I. Causative Agent and Disease

North American viral hemorrhagic septicemia virus (NA-VHSV) Type IVa is a bullet-shaped RNA rhabdovirus. It is molecularly distinct from a similar virus (Type IVb) found in the Great Lakes, USA that is pathogenic for a large number of non-salmonid fish species and different from the VHSV strains in Europe that are pathogenic for rainbow trout (Egtved virus) and much less so for some marine fish species.

II. Host Species

NA-VHSV Type IVa infects a wide range of marine host species in the northern Pacific Ocean including anadromous coho and Chinook salmon. In Alaska, the virus has been found in Pacific herring, Pacific cod, Pacific hake and walleye pollock but has not occurred naturally in salmonids. The virus has been associated with epizootic mortality, mostly in Pacific herring and sardines and is enzootic in populations of Northern Pacific herring. Experimental studies have shown that juvenile Alaskan Chinook, coho, pink and sockeye salmon are refractory to the virus by waterborne exposure.

III. Clinical Signs

Detection of NA-VHSV in anadromous salmonids in Washington and Oregon has generally been at very low levels and prevalences of virus and has not been associated with clinical disease. In Pacific cod, secondary VHSV infection has been detected at low levels in skin erosions and ulcers caused by other primary pathogens, but does not become systemic. In Pacific herring the virus can be acutely lethal for up to 100% of exposed juvenile herring with lower chronic mortality occurring in adult fish. 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 in some cases. Fin erosion and lethargic swimming behavior may be present as well. 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 the 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 of cells in cell monolayers occurs from virus infection. Virus identification is confirmed by PCR.

VI. Prognosis for Host

Susceptible juvenile herring sustain mortality up to 100% while mortal-
VIRUSES

It may not occur in adult fish or if so mortality is lower and more chronic. Herring that survive virus infection develop apparent immunity to reinfection. Noteworthy is that low-levels of VHSV are generally found in a small percentage of apparently healthy herring from most populations. Clinical disease and mortality from the virus is variable but generally low 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 often caused by VHSV

Pacific herring with typical VHS lesion

Ultrastructural section of virus particles (arrow) in cultured fish cell, TEM