or hardness. However, the oxygen difference of 0.6 to 1 ppm probably represents a real difference even though it is not statistically significant because fish died of oxygen deficiency in three aeration control ponds but

SUMMARY AND CONCLUSIONS

1. Recirculation and biofiltration of the water in fish ponds yielded a net production of catfish of over 19,300 lb/acre.
2. Channel catfish and white catfish responded similarly to the filtered water system.
3. Filtration improved the water quality in the pond by reducing ammonia, organic carbon, nitrite and turbidity and possible increasing dissolved oxygen.
4. At high stocking rates (4,000 lb/acre) a settling basin in series with the filter had no effect on the fish production but did cause a 20.6% increase in production at low stocking rates (2,000 lb/acre).
5. As a result of crowding of the fish, diseases and parasites were a major problem.

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CHANNEL CATFISH VIRUS DISEASE IN SOUTHERN UNITED STATES

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ABSTRACT

Channel catfish virus disease is caused by a specific virus which has been isolated from 23 epizootics reported from nine different states. This paper gives clinical signs, mortality patterns and susceptibility of different age and size fish to channel catfish virus. Recommended practices for controlling the disease are presented.

INTRODUCTION

Channel catfish virus is the etiological agent of channel catfish virus disease (CCVD) and is the only known virus disease that results in high mortalities in warmwater fish of North America. The virus appears to be limited to the Southern United States where mortalities as great as 95% have been reported from epizootics affecting cultured channel catfish, *Ictalurus punctatus* (Rafinesque).

The objectives of this paper are to make more people aware of the presence of CCVD, describe some of its characteristics and effects on catfish populations and to present some suggestions for corrective measures. The author has tried to bring together the available information about the disease which may aid in recognition and control of CCV epizootics.

HISTORY AND DISTRIBUTION

Channel catfish virus disease was first discovered in 1968 (Fijan, 1968; Fijan, Wellborn, and Naftel, 1970) when it was isolated from channel catfish epizootics in four southern states. Since that time it has been found in five additional states. Table 1 illustrates the distribution and relative frequency with which this disease has occurred.

TABLE 1. Geographic distribution and frequency of channel catfish virus disease, 1968-1971

State	Number of cases per year 1968	1969	1970	1971	Totals
Alabama	1	2	1	2	6
Arkansas	1	1	0	1	3
Georgia	0	1	0	2	3
Kentucky	1	0	0	0	1
Mississippi	0	0	2	1	3
Texas	1	0	2	1	4
West Virginia *	0	0	0	1	1
Kansas **	0	0	0	1	1
Oklahoma ** †	0	0	0	1	1
Totals	4	4	5	10	23

* K. E. Wolf, Eastern Fish Disease Laboratory. Kearneysville, West Virginia (personal communication).
** Bureau of Sport Fisheries and Wildlife, Region II, Division of Fish Hatchery personnel (personal communication).
† Fry transferred from hatchery in Kansas.

Twenty of the 23 cases reported in Table 1 were diagnosed at the Southeastern Cooperative Fish Disease Laboratory, Auburn University. There have been numerous earlier reports of catfish mortalities, accompanied by the described clinical signs of the disease, from which no virus isolations were attempted. It is possible that the disease has been occurring for some time and is just beginning to be recognized. The disease is widespread and coupled with its effect on a catfish population, it is a problem that warrants concern.

Fingerlings from commercial hatcheries or from recently stocked grow-out ponds have produced 61% of the CCVD cases thus far diagnosed (Table 2). Federal hatcheries have been responsible for 31% of

TABLE 2. Sources of channel catfish virus isolates, 1968-1971

Year	SOURCE Commercial	Federal	Other	Total
1968	1	2	1	4
1969	3	1	0	4
1970	5	0	0	5
1971	5	4	1	10
Total	14	7	2	23
Percent of total	61	31	8	..

the cases. It is apparent that the commercial catfish industry is the primary source of the disease and indiscriminate shipping of survivors of epizootics has undoubtedly increased the range of the disease. As far as is known only two of the affected commercial hatcheries have taken steps to eradicate the disease, whereas, all of the federal hatcheries have initiated corrective measures. On one commercial farm where CCVD appeared in 1968, the infected fingerlings and broodstock were destroyed and the ponds disinfected with chlorine. The disease has not reoccurred. This method of disinfection was successful for Fijan *et. al.* (1970) and more recently at a federal hatchery.[1]

EPIZOOTIOLOGY

CCV is a highly virulent, communicable disease of fry and fingerling channel catfish. The susceptibility of other species of fish to CCV has been tested in the laboratory. White catfish *Ictalurus catus* (Linnaeus), (Fijan and Naftel, in review) and blue catfish *Ictalurus furcatus* (Lesueur) (Plumb, unpublished data) have shown some experimental susceptibility, but the virus has not been isolated from these species under hatchery conditions and these species are probably refractive to the virus.

Wellborn, Fijan and Naftel (1969) and Fijan, *et. al.* (1970) reported that the disease can be transmitted in water, by contact or by swabbing the gills of susceptible fish with virus. The disease can be transmitted by intraperitoneal or intramuscular injection with bacteria free filtrate and by feeding infected feed. It is speculated that the virus may be transmitted vertically from broodstock to offspring via the egg, sperm, or ovarian fluid (Wellborn, *et. al.*, 1969; Plumb, 1971a). Several cases have occurred that strongly support this theory. In one hatchery situation, fry of the same lot of broodstock contracted the disease two successive years, and in a second instance, progeny left in the pond with their parents developed CCVD. The adult fish in each case exhibited very high serum neutralization indices against CCV. In view of this, surviving fish of epizootics should not be used as broodstock and should be considered potential virus carriers. It should be pointed out, however, that virus has not been isolated from adult fish despite numerous attempts.

Characteristically, fingerling fish infected with CCV are lethargic, sometimes swim eradically or hang head-up in the water, then fall to the bottom and expire. The fish have distended abdomens, exhibit exophthalmia and show varying degrees of hemorrhaging on the body and fins; the gills are pale and indicative of a general anaemia. Internally, the body cavity is filled with straw colored fluid and the intestine is usually devoid of food; the liver and kidney are pale or hemorrhagic with petechiae. All internal organs are swollen. However, it is important to remember that all of these clinical signs may not be present and they are not unique to CCVD. Positive identification of CCVD can only be made with the aid of cell culture.

Infection develops rapidly after injection of virus with mortality occurring as quickly as 32 hours later. Virus can be isolated from the kidney and blood 24 hours after infection and sequentially from the intestine, spleen, liver, and brain (Plumb, 1971b). The kidney of infected fish appears to be the primary organ affected, often yielding more than 1×10^6 $CCID_{50}$ per CC of tissue (Cell culture infectious doses—50% end point); all organs and tissues thus far examined are affected in some way.

Although mortalities are frequently acute with 95% loss of fish, there have been some cases where the mortality remained less than 50%. The degree of mortality is believed to depend upon the age and size of the fish (Fijan, *et. al.* 1970), but unfavorable environmental conditions such as high temperature, low oxygen, crowding, improper handling and secondary bacterial infections may play a large role in stimulating development of epizootics. Stress of some type has been implicated in

[1] K. E. Wolf, Eastern Fish Disease Laboratory, Kearneysville, West Virginia (personal communication).

many cases and all cases have been associated with water temperature in excess of 28 C.

Fijan and Naftel (in review) stated that acute mortality of 85 to 100% occurred at 30 C or higher; chronic mortality at 20 to 25 C and little or no mortality below 15 C. The Mammoth Spring National Fish Hatchery, Mammoth Spring, Arkansas, has successfully avoided reoccurring epizootics by holding sac fry in troughs with the temperature below 27 C and then immediately moving the swim-up fry into rearing ponds.[1] However, this is not a foolproof rearing technique, as many of the cases that have been diagnosed involved fish that were pond hatched and reared.

With one exception, natural epizootics in hatcheries have involved young of-the-year fish weighing less than 10 g (Fijan, *et. al.*, 1970). The exception involved 12-month-old fish weighing 10 to 12 g (Plumb, unpublished data).

Fijan and Naftel (in review) reported that fish weighing 10 g were artificially infected with CCVD, but fish that weighed 200 g were refractive. The limited data illustrated in Table 3 indicate that both size and age play a role in susceptibility. When fish exceed 1 year of age they are less susceptible, but fish up to 100 g may be experimentally infected. The 13 month old fish from a private hatchery were refractive to CCV even though they weighed only 6g. These fish were in poor physical condition which may have reduced their susceptibility. It has been observed that well fed, healthy catfish are more susceptible to CCV than fish in poor condition. A smilar phenomenon has been reported with infectious pancreatic necrosis virus of trout where the larger, more robust, fish were the first to become diseased. (Wolf, 1966.)

TABLE 3. Effects of channel catfish virus on different size and age channel catfish from 3 sources

Source of test fish	Age Mo.	Weight g.	Fish injected	Virus dilution *	Mortality Pct.
Marion National Fish Hatchery	5.5	15	15	10^{-2}	93
Auburn Agricultural Experiment Station	8	5	10	10^{-1}	100
	18	100	8	10^{0}	13
	24	200	7	10^{0}	0
Private Hatchery	10	4.3	10	10^{0}	100
	13	6.0	10	10^{-1}	0

* Each fish injected intraperitoneally with 0.1 ml of the virus dilution with an unknown titer.

CONCLUSIONS AND RECOMMENDATIONS

It is apparent that CCVD is a disease influenced by unsuitable environmental conditions. With this in mind the following recommendations are suggested in combating the disease on catfish farms with a history of CCVD:

1. Avoid moving fingerling channel catfish during hot weather.
2. Do not overcrowd the population.
3. Do not employ intensive trough culture, but place the fry in ponds as soon as possible.
4. Keep the water temperature below 27 C.

In view of the fact that CCVD is a highly communicable virus disease and that survivors of epizootics may be carriers of the disease, the shipping of diseased fish or survivors to waters where no history of the

[1] R. G. Bowker, Hatchery manager, Mammoth Spring National Fish Hatchery, Mammoth Spring, Arkansas.

disease exists must be avoided. Also, the utilization of survivors of CCV epizootics for broodstock is not recommended. The safest procedure to prevent reoccurence of the disease is to destroy the affected fish and the broodstock and disinfect the ponds with chlorine. New fish stocks should be acquired from a source that has no history of CCVD. An ethical attitude of the catfish grower is essential in limiting the continued spread of CCV.

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DISEASE IN FISH DUE TO THE PROTOZOAN Epistylis (CILIATA:PERITRICHA) IN THE SOUTHEASTERN U. S.*

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ABSTRACT

"Red-sore" disease of fishes caused by the stalked ciliate *Epistylis*, is very common and widespread in the Southeastern U. S. Epizootics occur most frequently during the winter and spring months. Research has shown that this species is not an obligate parasite but only uses the host fish as an attachment site. The disc-like attachment organelle, penetrating the skin of the fish, apparently secretes an enzyme that dissolves the fishes' scales or spines and produces pit-like inflamed lesions. Bacterial infections often occur secondarily to the *Epistylis* infestation. Observations of the life history have shown a formation of telotrochs which are characteristic of the order Peritricha. Preliminary laboratory tests show that a single treatment with potassium permanganate at a rate of 2 ppm or formalin at a rate of 15 ppm will control *Epistylis*.

INTRODUCTION

A characteristic "red-sore" disease of fish involving scale erosion and pit-like inflamed lesions is produced by the stalked ciliated protozoan *Epistylis*. This disease is very common in the Southeastern U. S. and cases of the disease have been reported to the Southeastern Cooperative Fish Disease Project Laboratory from virtually every state in the Southeast. Even though the disease is extremely common and appears to affect mainly species of sport fishes, no published reports of the occurrence of the disease are available in the U. S. other than that of Rogers

* Supported by the Southeastern Cooperative Fish Disease Project (In part by Sport Fish Restoration Funds).

(1970). The main problem with *Epistylis* infestation is not that of mortality of fish but of rejection by anglers because of the diseased appearance of the fish. The species of *Epistylis* reported in this paper is not an obligate parasite but apparently uses the host fish only as an attachment site.

THE ORGANISM

Epistylis is characterized by having an urn-shaped or elongate bell-shaped body attached to a dichotomously branched stalk (Fig. 1). A ring of cilia leading into the cytostome adorns the adoral end of the body. An elongated ribbon shaped macronucleus winds through the center of the body. The stalks are non-contractile and are attached to the skin of the host by a disc-like holdfast organ.

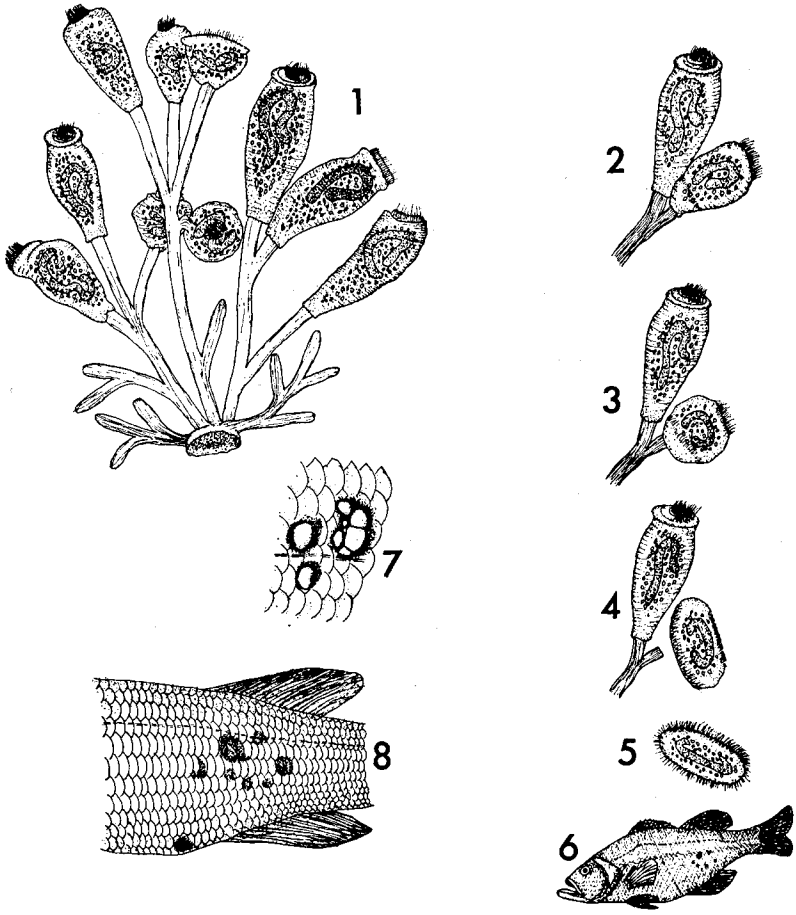

FIGURE 1. Colony of *Epistylis*. FIGURES 2-4. Formation of telotroch by individual *Epistylis*. FIGURE 5-8. Telotroch seeks new host and establishes new colony and characteristic pit-like lesions.

LIFE CYCLE

Epistylis is not an obligate parasite. Colonies of the organism were found to grow in the laboratory on food particles or other organic debris in fish holding tanks or attached to sides or bottoms of the tanks.

Observations of transmission of the organism showed the formation of telotrochs which are characteristic of the Peritricha. *Epistylis* is a primary invader and does not require breaks in the epithelium to become established. The telotroch developed in the following manner: the body of the organism contracted and rounded up and then a ring of cilia developed near the proximal end of the body (Fig. 2); the adoral ring of cilia was apparently re-absorbed into the body; the body then changed from a rounded to a dorsoventrally depressed, disc-like shape with a ring of cilia around the margin (Figs. 3-4); it then detached from the stalk and became a free-swimming telotroch (Fig. 4). The telotroch then seeks a new host or other attachment site, secretes a stalk and holdfast, elongates and divides by binary fission to produce a new colony and the characteristic lesions (Figs. 5-8).

PATHOLOGY

In transmission studies in the laboratory the teletroch almost always attached itself to the host at the end of spines or on epithelium overlying bones or spines. In severe infestations colonies were found all over the body.

The first detectable lesions on the fish were small protrusions of proliferated epithelium. Within this hyperplastic growth could be found one to several *Epistylis* cells. Apparently the telotroch would cause cell proliferation that would enclose the organism. The hyperplastic protrusions ranged in size from one to five millimeters in diameter. No hemmorhage was evident around the proliferated area at this stage. With subsequent colony development and formation of the disc-like holdfast, the epithelium would erode away from the top of the protrusion exposing the *Epistylis* colony.

Where colonies were overlying scales, spines, or bones erosion of these structures would become evident at this stage of colony development. The epidermis would be completely destroyed and the dermis would be hemorrhagic and inflamed. Bacteria were commonly associated with the lesions and rarely fungi were found. Extensive scale erosion was associated with the larger colonies and pit-like inflamed lesions ranging in size up to 2½ centimeters in diameter were observed. Rarely scale regeneration would begin within the center of the lesion. In some cases spines would be completely eroded away. It is though that enzymatic action caused erosion of spines and scales. Mortalities associated with *Epistylis* infestations were infrequent and were probably due to secondary bacterial infections.

OCCURRENCE

"Red-sore" disease due to *Epistylis* has been diagnosed at the Southeastern Cooperative Fish Disease Laboratory from virtually all the Southeastern states. It has been most commonly found in new reservoirs and on species of the family Centrarchidae. Apparently the organic enrichment present in new reservoirs enhances epizootics of *Epistylis*. The disease has been found every month of the year but is most prevalent during the winter and spring months.

CONTROL

Preliminary tests at our laboratory showed that a single treatment with potassium permanganate at a rate of 2 ppm or formalin at a rate of 15 ppm would control *Epistylis*. In a personal communication, H. R. Schmittou reported that field treatments with 15 ppm formalin gave control of *Epistylis* but it would recur within several weeks. Early laboratory tests by Dr. G. K. Krantz, formerly at our laboratory, showed that *Epistylis* had a high sensitivity to salt. Salt concentrations of 0.2 per cent caused complete mortality of *Epistylis* colonies within eight hours. A 2.0 per cent concentration gave complete mortality within five minutes. Based on this information a 2.0 per cent salt bath for five minutes should control *Epistylis*.

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FISH MORTALITIES ASSOCIATED WITH Goezia Sp. (Nematoda: Ascaroidea) in Central Florida [1]

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ABSTRACT

This is the first report of *Goezia sp.* from freshwater fish in North America. Extensive mortalities among striped bass *(Morone saxatilis)* resulted from the damage caused by these nematodes. A possible mode of infection is given and a discussion of the pathology associated with the worms is presented.

INTRODUCTION

The striped bass, *Morone saxatilis* (Walbaum), has in recent years enjoyed a come back in the Southeastern United States due to large scale stocking programs conducted by various states. In most cases the stocking is the basis for an extensive put-grow-and-take fishery. The state of Florida successfully stocked this fish in the summer of 1968 (Ware, 1970). Ware discussed the establishment of the striped bass and the growth of the fish during its first two years. He also discussed some of the difficulties caused by the nematode, *Geozia* sp., which caused large mortalities in the stocks of striped bass put into four Florida lakes. This nematode and its effects on the stocking program that will be discussed in this paper.

CASE HISTORY

The nematode, *Goezia* sp., appears to have been introduced into the striped bass populations in 1968 when, at Richloam State Hatchery, fry were fed ground up frozen marine herring. That this is the site of entry of the worm is evidenced by the facts that (1) the striped bass arrived at Richloam State Hatchery in the sac-fry stage and thus were not feeding; (2) the worm is known to infect marine herring (Yamaguti, 1961); (3) changes in the feeding procedures (namely, eliminating the raw fish meal), in subsequent years have prevented more recent stocks of striped bass from becoming infected at the hatchery, and (4) *Goezia* sp. can be found in adult striped bass on the Richloam Hatchery. These hatchery fish are part of the original stock of striped bass that were fed the raw herring in 1968.

There is precedence in the literature for this type of transmission for *Goezia* sp. Dollfus (1935) reported a very similar situation in which a three-year-old rainbow trout became infected with *Goezia ascaroides* by being fed raw fish meal made from marine fishes. This fish had been hatched and raised in an artificial environment.

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