



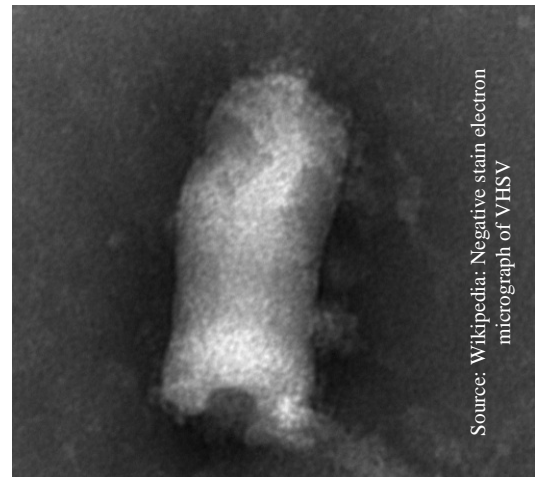
# viral hemorrhagic septicemia

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<b>Common Name</b>	Viral hemorrhagic septicemia, VHSv
<b>Genus &amp; Species</b>	<i>Novirhabdovirus</i>
<b>Family</b>	Rhabdoviridae
<b>Order</b>	Mononegavirales
<b>Group</b>	Group V RNA

**Diagnosis:** VHS is a ribonucleic acid (RNA) virus with bullet-shaped morphology typical of rhabdoviruses and a 11-12 kb nucleotide genome encoding five structural proteins. Viral particles are 170-180 nm in length and 60-70 in width (Skall et al. 2005; Elsayya et al. 2006). Clinical signs of VHS differ depending on the course of infection. In the latent manifestation of the disease, some mortality may occur and fish become hyperactive, sometimes displaying symptoms such as twisting of the body and behavior that involves swimming erratically in circles or in a corkscrew pattern (CFSPH 2003). Conversely, some carriers of the virus may show no symptoms. Histopathological changes occur in the liver, kidneys, spleen and skeletal muscle (McAllister 1990); the kidney and spleen appear to be the organs most often targeted by the VHS virus (Brudeseth et al. 2002). In the acute form of the disease, fish become lethargic, dark and anemic, with bulging eyes, congested kidneys, mottled liver, and with hemorrhage in the eyes, skin, gills, fin bases, skeletal muscle and viscera (McAllister 1990). Mortality is very high and the disease is short-lived (CFSPH 2003). In the chronic form, mortality is low and all the symptoms are similar to the acute form, except that hemorrhaging is not common. Instead, the liver, spleen and kidneys exhibit an accumulation of fluid such that the body becomes bloated and the liver and kidneys become very light in color. Survivors of infection can be carriers of the virus throughout the rest of their lives.



**Ecology:** VHS occurs in both marine and freshwater environments. It requires an incubation period of approximately 7 to 15 days, depending on water temperature (CFSPH 2003). It becomes inactivated in ether, chloroform, glycerol, formalin, sodium hypochlorite, sodium hydroxide, iodophors, UV radiation, or by desiccation, or exposure to pH levels lower than 2.4 or higher than 12.2 (CFSPH 2003; McAllister 1990). The optimum replication temperature is 14-15°C, whereas replication is low at 6°C and almost nonexistent at 20°C ( De Kinkelin et al. 1980, Bernard et al. 1983, McAllister 1990). The virus becomes inactive after 24 hours at 20°C in water, but can persist for five days at 4°C in water.

**Habitat & Distribution:** VHS is indigenous to eastern and western Europe, Japan, and the Pacific coast (from California to Alaska) and Atlantic coast of North America. It is not known how VHS was initially introduced to the Great Lakes-St. Lawrence River system; however, genetic evidence suggests that the virus originated from the Atlantic coast of North America, possibly via transport in ballast water or infected migratory fishes (Elsayad et al. 2006). Aquaculture activities are implicated in the spread of the virus (Skall et al. 2005; Fisheries Research Services 2006). The potential for transport with baitfish is demonstrated by the virus' recovery in cell culture from Pacific herring *Chupea pallasii* after two freeze/thaw cycles in a conventional freezer (Meyers et al 1994).



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**Status:** The North American strain of VHS virus is present in Lake Huron, Lake St. Clair, Lake Erie, Lake Ontario, and the St. Lawrence River (Elsayad et al. 2006; USDA 2006).

**NOAA Fact Sheet:**

<http://www.glerl.noaa.gov/seagrant/GLWL/Zooplankton/Malacostrans/Mysidacea/VHSVfactsheet31Jan2007.pdf>

**Photo Credit:** "Negative stain electron micrograph of VHSV"

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