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INFECTIOUS PANCREATIC NECROSIS

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Infectious pancreatic necrosis (IPN) is a viral infection primarily of trout and salmon, but the virus has also been isolated from a wide variety of other fish species. The infection is characteristically seen in trout as an acute disease causing high mortality in fry and fingerlings. However, it may also occur as a benign and inconspicuous infection (Bullock et al. 1976). Because high fish losses are often associated with IPN outbreaks, it is considered one of the major fish disease problems in the United States, Canada, and Europe (Desautels and MacKelvie 1975).

SIGNS OF INFECTION

The first sign of a typical IPN epizootic is a sudden increase in mortality. The largest and most vigorous fry or fingerlings usually are affected first. A whirling behavior is typical when the mortality rate is high; affected individuals swim in a rotating manner about their long axis. Abnormal movements may be slow and feeble or rapid and frantic. When not otherwise obvious, the whirling response may sometimes be elicited by a sharp rap on the trough. Moribund behavior may alternate between periods of quiescence, during which victims lie on the bottom and respire weakly, and convulsive frenzies. Whirling is a terminal sign, and death usually ensues within an hour or two although this characteristic behavior may be absent among very young fish or fish of poor quality (Wolf 1966). Other signs of infection that may be observed include an overall darkening of individual fish, exophthalmia, abdominal distension, and hemorrhages in ventral areas. Tiny hemorrhages may occur among the pyloric caeca, and the liver and spleen are usually pale. The digestive tract is usually devoid of food. A clear or milky

mucus may be found in the stomach and anterior intestine, a distinctive characteristic of IPN.

DIAGNOSIS

If young trout suffer a rapidly increasing mortality, exhibit some of the signs described, and are free of pathogenic bacteria and parasites, there is a possibility that the fish have IPN (Wolf 1966). Confirmation of IPN requires isolation of the virus in cell culture and identification by a serum neutralization test using polyvalent, anti-IPN virus serum. IPN diagnostic and inspection procedures and the methods for IPN confirmation are described in the Canadian Fish Health Protection Regulations "Manual of Compliance" (Anon. 1977), in "Procedures for the Detection and Identification of Certain Fish Pathogens" (McDaniel 1979), and in Ljungberg and Jorgenson (1973).

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

IPN virus has been isolated from fish in the United Kingdom (Wolf 1972), Scandinavia (Ljungberg and Jorgenson 1973), Europe, Japan, and North America (Hill 1977). IPN virus has been isolated in brook, brown, rainbow and cutthroat trout; Atlantic, coho, chinook, amago, and himemasu salmon; and eels. IPN-like viruses have also been isolated from carp, perch, roach, bream, pike, and white suckers (Hill 1977).

SOURCES AND RESERVOIRS OF INFECTION

Infected fish serve as reservoirs of infection. During epizootics, virus particles are shed into the water with feces, eggs, and seminal and ovarian fluids. High levels of virus are present during IPN outbreaks. Surviving fish become carriers and intermittently shed virus over a long period of time (Wolf 1966). It has also been demonstrated that viable virus can remain with eggs in spite of disinfection (Bullock et al. 1976). Yamamoto and Kilistoff (1979) have determined that brook trout infected with IPN virus at the time of stocking will harbor virus over a period of several years.

SUSCEPTIBILITY AND RESISTANCE FACTORS

Several factors affect the overall mortality rate in a population of salmonid fry infected with IPN virus. The susceptibility of salmonid fishes to IPN disease decreases with increasing age, the most susceptible fish being first-feeding fry. High resistance is usually, but not invariably, achieved at an age of 4-6 months. Another major factor determining the overall mortality from an IPN infection is the particular strain of IPN virus involved. It has been found that strains of IPN virus may differ in virulence and may produce mortalities among trout ranging from under 10% to over 90%. Most strains are able to produce high mortality but the susceptibility varies from species to species with higher mortality occurring in brook and rainbow trout than in brown trout and Atlantic salmon. The virus is also

pathogenic to amago and himemasu salmon, both species showing high mortalities (48.62%) in six and eight week old fry (Sane 1973). Although coho may serve as carriers of IPN virus, there is no report of a mortality caused by IPN in this species (Wolf and Pettijohn 1970). Other factors such as the dose of the virus, the route of infection, density of the fish population, water temperature, and the presence or absence of "stress" conditions all affect mortality (Hill 1977).

MODES OF TRANSMISSION

'An important feature of IPN disease, from an epizootiological point of view, is the fact that most survivors of infection become lie-long virus carriers and thus shed varying quantities of virus over a long period. This results in a typical transmission of the disease from parents to progeny via the egg, and is probably one of the main factors for the geographical spread of IPN" (Hill 1977).

According to Wolf (1966): "Quite likely, egg transmission is the normal means by which the virus is passed from one generation to another."

INCUBATION PERIOD

The incubation period of IPN infection is temperature dependent, ranging from 6 d at 125°C to several weeks at 4°C (Wolf 1966).

METHODS OF CONTROL

PREVENTION

Avoidance is the most effective control measure. This requires the incubation of virus-free eggs and the propagation of IPN-free stock in an uncontaminated water supply. This approach is the method of choice. Success depends on a rigorous fish health inspection program to prevent the introduction or inadvertent spread of IPN.

Under circumstances where avoidance is not possible, the mortality associated with IPN may be reduced by rearing fry for 4 months at cold water temperatures (less than 7°C). Even so, the problem of the carrier state in surviving fish persists. This procedure may reduce loss rates because the cold water temperatures during the period of greatest fry susceptibility are cold enough to prevent mortality. Cold water temperatures also appear to have a depressing effect upon infectivity and/or transmission of the virus (Frantsi and Savan 1971).

If a hatchery must operate with water from streams containing IPN virus carriers, the water should be treated to eliminate the IPN virus. Ozone appears to be effective for this (Wedemeyer et al. 1978).

THERAPY

There is no effective treatment for IPN but Economon (1972) has reported some success with povidone iodine treatments.

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

IMMEDIATE

As there is no effective therapy, the only immediate control of the virus is to eliminate infected stocks.

LONG TERM

Inspect all egg sources and hatchery fish populations at least annually for the presence of IPNV.

-Phase out all IPN virus infected broodstock and in the interim, do not transfer eggs from infected broodstocks to any hatcheries which are free of the disease.

Under no circumstances should infected fish be stocked into lakes, reservoirs, or streams that serve as water sources for hatcheries or that serve as broodstock sources. There is evidence that planting only IPN virus-free fish into areas previously planted with IPN virus carriers may ultimately lead to a carrier-free state (Yamamoto and Kilistoff 1979).

Any fry or fingerlings suffering from clinical IPN disease should be incinerated or buried with unslaked lime.

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