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VIRAL HEMORRHAGIC SEPTICEMIA

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Viral hemorrhagic septicemia (VHS) is an acute to chronic viral disease of salmonids that causes serious economic problems in rainbow trout cultured in several European countries. Although this disease has not been detected in North America, it is included here as a disease of significant concern. The disease was first recognized by Schaeperclaus in Germany in 1938. In 1949, the disease was named Egtved disease after an outbreak in Denmark near a village of that name. In 1966, the Office Internationale d'Epizooties recommended that the name be changed to viral hemorrhagic septicemia to reduce confusion due to the names used in the various European countries where it occurs (Roberts 1978).

Viral hemorrhagic septicemia is caused by a bullet-shaped virus similar in size and shape to the virus that causes infectious hematopoietic necrosis (IHN) in North American salmonids. The VHS virus can be distinguished from IHN by specific serum neutralization tests (McAllister et al. 1974). Losses to VHS among infected rainbow trout fingerlings often exceed 90%. If fish are exposed for the first time at older ages, the resultant disease is more chronic and has a more prolonged course. Losses can be severe in cold water under crowded, stressful situations, even among older fish. The disease is transmitted by contact and from fish to fish through the water (Rasmussen 1965). If parent to progeny transmission occurs, it is suspected that the virus is spread as a contaminant during spawning operations rather than by the virus being carried within the egg (Roberts 1978). Outbreaks of VHS are most common and most severe during the winter. Losses taper off in the spring as water temperatures rise, cease during the summer, then recur sporadically in the fall. As for other viral diseases, there is no therapy for VHS. Avoidance is the only successful control technique (Ghittino 1965).

SIGNS OF INFECTION

Viral hemorrhagic septicemia appears first in fingerlings which have been feeding for 40 d or more. Early clinical signs can easily be confused with other viral, bacterial or parasitic infections. Later in the course of the disease, signs of excitability become apparent, including erratic swimming similar to the behavior of trout dying of whirling disease caused by the parasite *Myxosoma cevebdis* (Roberts 1978).

EXTERNAL SIGNS

1. Rainbow trout involved in acute outbreaks of VHS are dark in color, lethargic, and exhibit hemorrhages in the fin sockets. Exophthalmia (pop-eye) is common and persists throughout the course of the disease.
2. As the disease progresses, affected fish become nearly black. An acute anemia develops, and the gills are pale in color.
3. After several months, the mortality finally may cease and some of the remaining fish often display whirling behavior, erratic swimming and nervousness.

INTERNAL SIGNS

1. During acute outbreaks, small hemorrhages are common in the musculature, gills, and visceral organs. Massive hemorrhages can often be found in the abdominal cavity of freshly dead fish (Roberts 1978).
2. During mid-stages of a disease outbreak, internal organs become very pale. Visceral, intramuscular, and gill hemorrhages develop as distinctive signs of the disease.
3. In late stages of the disease, kidneys become swollen and discolored.

DIAGNOSIS

A confirmed diagnosis of VHS can be made only by isolating and serologically identifying the causative virus in an appropriate cell culture system (Jorgensen 1974). The VHS virus is similar to the agent of IHN in that it can be isolated only from fish during an active epizootic or from ovarian fluid from adult carriers at the time of spawning or shortly thereafter. The virus can seldom be isolated from asymptomatic fish at other times.

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST Ranges

Numerous, severe outbreaks of VHS have occurred in Denmark, Germany, France, Italy, Switzerland and the Scandinavian countries. It has not been detected in the British Isles, in North or South America, or in the Far East (Roberts 1978).

VHS is especially serious among hatchery-reared rainbow trout. Cultured Atlantic salmon and brown trout have suffered VHS outbreaks. Brook trout have

been experimentally infected in the laboratory. All reported cases of VHS occurred among cultured fish. No natural outbreaks have been observed in wild fish populations (Roberts 1978).

SOURCES AND Reservoirs OF INFECTION

There is strong circumstantial evidence that survivors of VHS epizootics become asymptomatic carriers and serve as reservoirs of infection. The primary sources of VHS, therefore, involve the transfer of asymptomatic carriers to new areas, shipments of eggs contaminated by virus-laden ovarian fluids, or water-borne infectivity from upstream epizootics.

MODES OF TRANSMISSION

Experimental transmission of VHS has been accomplished by the injection of infected cell culture fluid (Jensen 1965), by brushing infected fish tissue suspensions on the gills of healthy fish (Jorgensen 1970), by injecting filtrates of organ homogenates, and from fish to fish through flowing water (Rasmussen 1965). The virus survives for more than 24 hr in 14°C water and for at least a week in the dry state (Jorgensen 1974). Potential transmission can occur through virus-contaminated nets, boots, egg crates or other equipment.

Jorgensen (1970) demonstrated that VHS virus could be transmitted as an external contaminant of eggs. Ghittino (1965) attributed a 1964 outbreak he observed to VHS-infected eggs. These findings are similar to those of Amend (1975) in his work with IHN virus.

SUSCEPTIBILITY AND RESISTANCE FACTORS

Although VHS is predominantly a disease of rainbow trout, other species can be affected. The course and severity of VHS appears to be age-dependent (Roberts 1978; Bellet 1965). Losses to VHS are most severe among fingerling rainbow trout 2-6 months of age; yearling fish often have a milder case and many survive. Rainbow trout older than two years are almost completely refractile to infection (Bellet 1965).

Stress plays a significant role in the development of epizootics. Overcrowding, malnutrition, handling, and transportation can lead to high losses due to VHS. Under optimal fish cultural conditions, few fish will show signs of the disease until a stressful condition finally occurs (Jorgensen 1974).

Water temperature also plays an important role in the course of VHS and the disease is a more serious problem at water temperatures colder than 15-16°C (Bellet 1965). This helps to explain why VHS is so severe in fingerling trout during their first winter (Roberts 1978).

Incubation PERIOD

Losses to VHS occur 6 days after gill exposures at 15°C and at 8-11 d if the virus is added to the water at 10°C (Jorgensen 1974). Losses usually continue for several months thereafter (Rasmussen 1965).

On Danish trout farms, the incubation period is reported to vary from 7-15 d. If fish cultural conditions are favorable for the fish, incubation periods of up to a year or more may occur following exposure to VHS virus through the water supply from upstream epizootics (Jorgensen 1974).

SEASONAL INCIDENCE

Viral hemorrhagic septicemia is a winter and spring time disease. Outbreaks are most severe when water temperatures are below 8-10°C. At hatcheries supplied with cold spring water or well water, the disease can be a year-round problem (Rasmussen 1965).

METHODS OF CONTROL

PREVENTION

Avoidance of VHS is by far the best approach to control (Ghittino 1965). No fish or eggs should be introduced from areas where VHS has been detected.

THERAPY

There is no cure for VHS. Before discovery of the viral etiology of VHS, several European veterinarians believed the disease was the result of nutritional deficiencies (Rasmussen 1965; Ghittino 1965). The finding of a viral agent explained why nutritional supplements and antibacterial treatments failed to control losses (Rasmussen 1965; Bellet 1963).

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

IMMEDIATE

Viral hemorrhagic septicemia has not been recognized outside of Europe. If VHS is detected in new areas, an immediate quarantine should be placed on the affected facility. Water flows should be turned off to prevent release of a contaminated effluent and possible spread of the virus downstream. Destruction and proper disposal of the fish, followed by prompt, thorough disinfection of all facilities and associated equipment should commence as soon as possible. Valuable time should not be wasted on deliberations on what to do when the disease has been confirmed. A serious problem associated with possible spread of the disease exists until the water supply has been turned off, the fish buried, and the facility disinfected. The source of the infection must be identified and corrective measures must be undertaken to ensure that reinfection does not occur.

LONG TERM

Danish authorities, prompted by catastrophic fish losses due to VHS, initiated a voluntary disease eradication plan in 1965. According to Jorgensen (1977), hatcheries were inspected and a register of disinfected or clean farms

was established. In 1969, the Danish Parliament made the voluntary program mandatory. Between 1965 and 1976, a total of 481 farms were depopulated of fish, disinfected, and repopulated with VHS-free fish. Reinfection, after disinfection, was common in Danish hatcheries unless neighboring facilities had also been treated. By June 1977, 380 farms had been registered as free of VHS.

According to Gbittino (1965), "The best prophylaxis against VHS is the perfect isolation of healthy fish from any possible source of infection." To date, hatcheries and fisheries resources in the Americas have been free of VHS. Vigilance, understanding, and strict control over introductions of salmonid eggs or fish from Europe are essential if this status is to be maintained.

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