



Spring Viremia of Carp¹

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Introduction

Spring viremia of carp (SVC) is a viral disease that can cause significant mortality of common carp (*Cyprinus carpio*). This species is raised as a food fish in many countries and has also been selectively bred for the ornamental fish industry, where it is known as koi. Historically, the disease has been a problem in Europe, the Middle East, and Russia. Recently, SVC has been reported in koi in the United States for the first time. This information sheet is intended to inform veterinarians, biologists, culturists, and hobbyists about SVC.

What is Spring Viremia of Carp?

Spring viremia of carp is caused by *Rhabdovirus carpio*, a bullet-shaped RNA virus. The disease has been reported in common carp (or koi) (*Cyprinus carpio*), grass carp (*Ctenopharyngodon idella*), bighead carp (*Aristichthys nobilis*), silver carp (*Hypophthalmichthys molitrix*), and Crucian carp (*Carassius carassius*), a close relative of the goldfish. Recent evidence suggests that common goldfish (*C. auratus*) are also susceptible.

The disease was initially diagnosed in Yugoslavia (Fijan et al. 1971). Since then, it has been identified in other European countries, Russia, and the Middle East. Mortality has reached 70% in yearling carp from European populations. Adult fish can also be affected but to a lesser degree.

What are the Signs of SVC?

Clinical signs of SVC are often non-specific and may include darkening of the skin, exophthalmia (pop-eye), ascites (dropsy), pale gills, hemorrhages in the gills, skin, and eye, and a protruding vent with a thick mucoid (white to yellowish) fecal cast.

Internally, edema (fluid build up in organs and in the body cavity), inflammation, and pinpoint hemorrhages in many organs, including the swim bladder, may be present.

The presence of pinpoint hemorrhages in the swim bladder is considered an important indicator of this disease. The intestine is often severely inflamed and may contain significant amounts of mucus.

The spleen is often enlarged. Concurrent infection with bacteria, particularly *Aeromonas* (*A. salmonicida* or *A. hydrophila*), may confuse the diagnosis as fish will show signs of systemic infection such as ascites and hemorrhages.

Behaviorally, infected fish may appear lethargic, exhibit decreased respiration rate, and loss of equilibrium. Moribund fish have been reported to lie on their sides, often on the bottom of the tank, and when startled swim up but then return to the bottom. Fish are also reported to congregate where there is slow water flow and near pond banks (Fijan 1999).

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Transmission of SVC

The rhabdovirus that causes SVC enters the fish through the gills, replicating in gill epithelium (Ahne 1978; Baudouy et al. 1980). The virus is spread via feces in the mucoid casts. Blood-sucking parasites, including leeches and the fish louse *Argulus*, have been implicated in spreading the disease (Pfeil-Putzien 1977; Ahne 1985). Mechanical transmission by birds and equipment is suspected because of the longevity of the virus in water, mud, or following desiccation (Ahne 1982a; Ahne 1982b).

Experimental transmission has been accomplished by co-habitation, intracranial and intraperitoneal injection, intubation of the virus into the intestine, and by immersion. However, direct application of the virus to scarified skin has been unsuccessful. (Fijan 1972; Fijan et al. 1971; Hill 1977).

The presence of the virus in ovarian fluids suggests that vertical transmission (female parent to offspring) may be possible (Fijan 1999).

Effect of Water Temperature

Although other factors, such as age, can determine how severely the disease will affect a population, the temperature at which fish become infected, temperature fluctuations during the infective period, and the ability of the fish to mount a timely immune response seem to be the most important components for SVC.

In natural outbreaks, mortalities were confirmed in spring of 1969 and 1970 in Yugoslavia when water temperatures ranged from 12°C to 22°C (54°F to 72°F). The optimum temperature for viral replication *in vitro* is 20-22°C (68-72°F), however, this is also an optimum temperature range for immune function of susceptible species (Fijan 1999). Clinical and experimental data indicate that maximum mortality can be expected at water temperatures below 18°C (64°F) (Fijan 1999; McAllister 1993).

These findings have led experts (Wolf 1988; Ahne 1980; Fijan 1999) to suggest that outbreaks of SVC can be prevented or stopped in mature fish by raising water temperatures above 20°C (68°F); however, the results of such attempts have not been well documented. Because of the potential severity of the disease, depopulation is recommended.

How is SVC diagnosed?

Diagnosis of SVC can be accomplished by several methods. Direct methods include virus isolation and identification using fathead minnow (FHM), epithelioma papillosum of carp (EPC), and primary carp ovary cells (COC) cell lines. Indirect tests for SVC include ELISA, virus neutralization and immunofluorescence of suspect tissue.

Laboratories approved by the USDA to test fish for SVC are listed in Appendix A.

How is SVC treated?

Antiviral drugs are not available to treat SVC or other viral diseases of cultured fish. Temperature manipulation is probably the most practical means of preventing or controlling mortality once an epizootic is in progress. Maintaining water temperature above 20°C (68°F) may prevent a potential outbreak.

In active outbreaks, efforts are directed at depopulating infected stock, and disinfecting all areas where infected fish were held. However, in some circumstances, this may be difficult. The virus can be infective in mud and water for up to 42 days (Plumb 1999).

The virus can be inactivated by formalin, ozone, sodium hypochlorite (chlorine at 500 ppm for ten minutes), organic iodophors, gamma and ultraviolet irradiation, pH extremes of < 4.0 or greater than 10.0, and heating at 60°C (140°F) for 15 minutes (Smail and Munro 1989; Fijan 1999). All equipment and tanks, raceways, and ponds should be disinfected.

Fish that are exposed to physiological stressors such as crowding, handling, poor water quality, malnutrition, and sudden temperature changes are most susceptible, because of resulting immune system suppression.

Vaccine development has been attempted in the Czech Republic (Macura et al. 1983) with promising results but further studies are necessary. The development of genetically resistant strains should also be pursued (Fijan 1999).

How can SVC be prevented?

In the face of infection, maintaining a water temperature of 20°C (68°F) or higher will increase the chances for infected fish to develop an immunity to SVC, reducing mortalities. It is unknown at this time whether fish that have been exposed to SVC, and subsequently become immune, will serve as a source of virus to unexposed fish.

New fish should be purchased from SVC-free suppliers and farms.

Regulatory Considerations

Spring viremia of carp is listed as a notifiable disease, by the Office International des Epizooties (OIE), in the International Aquatic Animal Health Code (OIE 1997a). The OIE has published a diagnostic manual that includes protocols required to confirm a diagnosis of SVC (OIE 1997b). It also lists criteria for “SVC-free” status for aquaculture facilities and geographic regions.

In the United States, suspect cases should be sent to one of the three USDA-approved labs listed in Appendix A for confirmation. SVC is considered a notifiable disease in the United States, therefore prompt notification of the State Veterinarian’s office and appropriate USDA-APHIS Veterinary Services officials is mandatory.

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Appendix A

USDA approved diagnostic laboratories capable of testing for Spring Viremia of Carp

1. University of Arkansas-Pine Bluff
Cooperative Extension Program
PO Box 4912 OR 1200 University Drive
Pine Bluff, AR 71611
Phone: (870) 543-8537
2. Pennsylvania Animal Diagnostic Laboratory
System
State Veterinary Laboratory
2305 North Cameron Street
Harrisburg, PA 17110
Phone: (717) 787-8808
3. Washington Animal Disease Diagnostic
Laboratory
College of Veterinary Medicine
Washington State University
PO Box 647034
Pullman, WA 99164-7034
Phone: (509) 335-9696