EFFECTS OF CONFINEMENT STRESS AND CORTISOL ON THE SUSCEPTIBILITY OF CHANNEL CATFISH TO INFECTION BY *ICHTHYOPTHERIUS MULTIFILIIS* AND CHANNEL CATFISH VIRUS

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Many diseases of fish are referred to as opportunistic to indicate that the pathology caused by the infectious agent usually occurs after the protective mechanisms of the fish have been suppressed. Immunosupression and disease outbreaks often follow an episode of stress, which can be induced by a variety of conditions frequently encountered in aquaculture. One of the primary stress responses is the secretion of cortisol. Cortisol has many biological activities including the induction of gluconeogenesis and the suppression of the inflammatory response. The role of cortisol in increasing disease susceptibility is difficult to assess when fish are stressed by physical means (such as handling) due to the other physiological responses which also occur. The objective of the present studies was to determine if confinement stress and the chronic administration of cortisol resulted in similar changes in susceptibility to the

protozoan parasite *Ichthyoptherius multifiliis* (ich) and to channel catfish virus (CCV).

Handling Stress- Yearling channel catfish were exposed for 15 minutes to 2,500 theronts/l of *I. multifiliis* after a low-water confinement stress of 0, 2 or 6 hours. Six days after exposure to ich, the trophont density on the skin was determined and reported as trophonts per cm^2. A second group of fish was used to determine the physiological stress response of fish exposed to the low-water confinement stress. Blood samples were taken before, 2 and 6 hours after confinement. Plasma cortisol, glucose and chloride concentrations were measured. Channel catfish fry about 4 weeks old were challenged with 0, $3.3X10^4$, $3.3X10^5$ or $3.3X10^6$ plaque forming units of CCV in 400 ml water after 0, 2 or 6 hours of confinement stress. Mortality was determined during the 14 days after exposure.

Plasma cortisol and glucose, but not chloride, were significantly higher after 2 and 6 hours of confinement. This is a typical response, which has been documented several times previously. Fish exposed to 6 hours of stress prior to ich exposure had a significantly higher infection rate than control fish. Fish stressed for 2 hours had an intermediate infection rate not significantly different from the non-stressed fish or those stressed for 6 hours. Mortality after CCV exposure was related to the dose of the virus but was not affected by confinement stress.

Dietary Cortisol- Yearling channel catfish were fed for 9 days at 1% of the body weight per day with feed containing 200 mg cortisol per kg of feed. Plasma samples were taken immediately before the last feeding and at 2, 4, 6 and 12 hours after the last feeding and analyzed for cortisol.

Yearling channel catfish were offered feed containing 0, 100 or 200 mg cortisol per kg feed for 13 days. An initial blood sample was taken 24 hours after the last feeding. Fish were then exposed to a low-water confinement as above for 0, 2 or 6 hours and blood samples were taken and analyzed as above. A second group of fish, fed the same diets was exposed to ich theronts and the infection determined as above. Channel catfish fry offered cortisol-containing feed were exposed to CCV and mortality evaluated as above.

Plasma cortisol concentrations of cortisol-fed unstressed fish were significantly higher than controls 2, 4, and 6 hours after feeding. The highest concentration occurred six hours after feeding. When fish were fed cortisol and exposed to a

confinement stress 24 hours after the last feeding, fish fed the control diet displayed a typical cortisol stress response. Stressed fish had cortisol concentrations about four times that of unstressed fish. However, fish fed 100 or 200 mg cortisol per kg feed displayed no increase due to confinement. The hepatosomatic indices of fish fed cortisol were significantly smaller than controls under all experimental conditions.

Conclusion

These data suggest that confinement-induced stress and orally administered cortisol increase the susceptibility of channel catfish to infection by ich, but not to mortality due to CCV. The defense mechanisms which protect fish from parasite infections may differ from defense mechanisms against virus infection and cortisol may differentially affect these defense mechanisms. The fish used in these studies had no previous history of exposure to ich or CCV, therefore, likely both innate and acquired immunity systems were involved. Several papers have shown both handling stress and exogenous cortisol increase the sensitivity of fish to bacterial infections. Plasma cortisol concentrations from cortisol-fed fish were highly variable and likely reflect different feeding success among the fish. Cortisol fed once daily likely results in a large single spike in the blood each day which clears the blood by the next feeding. Cortisol-fed fish were not able to mount a confinement-stress-induced increase in cortisol likely due to a negative-feedback-induced suppression of ACTH or CRF.