MARINE OPTION PROGRAM PROJECT

"PRAWN HISTOLOGY"

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A CASE OF SPONTANEOUS MUSCLE NECROSIS IN CULTURED FRESH WATER PRAWNS, MACROBRACIUM ROSENBERGII

INTRODUCTION

The commercial culture of fresh water prawns Macrobracium rosenbergii is a rapidly developing aquatic industry. Although culture methods are primitive and basic knowledge of the nutritional requirements, anatomy and physiology of these crustaceans are not well elucidated, non-intensive production systems (dirt ponds) presently yield 1500 to 2500 lbs/acre/year of prawns. In Hawaii prawn aquaculture is projected to become a $300 million per year industry for the state in the next 20 years, according to the Aquaculture Development Program.( ).

Attendant with the commercial culture of fresh water prawns is the emergence of several diseases. The purpose of this report is to present a case of Spontaneous Muscle Necrosis (SMN) and to review the present knowledge of this disease affecting crustaceans.

LITERATURE REVIEW

SMN (syn. muscle opacity, muscle necrosis) is a non-infectious disease reported from marine shrimp (panaeids spp) (Rigdon 70; Venkataramiah 70, 71; Lightner 73; Sindermann 77; Lakshmi 78) and fresh water prawns (Macrobracium rosenbergii) (Fujimura 72; Sanidifer 75; Sindermann 77) held under less than optimal culture conditions. The etiology and pathogenesis of the disease are undefined, but the condition is usually associated with adverse environmental conditions such as anoxia (Rigdon 70; Venkataramiah 71; Sindermann 77;

Lakshmi 78), sub-or supra-optimal salinity (Venkataramiah 70, 71; Sanidifer 75; Sindermann 77; Lakshmi 78), hyper-or hypotermic temperature (Rigdon 70; Venkataramiah 71), hyperactivity (Lakshmi 78), overcrowding (Rigdon 70; Venkataramiah 70, 71; Lakshmi 78), brief exposure to air and handling (Venkataramiah 71) amd by narcotizing with quinaldrine (Johnson 74). The incidence of SMN in captive shrimp populations has been reported to be as high as 60 % (Lakshmi 78).

The gross lesions include focal to confluent opaqueness of the striated musculature usually involving the cardial 1/3 of the abdomen. Microscopically, these "white areas" are composed of necrotic muscle cells with little to no inflamatory cell infiltration into the areas (Rigdon 70; Lakshmi 78). Muscle lesions of several days or longer in duration are typified by proliferation of sarcoleminal nuclei indicating regeneration of the cells in the necrotic foci (Rigdon 70).

Electron microscopy findings of spontaneous muscle necrosis lesions reported by Lakshmi in 1978 include disorganized myofibrils, loss of sarcomeres, peripheral mitochondria and cytoplasm, disintegration of inter-fibrillar mitochondria, and the swelling of the sarcoplasmic reticulum.

Bacteria, parasites or virus-like particles have not been observed in the necrotic foci and are not reported to be a cause of the observed muscle lesions (Rigdon 70; Venkataramiah 70; Sindermann 77; Lakshmi 78).

Transmission of this condition by one shrimp to another by feeding diseased shrimp tissue has been unsuccesful (Venkataramiah 70). Affected shrimp have been observed to recover but when musculature involvement is extensive the shrimp may die in 24 - 48 hours (Rigdon 70; Venkataramiah 70, 71; Sindermann 77; Lakshmi 78).

Other disease processed have been reported to result in muscle opacity in panaeid shrimps. Bacterial septisemias (Vibrio, Aeromonos, Pseudomonas) and microsporidians infections also produce a gross lesion of muscle opaqueness in shrimp (Sindermann 77). These diseases can be differentiated from SMN on the basis of microscopic examination of affected tissue.

CASE HISTORY

One hundred fifty Macrobracium rosenbergii Sarawak strain F_1 generation were stocked outdoors in a plastic lined swimming pool (3920 liters) on August 21, 1978 and held for eight months. During this time the water temperature ranged from 18° C to 22° C and the prawns were occassionally fed prawn pellets. Water was added to the pool to correct for losses that occured through evaporation. On April 9, 1979 the tank was drained and the entire population of 140 prawns were transferred to an indoor holding tank (1500 liters). During the initial 48 hours following transfer a 40 % mortality was observed in this population. Focal to confluent areas of abdominal muscle opaqueness were noted after 48 hours but surviving prawns were affected with focal areas of muscle opacity. Specimens were obtained for gross post-mortem and histopathological examination.

POST MORTEM EXAMINATION AND HISTOPATHOLOGY

The prawns examined ranged from 2 - 4 cm in orbital length and were representative of the size of the prawns in this population. Gross lesions were limited to focal areas of muscle opacity in the middle to distal segments of the abdominal flexor muscles. Gills, hepatopancreas, heart and

exoskeleton were grossly normal.

Representative samples of the organ tissue from the prawns were fixed in 10 % buffered formalin and processed in a routine fashion for histopathological examination. Sections of abdominal muscle, hepatopancreas, heart and gills were stained with hematoxylin-eosin, Gridley, Giemsa, Kinyon and gram-Weigert methods.

Microscopically, the muscles of the affected prawns had focal areas of necrosis characterized by marked proliferation of sarcolemnal nuclei, increased connective tissue elements, occasional nuclear pynosis and kasyorrhexis, and sporadic infiltration of monomuclear hemocytes. Bacteria, fungi, protozoans, or metazoan parasites were not observed in the stained impression smears of any organ tissue. Lesions were not observed in section of gills, hepatopancreas, heart and green gland from the prawns.

DISCUSSION

A tentative diagnosis of SMN in fresh water prawns can be made on the basis of a history of stressful conditions, gross lesions and stained impression smears of affected tissue. The differential diagnosis in this case included SMN and bacterial septisemia. A dianosis of SMN can be confirmed by histopathology.

All studies regarding SMN in paneads and macrobracium indicate the non-infectious etiology of this condition. The findings in this case support this hypothesis as no biological agents were observed in the muscle lesions.

The methods by which the prawns were reared indicate several possible stressors which predisposed this population to the occurence of SMN and the observed high mortality. Specifically, this population was held at sub-

optimal growing temperatures and fed irregularly. This resulted in marked stunting of prawns. Furthermore, the capture and transfer methods subjected these prawns to a period of hyperactivity and created probable anoxic conditions that resulted in a high incidence of SMN and mortality.

The high mortality in this population following transfer could have been prevented through rearing of prawns at optimal environmental temperature providing a constant supply of feed and through careful handling and transferring of prawns in well-oxygenated water.

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