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Fish Mortality in the Mississippi Catfish Farming Industry in 1988: Causes and Treatments

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ABSTRACT: The 1988 fish mortality summary for the catfish (*Ictalurus punctatus*) industry in Mississippi is presented. In 1988, 2,456 cases were submitted to Mississippi Cooperative Extension Service fish disease laboratories at Belzoni and Stoneville. Bacterial infection caused by *Edwardsiella ictaluri* was the leading cause of catfish mortality. Descriptions and treatments are presented for bacterial, parasitic, viral and other diseases affecting Mississippi farm-raised catfish in 1988.

Key words: Fish mortality investigations, channel catfish, *Ictalurus punctatus*, farm-raised catfish, diseases and treatment, aquaculture.

Mississippi leads the United States in aquacultural acreage with 91,583 acres devoted to channel catfish (*Ictalurus punctatus*) culture (Brunson et al., 1989), with an estimated value of over 2.1 billion dollars. Fish health care service provided by the Mississippi Cooperative Extension Service (MCES; Stoneville, Mississippi 38776, USA and Belzoni, Mississippi 39038, USA) saves the catfish industry several million dollars each year based on an estimated $1,000.00 to $10,000.00 savings to the farmer per diagnosed case. MCES fish disease diagnostic laboratories examined 2,456 catfish disease cases during 1988. Pathogens and other management factors contributing to fish mortalities were recorded and appear in Table 1.

The reported data represent the cases submitted to MCES diagnostic laboratories in 1988 and do not reflect actual disease occurrences in the catfish industry. Some diseases are submitted less frequently than others; for example, winter kill is often not submitted because many farmers feel that there is no effective treatment. All listed etiologies (Table 1) were regarded as contributing to the fish's diseased state and were not always solely responsible for mortalities. Insufficient amount of feed or crowding (Table 1), for example, were diagnosed as major contributors to the fish's diseased state, but were not regarded as the sole cause of mortality. Many cases involved multiple etiologies; this caused the total number of potential causes of mortality in Table 1 to be higher than the total case number.

Enteric septicemia of catfish (ESC) is caused by *Edwardsiella ictaluri* (Hawke, 1979; Hawke et al., 1981; MacMillan, 1985). *Edwardsiella ictaluri* occurred most frequently (48% of all cases) and was the leading contributor to channel catfish mortalities. ESC affects all sizes of channel catfish, but it occurs more often in fingerlings (up to 15 centimeters) (Freund et al., 1990). Epizootics of ESC occur between 22 and 28 C (Francis-Floyd et al., 1987) with peak outbreaks occurring in May, June, September and October. ESC epizootics may cause significant mortalities of 500 to 2,000 fish per day in ponds containing 80,000 to 1,000,000 fish. The pathogenicity of ESC in catfish has been described by Jarboe et al. (1984), Miyazaki and Plumb (1985), and Shotts et al. (1986).

The second major contributor to channel catfish mortality was external *Cytophaga* sp. bacteria. Internal *Aeromonas* sp. complex bacteria ranked third. *Cytophaga* sp. and *Aeromonas* sp. complex are opportunistic and cause mortalities mostly in the Fall and Spring. During hot summer temperatures (about 30 C) the catfish immune system is optimal making it more difficult for bacterial pathogens to thrive. Additionally, during cold winter temperatures (below 14 C) bacterial pathogens are seldom able to affect catfish and are

TABLE 1. Etiologies contributing to mortalities of catfish examined by the Mississippi Cooperative Extension Service in 1988.

Etiology	Number of cases
Bacteria	
Edwardsiella ictaluri	1,169
Cytophaga sp. external	637
Aeromonas sobria	356
Cytophaga sp. systemic	221
Cytophagaceae (other than *Cytophaga* sp.)	154
A. hydrophila	80
Plesiomonas shigelloides	17
Edwardsiella tarda	15
Pseudomonas sp.	12
Aeromonas sp.	6
Enterobacter sp.	4
Acinetobacter sp.	3
Flavobacterium sp.	2
Pseudomonas fluorescens	1
Escherichia coli	1
Citrobacter sp.	1
Shigella sp.	1
Hafnia sp.	1
Vibrio sp.	1
Parasites	
Trichodina sp.	376
Ambiphrya sp.	304
Proliferative gill disease	221
Ichthyobodo sp.	144
Trichophrya sp.	143
Chilodonella sp.	72
Ichthyophthirius multifilis	23
Heteropolaria sp.	12
Monogenea on gills	11
Henneguya sp.	8
Lernaea sp.	4
Apiosoma sp.	3
Gyrodactylus sp.	1
Bodomonas sp.	1
Virus	
Channel catfish virus disease	53
Fungus	
External *Saprolegnia* sp.	356
Water quality	
Ammonia	205
Nitrite	26
Low oxygen stress	7
Dissolved oxygen depletion	4
Insufficient hardness	4
pH elevation	1
Gas bubble disease	1
Nutritional	
Insufficient amount of feed	10
Miscellaneous	
Winter kill	278
Severe anemia	44
Crowding	26
Handling	9
Toxicity suspected	6
"Gas-in-gut" condition	2
Unknown	121
Inadequate sample	127

usually not part of a disease problem. Bacteriostatic antibiotics are incorporated into fish food for controlling bacterial infections. Romet-30 (sulfadimethoxine and ormetoprim, Hoffman-LaRoche Inc., Nutley, New Jersey 07110, USA) and Terramycin (oxytetracycline, Pfizer, Inc., Lee's Summit, Missouri 64081, USA) are the two antibiotics approved by the FDA for use in catfish. Bacteria recovered from sick fish are examined for sensitivity to these antibiotics before treatment recommendations are made. The efficacy of Romet-30 has been demonstrated by Plumb et al. (1987) and Bowser et al. (1986).

Most of the catfish parasites causing significant mortalities are protozoa (Rogers, 1985; MacMillan, 1985). *Trichodina* sp. and *Ambiphyrya* sp. are the most frequently encountered protozoans. However, *Ichthyophthirius multifilis* (Ich) results in highest mortalities on a per case basis. Potassium permanganate, copper sulfate and formalin are the only chemicals approved by FDA to be used on food fish. Copper sulfate is the preferred treatment for *Trichophrya* sp. Due to the complex life cycle of *I. multifilis*, it must be treated on alternate days with any of the above three chemicals until control is achieved.

Channel catfish virus disease (CCV) causes mortalities in fingerlings ≤15 cm in length when pond temperatures are above 20 C (Plumb, 1973, 1978). No treatment exists for this virus, but losses can be reduced significantly under proper management (Crosby and Durborow, 1988).

Saprolegnia sp. is usually associated with winter mortality syndrome (winter kill) (Durborow and Crosby, 1988).

In advanced stages of proliferative gill disease (PGD), gills have a red and white mottled appearance like ground hamburger meat, hence the name hamburger gill disease. This ground hamburger appearance results from swelling of the gill and loss of gill filament structure due to a breakdown of cartilage (Duhamel et al., 1986). The filaments are not well defined structurally and appear mashed together. A myxosporean parasite associated with the cartilage and other parts of the gill has been proposed as the causative agent of PGD (Hedrick et al., 1989; Groff et al., 1989). PGD occurs mostly when water temperatures are between 15 and 20 C, and occurs more often in Spring than in Fall. Although experimentally unproven, pumping water from a pond with good water quality into the affected pond frequently brings losses under control after the water level is increased about 0.3 m (approximately 48 hr of pumping with a re-lift pump into an 8 ha pond).

Ammonia occurs in catfish ponds frequently at levels that are considered adverse or even lethal under laboratory conditions. However, pond-raised catfish become acclimated to high ammonia, and diagnostic laboratories usually implicate ammonia as a disease-causing agent only when the concentration of the un-ionized form of ammonia exceeds 0.4 mg/l (Durborow, 1988). Frequency of high ammonia occurrences can be reduced by feeding at reasonable rates (not exceeding 112 kg/ha). High ammonia concentrations can be reduced slightly by diluting with water from an adjacent pond or well. In addition, fertilizing has been observed to reduce ammonia by increasing phytoplankton growth and activity, resulting in ammonia absorption.

High nitrite concentrations can cause brown blood disease (methemoglobinemia) in channel catfish especially when the chloride to nitrite ion ratio falls below 6:1 (Durborow and Crosby, 1989). Nitrite oxidizes hemoglobin to methemaglobin which prevents transport of oxygen in the blood to organs, thus suffocating fish in spite of adequate dissolved oxygen in the water. Adding salt (NaCl) to ponds elevates chloride concentrations and reduces nitrite ions entering fish through the gills. Catfish farmers in the Mississippi Delta strive to maintain at least a 6 to 1 chloride to nitrite ratio, and frequently keep at least a 20–25 mg/l chloride concentration in their ponds to provide protection against a sudden nitrite increase.

Total hardness of catfish hatchery water should be at least 5 mg/l $CaCO_3$. At hardness levels below 5 mg/l, fry have poorer growth and survival and eggs do not hatch as well, sometimes becoming enlarged and opaque due to insufficient level of calcium in the water. The deficiency can be corrected by adding a calcium chloride ($CaCl_2 \cdot 2H_2O$) solution to hatchery water (Durborow et al., 1989).

Severe anemia listed in Table 1 refers to a condition in channel catfish commonly called "white-lip" or "no-blood" disease. Hematocrits are often as low as 1 to 9% (Plumb et al., 1986). Contaminated feed (Butterworth et al., 1986) and high nitrite in the water (Tucker et al., 1989) have been implicated in causing severe anemia. However, other unknown etiologies probably exist. Treatment recommendations have included maintaining a high chloride to nitrite ratio and/or changing type of feed, although often neither of these recommendations solves the problem.

In catfish with "gas-in-gut" condition, the intestine was distended three to eight times the normal diameter (Table 1). In these cases, no infection or water quality-related problem was identified, so mortalities were attributed to the "gas-in-gut" condition. No treatment has been found.

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