Spring Viremia of Carp

Introduction

Spring viremia of carp (SVC) is a viral disease of fish, primarily common carp *(Cyprinus carpio)*. Although the disease and its causative agent, spring viremia of carp virus (SVCV) or *Rhabdovirus carpio*, was first described in 1971, there is evidence that the disease has been present in Europe for at least 50 years and, potentially, since the Middle Ages. Before the disease was recognized, it was variously called infectious dropsy, infectious ascites, hemorrhagic septicemia, or rubella.

In Europe, the disease has had substantial impact on the production of carp, with estimated losses of 10–15 percent of 1–year–old carp or about 4,000 tons annually. In some cases, mortality rates of young carp can reach 70 percent. This impact of the disease has led to its listing by the Office International des Epizooties (OIE) as notifiable.

Susceptible species and geographical distribution

Although common carp, which includes the variety called koi carp, is the main species of fish affected by SVC, there have been several other species that are susceptible to the disease under non–experimental (natural) conditions. These species include crucian carp *(Carassius carassius)*, grass carp *(Ctenopharyngodon idella)*, silver carp *(Hypophthalmichthys molitrix)*, bighead carp *(Aristichthys nobilis)*, goldfish *(Carassius auratus)*, tench *(Tinca tinca*), and sheatfish *(Silurus glanis)*.

Under experimental conditions, other species including roach *(Rutilus rutilus)*, pike *(Esox lucius)*, guppy *(Lebistes reticulates)*, pumpkinseed *(Lepomis gibbosus)*, goldfish *(Carassius auratus)*, zebra danios *(Brachydanio rerio)*, and golden shiners *(Notemigonus crysoleucas)* have been found to be susceptible to the disease.

Many species in the minnow family *(Cyprinidae)* are indigenous to the United States, including endangered species; the susceptibility of these has not yet been determined either under natural or experimental conditions. A SVC–like virus has been isolated from diseased penaeid shrimp *(Penaeus stylirostris* and *P. vannamei)*.

Historically, SVC has been reported from many countries in Europe, the Middle East, and Asia, but recently the disease has been reported in South and North America as well.

Clinical signs of SVC

First signs of the disease may be a change in behavioral patterns of the fish. Fish may congregate in slow–flowing water, near pond banks, or lie on the bottom. Over time, the rate of respiration will decrease, as will reaction to stimulation and swimming speed. As the disease progresses, the fish become sluggish and may swim and lie on their sides.

Externally, the fish can exhibit a number of non–specific physical signs including darkening of the skin, swollen abdomen, exophthalmia (pop–eye), hemorrhages in the skin, gills and anterior eye chamber, anemia and pale gills, and a protruding vent.

Internally, the signs are dominated by building up of fluid (edema) in all organs and in the body cavity, hemorrhages in the swim bladder, and inflammation of the intestines.

Temperature and seasonality

Research has shown that the optimal temperature for development of SVC in experimentally infected carp is between 16 and 17° C. At this temperature, 90 percent of the fish died within 5 to 17 days after being infected. At lower temperatures, 11–15° C, the percent of fish that died was similar but the mortality was delayed (2–3 weeks). Mortality was reduced at temperatures between 17 and 26° C. The optimum temperature for in vitro virus replication is 20–22° C. Other experiments have investigated the influence of increasing and decreasing temperatures on the rate of disease. Research has also demonstrated that a gradual decrease of temperature (11 down to 5° C) caused low mortality, while increasing temperature back to 20° C caused massive mortality as the temperature changed from 7 to 14° C.

These results correspond with the field observations that most SVC outbreaks occur in the spring with warming temperatures. After water temperatures rise above 15–18° C, the immune system of carp becomes capable of rapid interferon and neutralizing antibody synthesis that suppresses viral replication. Thus, in the countries where SVC has been reported, there are only sporadic reports in June and July. The temperature constraints make

tropical and subtropical climates unfavorable for SVC outbreaks. The replication of virus as temperatures rise also has implications for detecting virus in fish populations. All viral isolations for SVC were from samples taken in May when the water temperature was between 10 to 18° C. Virus detection likely would be more difficult during the other seasons of the year.

Transmission

Infected fish can shed virus in feces and possibly in urine and gill mucus. Horizontal transmission likely occurs when virus enters fish through the gills. Research has demonstrated that SVC was easily transmitted horizontally through water from experimentally infected fish to uninfected fish. Reservoir hosts include sick fish and fish that have survived an outbreak. In addition to carp, other cultured and wild fish may serve as reservoirs for the disease. Vertical transmission may be possible since SVC virus has been found in ovarian fluids, but the lack of outbreaks among fry and fingerlings suggests that it is not an important route of transmission.

Parasites such as the carp louse, *Argulus foliaceus*, and the leech, *Pisciola* geometra, have been shown to be passive vectors in the transfer of disease to healthy carp. Mechanical vectors can also be a problem since SVCV can maintain infectivity for a long time in water or mud or after becoming dry.

Diagnosis

In 2000, OIE set the international standards for diagnosing SVC virus. The diagnosis of SVC in clinically infected fish can be accomplished through virus isolation or by using an immunological test such as direct immunofluorescence (IF) test or an enzyme–linked immunosorbant assay (ELISA). A virus neutralization (VN) test is the confirmatory identification test. Immunofluorescence tests and ELISAs should be followed by virus isolation and a VN test.

The OIE has specified criteria for declaring countries, zones, and aquaculture establishments free of SVC. The International Aquatic Animal Health Code and the Diagnostic Manual for Aquatic Animal Diseases have complete details on all of the requirements so only some general criteria are presented here. The appropriate Web sites for these documents are listed at the end of the document. A country declared free must meet these conditions: 1) no recorded outbreak of SVC for at least 2 years; 2) no detection of virus in any of the susceptible fish species tested during an official surveillance scheme during the past 2 years; and 3) requirements met for importing live fish from other countries.

For a zone to be declared free of SVC, both aquaculture establishments and wild populations containing susceptible fish species must have been tested in an official surveillance scheme and SVC must not have been detected in the past 2 years. The zone must also be one or more entire catchment areas or be part of a catchment area where upstream migration of fish from downstream areas cannot occur.

For an aquaculture establishment to be declared free of SVC, it may be part of a free country or zone. An aquaculture establishment in an infected area can still be declared free if it: 1) has been tested under an official health surveillance scheme for at least 2 years without detection of SVCV; 2) is supplied by water from a spring, well, or borehole only and is free from wild fish; and 3) is not connected to a watercourse or there is a natural barrier that prevents the migration upstream of fish from downstream stretches of the waterway.

The OIE Diagnostic Manual for Aquatic Animal Disease has specifications for surveillance programs to achieve and maintain health status. Briefly, fish culture units on aquaculture establishments must be inspected twice annually for 2 years. Each inspection should be conducted in order to detect a 2 percent prevalence with 95 percent confidence level. This represents collection of approximately 150 appropriate–age fish at times of the year clinical signs are most likely to be observed and isolating pathogens is the easiest. Ovarian fluid samples can be used if available. To maintain free status, twice annual inspections of 30 fish are required. Wild fish populations need to be sampled only once a year for 2 years and 150 fish from different fish crops may be pooled. Maintenance of health status of wild fish can only be attained by annual sampling of 150 fish including as many broodfish as possible.

Prevention

There are several recommendations for preventing the disease from becoming established on commercial farms. Using a source of water that is free from disease such as a spring or a well is necessary, especially in an endemic disease area, to exclude disease. Other on–farm measures include disinfection of eggs by iodophore treatment, regular physical and chemical disinfection of ponds, disinfection of equipment, and proper disposal of dead fish. Also, new fish being brought onto farms should be purchased from an SVC–free source. Movement of ornamental fish to shows and returning to operations should be undertaken with caution.

Currently, no commercially available vaccine exists for SVC. However, some studies hold promise for the development of a vaccine.

Addition Information

For more information on SVC contact:
Otis Miller, Jr.
National Aquaculture Coordinator
USDA, APHIS, Veterinary Services
4700 River Road
Riverdale, MD 20737–1231
Phone: (301) 734–7697

For the OIE's International Aquatic Animal Health Code– 2001, visit their Web site at http://www.oie.int/eng/normes/fcode/A_summry.htm.

For the OIE's Diagnostic Manual for Aquatic Animal Diseases visit their Web site at http://www.oie.int/eng/normes/fmanual/A_summry.htm.
