

**LITERATURE REVIEW**  
**Viral Hemorrhagic Septicemia Virus**  
**VHSV**

***PELTON ROUND BUTTE HYDROELECTRIC PROJECT***  
***FERC No. 2030***

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## INTRODUCTION

Viral hemorrhagic septicemia virus (VHSV), European strain, is the causative agent of a severe affliction of rainbow trout (*Oncorhynchus mykiss*), other salmonids, and some non salmonid fishes. This disease has been associated with aquaculture of trout in Europe and was first recognized in Denmark. It appears that the virus was endemic in brown trout (*Salmo trutta*), Atlantic salmon (*Salmo salar*) and Danube salmon (*Hucho hucho*) in Europe. The disease was found throughout Europe by the 1930s. With higher densities of fish in culture and the introduction of rainbow trout, the virus proved to be highly virulent for this new host. Brook trout (*Salvelinus fontinalis*) were highly susceptible, and grayling (*Thymallus arcticus*) and whitefish (*Prosopium sp.*) were also observed to be infected by the virus (Ahne, W. and I. Thomsen. 1985).

In the mid 1960s VHSV was first observed by electron microscopy and appeared similar in morphology to the rhabdovirus, vesicular stomatitis virus (VSV). The F1 type strain was designated as the reference strain. Although similar in shape to Infectious Hematopoietic Necrosis Virus (IHNV), McAllister and co-workers determined the two were serologically unrelated (1974). Histological examinations of fish tissues infected with IHNV, VHSV, or infectious pancreatic necrosis virus (IPNV) supported the conclusion that these were all distinct and different viruses (Yasutake 1970).

Serological studies in Denmark in the 1970s revealed a second strain of VHSV. In the late 1970s, a third serotype of VHSV was discovered by de Kinkelin (1977). The first reports of VHSV's ability to infect saltwater species of fish were in the early 1980s. Sea bass (*Dicentrarchus labrax*) and turbot (*Scophthalmus maximus*) young were susceptible to waterborne challenge with VHSV (Castric and de Kinkelin 1984). In another instance, fish transferred to sea water exhibited 80% mortality from VHSV infection (Castric and de Kinkelin 1980). Feral rainbow trout were found to have culturable VHSV only during low temperature times of the year. Presumably, this is when the immune system of the fish was less responsive to infection (Jorgensen 1982).

In the late 1980s, VHSV was detected for the first time in North America among spawning chinook (*O. tshawytscha*) and coho salmon (*O. kisutch*). This discovery led to extreme measures being taken to disinfect the facilities and destroy all fish and eggs associated with the facility (Brunson et al. 1989 and Hopper 1989). Experimental work has since suggested that, although the North American VHSV (NA-VHSV) is biochemically and serologically similar to the European isolate, it is much less virulent (Winton et al. 1991).

## SIGNS OF VHSV DISEASES

The disease usually occurs in the absence of secondary bacterial infections. However, secondary viral infections do occasionally occur with IPNV. Three forms of VHSV have been noted: chronic, acute, and latent. Typically the fish go off feed and the behavior is erratic varying between lethargic and frantic. The weakened condition causes the fish to drift and lose orientation. The fish are noticeably darker, and exophthalmia is often exhibited. Gills are pale, and petechial hemorrhages are common in musculature, gills, and the base of fins (Yasutake 1970). Histopathology has shown that the kidneys are the principal target organ of infection. The spleen exhibits damage similar to that of the kidney. Necrosis and degeneration of the liver also commonly occur. In contrast to IHNV, the pancreatic tissue is less affected. Because of the damage to the hematopoietic tissues, blood cells are highly affected and the hematocrit values are depressed (Wolf 1988). These changes occur in trout, salmon, and saltwater species. Northern pike (*Esox lucius*), however, have much more severe involvement of the musculature (Yasutake 1970).

## PROPERTIES OF VHSV

Viral hemorrhagic septicemia virus was first described by Jensen in 1963. He noted that it was sensitive to ether and glycerol indicating a lipid enveloped virus. VHSV grows between 4 and 20°C. Moderate temperatures inactivated the virus as shown by the loss of infectivity at room temperature. Electron micrographs revealed a bullet shaped morphology of the virus particle with dimensions of 75 X 240 nm (de Kinkelin and Scherrer 1970). The genome of VHSV, like IHNV, is a single stranded RNA molecule of negative sense. It is composed of five genes and there are respectively five structural proteins in the virion coded by these genes. In many ways, it is biochemically similar to IHNV and has been placed in the same genus. However, molecular genetics and serological analysis of the two viruses has defined them as distinct agents (Winton et al. 1991).

## TRANSMISSION

Waterborne transmission occurs at temperatures of 1 to 15°C. In the laboratory, infection by immersion requires about 1 to 2 X 10<sup>4</sup> plaque forming units (pfu) per milliliter of water to cause disease. After an outbreak, survivors are known to become carriers of the virus especially at lower temperatures. These carrier fish continue to shed virus (Wolf 1988).

## HOST RANGE

Atlantic salmon, brook trout, brown trout, rainbow trout, golden trout (*O. aquabonita*), grayling, lake trout (*Salvelinus namaycush*), pike, sea bass, and turbot are all susceptible to infection by VHSV from Europe. Infection from the European strain of VHSV has not been demonstrated in chinook or coho salmon. The European strain is found throughout Europe and as far east as Russia, but surprisingly not in the British Isles (Wolf 1988).

## DISTRIBUTION AND POTENTIAL VIRULENCE IN NORTH AMERICA

Marine reservoirs of NA-VHSV may be important in the distribution of the virus to the Pacific Northwest. The NA-VHSV strain has been found in coho and chinook salmon from Alaska to the mouth of the Columbia River. It has also been detected in Pacific cod (*Gadus macrocephalus*) and herring (*Clupea pallasii*) and has been associated with fish stressed by oil pollution and lesions on herring. The collapse of the herring and cod fisheries has been attributed, in part, to NA-VHSV infection of these fish (Ted Meyers, Alaska Fish and Game, personal communication). Yearling coho salmon and winter run steelhead trout (*O. mykiss*) as well as fall run chinook fry have been found to be inapparently or transiently infected by NA-VHSV. Experimental infection of rainbow trout has produced contradicting results as to the susceptibility of this species. Winton and co-workers (1989) found that no disease was associated with the infection. While, Follett and co-workers (1997) found a 12% mortality when rainbow trout were exposed to  $10^5$  pfu of NA-VHSV. Differences in procedures and quantities of virus may account for these conflicting results.

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