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Channel Catfish Virus Disease

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Channel catfish virus disease (CCVD) was first recognized as a disease problem during the early days of commercial catfish farming. During the late 1960s, high mortalities were reported in channel catfish (*Ictalurus punctatus*) fingerlings and fry shortly after transfer from the hatchery to fry ponds. The causal agent was identified as a herpesvirus in 1971. Today the virus is present in all catfish growing regions of the United States.

The disease is specific to channel catfish and brood stock are believed to be the major source of infection to young fish. The disease is strongly influenced by environmental stressors. There are no effective preventive or treatment measures, but the effect of the disease can be minimized through optimal management practices.

Although CCVD has a small overall impact on the industry, the effects on individual farms can be significant, with mortalities approaching 100 percent in some production units. Collectively, CCVD typically accounts for only 1 to 2 percent of total disease losses in catfish. In the Mississippi Delta, CCVD accounted for 1.8 to 5.8 percent of cases received by the Aquatic Diagnostic Laboratory at the Thad Cochran National Warmwater Aquaculture Center in Stoneville, Mississippi, from 1997 to 2002.

Clinical signs of disease

Channel catfish virus disease occurs in fry and fingerlings less than a year old and less than 6 inches long, both in the hatchery and in ponds. The first sign is a slowing of feeding activity. Fish may be seen swimming erratically, often in an aimless spiral pattern. Brief episodes of hyperactivity may be seen when fish are disturbed, followed by extended periods of lethargy. Ultimately, large numbers of fish may congregate along the sides of hatching troughs or ponds and hang motionless in a head-up, tail-down position.

Visual inspection of diseased fish usually reveals some or all of the following signs: a swollen abdomen; distension of the vent area; and bulging eyes (Fig. 1). Pinpoint hemorrhages may be seen at the bases of fins, on the ventral abdomen, and within muscle tissue. Gills may be pale and may also contain pinpoint hemorrhages. Clear yellow to blood-tinged fluid is often present in the body cavities of diseased fish. The digestive tract contains no food, but may be filled with yellow fluid and mucus. The liver

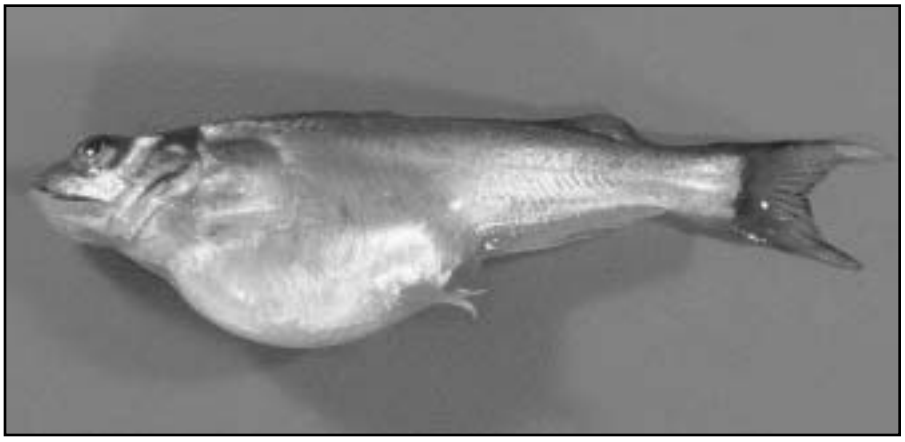

Figure 1. A channel catfish fingerling infected with CCVD. Note the abdominal swelling, hemorrhage, distension of the anal area, and bulging eyeballs.

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and kidney may be pale and contain small hemorrhages, while the spleen is often dark and enlarged (Fig. 2).

The primary damage caused by the virus can be found in the kidney when examined microscopically. Dead tissue and hemorrhage are seen in both the blood-forming and excretory components of the kidney. Kidney failure probably accounts for the accumulation of fluid in the abdominal cavity and other sites, while destruction of blood-forming tissues and hemorrhage result in the pale appearance of the gills, liver and kidney. Lesser damage may also occur in the spleen, liver, intestinal tract, pancreas and brain. Inflammatory cells invade affected areas in an attempt to remove the damaged and infected tissue.

Factors associated with disease outbreaks

The disease is highly host specific for the channel catfish, but susceptibilities vary among individual strains of fish. In early studies, the percent survival between two inbred strains exposed to the virus varied by almost 60 percent (29 percent for Falcon vs. 88 percent for Yazoo). Inbred strains were generally more susceptible to infection, while certain outbred strains had hybrid vigor and increased resistance to the disease. Blue catfish (*Ictalurus furcatus*) fingerlings can be experimentally infected, but are considered resistant under natural conditions. Only a single case of infection has been described. Channel catfish x blue catfish hybrids are reported to be as susceptible as the parental channel catfish strain. Bullhead (*Ameiurus nebulosis, A. natalis, A melas*), European (*Silurus glanis*), African (*Clarius gariepinus*) and Asian catfish (*Clarius batrachus*) are resistant to CCVD infection. The susceptibility of the *Pangasius* species of catfish, basa (*P. bocourti*) and tra (*P. hypophthalamus*), is unknown.

Overall mortality and the speed at which signs develop are highly variable and directly related to fish size, water temperature, and the amount of virus to which the fish are exposed. Concurrent infections with *Flavobacterium columnare* (columnaris disease), *Edwardsiella ictaluri* (enteric septicemia of catfish, or ESC), or both also can influence mortalities. Outbreaks in channel catfish fry and fingerlings usually occur during their first summer (June through September) when water temperatures are above 77 °F (25 °C). The greatest mortalities occur in the smallest fish and when temperatures exceed 86 °F (30 °C). Under farm pond conditions, clinical signs can develop in as little as 2 to 3 days following exposure at 77 to 86 °F (25 to 30 °C), and mortalities may approach 100 percent in less than a week. At 68 °F (20 °C) signs may not be seen for up to 10 days and mortalities are significantly lower. On individual farms, outbreaks may occur sporadically from year to year, but when they do take place, losses can be severe. Mortalities are strongly influenced by poor management practices and environmental factors that place undue stress on young fish.

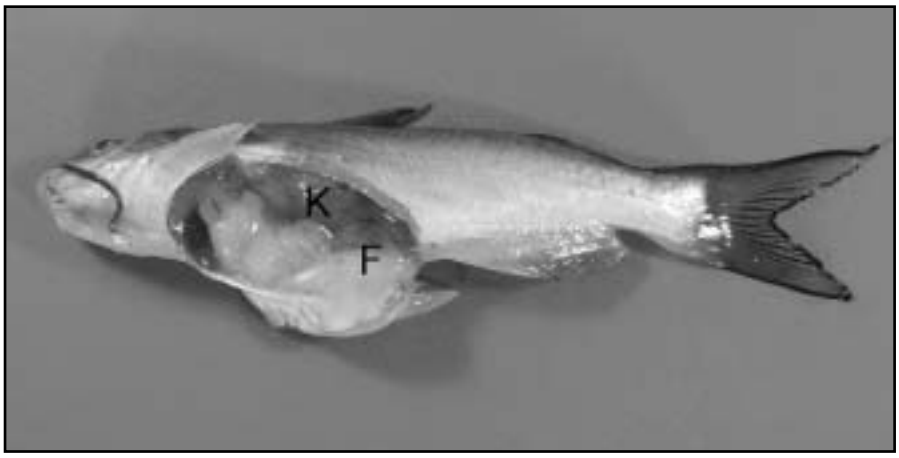

Figure 2. An infected channel catfish fingerling with pale, swollen kidney (K) containing pinpoint hemorrhages. Fluid, or ascites, is also present (F).

Transmission of the virus occurs by two routes–horizontally (from fish to fish) and vertically (presumably from brood stock to fry via eggs or semen). While fish are dying, transmission can occur between fish through the water and by direct contact. The virus enters fish by crossing the lining epithelium of the gill and intestinal tract. Deaths from CCVD are rarely seen after fingerlings reach 1 year of age, unless they are severely stressed.

Most significantly, the virus persists in a dormant form in fish that survive an outbreak. If these fish are subsequently used as brood stock, the infection can be passed without causing signs of disease. Most commercial brood stocks are believed to harbor the virus in this dormant state. The virus is passed, presumably in reproductive products, from brood stock to their offspring at or around the time of spawning. Brood fish may carry the virus even though they have no history of disease or detectable evidence of past infection (neutralizing antibodies) in their blood. It is not known whether food-size fish that have survived an outbreak in previous years can transmit the disease directly to susceptible fingerlings. However, considering the frequency of understocking in the catfish industry, evidence suggests that this probably does not occur to any significant extent.

The virus persists in pond water for only 2 days at 77 °F (25 °C), but up to 28 days at 39 °F (4 °C). The virus is inactivated almost immediately upon contact with pond mud, to which it readily adheres. In dechlorinated tap water survival times are considerably longer, pointing to the need for proper disinfection in hatchery troughs. The virus is highly sus-

ceptible to ultraviolet radiation and drying and is not believed to persist on netting and other equipment allowed to dry and exposed to sunlight for a day or two.

Diagnosis

Channel catfish virus disease should be suspected in fry or fingerlings if there are high mortalities during hot summer months when water temperatures are above 77 °F (25 °C) and clinical signs are consistent with infection. Keep in mind, however, that visible signs may not be distinguishable from ESC and that some temperature overlap exists between the two diseases. It is important to submit specimens for diagnosis while fish are dying, as the virus does not remain at detectable levels for more than a few days after deaths cease. At high summer water temperatures, the virus cannot be isolated from dead and decomposing fish after 48 hours. While diseased or freshly dead specimens are preferable, the virus can be recovered from specimens held on ice for up to 2 weeks and from frozen samples for several months.

Diagnosis involves inoculating filtered organ extracts onto living cell cultures, usually channel catfish ovary cells. The cell cultures are examined for characteristic changes called cytopathic effect, or CPE (Fig. 3). A presumptive, but highly reliable, diagnosis can usually be made in 24 to 48 hours, but can take up to 1 week. Because of the short time required for a diagnosis and the rapidity with which the disease can spread, it is imperative that samples be submitted for diagnosis as soon as a problem is suspected. More sophisticated confirmatory techniques are available, but not at most diagnostic laboratories.

Treatment

There are no effective treatments for CCVD or other viral infections of cultured fish. An accurate diagnosis should be sought, however, as signs may mimic bacterial diseases such as ESC, which can cause producers to make inappropriate or ineffective treatments. Furthermore, certain chemical treatments used to treat external parasites and infections, such as copper sulfate and formalin, can stress fish and precipitate or worsen mortalities from CCVD, primarily because they lower the dissolved oxygen level in the water. The use of antibiotic medicated feeds may reduce mortalities in some cases by controlling concurrent bacterial infections such as ESC and columnaris, which are common during CCVD outbreaks. An alternative to using medicated feeds is simply to stop feeding while fish are dying. This is believed to limit transmission because fish are not crowding together to feed. It also reduces stress by improving water quality. Reducing water temperatures below 66 °F (19 °C) stops deaths under experimental conditions, but is impractical in most commercial operations.

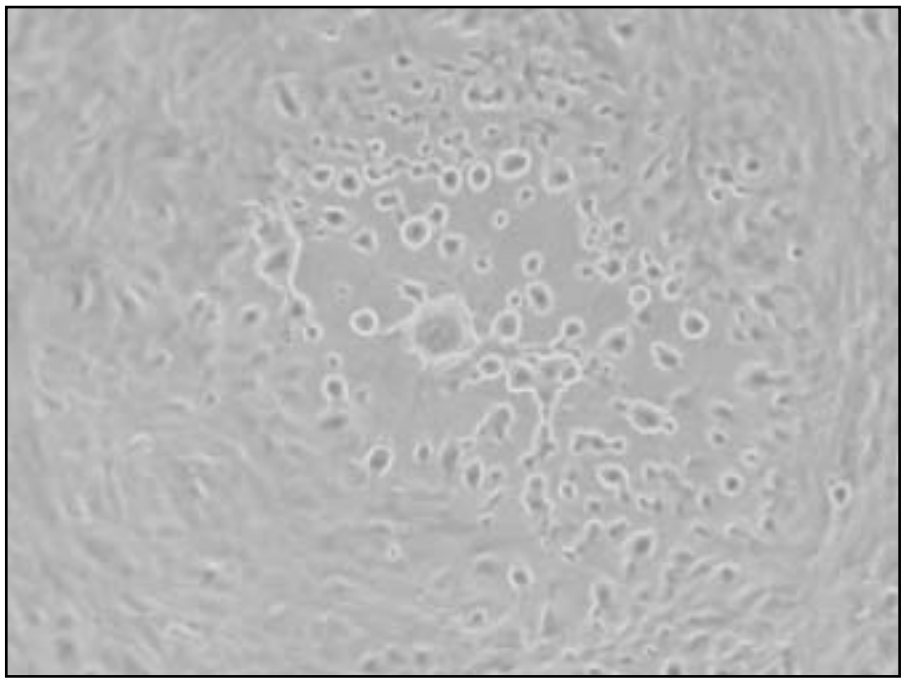

Figure 3. Diagnostic cell culture showing CCVD-infected channel catfish ovary cells exhigiting plaque formation or cytopathic effect (CPE).

Control and prevention

In the absence of effective treatments, good management practices are essential to limiting the frequency and severity of CCVD outbreaks. These practices are avoidance, containment and stress reduction. (Advances in molecular technology may some day make the screening of brood stock and individual egg masses for the presence of virus a practical method of avoiding the disease.)

Water supplies to hatcheries and ponds should not contain feral fishes that might harbor the virus. Newly acquired fry and fingerlings should be quarantined and never mixed with other groups of fish. Fish surviving an outbreak may be stunted, but will eventually grow to market size. However, these fish should not be mixed with susceptible fry or fingerlings or placed in ponds with no known history of the disease. It should be assumed that survivors carry CCVD so they should not be used as brood stock. When the disease cannot be avoided and has been a persistent problem, the use of strains with better resistance may help, though at present they are not widely available.

Containment involves sanitation and disinfection. Troughs in hatcheries should be cleaned, disinfected and dried between batches of fry. Fry from diseased troughs should be carefully removed and destroyed. Be careful not to conta-

minate adjacent troughs with splash over, nets, siphons or other implements. Disinfect equipment, troughs, and any surfaces that contact diseased fish with calcium hypochlorite HTH® to produce 200 mg/l (ppm) available chlorine (Table 1) or with 1.2 ounces (35 ml) of household bleach (5.25 percent sodium hypochlorite) per gallon of water for at least 1 hour. Afterward, neutralize residual chlorine with sodium thiosulfate (7.4 ppm thiosulfate/1 ppm chlorine), aerate vigorously, and flush with large amounts of dechlorinated water. Keep in mind that the presence of abundant organic matter decreases the effectiveness of chlorine-based disinfectants.

Quarantine ponds with diseased fish as well as possible and do not allow effluent to flow to other ponds. Seines, aerators and other equipment should be disinfected and allowed to dry thoroughly. Dead fish should be removed, as they may infect other fish or be moved to nearby ponds by birds and other animals. Susceptible fish should not be stocked into ponds with a history of CCVD unless the ponds have been disinfected, drained and allowed to dry first. Evidence suggests that thorough drying alone may be sufficient to eliminate the virus from ponds.

Maintaining optimal water quality, particularly high dissolved oxygen levels, in hatcheries, brood stock ponds and fingerling ponds is the best way to avoid the stress that may precipitate or worsen an outbreak of CCVD. In the hatchery, avoid crowding, low water flow and poor water circulation. Remove eggshell debris and provide good nutrition with a high protein diet. In fingerling ponds, do not overstock or overfeed. Whenever possible, avoid handling fry and fingerlings at temperatures above 68 °F (20 °C). As this is not practical for most operations, reduce stress by 1) harvesting during the cooler hours of the day, 2) not holding fish for extended periods under close confinement, 3) limiting grading activities, 4) not allowing fish to crowd during transport, and 5) always providing adequate oxygen. Never seine or transport fish during an active outbreak of disease.

The channel catfish can mount a protective immune response against the virus, probably in response to proteins on the outer surface of the viral particle. Despite this, 20 years have passed since the first attempts were made to develop a practical CCVD vaccine and none is available. Vaccine development is hampered by fear that CCVD will revert to a virulent form, difficulties in delivering a vaccine to eggs and large numbers of small fish, the reluctance of licensing authorities, the cost of development and production, and lack of knowledge regarding the existence of multiple, distinct CCVD strains.

Scientists have tried to vaccinate eggs and fry by exposing them to or inoculating them with a weakened CCVD strain, the protein components of the outer coat of the virus, and genetically altered strains. Most recently, a DNA vaccine has shown efficacy in protecting young fish against CCVD, but problems with the delivery system remain to be overcome. Modern laboratory techniques have made it possible to determine the entire genetic makeup of the virus, so it is likely that an effective vaccine will be developed, but only if the cost can be justified against the overall impact of the disease on the industry and if vaccination can be successfully integrated into typical production schedules.

Table 1. Amount of HTH needed to produce 200 ppm available chlorine in 100 gallons (378 liters) of water.

HTH (% chlorine)	Grams	Ounces
15	529	19.0
50	151	5.5
65	121	4.3

SRAC fact sheets are reviewed annually by the Publications, Videos and Computer Software Steering Committee. Fact sheets are revised as new knowledge becomes available. Fact sheets that have not been revised are considered to reflect the current state of knowledge.

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