

PART V

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INTRODUCTION

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Fish culturists have opportunities to control many of the environmental and host characteristics that are important determinants in the occurrence of fish diseases. These “producer options” have been discussed in earlier parts of this guide where emphasis was placed upon providing suitable rearing facilities, clean and adequate water supplies, good nutrition, immunization, sound genetics, and other factors that contribute to the welfare of the host and the aquatic environment. Throughout these earlier chapters, many diseases were mentioned but no attempt was made to provide detailed information or to identify and compare prevention and control techniques that could be more effective for one disease than another.

The following portion of this guide contains twelve chapters that describe the important diseases of salmonids. Similar outlines have been used to discuss each disease to assure that essential information was provided and to facilitate comparisons among diseases. Each chapter opens with a short introductory summary, a brief description of external and internal signs of the disease, and methods of diagnosis. A discussion of the epizootiology of the disease follows. Like epidemiology in public health, the epizootiology of fish diseases involves the collection and analysis of information that helps to explain when, where, why, and how diseases are introduced or transmitted, and why diseases may vary in severity. Each section on epizootiology includes information on the geographic and host ranges, sources and reservoirs of infection, modes of transmission, susceptibility and resistance factors, incubation period, and seasonal incidence. Methods of control and eradication, together with some of the problems involved, conclude each chapter. Finally, a list of references is given to provide access to the literature. Wherever possible, key review articles have been cited.

The practical value of the disease chapters goes beyond providing ready access to descriptive information and the literature. In contrast to earlier chapters, they provide a different viewpoint on the need for, and methods of, fish disease control with specific reference to the nature of the pathogen and the characteristics of the disease. A clearer understanding of each disease should

help to balance earlier material related to the host and the environment. Knowledge of the dynamic relationships between virulent pathogens and susceptible hosts in fish cultural environments is essential to the development and integration of effective fish health protection procedures.

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INFECTIOUS HEMATOPOIETIC NECROSIS

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Infectious hematopoietic necrosis (IHN) is an acute systemic viral disease of Pacific Coast rainbow and steelhead trout and sockeye and chinook salmon fry and fingerlings (Amend et al. 1969). IHN can quickly kill more than 90% of a population of young fish. More than 100 million cultured and wild trout and salmon fry have died of this disease during the past 10 yr (Mulcahy 1981a). Infectious hematopoietic necrosis is caused by a bullet-shaped virus described by McAllister et al. (1974). The pathogen can be transmitted from fish to fish and from parent to progeny via seminal fluids or infected eggs (Amend et al. 1969; Wolf et al. 1973). Asymptomatic adult carriers shed infective viral particles when spawning (Amend 1975). According to Amend and Pietsch (1972), the parent to progeny transmission of IHN virus can be interrupted by organic iodine disinfection of contaminated eggs. Mulcahy (D. Mulcahy, National Fishery Research Center, Seattle, WA, personal communication) reports, however, that there have been several outbreaks of the disease among fish hatched from disinfected eggs that had been spawned by adult carriers. No therapy will halt an outbreak once it has started.

SIGNS OF INFECTION

Infectious hematopoietic necrosis infections usually are characterized by a sudden, lethal onset but the signs of infection and the times of occurrence of epizootics vary with the host species. Wild sockeye salmon fry are most severely affected at emergence from the spawning bed and during the next two months. In chinook salmon, and steelhead and rainbow trout, losses may occur from the sac fry stage through yearlings. With the possible exception of Idaho rainbow trout, older fish rarely die from IHN. After the first signs of IHN appear, sockeye fry

generally survive for only a few hours; but chinook salmon, steelheads, and rainbows may live for a day or more.

EXTERNAL SIGNS

1. IHN should be suspected if sockeye salmon fry appear dark, lethargic, whirl through the water and suffer a sudden acute mortality. Chinook salmon and steelhead and rainbow trout may reach 100 to 30/lb before IHN strikes. Infected fish usually become dark, exophthalmic, avoid the water current, drift against the tail screen and die.

2. In quiet water, long, opaque, off-white fecal casts can be observed trailing from the vents of rainbow trout fingerlings.

3. Abdominal swelling and pale gills are additional signs commonly observed.

INTERNAL SIGNS

1. In infected sockeye salmon fry, the kidney is often translucent and speckled with pigment cells. Other organs are usually pale except for the spleen which is cherry red.

2. A clear, straw-colored fluid accumulates in the body cavity of some fish infected with IHN, but most internal signs are similar to those caused by other viral diseases.

DIAGNOSIS

During IHN outbreaks, a generalized systemic viremia develops in which the virus can be detected in almost any tissue (Amend 1974). A confirmed diagnosis requires isolation of the IHN virus from fish with typical signs of the disease and the identification of the virus by neutralization tests with anti-IHN serum (Amend 1970a). In Southern Idaho and Oregon, rainbow trout have been found to carry dual infections of IHNV and infectious pancreatic necrosis virus (Mulcahy and Fryer 1976) so care must be taken to check for other viruses.

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

Infectious hematopoietic necrosis is common in cultured and wild salmonid populations on the Pacific coast of the U.S. and Canada from California to Alaska and inland to Idaho. The disease is ubiquitous in sockeye salmon (Amend and Wood 1972; Grischkowsky and Amend 1976; Williams and Amend 1976). In wild sockeye salmon stocks, significant natural outbreaks may kill more than 40% of the emergent fry (Williams and Amend 1976).

In Idaho, IHN has become one of the most serious fish disease problems facing fish culturists in the large commercial rainbow trout hatcheries. Fry losses commonly exceed 40% in badly infected lots. As the disease becomes established, outbreaks may occur among rainbow trout of almost any age. Records show that this disease is now a threat to much older rainbows than in the past (D.

Mulcahy, National Fishery Research Center, Seattle, WA, personal communication).

Isolated IHN outbreaks have been reported in Colorado, Minnesota, New York, South Dakota, West Virginia, and possibly elsewhere (Wolf et al. 1973; Carlisle et al. 1979). In most instances, these epizootics have been associated with the shipment of infected rainbow trout eggs.

Sources and Reservoirs of Infection

Infected adult salmonids shed IHN virus at spawning time in ovarian and seminal fluids. The proportion of adults with detectable IHN virus increases as the spawning season progresses (Amend 1975; Mulcahy 1981a). During spawning, a higher proportion of sockeye salmon females than males have been found to be IHN virus carriers (Mulcahy and Fryer 1976). Ovarian fluid, collected from spawned-out females within a week after spawning, often carries more IHN virus than fluid collected from the same fish when eggs were taken. Although Amend (1975) considered IHN virus to be an external contaminant of eggs, outbreaks of the disease among fish hatched from eggs disinfected with an organic iodine disinfectant suggests that the virus may occur within eggs or as a surface contaminant (D. Mulcahy, National Fishery Research Center, Seattle, WA, personal communication). This hypothesis merits further research since egg disinfection is sometimes assumed to be an effective preventive measure.

MODES OF TRANSMISSION

Egg-associated transmission of IHN virus is the primary mode of transmission but the disease has been transmitted by several other means. Early work by Guenther et al. (1959) showed that the disease can be introduced by feeding raw, frozen salmon viscera from canneries. Amend et al. (1969; 1975) found that the virus can also be transmitted by adding infected cell culture medium to tanks containing susceptible experimental fish, by placing healthy fish in a screened-off section of a tank next to infected fish, and by feeding raw, ground, infected fry to healthy fish.

Susceptibility AND RESISTANCE FACTORS

Sockeye and chinook salmon and rainbow and steelhead trout are severely affected by IHN. Although other species of salmonids may occur in the same waters as these species, it appears that coho salmon and other trout species are more resistant. Early work on Sacramento River chinook disease (SRCD) (now recognized as a member of the IHN group of viral diseases) showed that SRCD did not occur at water temperatures over 15°C (Amend 1970b). This may indicate the existence of several geographic strains of IHN virus because this phenomenon applies only to chinook salmon in the Sacramento River basin (D. Mulcahy, National Fishery Research Center, Seattle, WA, personal communication). Work by Hetrick et al (1979) showed that rainbow trout were readily infected by IHN virus at 15 and 18°C. These differing responses could be caused by interactions between different geographical strains of virus, amounts of virus present, and the strain, species, and age of the host.

INCUBATION PERIOD

Experimental work with rainbow trout fingerlings has shown that the incubation period of IHN is directly related to water temperature (Hetrick et al. 1979). Hetrick and his co-workers found the mean number of days to death following exposure to the virus ranged from as little as 5.5 d at 21°C to as long as 15.8 d at 3°C. In sockeye salmon and steelhead trout the incubation period is similar. Increasing the water temperature to 18°C for 4-6 d protects chinook salmon fingerlings against the Sacramento River strain of IHN virus rather than shortening the incubation period (Amend 1970). If these same fingerlings are exposed later at lower water temperatures, they become infected.

SEASONAL INCIDENCE

IHN disease coincides with the occurrence of susceptible stages of host fishes thereby suggesting seasonality of the disease in some species and locations. Seasonal increases in population densities and the accompanying stresses also give rise to what appear to be seasonal changes in the incidence of IHN. Since trout and salmon are spawned over most of the year, fish culturists must be continually alert to the possible occurrence of IHN.

METHODS OF CONTROL

PREVENTION

Avoiding the introduction of IHN-infected eggs and fish is the only sure method of prevention. The transfer of salmon or trout eggs or fish from California, Oregon, Idaho, Washington, British Columbia and Alaska into areas where the disease is not known to occur should be done with caution. If eggs must be obtained from these areas, they should be obtained from properly inspected stocks and should be thoroughly disinfected with an organic iodine disinfectant both prior to and after shipment (Amend and Pietsch 1972). An organic iodine disinfectant solution that provides 100 ppm iodine at pH 6.0 or higher for 10 min has been recommended (Amend 1974). Organic iodine disinfection is not a guarantee against transfer of the virus.

The infectious hematopoietic necrosis virus can be reliably detected in carrier fish only at spawning time and during epizootics (Amend 1970a, 1975). Repeated inspections employing thorough virological sampling are the best means of detecting carriers of IHN virus. However, even in carefully inspected populations, the disease can appear unexpectedly (Mulcahy 1981b).

Temperature modification to prevent losses in chinook salmon is not recommended because of the frequent occurrence of temperature-resistant virus strains, high energy costs, and the post-exposure persistence of IHN carriers (Amend, 1974).

THERAPY

No drugs or chemicals are known that will halt outbreaks of IHN.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

Certain hatchery practices will aggravate an existing IHN problem. According to Mulcahy (National Fishery Research Center, Seattle, WA, personal communication), heavy loading of eggs or fry in incubators is an important factor contributing to the severity of outbreaks. Water reuse, handling stresses and other adverse conditions increase the impact of the disease on older fish.

When an IHN outbreak is suspected, immediate isolation (quarantine) of the affected lot of fish will ~~h~~spread of the disease to other rearing units or to other lots of fish. If the number of eggs or fry in individual rearing units can be reduced at the outset, fewer fish will contract the disease if an outbreak occurs. Upon confirmation of IHN, prompt destruction of the affected fish or lot, followed by intense chlorine disinfection of all associated incubators, rearing units and equipment, is recommended (Amend 1974).

LONG TERM

Procedures for the elimination of IHN in cultured rainbow trout differ from those that might be used in wild anadromous populations of sockeye and chinook salmon and steelhead trout. When a coldwater fish hatchery has been effectively disinfected and is supplied with an uninfected water source, the key to long-term IHN control lies in obtaining reliably inspected stocks of eggs or fish only from sources shown to be free of this disease. The importation of thoroughly disinfected eggs is probably safer than importing fish, but stocks originating from the West coast of North America and from Idaho should be avoided.

The elimination of IHN from anadromous fish populations is very difficult. There is no way to effectively deal with transmission of the disease in naturally spawning fish. Adults that return to hatcheries can be inspected for the presence of IHN and other diseases, but there is seldom enough time between the arrival of adults at a facility and spawn taking to allow for tests to be able to distinguish between uninfected fish and carriers. In any event, artificially spawned eggs from anadromous stocks should always be disinfected and incubated at the lowest feasible density. If hatcheries are stream-fed, a coordinated effort should be made to prevent carrier fish from migrating into the water supply stream. IHN-eradication procedures should begin with the uppermost hatchery in the watershed. As long as eggs are available from sources free of IHN, this disease should not be considered a problem that must be "lived with", especially in rainbow trout programs (D. Mulcahy, National Fishery Research Center, Seattle, WA, personal communication).

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