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CHANNEL CATFISH VIRUS DISEASE

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INTRODUCTION

Channel catfish virus disease (CCVD) is an acute infection of cultured fry and fingerling channel catfish (*Ictalurus punctatus*). The causative agent is the channel catfish virus (CCV), a member of the herpesvirus group. Since its first identification (Fijan 1968) the virus has been isolated from infected fish collected during epizootics at catfish hatcheries in the warmer latitudes of the United States. The disease is found primarily during the summer and, with a single known exception to date, in fish less than 4 months old. The virus has been isolated only from fish taken while an epizootic was in progress.

DIAGNOSIS AND IDENTIFICATION

A sudden increase in morbidity is the first indication of the disease. Infected fish swim erratically or convulsively, sometimes rotating about their longitudinal axis. Victims sink to the bottom, become quiescent and respire weakly but rapidly, and then die.

Clinical signs of diseased fish vary; some or all of the following may be present: distension of the abdomen due to the accumulation of a clear straw-colored fluid in the peritoneal cavity; exophthalmia; pale or hemorrhagic gills; hemorrhagic areas at the base of fins and throughout the skin, particularly on the ventral surface. A general hyperemia occurs throughout the visceral cavity, although the liver and kidneys may be pale. The spleen is generally very dark red and enlarged. The stomach and the intestine are devoid of food but filled with a mucoid secretion.

Histopathologic changes are similar in natural and experimental CCV infections (Wolf et al. 1972, Plumb et al. 1974, Major et al. 1975). Renal hematopoietic tissue is edematous and has extensive areas of necrosis and cellular dissolution coupled with an increase in macrophages. The liver develops regional edema, necrosis, and hemorrhage, and hepatic cells have eosinophilic intracytoplasmic inclusions. Pancreatic acinar cells are necrotic. The submucosa of the digestive tract is edematous and has focal areas of macrophage concentration and hemorrhage. The spleen becomes congested with erythrocytes and this change is coupled with extensive reduction of lymphoid tissue. Virus particles have been seen in electron micrographs of the liver, kidneys, and spleen of infected fish (Plumb et al. 1974).

A generalized viremia is established within 24 h after experimental infection. The kidneys, liver, spleen, and intestine become involved in virus replication 24 to 48 h after infection and virus can sometimes be isolated from brain tissue after 48 h. Peak virus titers occur in the kidneys and intestine 72 h after infection and in the spleen, brain, and liver after 96 h. Few virus particles are found in the musculature.

The virus is frequently found in fish with an apparent secondary bacterial infection of *Aeromonas hydrophila* or *Flexibacter columnaris*. Thus, it is imperative that virological examination of affected catfish be coupled with bacteriological examination.
CAUSE OF THE DISEASE

The etiological agent is a virus with DNA nucleoprotein. Wolf and Darlington (1971) described the virus as herpes-like, and 95 to 105 nm in diameter. Enveloped virions have a diameter of 175-200 nm. Infectivity is inactivated by 20% ether and by treatment with 5% chloroform. CCV is heat-labile at 60 C for 1 h. It survives for less than 24 h on dried concrete chips, and less than 48 h on dried fish netting or glass cover slips. It retains infectivity in pond water for about 2 days at 25 C, but for 28 days at 4 C. In dechlorinated tap water, infectivity is retained for 11 days at 25 C and for over 2 months at 4 C. Under experimental conditions, infectivity is immediately destroyed in pond bottom mud. Infectious virus could not be isolated from decomposing victim fish at 22 C, 48 h after death; however, it was recoverable for up to 14 days from iced fish, for 162 days from fish frozen at -20 C, and for 210 days from fish frozen at -80 C (Plumb et al. 1973).

Intranuclear CCV replication occurs at 10 to 33 C with 25 to 30 C being optimum. Replication takes place in BB cells and in cell cultures derived from channel catfish ovaries, but not in RTG-2, FHM cells, or several homeothermic cell lines (Wolf and Darlington 1971). In susceptible cell cultures the virus induces the formation of characteristic giant multinucleated (syncytium) cells. Initial cytopathic effect may be evident as early as 2 h in channel catfish cells at 25 C.

POSSIBLE SOURCE OF INFECTION

The virus has been isolated from fingerling channel catfish during epizootics only when mortality was occurring; its isolation from adults has not been demonstrated. However, some adults that produced CCV-diseased offspring had high virus neutralizing antibody titers, and consequently were suspected of being carriers.

MODE OF TRANSMISSION

Under experimental conditions, it is possible to transmit the virus through the water to healthy fish from infected moribund or dead fish. The virus can also be transmitted by intramuscular or intraperitoneal injection, by its incorporation into feed, or by swabbing the gills with saline solution containing virus. Circumstantial evidence points to vertical transmission of the virus from adult to offspring.

INCUBATION PERIOD

The incubation period is inversely related to water temperature. Experimental infection at 30 C was followed by clinical signs in 32 to 72 h, and by the first deaths several hours thereafter. At 20 C the incubation period is 10 days. Under field conditions at 25 to 30 C, healthy channel catfish fingerlings develop the disease within 72 to 78 h after exposure, and up to 100%
die within 6 days. A group of naturally infected fry held at 28 C developed clinical signs of CCVD when 21 days old; 72 h later all were dead. A sample assayed for CCV when these fish were 14 days old was negative.

PERIOD OF COMMUNICABILITY

Channel catfish virus is infective when fish show clinical signs or soon after their death. The virus begins to disappear in surviving artificially infected fingerlings 120 h after infection. It is difficult or impossible to isolate virus once the clinical signs of enlarged abdomen, exophthalmia, and hemorrhage have passed.

SUSCEPTIBILITY AND RESISTANCE

The channel catfish is apparently the principal species affected by CCV. Experimental infection was induced in fingerling blue catfish (*Ictalurus furcatus*) and in channel catfish x blue catfish hybrids by injection, but not orally or by cohabitation with virus-infected channel catfish fingerlings. Brown bullheads (*I. nebulosus*) or yellow bullheads (*I. natalis*) could not be infected by injection or feeding. Feeding virus to different strains of channel catfish fry has indicated variation in susceptibility (Plumb et al. 1975). Young fish resulting from breeding different strains of channel catfish were more resistant to CCV than were purebred strains. Channel catfish up to 1 year old or 10 to 15 cm long are susceptible under experimental conditions. Upon injection, virus kills fish weighing up to 50 g, and in one instance CCV was isolated from a 190-g fish that died 2 days after injection. There is evidence that the growth of survivors of CCV epizootics may be stunted (McGlamery and Cratzek 1974).

RANGE

Channel catfish virus has been reported from channel catfish in most southern states and from intensive culture systems in Nebraska, Colorado, Kansas, Oklahoma, and California. The virus was also isolated from a group of fry shipped from the United States to Central America.

OCCURRENCE

Cases of CCVD have been diagnosed throughout the period from June to September. A number of cases were preceded by handling and occurred at water temperatures above 25 C. Laboratory experiments and field studies indicate that temperatures between 25 and 30 C favor the development of CCVD.
METHODS OF CONTROL

There is no known chemotherapeutic treatment for viral diseases of fishes. At present, the only practical control measures are avoidance, isolation, and sanitation. The incidence of CCVD is closely correlated with temperature. In laboratory experiments, mortality decreased significantly when the water temperature was reduced from 28 °C to 19 °C or less, 24 h after infection. Although this procedure has only limited application, it may be useful in areas where cool water is available.

Ponds from which diseased fish are removed should be drained and dried or disinfected with 40 ppm or more of chlorine. Survivors of CCV epizootics may be grown to a marketable size, providing the fish are held in ponds that do not pose a threat to other channel catfish. Under no circumstances should survivors of CCV epizootics be stocked in uninfected waters, nor should they be used as brood stock.

ANNOTATED BIBLIOGRAPHY


This is the first published account of channel catfish virus disease.


Describes three of the four initial outbreaks of the disease. Clinical signs are described and experimental evidence of the viral etiology is presented.


Inoculation of channel catfish with Chondrococcus columnaris cells and CCV resulted in a rapid primary response to both antigens. Analysis of total serum proteins and selected isozyme systems by acrylamide gel electrophoresis revealed differences between normal and experimental fish sera in the number of protein components. Specific antibody activity of the immune serum occurred in the macroglobulin fraction. Partial antibody characterization indicated that specific channel catfish immunoglobulins are macroglobulins with characteristics like those of most fish immunoglobulins.

Two groups of fingerling channel catfish, one experimentally infected with CCV by transmission through water, and the other collected during an epizootic, were examined histologically. Previously unreported pathologic changes of pancreatic and brain tissue are described.


Stunting in channel catfish which survived a channel catfish virus infection under experimental conditions is reported.


This paper provides an overall discussion of CCV disease. History, distribution, and epizootiology of the disease are discussed. Recommended practices for controlling the disease are presented.


Describes the reduction of mortality of infected fish when the water temperature was reduced from 28 C to 18 C. Application of this technique may reduce the effects of CCV in some fish cultural operations.


A comprehensive study of CCV disease. Discusses the problems of identifying carrier fish, the clinical and immune response of subadult fish to CCV, and organ tropism of the virus. Describes the electron microscopy of CCV replication in selected organs. Provides information on variation in susceptibility in several strains of channel catfish and a method of reducing mortality by reducing water temperature. Field studies on virus transmission are also discussed.


The neutralization of CCV by sera of adult fish that have been naturally exposed and experimentally injected with CCV is described. Antibody response in experimentally infected fish reached a peak in 60 days, whereas neutralization by naturally exposed fish remained high for a year. Use of serum neutralization of sera from adult fish may be used to separate CCV-exposed from nonexposed populations.

The survival of CCV in decomposing and iced catfish and fish frozen at -20 C, -80 C, and on dry ice is reported.


A histologic and electron microscopic study was made on selected organs from channel catfish fingerlings that were experimentally infected with CCV.


Sequential virus titrations were made on kidneys, intestine, liver, spleen, brain, and blood of CCV-injected fish. Corresponding sequential histopathogenesis is described for each tissue except blood.


The susceptibility to CCV differed among six strains of channel catfish and two strain-hybrid groups tested. Hybrids were less susceptible than the pure strains.


An excellent general account of viruses as pathogens. Discusses six recommendations for the control of virus disease. Of special importance to fish culturists.


Discusses history, pathology, transmission, control methods, and classification of the known fish viruses. Of particular interest is a detailed section on the problems and possibilities of controlling virus diseases of fishes.

Describes replication of CCV in BB cell cultures at temperatures of 10 to 33 C. Provides the virus characteristics that place CCV in the herpesvirus group and describes the development of virus replication in BB cells as determined by electron microscopy.


Provides the first account of the histopathology caused by CCV. Describes pathologic changes found in kidneys, liver, intestine, and other tissues.