

Piscine Orthoreovirus (PRV)

I. Causative Agent and Disease

Piscine orthoreovirus (PRV), also known as Atlantic salmon reovirus, was identified in 2010 by next generation sequencing of tissues from farmed Atlantic salmon in Norway dying from the disease “heart and skeletal muscle inflammation” (HSMI). The virus has double-stranded RNA with 10 segments and is 72 nm in diameter. There are three strains of the virus (PRV1, 2, 3) possibly influencing disease outcome in different host species under different environmental conditions.

II. Host Species

PRV is reported from Norway, Denmark, Ireland, Chile, Japan and the Pacific Northwest (WA, AK, BC, Canada) infecting Atlantic salmon, Pacific salmon and trout (cutthroat, steelhead, sea-run brown). In Alaska, PRV was sequenced from three stocks of coho and one stock of Chinook and unconfirmed in one stock of chum salmon.

III. Clinical Signs

HSMI, described from Norway in 1999, causes anorexia, lethargy, and ascites with inflammatory lesions of the heart and skeletal muscle. It is a disease of farmed Atlantic salmon (PRV-1) and rainbow trout (PRV-3) in both freshwater and seawater. PRV-1 has been associated with jaundice, anemia and degenerative/necrotic lesions of the liver/kidney in healthy farmed Chinook salmon and PRV-2 is associated with EIBS and jaundice/anemia in farmed coho salmon in Japan. Stress may precipitate clinical disease.

IV. Transmission

PRV can be transmitted by injection and horizontally. Marine forage fish

species may be possible reservoirs.

V. Diagnosis

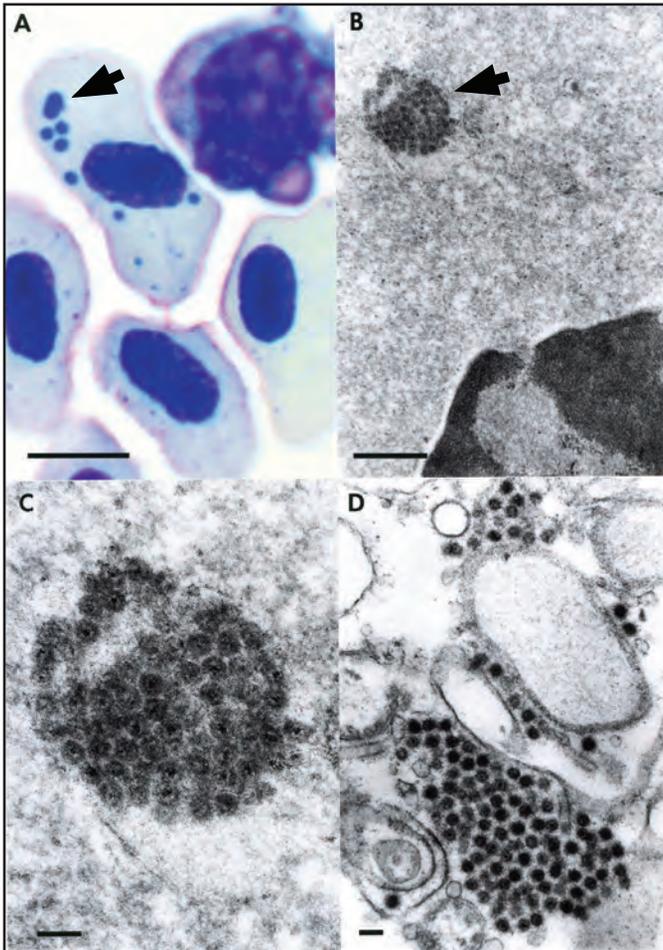
HSMI disease is diagnosed by histological changes of mononuclear inflammation and necrosis of the heart and red skeletal muscle with absence of pancreatic lesions. PRV replicates in the cytoplasm of red blood cells producing inclusion bodies similar to EIBS with or without anemia. This finding suggests a relationship of PRV with EIBS as well as HSMI. PRV does not replicate well in available fish cell lines, requiring molecular detection and sequencing for confirmation of the virus and strain.

VI. Prognosis for Host

Rarely, 20% mortality from HSMI has occurred in Atlantic salmon smolts 5-9 months after transfer to seawater. However, high levels of PRV genetic material are detected in asymptomatic wild and cultured salmonids with no evidence of HSMI disease. In one experiment, PRV was infectious for Chinook and sockeye salmon and persisted but did not cause fish mortality or HSMI, or other apparent disease. Testing of archived tissues from BC indicated PRV was present in asymptomatic wild and farmed Pacific salmon since 1987, possibly as early as 1977 before Atlantic salmon were imported for aquaculture. The ubiquity of PRV, apparent historic presence in wild Pacific salmon stocks in the PNW and lack of clear association with disease suggest the virus is of low risk to wild species of Pacific salmon.

VII. Human Health Significance

There are no human health concerns regarding infection of fish with PRV.



PRV and EBS viruses are likely related if not the same virus. EBS/PRV virus in Chinook salmon: (A) Peripheral blood smear with single and multiple cytoplasmic erythrocytic inclusion bodies (arrowhead), scale bar = 5 μ m; (B) TEM of a single virus inclusion body (arrowhead) in erythrocyte cytoplasm, scale bar = 0.5 μ m; (C) Higher magnification of virus particles (60-70 nm) in the inclusion body of (B), scale bar = 100 nm; (D) Extracellular virus particles associated with cell debris in peripheral blood, scale bar = 100 nm. (From Meyers 2007).