

# Spring Viremia of Carp

*Infectious Dropsy of Carp,  
Infectious Ascites, Hydrops,  
Red Contagious Disease,  
Rubella,  
Hemorrhagic Septicemia*

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The Center for  
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INSTITUTE FOR  
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IOWA STATE UNIVERSITY  
College of Veterinary Medicine



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## Importance

Spring viremia of carp (SVC) is a contagious viral disease mainly seen in farmed carp and related species. Outbreaks can cause substantial economic losses. SVC can be highly fatal in young fish, with mortality rates up to 90%. In Europe, where this disease has been endemic for at least fifty years, 10-15% of one-year-old carp are lost to SVC each year. The causative virus can be spread by fomites and parasitic invertebrates, and is difficult to eradicate; once it is established in a pond, elimination of the virus may require the destruction of all aquatic life. Since 2002, several SVC outbreaks have been reported in U.S., with both cultivated and wild species affected.

## Etiology

Spring viremia of carp is caused by the spring viremia of carp virus (SVCV), which is also known as Rhabdovirus carpio. This virus is a member of the family Rhabdoviridae and has been tentatively placed in the genus *Vesiculovirus*. SVCV is closely related to pike fry rhabdovirus, and these two viruses cross-react in some serologic tests.

SVCV strains vary in their pathogenicity. Isolates can be divided into four genetic groups. Genogroup Ia viruses originate from Asia. Genogroups Ib and Ic are comprised of isolates from Russia, Moldova and Ukraine. Genogroup Id mainly contains viruses from the U.K., although a few isolates in this group are from the former U.S.S.R. SVCV strains from recent outbreaks in the U.S. appear to be most closely related to genogroup Ia.

## Species Affected

SVC primarily affects carp and other species in the family Cyprinidae (minnow family), but it is also found in a few species from other fish families. Infections have been reported in common carp (*Cyprinus carpio*), koi carp (*Cyprinus carpio koi*), grass carp/ white amur (*Ctenopharyngodon idella*), silver carp (*Hypophthalmichthys molitrix*), bighead carp (*Aristichthys nobilis*), crucian carp (*Carassius carassius*), goldfish (*Carassius auratus*), tench (*Tinca tinca*), orfe (*Leuciscus idus*) and sheatfish/ European catfish/ wels (*Silurus glanis*). Common carp are the most susceptible species and are considered to be the principal host. Very young fish of various pond species, including pike and perch, are also susceptible. Experimental infections have been reported in roach (*Rutilus rutilus*), zebrafish (*Danio rerio*), guppies (*Lebistes reticulatus*), northern pike (*Esox lucius*), golden shiners (*Notemigonus crysoleucas*) and pumpkinseed (*Lepomis gibbosus*).

A SVC-like virus has also been found in diseased cultured shrimp (*Penaeus stylirostris* and *P. vannamei*).

## Geographic Distribution

Spring viremia of carp has been reported from Europe including the U.K., the western states of the former U.S.S.R. (Russia, Belarus, Georgia, Lithuania, Moldova and the Ukraine) and the Middle East. In 2004, an SVCV outbreak was confirmed in cultivated carp in China. Outbreaks have also been reported in the U.S. In 2002, SVC was found in cultivated fish in North Carolina and wild common carp in Wisconsin and Illinois. Outbreaks were reported in cultivated koi in Washington and Missouri in 2004, and in wild fish in the Upper Mississippi River (Wisconsin, Minnesota) in 2007. In the late 1990s, an outbreak was reported in imported goldfish in Brazil. An SVC-like virus has been found in cultured shrimp but not finfish in Hawaii.

## Transmission

SVCV is carried in clinically ill fish and asymptomatic carriers. This virus is shed in the feces and urine, as well as the gill and skin mucus of infected fish. It is also found in the exudate of skin blisters and edematous scale pockets. Transmission is by direct contact or through the water. The virus enters most often through the gills. SVCV has been found in ovarian fluids and "egg-associated" (vertical) transmission has not been ruled out; however, this does not appear to be an important route of spread.

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SVCV can also be spread by fomites and invertebrate vectors. Infectious virus can persist in 10°C water for more than four weeks and in 4°C mud for at least six weeks. Known vectors include the carp louse *Argulus foliaceus* and leech *Piscicola geometra*, but other aquatic arthropods might also transmit the virus. Fish-eating birds are also potential vectors.

## Incubation Period

Incubation periods from 7 to 15 days have been reported in experimental infections.

## Clinical Signs

Fish can carry SVCV with or without symptoms. Fish up to a year old are most likely to be affected, but illness also occurs in older animals. The clinical signs are nonspecific. In carp, the most common symptoms include abdominal distension, exophthalmia, inflammation or edema of the vent (often with trailing mucoid fecal casts), and petechial hemorrhages of the skin, gills and eyes. The body is often darkened with pale gills. Diseased fish tend to gather at the water inlet or sides of the pond, swim and breathe more slowly than normal, and react sluggishly to stimuli. Loss of equilibrium, with resting and leaning, are seen in the late stages. Concurrent bacterial infections (carp-dropsy complex) or parasitic infections influence the symptoms and mortality rate.

## Post-Mortem Lesions

The body is often darkened with pale gills, and petechial hemorrhages may be seen in the skin, gills or eyes. The abdominal cavity typically contains serous fluid, which may be mixed with blood or necrotic material. The muscles and fat may contain petechial or focal hemorrhages. Similar hemorrhages are also common on the internal organs, particularly on the walls of the swim (air) bladder. The intestines are often severely inflamed and dilated, and may contain necrotic material. The spleen is frequently swollen, with a coarse surface texture. Other lesions may include degeneration of the gill lamellae, edema of other internal organs, hepatic necrosis, jaundice, cardiac inflammation and pericarditis. In fish that die suddenly, gross lesions may be absent.

## Morbidity and Mortality

SVC outbreaks are most common in farmed carp, but can also occur in wild fish. Although fish of any age can become ill, disease is most common in young fish up to a year of age. The morbidity and mortality rates vary with stress factors and population density, as well as the species, age and condition of the fish. Water temperature affects the development of disease. In Europe, susceptible species usually become infected in the fall and winter as the water temperature falls. Some populations may become ill then, but most outbreaks occur in the spring as temperatures rise. Although the relationship between water temperature and illness is complex, clinical signs are most common at 17°C

(63°F) or below. Fry may become ill at temperatures as high as 22-23°C (71-73°F).

The mortality rate is highest in young fish. Mortality rates up to 70% have been reported in young carp during outbreaks. In experimentally infected fish, the mortality rate can be as high as 90%. Yearly losses in older fish are usually under 30%. Water temperatures affect the mortality rate. In experimental infections, the cumulative mortality rate is similar at all temperatures from 11°C to 17°C (52 to 63°F), but the fish die more quickly at 17°C than 11-15°C (52-59°F). The mortality rate decreases at temperatures between 17°C and 26°C (78.8°F). Recovery from infection usually results in strong immunity.

## Diagnosis

### Clinical

SVC should be suspected in cyprinid fish with signs of a systemic infection and an increased mortality rate, when water temperatures are below 20°C (68°F). A particularly high incidence of disease in common carp, with reduced susceptibility in carp hybrids and lower disease prevalence in other cyprinids, is also suggestive. The clinical signs and lesions are not pathognomonic and must be confirmed by laboratory diagnosis.

### Differential diagnosis

The differential diagnosis includes enteric septicemia of catfish and infection with atypical *Aeromonas salmonicida*.

### Laboratory tests

Spring viremia of carp can be diagnosed by virus isolation in cell cultures; appropriate cell lines include EPC (*Epithelioma papulosum cyprini*) cells, FHM (fathead minnow) cells and carp leukocyte cultures. SVCV will also replicate in some mammalian cell lines. The identity of the virus is confirmed by virus neutralization or polymerase chain reaction assay (PCR) and nucleotide sequence analysis of PCR products. Immunofluorescence or an enzyme-linked immunosorbent assay (ELISA) can be used for rapid presumptive identification of the virus in cultures.

PCR can also be used to detect SVCV in tissue extracts. Viral antigens can be identified directly in tissues by immunofluorescence or ELISA. Ideally, a diagnosis made by immunofluorescence or ELISA should be confirmed by virus isolation, but this may not always be possible. Transmission electron microscopy can also be used for a presumptive diagnosis.

Serology may become effective in screening fish populations, but it has not yet been validated for routine diagnosis. Antibodies to SVCV can cross-react with other rhabdoviruses, particularly pike fry rhabdovirus.

### Samples to collect

**Before collecting or sending any samples from animals with a suspected foreign animal disease, the**

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**proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease.**

The samples to collect from symptomatic animals vary with the size of the fish. Small fish (less than or equal to 4 cm) should be sent whole. The viscera including the kidney and encephalon should be collected from fish 4 to 6 cm long. The kidney, spleen, liver and encephalon should be sent from larger fish. Samples from asymptomatic animals should include the kidney, spleen, gill and encephalon.

Samples should be taken from ten diseased fish and combined to form pools with approximately 1.5 g of material (no more than five fish per pool). The pools of organs should be placed in sterile vials. The samples may also be sent in cell culture medium or Hanks' balanced salt solution with antibiotics. They should be kept cold [4°C (39°F)] but not frozen. If the shipping time is expected to be longer than 12 hours, serum or albumen (5-10%) may be added to stabilize the virus. Ideally, virus isolation should be done within 24 hours after fish sampling.

## Recommended actions if spring viremia of carp is suspected

### Notification of authorities

Spring viremia of carp should be reported to state or federal authorities immediately upon diagnosis or suspicion of the disease.

Federal Area Veterinarians in Charge (AVIC):

<https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/contact-us>

State Animal Health Officials:

<http://www.usaha.org/federal-and-state-animal-health>

## Control

In the U.S., the USDA currently restricts the importation of live fish, fertilized eggs and gametes from susceptible species including common carp, koi, grass carp, silver carp, bighead carp, Crucian carp, goldfish, tench and sheatfish. In areas where SVC is not endemic, it is controlled by culling, disinfection, quarantines and other measures. Once SVCV is established in a pond, it can be very difficult to eradicate unless all forms of aquatic life at the site are destroyed.

In areas where SVC is endemic, good biosecurity and sanitation are necessary to prevent the virus from entering a farm. The use of spring or well water helps prevent entry in water or parasitic arthropods. New fish stocks should come from an SVC-free source. Additional control measures include iodophore treatment of eggs, regular physical and chemical disinfection of ponds, disinfection of farm equipment, and safe disposal of dead fish to prevent spreading disease. SVCV is susceptible to oxidizing agents, sodium dodecyl sulphate, non-ionic detergents and lipid solvents. It can be inactivated with formalin (3% for 5 minutes), chlorine (500 ppm), iodine (0.01%), NaOH (2%

for 10 minutes), UV irradiation (254 nm) and gamma irradiation (103 krad). SVCV can also be inactivated by heating to 60°C (140°F) for 30 minutes, as well as pH 12 for 10 minutes, or pH 3 for 3 hours.

Good sanitation and management techniques also decrease the incidence of disease on infected farms. Reducing the fish stocking density in winter and early spring can decrease virus spread. Stress should be minimized. In facilities with controlled environmental conditions, increasing the water temperature to at least 19-20°C (66-68°F) may prevent or halt outbreaks, or reduce the mortality rate. Antibiotics can be used to control the bacterial component of carp-dropsy complex (co-infection with *Aeromonas* or other systemic bacteria). Vaccines for SVC are in development, but are not yet available.

Hobbyists who show fish should only participate in events where each participant's fish are kept in separate tanks. Anglers should avoid transferring fish or fish parts between bodies of water, to prevent spreading disease. They should also notify their local fish and game department if they see unusually high numbers of dead or dying fish.

## Public Health

There is no indication that SVC is a threat to human health.

## Internet Resources

USDA APHIS Aquaculture Disease Information

[http://www.aphis.usda.gov/animal\\_health/animal\\_dis\\_spec/aquaculture/](http://www.aphis.usda.gov/animal_health/animal_dis_spec/aquaculture/)

World Organization for Animal Health (OIE)

<http://www.oie.int>

OIE Manual of Diagnostic Tests for Aquatic Animals

<http://www.oie.int/international-standard-setting/aquatic-manual/access-online/>

OIE Aquatic Animal Health Code

<http://www.oie.int/international-standard-setting/aquatic-code/access-online/>

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\*Link defunct