su eliminación en un punto alejado de cualquier curso de aguas naturales.

Palabras clave
Agentes patógenos – Análisis de riesgos – Desinfección – Eliminación de desechos – Pescado – Procesado.

■

Appendix

Survival of viral and bacterial pathogens

Viruses

General: 5 min exposure to an iodophore solution containing 25 ppm I_2 will destroy all known salmonid viruses (2).

Channel catfish virus

Temperature	Inactivated by heat (60°C for 1 h) (63, 68), survives longer at 4°C than 25°C (63, 75), and preserved by freezing (75) in fish tissues (64)
Other	Survives < 1 day on dried concrete, < 2 days on netting and glass. Infectivity lost in dead fish after 2 days at 22°C, and in mud (63)

***Oncorhynchus masou* virus**

Temperature	Survives > 2 weeks at 0°C-5°C, but infectivity lost in < 17 days at –20°C (34)
Other	Inactivated by UV at 3.0×10^3 µW sec/cm^2 (34)

Rhabdovirus olivaceus

Temperature	Survives > 1 month at 0°C-5°C, for > 2 years at –20°C and below (35)
Other	Inactivated by UV at 2.0×10^4 µW sec/cm^2 (35)

Spring viraemia of carp

Temperature	Well preserved at –80°C; inactivated in 30 min at 56°C (75)

Infectious haematopoietic necrosis virus

Temperature	Survives freezing, and dehydration at 4°C-10°C (62). Inactivated by heat (24, 90), and in tissue after 7 days at 4°C-10°C (62)
Salinity	Viable in freshwater, estuarine water, and seawater, longer at 15°C than 20°C (85)
pH	Survives at pH 6-8 (62), and longer at pH 4 at 22°C than 30°C-35°C (91)
Other	Inactivated by ozone, chlorine (90), alcohol, cresol, iodophor and chlorine (31)

Viral haemorrhagic septicaemia virus

Temperature	Inactivated by high temperature (< 1 min at 70°C, < 10 min at 50°C), preserved by low temperatures. Inactivated more rapidly in tissues, < 48 h at 20°C and 1 week at 4°C (75)
Other	Inactivated in 5 min by quarternary ammonium compounds @ 125 mg/l (13)

Infectious pancreatic necrosis virus

Temperature	Survives 3-4 h at 65°C to ~ 10 min at 80°C (91), but inactivated after 16 h at 60°C (24, 45), and in silage at 60°C (76). Well preserved at –80°C, resistant to drying (45)

Salinity	Viable in freshwater, estuarine water, and seawater, longer at 15°C than 20°C (85). Best survival in estuarine water but slowly inactivated, probably by microflora (86), in aquatic environments (12, 75, 95, 99)
pH	Viable 14 days at pH 3.8-4.3, survival reduced by heat. Survives < 5 min in fish silage (pH 3.8-4.3) and pH 4 at pasteurisation temperatures (91). Thermal inactivation faster at pH 3 and pH 10 (45)
Other	In 1:4000 formalin, infectivity reduced over 14 days (45). Inactivated by chlorine and iodine (12, 90), ozone (39, 90), UV (39, 45), and quarternary ammonium compounds (13)

Bacteria

Aeromonas salmonicida

Temperature	Viable < 2 min at 50°C; 48 h at 35°C (91). Inactivated at 44°C after 3 h, 46°C for 1 h, or 48°C for 30 min (28). Less heat resistant at low pH (91)
Salinity and sediments	Reviewed in detail (4). Long survival in sterile sediments (51, 94), but loses pathogenicity after 8-9 months (51). May multiply in sterile sediments (15). Enters unculturable condition in seawater (70) and lake water (1, 53), but recoverable by addition of nutrients (1, 29). Survives longer in sterilised than in unsterilised seawater (16, 21). Atypical *A. salmonicida* survives in brackish water, seawater and sediment, longer at 4°C than 15°C (94)
pH	Survives < 90 min at pH 4.0. Undetectable after 3 min in fish silage (pH 3.8-4.3) (91)
Other	Viable for < 32 days in dead fish, 40 days in tank water (43), < 49 days in fish at –10°C, and < 28 days in fish at 4°C (10). Inactivated by chlorine, iodine or ozone (39, 40)

Edwardsiella ictaluri

Temperature	Recoverable from fish frozen after –20°C for 20 days (8)
Salinity and sediments	Survives in mud and pond water (65). Mortalities higher at 0 mg/l^{-1} and at 100 mg/l^{-1} than at 1000-3000 mg/l^{-1} NaCl (66)

Edwardsiella tarda

Temperature and sediments	Survives in fish at –20°C for 50 days (8). Can be isolated from dressed fish; prevalent in farm water and mud (97)

Vibrio salmonicida

Salinity and sediments	Survives > 14 months in seawater; lower lethal salinity of ~ 10 ppt (27). Viable in fish farm sediments for several months (18, 20)
Other	Survives starvation for > 60 weeks (19). Inactivated by ozone (39)

Vibrio anguillarum

Temperature	Inactivated by heating to 44°C for 3 min (32)
Salinity	Survives > 50 months in seawater, with lower lethal salinity of 5 ppt (27)
Other	Survives starvation for > 60 weeks (19). Inactivated by UV (80) and ozone (39)

Yersinia ruckeri

Temperature	Probably inactivated after 7-8 h at 40°C or 1 h at 49°C (28)
Salinity and sediments	Survives in water > 4 months; better at 0-20 ppt than 35 ppt salinity (82). Viable < 2 months in mud (37), and in filtered seawater < 26 days at 20°C and > 4 months at 10°C (21)
Other	Inactivated by ozone (39). Rapidly destroyed in fish silage (76)

Pasteurella piscicida

Temperature	Does not survive > 52 months at –80°C (25)
Salinity	Does not survive > 4-5 days in estuarine waters (84)
Other	Inactivated by UV at 20,000 µW/sec (80)

Streptococcus sp.

Temperature	May survive freezing for > 6 months (52, 81)
Sediment	May be isolated from water and mud near fish farms (36)
Other	Inactivated by UV at 20,000 µW/sec (80)

Renibacterium salmoninarum

Temperature	May survive > 3 h at 55°C, inactivated at pasteurisation temperatures (91)
Salinity and sediments	Not found in water and sediment near fish farms, but survives < 21 days in tank sediment/faeces, and < 28 days in sterilised river water (3)
pH	Survives in low numbers for > 3 h at 55°C, and > 4 h at pH 4.0, but undetectable after 30 min in fish silage (pH 3.8-4.3) (91)
Other	99% inactivation by chlorine at pH 7.0 and 15°C (60)

Mycobacterium spp.

Temperature	Survive 1-4 min at 60°C-65°C (72, 91)
pH	*M. chelonei* survives 90 min in fish silage (pH 3.8-4.3) and > 14 days at pH 4 (91)

■

References

1. Allen-Austin D., Austin B. & Colwell R.R. (1984). – Survival of *Aeromonas salmonicida* in river water. *FEMS Microbiol. Lett.*, **21**, 143-146.

2. Amend D.F. & Pietsch J.P. (1972). – Virucidal activity of two iodophores to salmonid viruses. *J. Fish. Res. Board Can.*, **29**, 61-65.

3. Austin B. & Rayment J. (1985). – Epizootiology of *Renibacterium salmoninarum*, the causal agent of bacterial kidney disease in salmonid fish. *J. Fish Dis.*, **8**, 505-509.

4. Austin B. & Austin D.A. (1993). – Bacterial fish pathogens. Ellis Horwood, New York, London & Toronto, 384 pp.

5. Batts W.N., Landolt M.L. & Winton J.R. (1991). – Inactivation of hematopoietic necrosis virus (IHNV) by low levels of iodine. *Appl. environ. Microbiol.*, **57**, 1379-1385.

6. Baxa D.V., Groff J.M., Wishkovsky A. & Hedrick R.P. (1990). – Susceptibility of nonictalurid fishes to experimental infection with *Edwardsiella ictaluri*. *Dis. aquat. Organisms*, **8**, 113-117.

7. Beutling D. & Peconek J. (1995). – Eigenschaften halophiler Bakterien isoliert von Salzheringen. *Arch. Lebensmittelhyg.*, **46**, 36-40.

8. Brady Y.J. & Vinitnantharat S. (1990). – Viability of bacterial pathogens in frozen fish. *J. aquat. Anim. Health*, **2**, 149-150.

9. Cairoli A.B. & Conroy D.A. (1987). – An outbreak of vibriosis and its control in *Trachinotus* spp. cultured in floating cages in Venezuela. *Riv. Ital. Piscic. Ittiopatol.*, **22**, 19-20.

10. Cornick J.W., Chudyk R.G. & McDermott L.A. (1969). – Habitat and viability studies on *Aeromonas salmonicida*, causative agent of furunculosis. *Prog. Fish Cult.*, **31**, 90-93.

11. Crespo S., Grau A. & Padros F. (1994). – The intensive culture of 0-group amberjack in the western Mediterranean is compromised by disease problems. *Aquacult. Int.*, **2**, 262-265.

12. Desautels D. & MacKelvie R.M. (1975). – Practical aspects of survival and destruction of infectious pancreatic necrosis virus. *J. Fish. Res. Board Can.*, **32**, 523-531.

13. Dorson M. & Michel C. (1987). – An evaluation of the activity of five quarternary ammonium compounds on main viruses and bacteria pathogenic for salmonids. *Bull. fr. Pêche Pisci.*, **305**, 61-66.

14. Dorson M. & Torhy C. (1994). – Viral haemorrhagic septicaemia virus replication and interferon production in organs excised from resistant and susceptible trout. *In* Program and abstracts. International Symposium on Aquatic Animal Health, University of California School of Veterinary Medicine, Davis, California, 4-8 September. W-12.1.

15. Dubois-Darnaudpeys A. (1977). – Épidémiologie de la furonculose des salmonidés. III. Écologie de *Aeromonas salmonicida*, proposition d'un modèle épidémiologique. *Bull. Fr. Pisci.*, **50**, 21-32.

16. Effendi I. & Austin B. (1991). – Survival of the fish pathogen *Aeromonas salmonicida* in seawater. *FEMS Microbiol. Lett.*, **84**, 103-106.

17. El-Matbouli M. & Hoffmann R.W. (1991). – Effects of freezing, aging, and passage through the alimentary canal of predatory animals on the viability of *Myxobolus cerebralis* spores. *J. aquat. Anim. Health*, **3**, 260-262.

18. Enger Ø., Husevåg B. & Goksøyr J. (1989). – Presence of the fish pathogen *Vibrio salmonicida* in fish farm sediments. *Appl. environ. Microbiol.*, **55**, 2815-2818.

19. Enger Ø., Hoff K.A., Schei G.H. & Dundas I. (1990). – Starvation survival of the fish pathogenic bacteria *Vibrio anguillarum* and *Vibrio salmonicida* in marine environments. *FEMS Microbiol. Ecol.*, **74**, 215-220.

20. Enger Ø., Husevåg B. & Goksøyr J. (1991). – Seasonal variation in presence of *Vibrio salmonicida* and total bacterial counts in Norwegian fish-farm water. *Can. J. Microbiol.*, **37**, 618-623.

21. Fernandez A.I.G., Rodriguez L.A. & Nieto T.P. (1992). – Survival of bacterial fish pathogens in sea water. *Aquaculture*, **107**, 271-276.

22. Flick W.A. (1983). – Observations on loons as predators on brook trout and as possible transmitters of infectious pancreatic necrosis (IPN). *N. Am. J. Fish. Management*, **3**, 95-96.

23. Giorgetti G. & Ceschia G. (1994). – Pasteurellosis in the Mediterranean area: experiences from Italy. *In* Proc. 3rd International Marine Biotechnology Conference, University of Tromsø, Norway, 7-12 August. Programme and abstracts, 63.

24. Gosting L.H. & Gould R.W. (1981). – Thermal inactivation of infectious hematopoietic necrosis and infectious pancreatic necrosis viruses. *Appl. environ. Microbiol.*, **41**, 1081-1082.

25. Hashimoto S., Muraoka A. & Kusuda R. (1989). – Long-term preservation of *Pasteurella piscicida* by freezing or freeze-drying methods. *Nippon Suisan Gakkaishi*, **55**, 979-984.

26. Hjeltnes B., Andersen K., Ellingsen H.M. & Egidius E. (1987). – Experimental studies on the pathogenicity of a *Vibrio* sp. isolated from Atlantic salmon, *Salmo salar* L., suffering from hitra disease. *J. Fish Dis.*, **10**, 21-27.

27. Hoff K.A. (1989). – Survival of *Vibrio anguillarum* and *Vibrio salmonicida* at different salinities. *Appl. environ. Microbiol.*, **55**, 1775-1786.

28. Humphrey J.D., Smith M.T., Gudkovs N. & Stone R. (1991). – Heat susceptibility of selected exotic viral and bacterial pathogens of fish. Report of a study undertaken for the Australian Quarantine and Inspection Service. Australian Fish

Health Reference Laboratory, Commonwealth Scientific and Industrial Research Organisation (CSIRO), Australian Animal Health Laboratory, Geelong, 16 pp.

29. Husevåg B. (1995). – Starvation survival of the fish pathogen *Aeromonas salmonicida* in seawater. *FEMS Microbiol. Ecol.*, **16**, 25-32.

30. Hyatt A.D., Hine P.M., Jones J.B., Whittington R.J., Wise T.G., Crane M.S. & Williams L.M. (1997). – Epizootic mortality in the pilchard (*Sardinops sagax neopilchardus*) in Australia and New Zealand. II. Identification of a herpesvirus within the gill epithelium. *Dis. aquat. Organisms*, **28**, 17-29.

31. Inouye K., Ikeya F., Yamazaki T. & Hara T. (1990). – Virucidal activities of various germicides to infectious pancreatic necrosis virus (IPNV). *Fish Pathol.*, **25**, 81-86.

32. Jacobsen P. & Liltved H. (1988). – Thermal disinfection of seawater for aquacultural purposes. *Aquacult. Eng.*, **7**, 443-447.

33. Jacobsen P., Liltved H. & Efraimsen H. (1989). – Disinfection of effluent from fish slaughteries. *Aquacult. Eng.*, **8**, 209-216.

34. Kimura T. & Yoshimizu M. (1989). – Salmon herpesvirus: OMV, *Oncorhynchus masou* virus. *In* Viruses of lower vertebrates (W. Ahne & E. Kurstak, eds). Springer Verlag, Berlin, Heidelberg & New York, 171-183.

35. Kimura T., Yoshimizu M., Oseko N. & Nishizawa T. (1989). – *Rhabdovirus olivaceus* (hirame rhabdovirus). *In* Viruses of lower vertebrates (W. Ahne & E. Kurstak, eds). Springer Verlag, Berlin, Heidelberg & New York, 388-395.

36. Kitao T., Aoki T. & Iwata K. (1979). – Epidemiological study on streptococcicosis of cultured yellowtail (*Seriola quinqueradiata*). I. Distribution of *Streptococcus* sp. in sea water and muds around yellowtail farms. *Bull. Jpn. Soc. scient. Fish.*, **45**, 567-572.

37. Klontz G.W. & Huddleston T.R. (1976). – Control of enteric redmouth disease: a twelve month activity report on contract 10690004 DEMO-1014. University of Idaho, Moscow, Idaho.

38. Langdon J.S., Thorne T. & Fletcher W.J. (1992). – Reservoir hosts and new clupeoid host records for the myoliquefactive myxosporean parasite *Kudoa thyrsites* (Gilchrist). *J. Fish Dis.*, **15**, 459-471.

39. Liltved H., Hektoen H. & Efraimsen H. (1995). – Inactivation of bacterial and viral fish pathogens by ozonation or UV irradiation in water of different salinity. *Aquacult. Eng.*, **14**, 107-122.

40. Liltved H. & Landfald B. (1995). – Use of alternative disinfectants, individually and in combination, in aquacultural wastewater treatment. *Aquacult. Res.*, **26**, 567-576.

41. Lönnström L., Wiklund T. & Bylund G. (1994). – *Pseudomonas anguilliseptica* isolated from Baltic herring *Clupea harengus membras* with eye lesions. *Dis. aquat. Organisms*, **18**, 143-147.

42. McAllister P.E. & Owens W.J. (1992). – Recovery of infectious pancreatic necrosis virus from the faeces of wild piscivorous birds. *Aquaculture*, **106**, 227-232.

43. McCarthy D.H. (1980). – Some ecological aspects of the bacterial fish pathogen – *Aeromonas salmonicida*. *In* Aquatic microbiology. *Symp. Ser., Soc. Appl. Bacteriol.*, **6**, 299-324.

44. MacDiarmid S.C. (1994). – The risk of introducing exotic diseases of fish into New Zealand through importation of ocean-caught Pacific salmon from Canada. Ministry of Agriculture and Fisheries (MAF) Regulatory Authority, Ministry of Agriculture, Wellington, 161 pp.

45. MacKelvie R.M. & Desautels D. (1975). – Fish viruses: survival and inactivation of infectious pancreatic necrosis virus. *J. Fish. Res. Board Can.*, **32**, 1267-1273.

46. Markiw M.E. (1992). – Experimentally induced whirling disease. I. Dose response of fry and adults of rainbow trout exposed to the Triactinomyxon stage of *Myxobolus cerebralis*. *J. aquat. Anim. Health*, **4**, 40-43.

47. Meyers T.R., Hauck A.K., Blankenbeckler W.D. & Minicucci T. (1986). – First report of viral erythrocytic necrosis in Alaska, USA, associated with epizootic mortality in Pacific herring, *Clupea harengus pallasi* (Valenciennes). *J. Fish Dis.*, **9**, 479-491.

48. Meyers T.R., Sullivan J., Emmenegger E., Follett J., Short S. & Batts W.N. (1992). – Identification of viral hemorrhagic septicaemia virus isolated from Pacific cod *Gadus macrocephalus* in Prince William Sound, Alaska, USA. *Dis. aquat. Organisms*, **12**, 167-175.

49. Meyers T.R., Short S., Lipson K., Batts W.N., Winton J.R., Wilcock J. & Brown E. (1994). – Association of viral hemorrhagic septicemia virus with epizootic hemorrhages of the skin in Pacific herring *Clupea harengus pallasi* from Prince William Sound and Kodiak Island, Alaska, USA. *Dis. aquat. Organisms*, **19**, 27-37.

50. Meyers T.R. & Winton J.R. (1995). – Viral hemorrhagic septicemia in North America. *Ann. Rev. Fish Dis.*, **5**, 3-24.

51. Michel C. & Dubois-Darnaudpeys A. (1980). – Persistence of the virulence of *Aeromonas salmonicida* kept in river sediments. *Ann. Rech. vét.*, **11**, 375-386.

52. Minami T. (1979). – *Streptococcus* sp. pathogenic to cultured yellowtail, isolated from fishes for diets. *Fish Pathol.*, **14**, 15-19.

53. Morgan J.A., Rhodes G. & Pickup R.W. (1993). – Survival of nonculturable *Aeromonas salmonicida* in lake water. *Appl. environ. Microbiol.*, **59**, 874-880.

54. Mqolomba T.N. & Plumb J.A. (1992). – Longevity of *Edwardsiella ictaluri* in the organs of experimentally infected channel catfish, *Ictalurus punctatus*. *Aquaculture*, **101**, 1-6.

55. Munday B.L., Langdon J.S., Hyatt A. & Humphrey J.D. (1992). – Mass mortality associated with a viral-induced vacuolating encephalopathy and retinopathy of larval and juvenile barramundi, *Lates calcarifer* Bloch. *Aquaculture*, **103**, 197-211.

56. Nelson R.T., McLoughlin M.F., Rowley H.M., Platten M.A. & McCormick J.I. (1995). – Isolation of a toga-like virus from farmed Atlantic salmon *Salmo salar* with pancreas disease. *Dis. aquat. Organisms*, **22**, 25-32.

57. Nylund A., Hovland T., Watanabe K. & Endresen C. (1994). – Infectious salmon anaemia (ISA): a multiorgan infection. *In* Proc. International Symposium on Aquatic Animal Health, University of California School of Veterinary Medicine, Davis, California. Program and abstracts, W-19.1.

58. Oseko N. (1994). – Pathology for hirame rhabdovirus (*Rhabdovirus olivaceus*). *Bull. Nansei Natl Fish. Res. Inst. Nanseisuikenho*, **27**, 129-231.

59. Parry L. & Dixon P.F. (1997). – Stability of nine viral haemorrhagic septicaemia virus (VHSV) isolates in seawater. *Bull. Eur. Assoc. Fish Pathol.*, **17**, 31-36.

60. Pascho R.J., Landolt M.L. & Ongerth J.E. (1995). – Inactivation of *Renibacterium salmoninarum* by free chlorine. *Aquaculture*, **131**, 165-175.

61. Peters F. & Neukirch M. (1986). – Transmission of some fish pathogenic viruses by the heron, *Ardea cinerea*. *J. Fish Dis.*, **9**, 539-544.

62. Pietsch J.P., Amend D.F. & Miller C.M. (1977). – Survival of infectious hematopoietic necrosis virus held under various environmental conditions. *J. Fish. Res. Board Can.*, **34**, 1360-1364.

63. Plumb J.A. (1989). – Channel catfish herpesvirus. *In* Viruses of lower vertebrates (W. Ahne & E. Kurstak, eds). Springer Verlag, Berlin, Heidelberg & New York, 198-216.

64. Plumb J.A., Wright L.D. & Jones V.L. (1973). – Survival of channel catfish virus in chilled, frozen, and decomposing channel catfish. *Prog. Fish Cult.*, **35**, 170-172.

65. Plumb J.A. & Quinlan E.E. (1986). – Survival of *Edwardsiella ictaluri* in pond water and bottom mud. *Prog. Fish Cult.*, **48**, 212-214.

66. Plumb J.A. & Shoemaker C. (1995). – Effects of temperature and salt concentration on latent *Edwardsiella ictaluri* infections in channel catfish. *Dis. aquat. Organisms*, **21**, 171-175.

67. Ransom D.P., Lannan C.M., Rohovec J.S. & Fryer J.L. (1984). – Comparison of histopathology caused by *Vibrio anguillarum* and *Vibrio ordalii* in three species of Pacific salmon. *J. Fish Dis.*, **7**, 107-115.

68. Robin J. & Rodrique A. (1980). – Resistance of herpes channel catfish virus (HCCV) to temperature, pH, salinity and ultraviolet irradiation. *Rev. Can. Biol.*, **39**, 153-156.

69. Rose A.S., Ellis A.E. & Munro A.L.S. (1989). – The infectivity by different routes of exposure and shedding rates of *Aeromonas salmonicida* subsp. *salmonicida* in Atlantic salmon, *Salmo salar* L., held in sea water. *J. Fish Dis.*, **12**, 573-578.

70. Rose A.S., Ellis A.E. & Munro A.L.S. (1990). – The survival of *Aeromonas salmonicida* subsp. *salmonicida* in sea water. *J. Fish Dis.*, **13**, 205-214.

71. Ross K., McCarthy U., Huntly P.J., Wood B.P., Stuart D., Rough E.I., Smail D.A. & Bruno D.W. (1995). – An outbreak of viral haemorrhagic septicaemia (VHS) in turbot (*Scophthalmus maximus*) in Scotland. *Bull. Eur. Assoc. Fish Pathol.*, **14**, 213-214.

72. Schulze-Robbecke R. & Buchholtz K. (1992). – Heat susceptibility of aquatic mycobacteria. *Appl. environ. Microbiol.*, **58**, 1869-1873.

73. Sindermann C.J. & Chenoweth J.F. (1993). – The fungal pathogen *Ichthyophonus hoferi* in sea herring, *Clupea harengus*: A perspective from the western North Atlantic. *In* Proc. International Council for the Exploration of the Sea (ICES), Comité Mariculture (CM), F:41, 39 pp.

74. Smail D.M. (1995). – Isolation and identification of viral haemorrhagic septicaemia (VHS) virus from North Sea cod (*Gadus morhua* L.). *In* Proc. International Council for the Exploration of the Sea (ICES), Comité Mariculture (CM), F:15, 6 pp.

75. Smail D.M. & Munro A.L.S. (1989). – The virology of teleosts. *In* Fish pathology (R.J. Roberts, ed.). Baillière Tindall, London, Philadelphia & Sydney, 173-241.

76. Smail D.M., Huntly P.J. & Munro A.L.S. (1990). – Fate of four fish pathogens after exposure to fish silage containing fish farm mortalities and conditions for the inactivation of infectious pancreatic necrosis virus. *Aquaculture*, **113**, 173-181.

77. Smail D.M., Irwin N., Harrison D. & Munro A.L.S. (1993). – Passage and survival of infectious pancreatic necrosis (IPN) virus in the cow's gut after feeding a silage mixture containing IPN virus. *Aquaculture*, **113**, 183-187.

78. Sohn S.G., Park M.A. & Lee S.D. (1992). – A histopathological study on masu salmon, *Oncorhynchus masou* fry infected with the infectious hematopoietic necrosis virus. *Bull. Natl Fish. Res. Dev. Agency, Korea*, **46**, 145-150.

79. Sugita H., Asai T., Hayashi K., Mitsuya T., Amanuma K., Maruyama C. & Deguchi Y. (1992). – Application of ozone disinfection to remove *Enterococcus seriolicida*, *Pasteurella piscicida*, and *Vibrio anguillarum* from seawater. *Appl. environ. Microbiol.*, **58**, 4072-4075.

80. Sugita H., Mitsuya T., Amanuma K., Hayashi K., Asai T., Maruyama C. & Deguchi Y. (1992). – Ultraviolet susceptibility of three marine fish pathogens. *Nihon Univ. Nichidai Nojuho*, **49**, 117-121.

81. Taniguchi M. (1983). – Progress of streptococcicosis in peroral inoculation. *Bull. Jpn. Soc. scient. Fish.*, **49**, 1171-1174.

82. Thorsen B.K., Enger Ø., Norland S. & Hoff K.A. (1992). – Long-term starvation survival of *Yersinia ruckeri* at different salinities studied by microscopical and flow cytometric methods. *Appl. environ. Microbiol.*, **58**, 1624-1628.

83. Thrusfield M. (1995). – Veterinary epidemiology, 2nd Ed. Blackwell Science, Oxford, 28-29.

84. Toranzo A.E., Barja J.L. & Hetrick F.M. (1982). – Survival of *Vibrio anguillarum* and *Pasteurella piscicida* in estuarine and fresh waters. *Bull. Eur. Assoc. Fish Pathol.*, **3**, 43-45.

85. Toranzo A.E. & Hetrick F.M. (1982). – Comparative stability of two salmonid viruses and poliovirus in fresh, estuarine and marine waters. *J. Fish Dis.*, **5**, 223-231.

86. Toranzo A.E., Barja J.L., Lemos M.L. & Hetrick F.M. (1983). – Stability of infectious pancreatic necrosis virus (IPNV) in untreated, filtered and autoclaved estuarine water. *Bull. Eur. Assoc. Fish Pathol.*, **3**, 51-53.

87. Torgersen Y. & Håstein T. (1995). – Disinfection in aquaculture. *In* Disinfectants: actions and applications, Part Two (H.A. McDaniel, ed.). *Rev. sci. tech. Off. int. Epiz.*, **14** (2), 419-434.

88. Totland G.K., Nylund A. & Holm K.O. (1988). – An ultrastructural study of morphological changes in Atlantic salmon, *Salmo salar* L., during the development of cold water vibriosis. *J. Fish Dis.*, **11**, 1-13.

89. Traxler G.S. & Rankin J.B. (1989). – An infectious haematopoietic necrosis epizootic in sockeye salmon *Oncorhynchus nerka* in Weaver Creek spawning channel, Fraser River system, BC, Canada. *Dis. aquat. Organisms*, **6**, 221-226.

90. Wedemeyer G.A., Nelson N.C. & Smith C.A. (1978). – Survival of the salmonid viruses infectious hematopoietic necrosis (IHNV) and infectious pancreatic necrosis (IPNV) in ozonated, chlorinated and untreated waters. *J. Fish. Res. Board Can.*, **35**, 875-879.

91. Whipple M.J. & Rohovec J.S. (1994). – The effect of heat and low pH on selected viral and bacterial fish pathogens. *Aquaculture*, **123**, 179-189.

92. Whitaker D.L. & Kent M.L. (1991). – Myxosporean *Kudoa thyrsites*: a cause of soft flesh in farm-reared Atlantic salmon. *J. aquat. Anim. Health*, **3**, 291-294.

93. Whittington R.J., Jones B., Hine P.M. & Hyatt A.D. (1997). – Epizootic mortality in the pilchard (*Sardinops sagax neopilchardus*) in Australia and New Zealand. I. Pathology and epizootiology. *Dis. aquat. Organisms*, **28**, 1-16.

94. Wiklund T. (1995). – Survival of 'atypical' *Aeromonas salmonicida* in water and sediment microcosms of different salinities and temperatures. *Dis. aquat. Organisms*, **21**, 137-143.

95. Wolf K., Quimby M.C. & Carlson C.P. (1969). – Infectious pancreatic necrosis virus: lyophilization and subsequent stability in storage at 4°C. *Appl. Microbiol.*, **17**, 623-624.

96. Wolf K. & Markiw M.E. (1982). – *Myxosoma cerebralis*: inactivation of spores by hot smoking of infected trout. *Can. J. Fish. Aquat. Sci.*, **39**, 926-928.

97. Wyatt L.E., Nickelson R. & Vanderzant C. (1979). – *Edwardsiella tarda* in freshwater catfish and their environment. *Appl. environ. Microbiol.*, **38**, 710-714.

98. Yasunaga N. (1982). – Occurrence of *Streptococcus* sp., a pathogen of cultured yellowtail, in muscle of sardine for diets. *Fish Pathol.*, **17**, 195-198.

99. Yoshimizu M., Takizawa H., Kamei Y. & Kimura T. (1986). – Interaction between fish pathogenic viruses and micro-organisms in fish rearing water: survival and inactivation of infectious pancreatic necrosis virus, infectious haematopoietic necrosis virus and *Oncorhynchus masou* virus in rearing water. *Fish Pathol.*, **21**, 223-232.