AQUACULTURE

ENCAPSULATION OF TREMATODE LARVAE METACERCARIAE IS LINKED TO MORTALITY IN CHANNEL CATFISH

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"BOLBOPHORUS DAMNIFICUS CAN CAUSE SIGNIFICANT LOSSES IN CATFISH PRODUCTION. HOWEVER, ONCE THE SOURCE OF INFECTION IS REMOVED AND THE PARASITE IS ENCYSTED, THE IMPACT ON PRODUCTION IS SIGNIFICANTLY REDUCED AND FISH RAPIDLY RECOVER."

David Wise

A recent decline in water acreage within the catfish industry has resulted in increased bird pressure on catfish operations. Land previously dedicated to aquaculture has been drained and dried for cultivation of row crops or devoted to conservation and wetland reserve programs. This has resulted in a significant reduction in available forage and loafing sites for pelicans and other piscivorous birds. As a result, trematodiasis in farm-raised catfish will continue to pose a challenge to catfish production for the foreseeable future.

B. damnificus is a digenetic trematode of the American White Pelican (AWP) that causes significant economic losses in catfish aquaculture in the southeastern United States. Trematode eggs are released into the pond by foraging birds. The eggs hatch and release a motile stage (miracidia) that infects snails, which in turn shed a cercaria stage that infects fish. The parasite encysts within the fish and when infected fish are eaten by an AWP, the encysted parasites (metacercariae) develop into new adults and complete the life cycle.

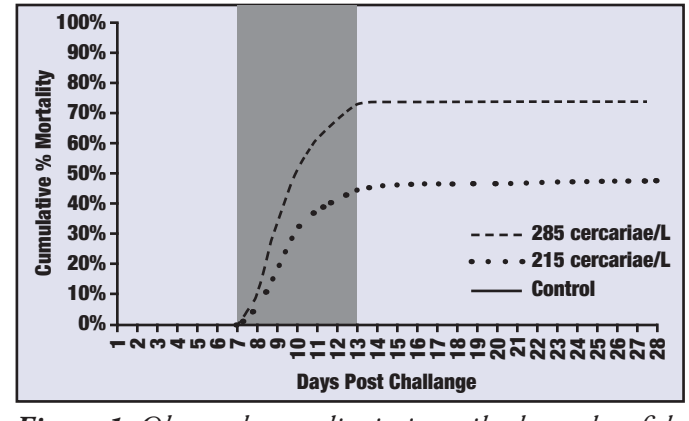

Figure 1. *Observed mortality in juvenile channel catfish (7-13 cm) exposed to Bolbophorus damnificus cercariae. Values represent the cumulative percent mortality for each treatment over the 28 day observational period. Gray area designates period of parasite encapsulation, see figure 3F-3L.*

At present, the pathophysiological effects of the parasite on the fish are poorly understood. Field studies investigating the impacts of *B. damnificus* on catfish production found the parasite had significant deleterious effects on production. However, once the source of infection was removed, weight gain, growth rate, feed consumption, and feed efficiency in parasitized fish were equal to or greater than trematode-free fish. This work suggested that early *B. damnificus* stages of infection severely impact production, however, once the metacercarial cysts are fully developed, these effects are minimal.

To gain a better understanding of the host-parasite

Figure 2. *Moribund juvenile channel catfish eleven days post-challenge (~215 B. damnificus cercariae/L). Fish presented with signs consistent with B. damnificus infection, including distended, fluid filled abdomens, multifocal erythema on the ventral surface, exophthalmia and raised papular lesions associated with underlying metacercariae.*

relationship and effects of *B. damnificus* metacercarial development on the host, three separate infectivity challenges were conducted. In the first two challenges, channel catfish fingerlings were exposed to two different doses of *B. damnificus* cercariae and observed for morbidity and mortality over 28 days. In both studies, mortality first occurred seven days post-challenge and peaked at nine and 10 days post challenge, respectively. Mortality had subsided by 14 days post-challenge, with only sporadic death occurring >15 days post-challenge (Figure 1). All dead and moribund fish presented with lesions consistent with *B. damnificus* infection (Figure 2).

A third challenge was performed to record histological changes associated with parasite development from initial exposure up to the cessation of mortality at 14 days post-challenge. Catfish (2-3 cm) were exposed to 215 cercariae per liter and observed twice daily for morbidity and mortality. Each day, two fish were arbitrarily sampled from each treatment and archived in 10% neutral buffered formalin. Mortality trends were consistent with previous challenges, beginning around day seven, peaking at day 10 and abating by day 13. No control fish died and no metacercariae were observed in any control fish. The observed mortality window coincided histologically with parasite encapsulation and the development of the host derived cyst (Figure 3). This would suggest that mortality and morbidity associated with *B. damnificus* in channel catfish is linked to host pathophys-

iological changes in response to the encapsulation of the metacercariae. However, once the encapsulation process is complete, the fish rapidly recover. While management strategies focused on the eradication of snail populations in catfish ponds are available and can mitigate losses, continued research efforts focusing on the pathophysiological effects of *B. damnificus* on the catfish host, and identification of methods to reduce these effects or promote a more rapid recovery, is warranted.

Figure 3. *Progressive development of Bolbophorus damnificus cercaraie in juvenile channel catfish. A) Day 2 post-infection, bar = 50 µm, B) Day 3 post-infection, bar = 100 µm, C) Day 4 post-infection, 100 µm D) Day 5 post-infection, bar = 100 µm; E) Day 6 post-infection, bar = 100 µm; F) Day 7 post-infection, bar = 100 µm; G) Day 8 post-infection, bar = 200 µm; H) Day 9 post-infection; bar = 200 µm; I) Day 10 post-infection, bar = 200 µm; J) Day 11 post-infection; bar = 200 µm, K) Day 12 post-infection; bar = 200 µm; L) Day 13 post-infection; bar = 200 µm*