

2.2.9 Spring Viremia of Carp

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A. Name of Disease and Etiological Agent

Spring Viremia of Carp (SVC) is caused by the fish rhabdovirus, Spring Viremia of Carp Virus (SVCV). The following names have historically been used for diseases that may have been caused by SVCV: swim-bladder inflammation (SBI), infectious dropsy, rubella, infectious ascites, hemorrhagic septicemia, and red contagious disease. For reviews of the disease and the causative virus see Ahne et al. (2002), Fijan (1999), and Wolf (1988).

B. Known Geographical Range and Host Species of the Disease

1. Geographical Range

Most reports of Spring Viremia of Carp are from Europe, but the virus has been isolated in other regions of the world including the Middle East, China and, in one incomplete report, South America. The SVCV has been reported from cultured shrimp in Hawaii, but not from Hawaiian finfish (Johnson et al. 1999). The virus was first reported in North America in 2002 when it was isolated from farmed koi in North Carolina and from feral common carp in Cedar Lake, Wisconsin. The DNA sequences of these North American isolates are very closely related to those from China.

2. Host Species

Natural infections have occurred in bighead carp, common carp, crucian carp, goldfish, koi, penaeid shrimp, pike, silver carp, tench, and the wells catfish (sheatfish). Experimental infections have produced disease or demonstrated viral replication in golden shiners, grass carp, guppies, pike, pumpkinseeds, roach, zebra danios, and fruit flies. Few North American species have been tested, but it is probable that many, especially cyprinids, will prove susceptible.

C. Epizootiology

In typical outbreaks, SVCV spreads horizontally during the winter when water temperatures are low and host immune systems are less active. When spring approaches and temperatures rise toward 10°C, fish develop clinical signs of SVC and begin to die. The peak mortality for SVC occurs at 15 to 17°C. Above 20°C, most fish are able to develop immunity to the virus and mortality ceases. Fish may also develop SVC in the fall if temperatures are in the right range for virus transmission and disease. The virus has not been successfully isolated from infected populations of fish following periods of water temperatures in the 25 to 30°C range. In experimental infections, it has been shown that immunosuppression triggered by cold temperatures is critical to the development of SVC. The actual temperature required to produce that immunosuppression is apparently related to the thermal optimum of the infected fish species. It is not known if stressing coolwater fish with high temperatures (temperatures well above optimal for the fish, but still below the upper limit for virus replication in culture) will induce clinical SVC.

Most reported losses from SVC have occurred in farmed carp during their first winter and spring. Little mortality has been reported in older fish in Europe, but this may be due to immunity in older fish previously exposed to the virus. The first outbreak reported in Wisconsin occurred in large mature carp, perhaps indicating their naïve status.

While SVCV has been isolated from fish lice (*Argulus sp.*) and leeches (*Pisicola sp.*) feeding on infected fish, there is no evidence that these mechanical vectors are important in the spread of SVCV. It has not been shown that vertical transmission is a significant mechanism for the spread of SVCV. However, there is one report of the virus being found in ovarian fluid from a population of common carp broodfish tested on farms in Europe with a history of SVC.

D. Disease Signs

The clinical signs of SVC are not very significantly different from those associated with bacterial septicemias in carp. Infected fish typically have hemorrhages of the skin, exophthalmia, abdominal distension, and a discharge of bloody mucoid material from the vent (Figure 1). Internal signs include petechial hemorrhages of the swim bladder and muscle, ascites, edema of the kidney, liver and spleen, and catarrhal and hemorrhagic enteritis.

E. Disease Diagnostic Procedures

1. Presumptive Diagnosis

To aid in the diagnosis of SVCV, certain key features such as life stage and species of fish, water temperature, season, clinical signs, and disease history of the facility are evaluated. To isolate SVCV, kidney and spleen homogenates are examined by standard cell culture techniques. Processed specimens may be inoculated onto the *Epithelioma papillosum cyprini* (EPC) or fathead minnow (FHM) cell lines and incubated at 22 to 25°C. Some isolates may be easier to detect on bluegill fin (BF-2) cells. Many cell lines are susceptible to SVCV and the virus will produce cytopathic effects (CPE) in fish cell lines inoculated with SVCV and incubated at a broad range of temperatures (15 to 30°C). Cytopathic effects include focal patches of round refractile cells that can be observed 24 to 96 hours post-inoculation depending on the concentration of virus in the inoculum and the temperature (Figure 2). Some clinical cases may have such high titers that focal CPE is not evident, and the entire monolayer may be destroyed. Appropriate clinical signs, fish species, season, water temperature, and observation of typical CPE provide the best evidence for a presumptive diagnosis.

Spring Viremia of Carp can easily be distinguished from Koi Herpes Virus (KHV). The KHV produces disease at temperatures above 20°C, does not cause CPE in EPC, FHM, or BF-2 cells, and commonly produces focal necrosis of the gill filaments, not the septicemia signs common with SVCV.

2. Confirmatory Diagnosis

Confirmation of SVCV has been achieved through a variety of immunological methods including serum neutralization, ELISA, and immunocytochemistry using both monoclonal and polyclonal antibodies. However, it has been shown that SVCV isolates similar to those recovered from fish imported from Asia may bind poorly to SVCV monoclonals and antisera. Additionally, care must be taken to insure that antibodies used do not cross react with Pike Fry Rhabdovirus (PFRV) and that appropriate PFRV controls are included in the assays. For these reasons, a reverse transcriptase, polymerase chain reaction (RT-PCR) assay is now regarded as the best confirmatory technique for virus isolated in cell culture. The RT-PCR works well with both European and Asian-related isolates and has the advantage that the PCR product can be sequenced and compared to other isolates for epidemiology. The RT-PCR protocol for SVCV is detailed in Section 2, 4.6.F.2.b “Polymerase Chain Reaction (PCR) Method for Confirmation of SVCV.”

F. Procedures for Detecting Subclinical Infections

Carp infected with SVC at water temperatures within the range of viral replication, but below that where overt disease begins to occur (4 to 10°C), routinely test positive for SVCV by tissue culture. The virus generally cannot be isolated from survivors of previous SVC outbreaks or from fish tested during the summer when water temperatures are greater than 20°C. There is little known about asymptomatic carrier fish other than one report of detection of SVCV by tissue culture of ovarian fluids from brood fish sampled during the spring. There has been no work investigating the detection of carrier fish by PCR.



Figure 1. Common carp experimentally infected with SVCV. Note the exophthalmia and hemorrhage of the eye and petechial hemorrhages on the skin.

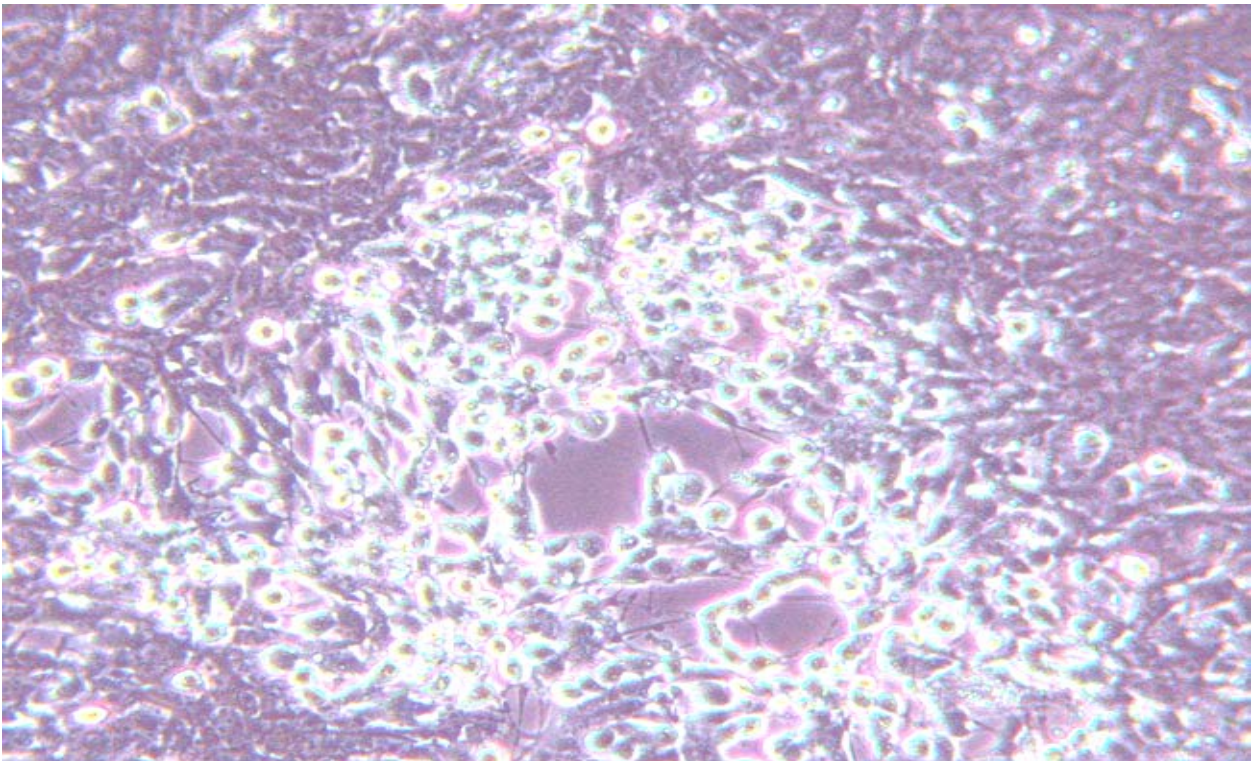


Figure 2. Cytopathic effects of SVCV in a monolayer of EPC cells. Note the foci of rounded and dead cells. Phase contrast.

G. Procedures for Determining Prior Exposure to the Etiological Agent

Prior exposure of common carp to SVCV can be detected by a competitive ELISA for antibodies to SVCV (Dixon et al. 1994). This assay is well accepted for common carp and koi but not for other species. The assay relies on polyclonal antibodies that may be difficult to produce and standardize.

H. Procedures for Transportation and Storage of Samples to Ensure Maximum Viability and Survival of the Etiologic Agent

See Section 2, 2.2.A.2.b “Guidelines for Preparation of a Fish Health Inspection” .

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