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**Infectious Salmon Anemia (ISA): Literature Review and implications for wild salmon**

by

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## **Introduction**

This paper provides a literature review about what is known about the taxonomy, distribution, infection routes, and pathologies of the Infectious Salmon Anaemia (ISA) virus. This disease has caused major losses in the Atlantic salmon farming industry on both sides of the Atlantic Ocean, and has now been detected in wild fish in Canada (New Brunswick), and Scotland. While this review considers the case of ISA, the lessons learned may help us to plan and cope for future disease epidemics.

## **Taxonomy of the virus**

The ISA virus was unknown to science prior to its surfacing in epidemic proportions in the Norwegian salmon farming industry in 1984. Nylund (1997) suggested that ISA might have first arrived in Norway via the importation of rainbow trout (*Oncorhynchus mykiss*), although he provided no evidence to support conjecture.

This virus has not yet been completely described, or named. However, it is most probably a member of the Orthomyxoviridae family (*i.e.*, a member of the flu group; Krossøy *et al.* 1999, Falk *et al.* 1997, Mjaaland *et al.* 1997), and may be the first detected species in a new genus within this family (Krossøy *et al.* 1999).

## **Tests used to detect the virus**

Three tests are used to identify the presence of ISA virus in Atlantic salmon (Lovely *et al.* 1999). They are: 1) an ISAV specific immunofluorescence antibody test (IFAT), 2) a reverse transcriptase polymerase chain reaction (rt-PCR) test, and 3) a virology test which cultures the virus and tests for cytopathic effects (CPE's) on a cell line (salmon head kidney line SHK-1 is preferred, but others like Chinook salmon embryo line CHSE-214 can be used ; Bouchard *et al.* 1999).

The IFAT test gives false positives, which is discouraging its use. The rt-PCR test provides the fastest result, but most investigators also prefer to have a final confirmation of the presence of the virus by a documentation of CPE's in a cell culture line. This, however, is time consuming and test results can take a month or more to obtain.

## **Virus detection and chronology for the epidemics in the aquaculture industry**

ISA was first reported in a hatchery on the Southwest coast of Norway, where it was associated with a mortality of parr (Håstein 1997). Clinical signs began to break out at sea cage sites in subsequent years, and smolt survival at some farms dropped from 86%, to 18% by 1988. The spread of the disease was extremely rapid. From a level of zero infections prior to 1984, it grew to the point in 1990 where 101 new farms were infected in a year. Following the implementation of aggressive control programs, infection levels fell to the point that generally, <10 sites become infected per year (Jarp and Karlsen 1997), although in 1998 about 15 sites were hit.

Outbreaks of ISA were first observed in Canada in 1996, where the disease was initially termed Haemorrhagic Kidney Syndrome (HKS) (Loevely *et al.* 1999; Mullins 1998; Byrne *et al.* 1998). The disease spread rapidly, and twenty-one sites tested positive for ISA in 1997, > 35 in 1998 (about 40% of all sites), and at present there are 17 that are positive for the disease. Of these 17, 10 hold unvaccinated fish from the 1998 year class and seven contain smolts that were vaccinated against ISA and went to sea in the Spring of 1999 (Stewart, P. *Has the ISA eradication program worked?* Fish Farming 12(9) p.1).

This disease became the first of the Canadian sea cage industry where an eradication order was issued as a control measure. The industry was put in a very difficult position as initially no compensation was offered for the fish that were slaughtered. At present the number of sites testing positive for ISA is decreasing, however, the virus is still causing severe damage. So far, \$20 million (Canadian) has been spent to compensate farmers for ordered eradications, and uncompensated losses may total as much as \$40 million (Canadian)(DFA 1999). The Canadian East coast salmon industry is annually worth about \$120 million (Canadian).

In Scotland, ISA was first reported in 1998. It spread rapidly to farms on the west coast Skye, and Shetland Islands, and by December 1999 11 sites had confirmed infections and a further 24 were suspected of being infected (about 10% of all Scottish sites; Scottish Executive Press Release, 15 December 1999).

The first reports of ISA in the Chilean salmon industry surfaced in March 2000. Scientists found the virus in farmed coho salmon (*Oncorhynchus kisutch*) at a single site. The Association of Chilean Salmon and Trout Farmers is disputing the identification (Rick Ramseyer, Fishmonger News Network, March 16, 2000. *Chilean salmon official downplays ISA virus report but scientists at Atlantic Veterinary College stand by their research.*).

No outbreaks of ISA have yet been reported from the USA, although infected Canadian sites occur in close proximity (< 10 km) to American farms.

### **Strains of ISA**

A recent comparison of a limited portion of the genome of a small number of ISA isolates from Norwegian and Canadian farmed Atlantic salmon showed that the Canadian version of the virus was about 80-90% identical with that of the Norwegian version (Blake *et al.* 1999). This is considered a fairly significant difference, and led the authors to suggest that the Canadian epidemic may not have resulted from a recent importation of the infection from Norway, but rather from a distinct North American genomic variant. I have not encountered any studies that report on comparisons of the Scottish strain of ISA to those of either Norway or Canada.

### **Symptoms of ISA in Atlantic salmon**

Clinical and disease symptoms include the fish becoming lethargic or moribund, lifting of scales off of the body, a protuberance of the eyes, skin lesions, pale gills, swollen livers, petechiae,

agglutination of the red blood cells, anemia, necrosis and/or hemorrhages in the pyloric caecae, intestine, liver and the kidneys (Bouchard *et al.* 1999, Rodger 1998). After experimental injections, in Atlantic salmon the virus seems to show up fastest and most consistently in the head and mid-kidney (Rimstad *et al.* 1999).

### **Transmission**

The disease is “highly contagious and lethal” (Totland *et al.* 1996, p. 25), and horizontal transmission can occur in the laboratory in both fresh water and seawater. The virus is believed to be carried in skin mucous, faeces, urine, and blood of infected fish (Totland *et al.* 1996, Nylund *et al.* 1994), and enters the body through the gills, or wounds such as those caused by sea lice (Totland *et al.* 1996, Nylund *et al.* 1994). Consumption of virus-contaminated food by salmon did not result in infections, so gut-passage apparently destroys the virus (Totland *et al.* 1996). Virus particles have been shown to retain their infectivity outside the body of the host in seawater (at 6 C) for at least 20 days (Nylund *et al.* 1994).

The disease does not appear to be vertically transmitted from infected parents to their offspring (Melville and Griffiths 1999). There is one report of an outbreak of ISA in first feeding fry at a site in Norway that could have implied vertical transmission of the disease. However, it is believed that the virus got into the facility by another but as yet unidentified route (Nylund *et al.* 1999).

At present, the Atlantic salmon is the only salmonid that has shown large-scale mortalities when infected with this virus. Rainbow trout (Nylund 1997, Nylund *et al.* 1997), and brown trout (*Salmon trutta*) are asymptomatic, and presumed to become life-long carriers of the ISA virus once infected (Rolland and Nylund 1998, Nylund 1997, Nylund and Jakobsen 1995, Nylund *et al.* 1995). The European eel (*Anguilla anguilla*) has also recently been identified as a carrier, and Canadian and European agencies are testing other marine species to see if they harbor the virus.

Rolland and Nylund (1998) used a combination of injections of blood from infected to fish to uninfected fish, and cohabitation experiments of infected fish with uninfected fish, to show that the disease could be transmitted under laboratory conditions from anadromous brown trout to Atlantic salmon. This confirmed earlier work by Nylund and Jacobsen (1995). Mortalities for salmon in these trials went to 100% in 50 % of their experiments.

### **Mortality patterns**

Laboratory trials, which often involved artificial injections of fish with virus, under controlled conditions, frequently showed infections beginning at about two weeks after exposure and high rates (>70%) of mortality in the next few weeks (e.g., Totland *et al.* 1996, Nylund 1997, Jones *et al.* 1999).

By contrast, mortality patterns in sea cages showed seasonal patterns and were less severe. In New Brunswick (Hammell and Dohoo 1999), there were distinct temporal peaks in mortality rates in early July, early September and late October. The approximately eight-week latency

period for the infection may drive this pattern subsequent to the initial outbreak. Mortality rates did not decrease as water temperatures declined in October. However, outbreaks were also recorded throughout the year.

Hammell and Dohoo (1999) defined outbreaks as mortalities within a given cage of greater than 1 fish/1000/day. In the 78 cages they considered, outbreaks lasted a mean of about 37 days, and resulted in losses of a mean of about 12.2% of the fish present. These values may be conservative, because they do not take into account situations where farmers “prematurely” terminated the outbreak by slaughtering the fish. Considering only sites where the outbreak ran its full course, about 45% of cages lost 5% of the fish present; about 15% of cages lost >5% but < 10%; and 78% of the cages had total mortalities of less than 20%.

## **Resistance**

Animals may be “resistant” to a disease because they have high tolerance (*i.e.*, are infected, but do not show clinical signs), or because they are not susceptible (*i.e.*, do not become infected).

Fish vary in their susceptibility to disease, in some cases due to their genetics (*e.g.* GjØen *et al.* 1997). When genetic variation for disease resistance exists, it may be possible to select for disease resistance. GjØen *et al.* (1997) evaluated the heritabilities for survival of 121 full sib groups of Atlantic salmon challenged with furunculosis (*Aeromonas salmonicida*), cold water vibriosis (*Vibrio salmonicida*), vibrio (*V. anguillarum*) and ISA. In these experiments, in the ISA treatment the cumulative mortality of Atlantic salmon was highest and heritabilities for survival were the lowest, indicating that salmon may have the least potential of the species tested for development of resistance to ISA through selection. In addition, the authors presented evidence that survival after challenge with the other diseases tended to be negatively correlated with survival after a challenge with ISA. In other words, if you select for resistance to these other disease, you may increase susceptibility to ISA.

## **Epidemiological studies**

Two studies, one each for Norway (Jarp and Karlsen 1997) and Canada (Hammell and Dohoo 1999), have examined the epidemiology of the outbreak of ISA in their respective salmon farming areas. The goal of both was to identify factors that could contribute to the eruption and maintenance of ISA epidemics, and if possible to quantify the disease risk each of these factors posed.

In Norway, risk factors identified included:

- Siting farms < 5 km from a processing plant, which increased the risk of ISA by a factor of 13.
- Having your farm located < 5 km from an infected farm, which increased the risk by eight times. Further reductions in this risk were not achieved when farms were sited more than 10 km away from other sites.

- Taking smolt deliveries from a number of different producers, which increased risk to an undetermined degree.

In New Brunswick, risk factors included:

- Stocking cages with > 12,000 fish, which increased risk by a factor of 3.92 compared to cages receiving < 5000 fish, and those receiving 5000-12000 fish were 2.4 times more likely to test positive for ISA than those with < 5000 fish. This risk was evaluated independently of fish density.
- Increased density of fish in the cages increased ISA risk, but the relation was complex and appeared to be more of a threshold effect than a continuing increase in risk with continuing increases in densities. Cages containing 2-5 fish/m<sup>3</sup> were 3.6 times more likely to develop ISA than those with less than 2.5 fish/m<sup>3</sup>, whereas those with > 5 fish/m<sup>3</sup> were 2.7 times more likely to become ISA cages compared to those with < 2.5 fish/m<sup>3</sup>.
- Sites with fewer sea lice treatments got more ISA.
- Increased site traffic (shared divers, shared barges, increased numbers of smolt deliveries, etc.), increased risks.

Overall, these studies highlighted the importance of developing procedures and regulations that would reduce the risk of a farm encountering infective particles, and eliminating factors which stress fish and reduce their natural immunity. By inference, any factor that could contribute to either of these problems is worth avoiding at a cage site.

### **Remedial measures**

ISA is not going to be eliminated from regions where it has now established itself. Industry control strategies require rigorous measures to reduce the number of infective particles in the environment, good husbandry to reduce stress in the fish which could predispose them to epidemics, the development of vaccines, and possibly the implementation of selective breeding programs to develop lines of farm fish that are resistant to ISA.

Once sites test positive for ISA, eradication of fish at infected farms is done in all jurisdictions. In addition, husbandry measures being introduced include providing good growing conditions at the farms, the fallowing of sites after a culture cycle for a period of at least six months, and the growing of single year classes of smolts at each site. Site traffic issues are being examined and dealt with through a combination of regulations and individual and collective initiatives. Zoning is also being employed in all jurisdictions, with severe restrictions being imposed on fish farming in areas where ISA is present compared to disease free areas.

For the industry, disinfection may be an option (Fraser 1999, Torgensen and Håstein 1997). This could be done to equipment that is moving between or among sites, or at processing plants to effluent and wastes. Available disinfectants include sodium hypochlorite (100 – 1,000 mg/l in fresh water for 30 min), formaldehyde (0.5% for 16h), formic acid (pH < 4 for 24h), heat (50 C for > 5 min), ozone (8mg/l/min for 4 min), and UV radiation (5mj/cm<sup>2</sup>), and iodophor (100mg/l for a 20 min bath; 200mg/l for low pressure spray).

New Brunswick salmon farmers inoculated their smolts in 1999 with a new ISA vaccine. Data are still being compiled, but it is clear that the vaccine did not, by itself, stop the epidemic. New farms have tested positive for ISA during 1999, including seven which had used the vaccine (Stewart, Paul. *Has the ISA eradication program worked?* Fish Farming V. 12, No. 9, p. 1). Joint government/industry research has just begun at Canada's Department of Fisheries and Oceans Biological Station in St. Andrews, New Brunswick, evaluating the efficacy of the present vaccine. Vaccines are not used in either Scotland or Norway, due to EU regulations that call for eradication rather than vaccination.

Selection for natural resistance through breeding programs may be problematic. Only one study (Nylund 1997) has investigated the resistances of different farm strains or wild populations of salmon to ISA. In this laboratory experiment fish were injected with ISA, and while more of the farm strain fish died and did so faster than their wild counterparts, mortality of the wild fish was still high (> 70%) at the point the experiment was terminated. There was also evidence for a negative correlation between resistance to bacterial diseases, and resistance to viral diseases. In other words, genetic selection for viral resistance could make the fish more susceptible to bacterial diseases, and *vice versa*.

### **Danger to people**

The virus does not cause agglutination of erythrocytes from mammals (including humans) or birds, and is unable to replicate at temperatures above 25 C (Falk *et al.* 1997). Thus it does not appear to pose a threat to humans, and the sale of fish slaughtered in eradication programs is permitted.

### **ISA in wild Atlantic salmon and farm escapees**

The first reports of ISA in escaped farmed Atlantic salmon, and wild Atlantic salmon occurred in 1999. In New Brunswick, Canada, four escaped farm salmon of 58 that were sampled entering the fish ladder in the Magaguadavic River were confirmed as positive for ISA. Prior to that, disease screening of escapees in this river in 1998 (N = 61) were all negative for the virus. In 1997, based on visual inspections, five escapees (N = 35) were diagnosed as suspect for the virus, but confirmation was not obtained because disease testing laboratories were overloaded with samples as the ISA epidemic took off.

Fifteen wild salmon were collected as broodstock from the Magaguadavic River in summer of 1999. They were held in three tanks in an isolated facility on a brackish well water supply. Subsequently, three fish held in the same tank showed signs of illness and died. Disease screening confirmed the cause of death was ISA. The remaining 12 fish then had gill mucous smears and blood samples withdrawn for an initial ISA screening, and tissue samples were taken after spawning for a final round of testing. Only one fish was found to be disease-free in all tests.

It is unlikely that the well water used in the holding facility was the source of the infection. It is also unlikely that all these fish had ISA at the time they were moved into the holding facility. It is more probable that at least one individual in each tank had the disease, and passed it along to uninfected tank mates.

These fish were artificially spawned and the eggs reared in quarantine. Resultant first-feeding fry have been tested for ISA(17 January 2000, 60 fry), and all tests were negative.

Shortly after the Magaguadavic results were reported, on 4 November 1999 The Scottish Executive issued a press release announcing that ISA had been found in salmon parr in the Rivers Conan, Easaidh and Tweed, in brown trout in the Conan and Easaidh, in sea trout in Laxo Voe, Shetland, and River Snizort in Skye, rainbow trout freshwater farms in Aberdeenshire and Kinnrosshire, and European eel in Loch Uisg, Mull. The parr were not reported as having any disease symptoms.

These results confirm that wild salmon may harbor the virus and get ill from it.

### **Implications for wild fish**

In North America, the ISA epidemic could not have occurred in a worse place or at a worse time. In the area where aquaculture is practiced wild salmon stocks are severely depressed and in many cases on the brink of biological extinction (Anon 1999, DFO 2000). There are no positive scenarios that can be attributed to the presence of this disease.

Given the heavy degree of mortality caused by the virus in farmed Atlantic salmon, it may be that the Atlantic salmon is a new and vulnerable host. Presumably the other species that are asymptomatic carriers have coevolved with ISA virus, and can now tolerate it. Given that only a few tens of fish are returning to North American rivers in the area where the virus is ravaging the salmon farming industry, the chances of a similar coevolution in Atlantic salmon appear slim.

Salmon farming did not “create” the ISA virus. Clearly, it existed before. However, in salmon farming, where large numbers of fish are confined in close proximity to each other, ideal conditions prevail for a quick spread of an infection. Diseased fish shed virus particles into the water, where they can reach new hosts. Both wild and farmed salmon contract the disease. Either group could now serve as a source of infection for the other.

The best protection against ISA for wild salmon populations is to minimize their probability of encountering the virus. Clearly there is no hope of vaccinating all wild parr in rivers. This argues for an aggressive program of good husbandry at fish culture stations and eradication of fish at cage sites when the virus appears. This concords with the industry’s desire to stop the spread of ISA among cage sites.

Atlantic salmon may carry the virus without immediately showing symptoms of the disease. However, when the infected individuals become stressed by environmental conditions, their immune system may succumb and they will then manifest symptoms. Thus, it is important to limit the spread of the disease as much as possible. The absence of the symptoms in fish having this virus does not guarantee that there will not be a future problem.

For those fish that do become infected, it will be critical to maintain healthy freshwater and ocean habitats in order to reduce stress and give their immune systems the best possible chances

of fighting off the virus. If certain human activities are fostering the spread of potential pathogens, we may have to curtail other human activities to compensate for the risks that we are introducing to wild salmon populations.

Given the occurrence of the disease in wild salmon populations, and its potential to spread in seawater, it would be prudent to initiate monitoring for the disease in the wild fish harvested at Greenland. This could provide an early warning of the potential scope and rate of spread of the disease in wild populations.

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