

Viral haemorrhagic septicaemia



AETIOLOGY

CLASSIFICATION OF THE CAUSATIVE AGENT

Egtved virus or viral haemorrhagic septicaemia virus (VHSV) of the virus family Rhabdoviridae, genus *Novirhabdovirus*.

RESISTANCE TO PHYSICAL AND CHEMICAL ACTION

Temperature: Inactivation at 50°C for 10 minutes.

pH: Inactivated by pH 12.2 for 2 hours and pH 2.5 for 10 minutes.

Chemicals: Inactivated by oxidising agents, sodium dodecyl sulfate, non-ionic detergents, and lipid solvents (ethyl ether and chloroform sensitive).

Disinfectants: Inactivated by 3% formalin for 5 minutes; 2% sodium hydroxide for 10 minutes; 540 mg/litre chlorine for 20 minutes and iodine compounds (I₂, 100 ppm) for 5 minutes.

Survival: Depends on temperature, long survival at temperatures below 15°C. Remains viable for up to 10 days in mud at 4°C. After 14 days only 90% inactivation in tap water or stream water at 10°C.

EPIDEMIOLOGY

HOSTS

- Viral haemorrhagic septicaemia (VHS) is a highly infectious virus disease predominantly affecting rainbow trout (*Oncorhynchus mykiss*) in aquaculture.
- Natural infection also occurs spontaneously in brown trout (*Salmo trutta*), brook trout (*Salvelinus fontinalis*), grayling (*Thymallus thymallus*), white fish (*Coregonus* sp.), pike (*Esox lucius*), and turbot (*Scophthalmus maximus*), Pacific herring (*Clupea pallasii*), Pacific sardine (*Sardinops sagax*), blackcod (*Anaplopoma fimbria*), Pacific hake (*Merluccius productus*); whereas infection has only been demonstrated in Atlantic salmon (*Salmo salar*), golden trout (*Salmo aguabonita*), lake trout (*Salvelinus namaycush*) and sea bass (*Dicentrarchus labrax*) by experimental infection (injection).
- Since the mid-1980s, a virus serologically identical to VHSV has been isolated from a large range of free-living marine fish species from the Pacific and Atlantic North American coast, the seas around the United Kingdom, the Baltic Sea, Skagerrak and Kattegat, and in the waters around Japan. The known range of susceptible wild marine species include: Pacific herring (*Clupea pallasii*), Pacific salmon (*Oncorhynchus* spp.), Pacific cod (*Gadus macrocephalus*), Atlantic Cod (*Gadus morhua*), black cod (*Anaplopoma fimbria*), English sole

(*Paraphrys vetulus*), Greenland halibut (*Reinhardtius hippoglossius*), haddock (*Gadus aeglefinus*), poor cod (*Trisopterus minutus*), rockling (*Rhinonemus cimbrius*), sprat (*Clupea sprattus*), Atlantic herring (*Clupea harengus*), whiting (*Merlangius merlangus*), blue whiting (*Micromesistius poutassou*), lesser argentine (*Argentina sphyraena*), Norway pout (*Trisopterus esmarki*), Dab (*Limanda limanda*), flounder (*Platichthys flesus*), Japanese flounder (*Paralichthys olivaceus*), pilchard (*Sardinops sagax*), Pacific hake (*Merluccius productus*), Pacific mackerel (*Scomber japonicus*), Pacific sandlance (*Ammodytes hexapterus*), smelt (*Thaleichthys pacificus*), surf smelt (*Hypomesus pretiosus pretiosus*), walleye pollock (*Theragra chalcogramma*), shiner perch (*Cymatogaster aggregata*), threespine sticklebacks (*Gasterosteus aculeatus*) and plaice (*Pleuronectes platessa*).

VIRAL GENOTYPES

There are four main genotypes of VHSV that appear to be more strongly related to geographical location than to year of isolation or host species:

Genotype Ia: Mainly isolates from farmed rainbow trout in continental Europe

Genotype Ib: Mainly isolates from marine hosts in the Baltic Sea, Skagerrak and Kattegat, related to Ia.

Genotype II: Isolates from marine hosts in the Baltic Sea, unlike Ib, with no strong link to Ia

Genotype III: Mainly isolates from around the British Isles

Genotype IV: Isolates from the Pacific Northwest (North American) and Japan

TRANSMISSION

- Natural infections occur by horizontal transmission of water-borne virus or by direct contact with secretions (urine) from infected fish.
- Water-borne virus can be carried downstream 10–20 km to infect susceptible rainbow trout stocks.
- Fish-eating birds (especially herons) can act as mechanical vectors from one facility to another.
- Transfer of infected fish in the incubation phase (i.e. before onset of visible signs) or of infected transport water is a well-known route of infection.
- Vertical transmission does not occur or is extremely rare. Nondisinfected green or eyed eggs from infected parent fish, however, are considered to be being very infectious.

SOURCE OF VIRUS

- Virulent virus is shed via urine and probably sexual fluids, gill and skin epithelia.

OCCURRENCE

VHS occurs in the continental part of Europe, including Russia. Outside this area infections with VHSV have caused significant mortality in turbot in aquaculture in Europe and UK, and Japanese flounder in aquaculture in Japan.

Mass mortality in wild marine species along the Pacific coast of Washington, United States of America (USA), Canada and Alaska, USA, has been associated with VHSV infections.

The disease is not endemic in all of the countries from which it has been reported.

Disease generally occurs at temperatures between 4°C and 14°C. Low water temperatures (1–5°C) generally results in an extended course with low daily mortality but high accumulated mortality. At high water temperatures (15–18°C), the disease generally takes a short course with high acute mortality but a modest accumulated mortality. VHS outbreaks occur during all seasons, but are most common in spring when water temperatures are rising or fluctuating.

For detailed information on occurrence, see recent issues of *World Animal Health* and OIE Web site.

DIAGNOSIS

CLINICAL DIAGNOSIS

- Increase in mortality in the population
- Fish become lethargic, separate from the shoal and gather at the water outlet or sides of a pond
- Fish may experience loss of equilibrium
- Haemorrhages on the skin, base of the fins and the vent
- Exophthalmia
- Overall dark coloration
- Pale gills

INTERNAL LESIONS

- Lesions may be absent or rare in cases of sudden mortality.
- Excess ascitic fluid in the abdominal cavity usually containing blood.
- Intestines that contain mucus instead of food. Flabby, pale rectum.
- Petechial haemorrhage of the visceral organs.
- Petechial haemorrhages in the muscle and fat tissue.
- Petechial haemorrhages in the swim bladder.
- Final diagnosis must await direct identification by immunotechniques or virus isolation and identification.

DIFFERENTIAL DIAGNOSIS

- Bacterial septicaemia as enteric redmouth disease, furunculosis, vibriosis.
- Infectious hematopoietic necrosis.
- Environmental stress factors e.g. osmotic shock.

- Transportation and handling stress.

LABORATORY DIAGNOSIS

Procedures

Isolation of the agent

- Inoculation of susceptible cell lines such as BF-2 or RTG-2 followed by microscopic examination

Identification of the agent

- Virus neutralisation
- Immunofluorescence tests
- Enzyme-linked immunosorbent assay (ELISA)
- polymerase chain reaction (PCR), immunohistochemical staining

Samples

- Whole fish
- Specimens of spleen, kidney, heart and encephalon are placed in transport medium for virus isolation, e.g. Eagles MEM with calf serum and antibiotics

PREVENTION AND CONTROL

- No treatment available

CONTROL MEASURES

- Raising water temperature and lowering stocking density combined with minimal handling stress can reduce the mortality.
- Strict isolation of outbreaks (= the infected site + all down-stream situated farms) with movement controls and control of human traffic.
- Destruction or slaughtering of all fish in infected and suspected farms.
- Thorough cleaning and disinfection.
- Allowing for at least 4 weeks at temperatures above 15°C.
- Restocking with certified VHS-free material.

SANITARY PROPHYLAXIS

- Virus-free water supply.
- Fencing against birds, animals and unauthorised people.
- Stocking with fish of known health status.
- Fish loading facilities are separated from aquaculture establishments.
- Effective prevention of escape.
- Avoid mixing fish from different sites.

MEDICAL PROPHYLAXIS

- No reliable vaccine is commercially available yet. Promising results have been obtained using DNA based vaccines under experimental conditions.

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