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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

	Etiologies contributing to mortalities of	
catfish exan	nined by the Mississippi Cooperative Ex-	
tension Serv	vice in 1988.	

TABLE 1.	Continued.	
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	Number of	
Etiology	cases	Misce
Bacteria		Wi
Edwardsiella ictaluri	1,169	Sev Cro
Cytophaga sp. external	637	Ha
Aeromonas sobria	356	Тох
Cytophaga sp. systemic	221	"Ga
Cytophagaceae (other than		Un
Cytophaga sp.)	154	Ina
A. hydrophila	80	
Plesiomonas shigelloides	17	
Edwardsiella tarda	15	116110
Pseudomonas sp.	12	usua
Aeromonas sp.	6	teric
Enterobacter sp.	4	fish
Acinetobacter sp.	3	tions
Flavobacterium sp.	2	mete
Pseudomonas fluorescens	1	ley,
Escherichia coli	1	Terr
Citrobacter sp.	1	
Shigella sp.	1	Lee
Hafnia sp.	1	the
Vibrio sp.	1	for ι
Parasites		sick
Trichodina sp.	376	these
Ambiphrya sp.	304	
Proliferative gill disease	221	men
Ichthyobodo sp.	144	met
Trichophrya sp.	143	et al
Chilodonella sp.	72	Μ
Ichthyophthirius multifilis	23	nific
Heteropolaria sp.	12	1985
Monogenea on gills	11	-
Henneguya sp.	8	and
Lernaea sp.	4	quei
Apiosoma sp.	3	er, <i>I</i>
Gyrodactylus sp.	1	in h
Bodomonas sp.	1	Pota
Virus		and
Channel catfish virus disease	50	
Channel catusit virus disease	53	prov
Fungus		Cop
External Saprolegnia sp.	356	for
Water quality		life o
	0.07	on a
Ammonia	205	-
Nitrite	26 7	thre
Low oxygen stress	7	С
Dissolved oxygen depletion	4	caus
Insufficient hardness pH elevation	4	in le
1	1	aboy
Gas bubble disease	1	
Nutritional		men
Insufficient amount of feed	10	redu
		ager

Etiology	Number of cases	
Miscellaneous		
Winter kill	278	
Severe anemia	44	
Crowding	26	
Handling	9	
Toxicity suspected	6	
"Gas-in-gut" condition	2	
Unknown	121	
Inadequate sample	127	

Illy not part of a disease problem. Bacostatic antibiotics are incorporated into food for controlling bacterial infecs. Romet-30 (sulfadimethoxine and oroprim, Hoffman-LaRoche Inc., Nut-New Jersey 07110, USA) and amycin (oxytetracycline, Pfizer, Inc., 's Summit, Missouri 64081, USA) are two antibiotics approved by the FDA use in catfish. Bacteria recovered from fish are examined for sensitivity to e antibiotics before treatment recomdations are made. The efficacy of Ro--30 has been demonstrated by Plumb . (1987) and Bowser et al. (1986).

lost of the catfish parasites causing sigant mortalities are protozoa (Rogers, 5; MacMillan, 1985). Trichodina sp. Ambiphyrya sp. are the most frently encountered protozoans. Howevchthyophthirius multifilis (Ich) results ighest mortalities on a per case basis. ssium permanganate, copper sulfate formalin are the only chemicals aped by FDA to be used on food fish. per sulfate is the preferred treatment Trichophrya sp. Due to the complex cycle of *I. multifilis*, it must be treated lternate days with any of the above e chemicals until control is achieved.

hannel catfish virus disease (CCV) ses mortalities in fingerlings ≤15 cm ength when pond temperatures are ve 20 C (Plumb, 1973, 1978). No treatt exists for this virus, but losses can be uced significantly under proper manment (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 CaCO₃. 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 (CaCl₂·2H₂O) 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 qualityrelated problem was identified, so mortalities were attributed to the "gas-in-gut" condition. No treatment has been found.

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