

INFECTIOUS HEMATOPOIETIC NECROSIS

ANIMAL GROUP AFFECTED	TRANSMISSION	CLINICAL SIGNS	FATAL DISEASE?	TREATMENT	PREVENTION & CONTROL
Salmonids in fresh water between 8°C and 15°C.	Horizontal transmission may be direct, via secretions or vectorial. Vertical or egg-related transmission is believed to occur.	Age dependent. Darkening and exophthalmia. Ascites, long white trailing fecal cast, pale gills. Hemorrhages at the bases of fins and at the vent.	Very high mortality can occur in 3-week to 6-month-old fish. Mortality also occurs in 6 to 14 months old fish.	No treatment. Epidemics can be controlled by quarantine, hygienic measures and disinfection, raise of temperature above 15 °C.	<i>in zoos</i> Stocking with fish of known health status. Disinfection and quarantine. Destruction of infected and exposed fish. Vaccination is at an experimental stage.

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Susceptible animal groups Salmonids in fresh water. Rainbow and steelhead trout, sea-run cutthroat and Kamloops rainbow trout, brown trout. Atlantic, Chinook, pink, coho and sockeye salmon. Chum, amago and yamame salmon.	
Causative organism Rhabdovirus, infectious hematopoietic necrosis virus.	
Zoonotic potential No	
Distribution North America, continental Europe and the Far East.	
Transmission Direct contact with clinically infected fish or contact with mucus from gill and skin, feces, urine, sexual fluids or eggs of asymptomatic carriers. Ingestion of infected tissue. Virus has also been isolated from leeches, copepods and mayflies. Fish-eating birds may also be a vector. Virus is not recovered as a latent infection from the marine phase of fresh water fish.	
Incubation period 5 to 14 days, depending on the water temperature.	
Clinical symptoms Very high mortality in 3-week to 6-month-old fish and mortality in 6 to 14 months old fish. Yolk sac hemorrhages. Darkening and exophthalmia, abdominal distension, pale gills, white fecal casts trailing from the vent. Usually lethargy but sometimes hyperactivity and erratic swimming. Hemorrhages at the bases of the pectoral and pelvic fins and at the vent. Up to 5 % of the survivors show scoliosis and lordosis.	
Post mortem findings See also clinical symptoms. Degeneration of gill lamellae. Visceral pallor and edema, ascites, gastrointestinal tract filled with translucent mucoid fluid. Sometimes petechiation of visceral fat, mesenteries, peritoneum, swim bladder, meninges, pericardium and skeletal muscle. Lesions may be absent in cases of sudden mortality. Till 6 months old: severe necrosis of hematopoietic tissue of the spleen and the kidney. Intracytoplasmic and intranuclear inclusions are visible in the acinar and islet cells of the pancreas. Focal necrotic changes can occur in the liver. Necrosis of granular cells in the lamina propria, stratum compactum and stratum granulosum of the alimentary tract is typically seen in fish 3 to 4 month old. In fish 6 to 14 months old histopathologic changes are not severe. Anterior kidney and spleen show focal areas of cellular degeneration and necrosis. Intracytoplasmic droplets can be seen in kidney tubule epithelium cells. Kidney imprints show necrotic bodies. Moderate sloughing of the epithelial lining of the small intestine.	



Diagnosis Direct serological or molecular identification or virus isolation and confirmatory identification by use of immunological (neutralisation, indirect fluorescent antibody test or ELISA), or molecular (DNA probe or RT-PCR) methods.
Material required for laboratory analysis Carriers (adult fish at spawning): gills, ovarian fluid, lower gut, spleen, kidney, heart and pyloric caeca. Young-of-the-year fish: spleen, kidney, heart. Whole alevin. Pieces of tissue are fixed for histopathological examination and/or immunostaining. A portion is placed in transport medium for virus isolation and a portion is placed in extraction buffer for ELISA or RT-PCR.
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OIE Reference Laboratories <ul style="list-style-type: none">• Dr James R. Winton Western Fisheries Research Center 6505 N.E. 65th Street, Seattle, Washington 98115 UNITED STATES OF AMERICA Tel: (1.206) 526.65.87 Fax: (1.206) 526.66.54 Email: jim_winton@usgs.gov
Treatment No treatment available.
Prevention and control in zoos Stocking with fish of a known health status. Surface disinfection of eggs with an iodophor solution. Quarantine, sound hygiene practices, disinfection. Ideally virus free water supply. Destruction of infected and exposed fish. Vaccination is at an experimental stage.
Suggested disinfectant for housing facilities The virus is inactivated by 3 % formalin in 10 minutes, 0.5 ppm chlorine in 10 minutes, 2 % sodium hydroxide in 10 minutes, 25 ppm iodine in 5 minutes, extremes of pH (below pH 4 or above pH 10) and heating (15 minutes at 60°C or 8 hours at 32°C). The virus is also inactivated by sodium dodecyl sulphate, non-ionic detergents, oxidising agents (ozone, ultraviolet radiation) and lipid solvents.
Notification Yes
Guarantees required under EU Legislation
Guarantees required by EAZA Zoos
Measures required under the Animal Disease Surveillance Plan
Measures required for introducing animals from non-approved sources
Measures to be taken in case of disease outbreak or positive laboratory findings Notify national veterinary authorities.
Conditions for restoring disease-free status after an outbreak Consult national veterinary authorities.
Contacts for further information
References <ol style="list-style-type: none">1. Ferguson, H.W. 1989. Systemic Pathology of Fish. Iowa State University Press/ Ames.2. Noga E.J. Fish Disease, Diagnosis and Treatment. Mosby-Year Book Inc., St Louis. 1996.3. OIE Diagnostic Manual for Aquatic Animal Diseases, Chapter 1.1 and 2.1.2., OIE, Paris, France.4. Roberts R.J. and Schlotfeldt, H.-J. Grundlagen der Fischpathologie. Verlag Paul Parey. 1985.5. Schlotfeldt, H.-J. and Alderman, D.J. What should I do? A Practical guide for the Fresh Water Fish Farmer. Ed. European Association of Fish Pathologists. 1995.6. Stoskopf M.K. 1993. Fish Medicine. W.B. Saunders Company, Philadelphia, London, Toronto, Montreal, Sydney, Tokyo.