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NEW ZEALAND FRESHWATER FISHERIES MISCELLANEOUS REPORT NO. 21

INFECTIOUS PANCREATIC NECROSIS (IPN)

by

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Report to: Department of Conservation

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INFECTIOUS PANCREATIC NECROSIS (IPN)

A synopsis prepared for the Department of Conservation

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Note that the following is not a comprehensive literature review but summarises some of the relevant literature on hand, and provides details of IPN found in New Zealand. Reviews of IPN are cited in the references of this report.

1 Cause

IPN refers to the disease, most commonly occurring in trout, caused by an IPN virus.

There is some confusion over the use of the terms IPN and IPN virus. In some uses IPN refers to the virus which causes the disease, but it also refers to the large number of IPN-like viruses that have been isolated from a wide range of freshwater and marine fish, often without any signs of disease. IPN viruses belong to a new group of viruses called birnaviruses. These are classified into several different serotypes including Ab, Sp, and VR-299 strains. There are marked differences in pathogenicity. In Europe the most important strain is Sp. VR-299 is most significant in America.

The Ab strain is of low pathogenicity but occurs widely in wild fish. Although not very important as a cause of disease,

> "it has great significance in relation to diagnostic and certification procedures since it is very difficult to distinguish from other more pathogenic strains without extensive laboratory studies"(1).

2 Detection in New Zealand

IPN virus was first found in 1986 and then twice in 1989. The virus was found at different locations in three separate catchments. In each case the virus was found by MAF Quality Management in apparently healthy fish during export certification tests. The infected fish were all "ocean ranched", returning to hatcheries after spending several years

at sea. Results of examinations of other stock at these sites have been negative.

There have been no disease outbreaks of IPN disease in New Zealand nor has there been any evidence of IPN disease revealed by the research, surveillance and diagnostic work carried out by MAF.

The IPN virus found in 1986 was the Ab strain which is of low pathogenicity. The strains of virus found in 1989 are still being determined.

The detection of IPN virus coincides with the introduction of export tests for fish viruses. This work is carried out by MAF Quality Management at the request and expense of some salmon farmers who seek disease certification for salmon exports to some countries. Each year many hundreds of salmon are used as broodstock although sampling is limited to usually sixty fish from each location. Given the low incidence of IPN and relatively small proportion of fish examined, it would not be unreasonable to expect the virus to be present in other fish and at other locations. Nevertheless the annual testing of virtually all farmed salmon stocks has resulted in thousands of fish being examined over the last few years with no other instances of IPN being found. Most of the testing has been carried out for the VHS virus, with negative results, and IPN has been revealed in the course of those tests. Note that testing of salmon stocks usually involves testing 60 fish out of that group and does not involve testing of all fish in the stock which may number thousands of fish.

There is no evidence to indicate whether IPN is a recent introduction into New Zealand, was introduced up to 100 years ago with the introduction of exotic fish, or has always been present in some native species, either marine or freshwater. However, as the virus has been found only in salmon returning from the sea this suggests the virus is being contracted at sea, possibly through the food chain.

During 1972 it was reported in the press that a viral disease had been found by Dr G C Hewitt in rainbow trout reared by the South Canterbury Acclimatisation Society at the Temuka Hatchery. The basis for that diagnosis is slight and cannot be verified. The hatchery was restocked in 1973 and 1974 under the supervision of MAF. Losses similar to those of 1972 did not recur although bacterial gill disease did cause some losses in 1974 (2). On this evidence it is unlikely that the 1972 losses in the Temuka Hatchery were caused by a virus.

3 Effect of IPN in New Zealand

This finding of IPN in salmon does not affect the export of killed salmon to existing markets.

It may affect export of live ova to some markets from broodstock from which the virus is isolated. MAF Quality Management have responsibility for export certification thus these matters are dealt with by them. The finding of IPN may also affect the negotiations concerning salmon exports to Australia, this responsibility also rests with MAF Quality Management.

The potential impact on trout culture will depend upon the strain and pathogenicity of the isolates as well as the chance of introduction into trout rearing facilities. As the strain identified to date is the Ab strain, and this is considered to be of low pathogenicity, the impact of the IPN found may not be great. Furthermore the strain found in 1986 was tested by experimental exposure to rainbow trout and found to be not pathogenic. (pers. comm. C. Anderson, Wallaceville Animal Research Centre.)

4 Effect of IPN in overseas salmon and trout culture

IPN is present in most if not all the major trout farming countries of Europe, North America, and Japan. Isolates have been found in at least 14 countries including Norway. It was reported in rainbow trout, certified as disease free, imported into Chile. Circumstantial evidence suggests that the IPN virus found in Chile was imported with eggs. The lot of fish from which the virus was isolated was destroyed and the hatchery area disinfected (3). IPN has not been found in Australia despite a survey of over 6000 salmonids from eleven freshwater salmonid hatcheries (4). This survey did not appear to include anadromous fish such as those from which IPN was found in New Zealand.

The present situation in Britain is as follows:

> "As for IPN this disease was first detected in GB in the early 1970s when there were heavy mortalities in affected hatcheries. An increasing number of trout farms became infected over the following years and some of these also experienced high losses in fry. However, today the mortality due to IPN is very variable. In the worst cases, mortalities can persist for weeks and total over 90% in the infected group, but usually the losses are much less and infections without significant mortalities are not uncommon. For the most part in GB, IPN is not now regarded seriously with most farmers considering it an occasional nuisance. A few years ago we amended out (sic) control policy such that although IPN remains a notifiable disease, we do not apply movement restrictions on any trout farms found affected but simply designate them for public information to assist the industry on a "buyer beware" basis. Thus farmers are free to decide for themselves whether to obtain trout and/or salmon ova from IPN affected sites if they so wish. For Atlantic salmon the position is still that movement

restrictions may be applied by MAFF or DAFS, depending upon the receiving site" (5).

5 The disease

IPN disease occurs mainly in one to four month old trout fry where it can cause losses of up to 90% although it can also occur without causing significant mortalities.

Stressful rearing conditions, such as excessive stocking density or handling, enhance the probability of disease outbreaks and may exacerbate their occurrence. Whether mortality will occur in the presence of the virus is likely to depend upon: the type of IPN, the concentration of the virus, method of exposure, age, species and physiological condition of the fish. Fish aged six months or older may become infected without apparent effect and without significant mortality. The Ab strain, while regarded as of low pathogenicity, has caused small losses in hatcheries and in scientific experiments. In his review Hill stated, concerning the disease in wild salmonids:

> "The clinical disease has been observed only under circumstances of hatchery cultivation of fish. To date [1982] there have been no reports of epizootics of IPN disease in wild juvenile salmonids, although this could merely be a reflection of the difficulty in monitoring the health of such populations" (6).

This quotation refers to an epizootic of IPN. Infection of wild fish is well known and is described in section 6, host range.

Survivors from an epizootic generally become life long carriers and may have reduced growth rates.

6 Host range

IPN viruses have been isolated, frequently without apparent effect, from 51 species of fish and shellfish (7). These include 13 species of salmon and trout, 18 freshwater non salmonids including eels, carp, pike, perch and rudd, eight marine fish species, including flounder, and 12 marine shellfish species including Pacific and American oysters, mussels and clams. It remains unresolved whether IPN in shellfish are truly infecting their hosts or are merely contaminating the host tissues as a result of ingestion of infected or contaminated material (6).

Freshwater crayfish exposed to IPN may become carriers and a vector for the virus (8).

IPN virus is pathogenic for rainbow trout, brook trout, and

brown trout. In Atlantic salmon there have been numerous isolations from carrier fish and the Sp strain has caused mortalities in farmed salmon fry in Norway. Few studies have been carried out with chinook. A study in 1963 using an isolate virulent for brook trout found that chinook and kokanee were resistant to the disease.

IPN viruses have been associated with epizootics in Atlantic menhaden (a marine herring) and Japanese eels. The Ab strain was found in fish kills of flounder and several other marine species in the US, however the diversity of species affected suggested that IPN might not have been the specific cause of mortality (9).

7 Transmission

IPN virus can be transmitted horizontally (from fish to adjacent fish) through virus being shed with faeces and urine. Egg-associated transmission can also occur as a consequence of infected fish shedding virus at spawning. Note that the virus isolated in one case in 1989 in New Zealand was found in tissues (pooled kidney and spleen), it was <u>not</u> found in ovarian fluid (pers. comm. C. Anderson). Isolation details from the other cases have not been advised. Routine iodophor disinfection of eggs does not prevent transfer of IPN. The virus could also be disseminated via nets or fish transporters if they are improperly decontaminated. Chlorine disinfection at 200 mg/l for one hour is effective, if properly carried out.

8 The Law concerning IPN in New Zealand

IPN is included under disease control provisions of both the Freshwater Fish Farming Regulations administered by MAF Fisheries and the Animals Act administered by MAF Quality Management. Provisions of that legislation allows MAF wide powers to take appropriate action ranging from doing nothing to destruction of stock.

Following the detection of the virus in 1986 advice of the finding of IPN virus was conveyed, by MAFQual, to the Salmon Farmers Association, Wildlife Service (now the Department of Conservation) and the National Executive of Acclimatisation Societies. MAFQual advised that the finding of IPN in 1986 was not considered to be significant, did not indicate a serious disease risk to the fishing industry or recreational fishing. MAF continued to monitor the situation and no other action was deemed necessary. The 1989 findings were also to be notified to the same interested parties.

9 Control

To date, the isolate identified from New Zealand has no apparent effect in salmon or trout culture or marketing of dead salmon. If found in broodstock it may affect export of those live salmon ova to some markets. Thus individual farms intending to export salmon eggs will need to consider precautions.

In outbreaks of disease from the Sp or VR299 strains there is no effective treatment. Avoidance is the only form of control and is achieved by preventing contact between host fish and virus. This has considerable practical difficulties. Rearing eggs on water supplies from a well or enclosed spring, and obtaining stock from tested broodstock can be considered. Success would depend upon a vigorous and expensive monitoring program and would be difficult for facilities using their own broodstock as the tests currently used take several weeks to complete. Good fish husbandry and reduction of stress may assist in reducing the severity or outbreaks of IPN.

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