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Epizootiology of Channel Catfish Virus Disease

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ABSTRACT—Channel catfish virus disease (CCVD) is a highly pathogenic herpesvirus disease of cultured channel catfish, Ictalurus punctatus, *fry and fingerlings. The disease is confined to the southern United States, but occurs sporadically in other areas where young channel catfish are intensively cultured. Blue catfish,* I. furcatus, *have been experimentally infected with CCV, but other ictalurids appear to be refractory. The causative virus can be isolated only from fish showing clinical signs and morbidity. The clinical signs resemble those of other fish viral diseases. Histopathology is characterized by hemorrhage, necrosis, and edema in most tissues. It is possible to isolate CCV in brown bullhead,* I. nebulosis, *cells or cell cultures derived from channel catfish ovaries. The immunological response of channel catfish is discussed and theoretical control measures are suggested.*

EPIZOOTIOLOGY

Channel catfish virus disease (CCVD) is a highly communicable viral disease of juvenile channel catfish, *Ictalurus punctatus,* in North America. The etiology of CCVD is a herpesvirus which results in hemorrhagia and edema in very young fish. It is primarily confined to cultured channel catfish populations in the southern United States where it occurs during the warmer months of June through September. Since the initial isolations (Fijan, 1968), CCVD has been confirmed in numerous epizootics in most southern states and several other localities where channel catfish are intensively cultured, including one Central American country.

Results of channel catfish virus (CCV) infections have varied from mild to catostrophic in different populations. The potential severity of the disease among susceptible channel catfish populations makes CCV a serious threat to culture systems already containing the virus, but its effect upon the catfish industry is not yet clear. Channel catfish virus has generally been found in fry or young-of-the-year fingerling channel catfish, although the virus was isolated from one yearling group of 18-cm fingerlings. Younger fish are much more susceptible, and in some cases 100 percent of infected fry have died involving individual lots of 8,000 to 3 million fish. In the fingerling stage (up to 5-10 cm and less than 5 months old) mortality may exceed 90 percent. However, size and age of fish alone do not determine the degree of mortality: environmental stresses such as low dissolved oxygen levels, high water temperature, crowding, and the stress of handling or transporting infected fish may influence the rate of mortality and may serve as triggering mechanisms for epizootics. Optimum water temperature for CCVD is above 25°C. Mortality may occur at 20°C, but below this temperature the effects of CCV are diminished. Secondary bacterial infections of *Aeromonas hydrophila* and *Flexibacter columnaris* also serve as synergistic forces in CCV infections and probably contribute to mortality.

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TRANSMISSION

Channel catfish virus can be readily transmitted from infected fish to healthy fry or fingerlings by placing the healthy fish in water outfall from tanks or ponds holding infected stocks. The virus can also be readily transmitted to channel catfish by intramuscular or intraperitoneal injection, placing infected and noninfected fingerlings together in the same tank, swabbing the gills with virus, or by feeding infected materials. The possibility of vertical transmission from carrier adult fish to their offspring exists. There has been a tendency for epizootics to be associated with young from certain groups of broodfish, but the pattern of epizootic occurrences has been sufficiently erratic to shed some doubt on the theory of a definite adult carrier. In spite of numerous attempts, CCV has not been isolated from adult or subadult fish selected from a suspect carrier population.

Blue catfish, *Ictalurus furcatus,* can be experimentally infected by intraperitoneal injection. The virus could

not be transmitted horizontally from infected blue catfish fingerlings to non-infected fingerlings, or by feeding contaminated feed. White catfish, *Ictalurus catus*, and brown bullhead, *Ictalurus nebulosis*, fingerlings could not be experimentally infected.

Plumb et al. (1975) presented evidence that genetically different strains of channel catfish vary in their susceptibility to CCV (Fig. 1). Under controlled conditions the survival of experimentally infected 1- to 3-month old fingerlings ranged from 10 to 71 percent. Heterogenetic F_1 fingerlings had lower mortality than most homogenetic groups.

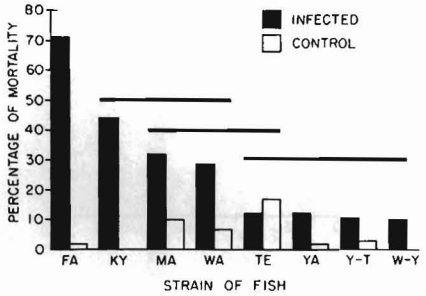


Figure 1.—Mortality of different strains of channel catfish fingerlings fed channel catfish virus. The strains infected were: Falcon (FA), Kentucky (KY), Marion (MA), Tennessee (TE), Warrior (WA), Yazoo (YA), Warrior-Yazoo (W-Y), Yazoo-Tennessee (Y-T). The means of any two strains that do not fall under the same line are significantly different (0.05). From Plumb et al., 1975.

CLINICAL SIGNS AND PATHOGENESIS

The clinical signs of CCVD were described by Fijan et al. (1970) and Plumb (1971a). Typically infected fish are lethargic, with hemorrhages in the skin and at the base of fins, bilateral exophthalmia, distended abdomens, pale gills, and dark pigmentation. The body cavity may be filled with a clear yellowish fluid; the liver is generally pale; the kidney is enlarged and pale, but the spleen is deep red; the stomach and intestine are pale and void of food, but filled with yellow mucoid fluid. Some affected fish swim erratically and immediately prior to death may hang at the water surface in a head-up position, but this latter sympton may be caused by other diseases or environmental disorders, especially when very young fish are involved.

After experimental CCV infections of fingerlings, a viremia quickly develops (Plumb, 1971b; Plumb and Gaines, 1975). Virus was isolated from the kidney, liver, and intestine 24 hours after infection, but the kidney consistently produced higher titers than other organs (Fig. 2). Virus was also isolated from the blood, brain, and spleen (Fig. 3). Very little virus was isolated from the muscle. Virus titers reached peaks at 72 to 96 hours after infection and then subsided. Seven days after injection the virus levels in kidney, intestine, liver, and blood were 2-4 logs below the peak levels. However, virus titers in spleen and brain increased by 1-2 logs from day 6 to day 7 postinjection. The reason for these differences is not clear, but if infections were becoming dormant 6-7 days after infection, these two organs may be sites for development of latent infection.

Histologic findings of CCVD have been described primarily from experimentally infected fish (Wolf et al., 1972; Plumb et al., 1974; Plumb and Gaines, 1975; Majors et al., 1975). Internally, diseased fish exhibit: General hemorrhagia; liver epithelium and pancreatic tissue have focal necrosis; renal hematopoietic and excretory tissues are necrotic and edematous; while white and red pulp of the spleen are completely destroyed. Tissues of the diges-

Figure 2.—Postinjection titers of channel catfish virus in kidney, liver, and intestine of experimentally infected channel catfish fingerlings. From Plumb and Gaines, 1975.

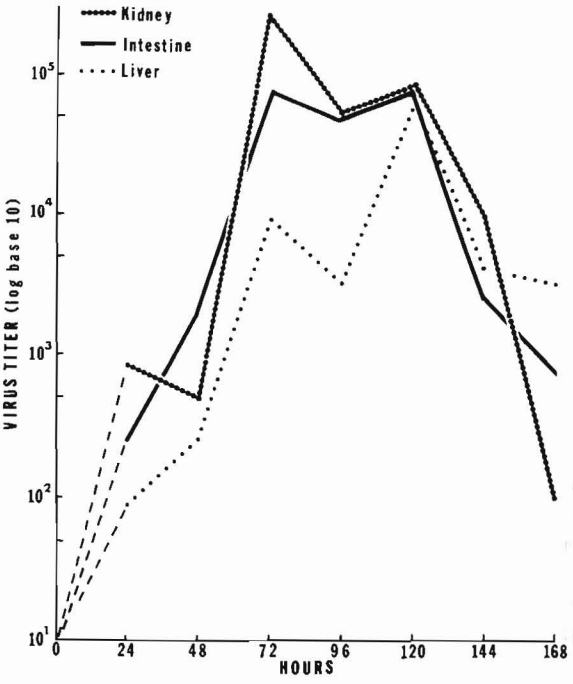
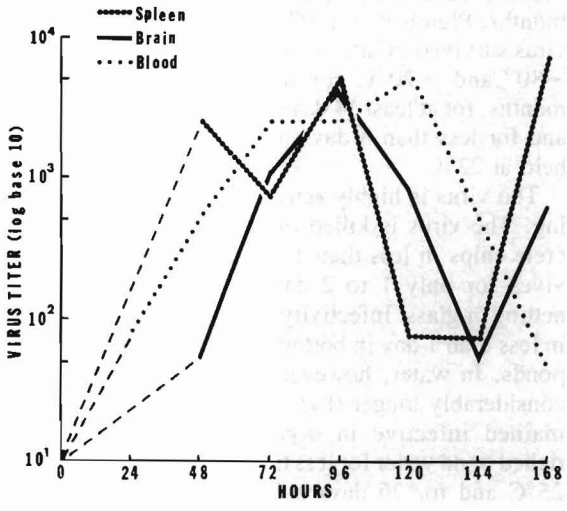


Figure 3.—Postinjection titers of channel catfish virus in blood, brain and spleen of experimentally infected channel catfish fingerlings. From Plumb and Gaines, 1975.



tive tract are edematous and hemorrhagic, and the mucosal layer of the intestine sloughs into the lumen. Skeletal muscle may have focal extravasation of blood, and brain tissue may be edematous. Histopathology of CCVD is very similar to that resulting from salmonid viruses in spite of their etiological differences (Yasutake, 1975).

Electron micrographs of tissues from experimentally infected fingerlings showed that CCV replicates in the nuclei of kidney, liver, and spleen cells (Plumb et al., 1974). Virus replication was characterized by the presence of intranuclear crystalline arrays and lamellar inclusions. Organ viral assay, and light and electron microscopy showed that CCVD is a systemic infection with generalized viremia. However, it appears that the kidney is the first and most severely affected organ.

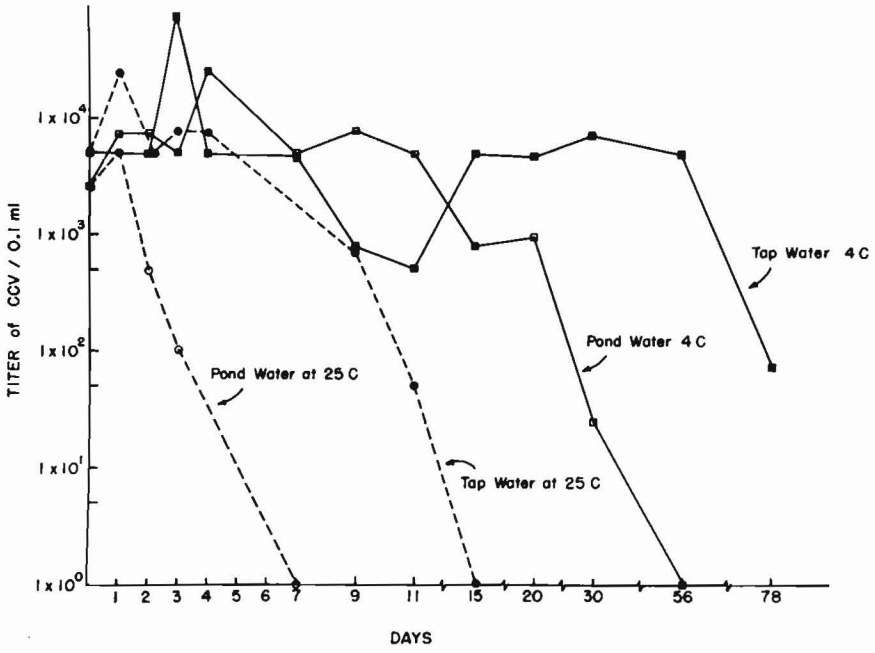


Figure 4.—Survival of channel catfish virus in pond and tap water at 4° and 25°C.

MORPHOLOGY AND BIOPHYSICAL NATURE

Based on evidence presented by Wolf and Darlington (1971) CCV is an icosahedral herpesvirus with 172 capsomeres. The nucleocapsid measured 95-105 nm in diameter, but the enveloped particle has a diameter of 175-200 nm. The virus is ether, chloroform, and glycerol labile.

Channel catfish virus is very stable under frozen conditions in vitro and in vivo. Wolf (1973) reported that at −80°C in tissue culture media, no infectivity was lost after 9 months and at −20°C infectivity persisted for 4 months. Plumb et al. (1973) found that virus survived in infected fish frozen at −80° and −20°C for more than 6 months, for at least 14 days in iced fish, and for less than 3 days in specimens held at 22°C.

The virus is highly sensitive to drying. The virus is killed on dried concrete chips in less than 1 day and survives for only 1 to 2 days on nylon netting or glass. Infectivity is destroyed in less than 1 day in bottom muds from ponds. In water, however, survival is considerably longer (Fig. 4); CCV remained infective in organically enriched pond water for less than 4 days at 25°C and for 26 days at 4°C, but in dechlorinated municipal water, infectivity was prolonged to approximately 11 days at 25°C and to more than 78 days at 4°C. Channel catfish virus does not survive well in water or on equipment at the temperature one would expect the disease to occur, especially if facilities and equipment are dried.

CELL CULTURE CHARACTERISTICS

The brown bullhead (BB) cell line (ATC-59) is the line of choice for CCV isolation and replication; however, cell culture derived from channel catfish ovaries are equally suitable. Wolf and Darlington (1971) failed to detect CCV replication in 15 other cell types including cells from other groups of fish, amphibia, birds, and mammals. Optimum replication temperature in BB cells is 30°−33°C, although replication will occur at 10°C (Wolf and Darlington, 1971). They found that at 30°C new virus was released 4 hours after cell culture inoculation, and cell associated virus reached a peak at 12 hours postinfection. The rate of virus replication in cell cultures is more rapid than in the fish as previously discussed (Plumb and Gaines, 1975). However, the cellular ultrastructure changes are similar in vitro and in vivo.

Cytopathic effect of CCV in BB cell cultures is characterized by the development of syncytia resulting from coalescing of many infected cells and inclusion of their nuclei. Prior to syncytial formation, cells become pyknotic, basophilic, show aggregation of chromatin, and lamellar-like, intranuclear inclusion bodies appear (Wolf and Darlington, 1971). Karyorrhexis and karyolysis follow the appearance of syncytia.

IMMUNOLOGICAL RESPONSE

The immunological response of fish is temperature dependent (Bissett, 1948; Snieszko, 1970). Trout respond to antigens slowly at their optimum growth temperatures, but warm-water species, especially channel catfish, respond very rapidly under their higher optimum growth temperature. McGlamery et al. (1971) reported a positive antibody response in 2-4 weeks in channel catfish inoculated with stomatitis virus when the fish were held above 25°C. Heartwell (1975) detected CCV antibody at 28°C in channel catfish 1 week after injection of viable virus.

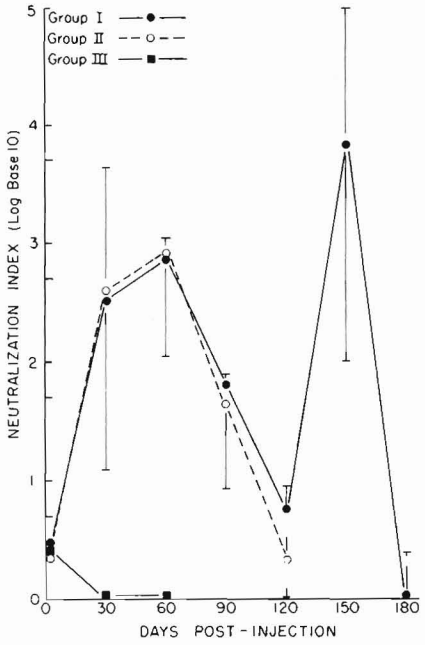


Figure 5.—Neutralization indices (log base 10) of 1-year-old channel catfish injected with channel catfish virus. Groups I and II were inoculated with infectious virus and Group III was inoculated with heat-killed virus. Group I was given a booster injection 120 days after the initial inoculation. From Plumb, 1973b.

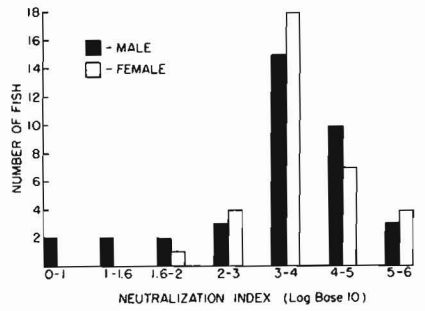


Figure 6.—Channel catfish virus (CCV) neutralization indices of sera from adult channel catfish with a history of CCV-diseased progeny.

Peak CCV specific antibody titers occur in adult channel catfish approximately 9 weeks after a single inoculum of virus (Fig. 5). A rapid anamnestic response will occur with a second injection after the initial peak titer, but it is weak and of short duration (Heartwell, 1975). Injection of heat-killed CCV elicits no immunological response. There is no information on the immunocompetence of fry or fingerling channel catfish to CCV.

The neutralization index (NI) was determined for sera from 67 adult channel catfish which had produced CCV diseased offspring for two consecutive years (Plumb, 1973b). The NI of these fish ranged from 1×10^1 to 1×10^5 with 76 percent of the NI values between 1×10^3 and 1×10^5 (Fig. 6). The NI of sera from a control group of adult channel catfish which had no history of CCV were all less than $1 \times 10^{1.25}$. Also, the NI from the CCV suspect fish remained at a high level throughout the year in contrast to the experimental immunological response reported by Plumb (1973b) and Heartwell (1975).

CONTROL

There is no known control of CCV; however, Plumb (1973a) reduced the water temperature from 28° to 18°C on infected fish 24 hours after infection and reduced the mortality from 95 to 24 percent. Reduction of temperature at the onset of clinical signs and death reduced mortality to 58-78 percent. In some instances where a fish farmer has cool water available and recognizes the signs of CCV, it may be possible to reduce the mortality by introducing cool water onto the population. However, the best means of combating CCVD is through avoidance of infected stocks. Detection of potential carriers is not possible through direct virus isolation. Broodfish previously associated with CCV epizootics had high levels of CCV specific antibody, therefore a serological approach may be possible to detect potential reservoirs of CCV.

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