

North American Viral Hemorrhagic Septicemia Virus (NA-VHSV)

I. Causative Agent and Disease

North American viral hemorrhagic septicemia virus (NA-VHSV) Type IVa is a bullet-shaped RNA novirhabdovirus. It is molecularly distinct from the similar Type IVb in the Great Lakes, USA that is pathogenic for a large number of nonsalmonid species. It is also different from Type IVc in marine/freshwater species of Atlantic Canada and most VHSV strains in Asia (IVa occurs in Japan/Korea) and Europe (Types I, II, III) that are pathogenic for some marine species and rainbow trout.

II. Host Species

NA-VHSV Type IVa infects many marine fish species in the northern Pacific Ocean including anadromous coho and Chinook salmon. In Alaska, the virus is reported from Pacific herring, Pacific cod, Pacific hake and walleye pollock. Two isolates from pink salmon have been the only occurrences of VHSV from a free ranging Alaskan salmonid. The virus is enzootic in populations of Northern Pacific herring and sardines causing epizootic mortality. Experimental studies indicate that juvenile Alaskan Chinook, coho, pink and sockeye salmon are refractory to the virus by waterborne exposure.

III. Clinical Signs

Detection of NA-VHSV from anadromous salmonids in Washington and Oregon has generally been at very low levels and prevalences and not associated with clinical disease. In Pacific cod, secondary VHSV infection can be detected at low levels in skin erosions and ulcers caused by other primary pathogens. Septicemia with skin hemorrhages may also occur. In Pacific herring, the virus can be acutely lethal for up to

100% of exposed juvenile fish with lower chronic mortality occurring in adults. Infected juvenile herring develop hemorrhages of the skin around the mouth and isthmus and/or at the base of fins while occasional hemorrhages occur in adult fish along the flanks that may progress to ulcers. Fin erosion and lethargic swimming behavior may also be present. Experimentally infected juvenile rainbow trout exhibited darkened body color and hemorrhaging at the base of fins and vent associated with low mortality.

IV. Transmission

Transmission of VHSV is horizontal through ambient seawater from fish to fish and likely by ingestion of infected fish. Individual infected juvenile Pacific herring can shed up to $10^{6.5}$ plaque forming units (PFU) of virus per ml. Primary virus infection is through the epidermis and possibly gill tissues followed by systemic infection (viremia). Because VHSV in the Pacific Northwest is indigenous to Pacific herring and other forage species utilized by salmon, these prey are a likely source of VHSV periodically detected in adult coho and Chinook salmon in Washington State.

V. Diagnosis

Cultures of susceptible fish cell lines are inoculated with kidney, spleen, liver, ovarian fluids or epidermal lesions from suspect fish. Presumptive diagnosis is made when characteristic cytopathic effect (CPE) or lysis occurs in cell monolayers from virus infection. Virus identification is confirmed by PCR.

VI. Prognosis for Host

Susceptible juvenile herring sustain up to 100% mortality which may not occur in adult fish or is lower and more

VIRUSES

chronic. Herring that survive virus infection develop apparent immunity to re-infection. Noteworthy, is that low levels of VHSV can occasionally be detected in a small percentage of apparently healthy herring from most populations. Clinical disease and mortality from the virus is

variable but generally lower in other forage species.

VII. Human Health Significance

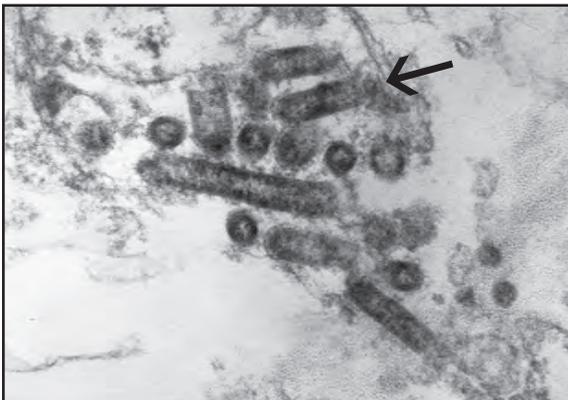
There are no human health concerns associated with NA-VHS virus.



Skin hemorrhaging in infected Pacific herring



Left: Pacific herring with typical VHS hemorrhage; **Right:** Skin hemorrhaging in infected Pacific cod (photo: NMFS staff)



Electron micrograph of VHSV particles (arrow) in a cultured EPC fish cell, X 56,500