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OCCURRENCE OF ACUTE FISH MORTALITY IN BATTICALOA LAGOON, EASTERN SRI LANKA - A DIAGNOSTIC STUDY

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INTRODUCTION

The Batticaloa lagoon is the fourth largest lagoon among lagoons found island wide. The occupying area is about 168 square kilometer and extending from Thraineelawannai in the south and Eravur to the North, and opening into the sea at Batticaloa and Kallar . The lagoon itself is 56km long and nearly 13 fathoms deep in some parts. This wild system is itself sharing with other land based farming systems. It also receives land run off from west and south banks of lagoon from heavily populated communities. There are 80% of fish species in this lagoon are both residence and migratory species.

Dramatic die-offs of wild fish have often been associated with abrupt environmental changes, although outbreaks of infectious diseases have also been reported (Sindermann,1963). The lagoon water becomes stagnant water body since march by sand bar formation across the lagoon outlet. Under this situation, the lagoon and its surrounding is sometimes experienced heavy shower in late March .This phenomenon would have resulted subsequent flow of flood water runoff from the hill side areas especially areas on the western side of the lagoon which predominantly agricultural lands and cattle rearing entering into the lagoon (ADB report, 2001).

Further there are eight streams and rivers opening into the Batticaloa lagoon In addition untreated effluents from shrimp farms , small industries such as rice mill, and butchers' shops

around the lagoon also enters into the lagoon. These nutrients rich water inputs into the lagoon creates conditions for algal blooms. The occurrence of algal bloom could be an annual event which is confirmed by the fishermen and villagers.

The main reason for the uncertainty regarding the role of disease in fish population is that the outcome of interaction between a fish and a disease causing organism is under the influence of a large variety of ecological and environmental factors. The variability leads to often unpredictable outcomes of the fish-agent-environment relationships. Disease can also act as a density-dependent factor that regulates and restricts the number and type of organisms that can thrive in a given environment (Croze,1981).

Efforts to understand wild fish diseases are further complicated by the lack of knowledge of the pathophysiology of many wild species (Kent and Fournie, 1993). A variety of studies have shown that sick fish tend not to keep up with their cohorts due to behavioural or physiological limitations (Stephen and Ribble, 1994)

MATERIALS AND METHODS

Fish

Wild fish from four different landing places between Plliyaraddy and Kalady bridge in the northern half of Batticaloa lagoon were collected biweekly from November 2002 to May 2002 and were subjected to gross pathological and histopathological examination. Clinical examination was also carried out at the field. The wild fish included naïve and moribund fish.

Histopathology:

Tissues particularly, gills, liver, kidney, muscle and heart were dissected out from the fish and fixed in 10 % neutral buffered formalin for one night. After fixation and dehydration of the specimens, tissues were embedded in liquid paraffin. Serial sections were cut longitudinally and sagitally by a sliding

microtome, (Hn-40, Germany and stained with hematoxlin and eosin. Giemsa stain was also used to stain some of the sections. After dehydration with series of ethanol and xylene, the tissues were embedded in canada bolsam and examined by light microscopy.. In the histological examination, each tissue sample was examined for the presence of histopathological lesions

Measurement of Ecological parameters:

Salinity (ppt) of the water where the fish were collected was measured by a refractosalinometer (S/Mill, Japan). Then the graph was plotted against the time of collection. Dissolved oxygen concentration was measured using the titration method and temperature was also noted at the time of collection.

RESULTS

Clinical and gross pathological changes in the affected catfish:

Among the death of fish , catfish (*Arius bilinatus*)was in significant number (75%) . All death fish were seen either in the shore or were floating in water coloum. However some of the severely affected catfish were moving along shore region. Fig I shows the death fish on the shore in which it can be seen other fishes like *Tilabia niloticus, Etroplus sp,* and *Mugil sp.* were common.

The clinical symptoms of catfish were the slow movement on the shore, and had orientation difficulties. Fig 3.2 &3.3 showed the gross pathological symptoms of affected fish. The external hemorrhage which was seen at the base of anus and caudal fin and the petechial hemorrhage on the surface of belly (Fig3.2). Swollen belly and protruded anus were common to all affected cat fish as in the fig 3.3. Internal gross pathological symptoms showed the extensive hemorrhage in the gill and body cavity and inflamed kidney as shown in fig3.4.

Histopathological changes in white cat fish (*Arius bilinatus*)

Gill kidney, heart and liver were subjected to histopathological study Among all, kidney was the mostly affected organ in white cat fish. In which it could be seen extensive necrosis and melanin infiltration as show in the figure 3.5. Histological section of gill showed the extensive necrosis and hemorrhage as in the figure 3.8. Also the fusion of secondary lamella were common in all affected and moribund white catfish. Liver cells showed extensive lipid degeneration and vacuolated hyaline cells throughout the sections observed((Fig 3.7).

Ecological parameters:

The variation of salinity in north bank of Batticaloa lagoon was observed between November 2001 to May 2002. Salinity was 25±3ppt in all four sampling places from November to December. It was dropped to 15±1ppt.

from January to mid march 2002. A sudden drop of salinity was observed in later part of march 2002 as shown in the fig 3.9. A mass number of fish death occurred when the salinity was 1±1ppt in the Urani and Sathrukunddan sampling places. The fish death was continued until mid may 2002. Dissolved oxygen concentration was range from $1.6x10^{-6}$ to $16x10^{-6}$ in Urani and Sathrukundan (fig 3.10). When the death occurred in lagoon, the north bank of lagoon was entirely separated by sand bar formation at the sea mouth. There was no water mixing occurred in the lagoon.

DISCUSSION

From the observations made from the field, particularly clinical, gross pathological and histopathological symptoms of cat fish, the ecological parameters of lagoon water and the water appearance at the time of fish mortality occurred , it is suggested that the mortality of the fish could have been due to various scenarios.

It is suggested that the combination of climate change and environmental degradation has created ideal conditions for the emergence, resurgence and spread of infectious diseases in fish. Not all fish are the same when it comes to disease. The ability to ward off infectious diseases may differ between fish species as well as between strains of the same species. In the six months of study, cat fish population was the most affected fish species. The natural history, behavioural and geographical distribution would also seem to he considered in a diagnostic study of fish death. It is therefore not reasonable to assume that all fish in a given area will face the same disease risk.

From the observation, It can be stated that with the condition of closed lagoon outlet and inflow of freshwater in large amount has created drastic decline of salinity levels (1±1ppt) The decline of salinity level to drastic levels in the lagoon would have been exerted severe stress to the brackish animals (Fig 3.9). Since the brackish animal would easily adapt the changes of salinity in the water, only catfish was subjected to mass mortality.

It was observed that significant number (80%) of white catfish of the total dead fish observed. The clinical and pathological study reveal that all the symptoms observed in the moribund white catfish were most common for viral diseases (Fijan, 1999). Commonly, white catfish are most susceptible for viral infection during warm weather as reported by Plumb 1971a. and outbreaks are more frequent in years with high water temperatures. As shown in fig 3.2. the external hemorrhage at base of anal fin and caudal fin is reported for common for viral infection(Wolf et al., 1972) although the fishers believe that might be caused by physical injury. Internal hemorrhage and inflamed kidney shows in fig 3.3, suggesting that this might be caused either by viral toxin or other lethally detrimental toxins. The same symptoms has been reported in Spring Viraemia of carp and channel cat fish virus (Woo & Bruno, 1999) . Among the organs affected , kidney which is the first and most severely affected organ as shown in figure 3.4,extensive necrosis of

haemopoetic tissue, collapsed renal tubules and melanin infiltration are common for viral infected fish (Fijan, 1999).

Liver pathology reveals extensive vacuolation and fatty degeneration are common symptoms not only for viral toxin but also for parasitic and bacterial toxins (fig3.6). Heart muscle also shows the pathological changes which are common to all pathogenic and toxic infection (fig 3.7). Gill of white catfish were also severely affected (fig 3,8), suggesting that lamellar fusion and necrosis are common to wide range of pathogenic and toxic infection (Robert, 1978). As reported in ADB report, Batticaloa lagoon is the highly nutritious lagoon and wide range of factors seem to be involved in the mass mortality of white cat fish. From the pathological observation so far, the mortality of white catfish might be caused by a toxin which either may be produced from viral pathogen or any other lethal toxin. It is impossible to confirm the causative agent would be virus at the time of death occurred due to lack of facilities for viral diagnosis. Attempts to study diseases in wild fish are fraught with methodological difficulties, perhaps the most significant being our inability to ready or regularly detect sick fish in the wild. Methods used to capture wild fish are often based the fishing methods. Further detail biological and ecological study of north bank of lagoon is necessary to understand the cause of this acute mass mortality of white cat fish.

Under one set of conditions, a microorganism may be harmless to a group of fish, whereas in other situations, the organism may result in mass mortality. The detection of an agent theoretically capable of causing disease or the detection of a particular symptom in some fish rarely provide the basis from which to reliably predict the nature of population effects that will occur.

Therefore, non-random samples of wild populations have the potential of seeing only a portion of the spectrum of disease or infections that may affect a wild population. The spectrum of disease that is observed might over or under -estimate the extent or impact of disease in wild stocks unless it is confirmed with further detail study in all aspects that may cause mass fish mortality .

REFERENCES

1. ADB TA NO 3479-SRI, Eastern Province Coastal Com munity Development project, Final inception report, ARCADIS Euro consult, June 2001.
2. Fijan.N. (1999). Spring Viraemia of Carp and other Viral Diseases and Agents of Warm water fish, (In: Fish Diseases and Disorders, Vol.3. Viral, Bacterial and Fungal infections, eds P.T.K Woo and D.W.Bruno), *CAB international,* pp-177-200)
3. Fijan, N. (1975). Advances in knowledge of viral diseases of fish.
4. Page. L.A.(ed.), *Wildlife* Diseases, Plenum Press, New York,pp, 127- 133.4. 4. .Plumb.J.A. (1971a). *Channel Catfish Virus Research* at Auburn University Progress Report series, No.95, Agricultural Experimental Station, Aubyrn University, Alabama.
5. Plumb.J.A.. (19971b). Tissue distribution of channel catfish virus, *Journal of Wildlife Diseases,* 7, 213-216.
6. Roberts,R.J.(1978). Fish Pathology: Macmillan Publishing Co .Inc: New York, USA.
7. Wolf, K., Herman. R. L and Carlson, C. P. (1972). Fish viruses: histopathologic changes associated with experimental channel cat fish virus disease: *Journal of the Fisheries Research Board of Canada* 29, 149-150.
8. Wolf, K. and Darlington, R.W.(1971). Channel catfish virus: a new herpesvirus of ictalurid fish, *Journal of virology* 8, 525-533.
9. Woo,P.T.K. and Bruno,D.W. (1999). Viral, Bacterial and Fugal infections, (In fish Diseases and Disorders, Vol.3. CAB International

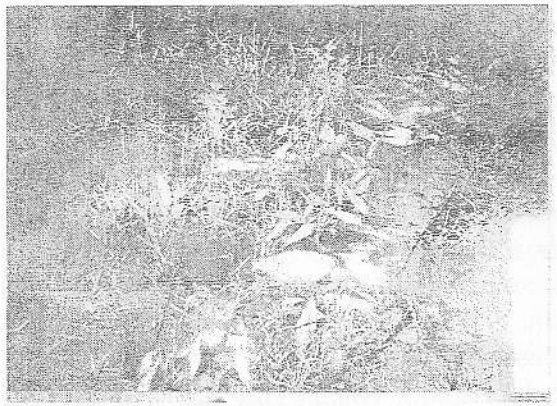

Fig 3.1. The mass number of fish death along the shore line of the lagoon. Of these, cat fish (Arius bilinatus) death are significantly high (75%) (H&E)

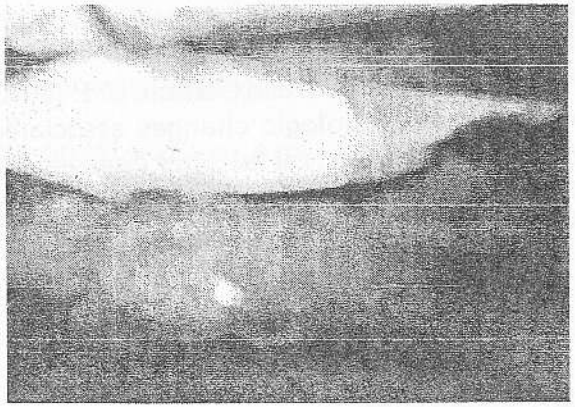

Fig3.2. A catfish with swollen stomach and protruded anus (x1)

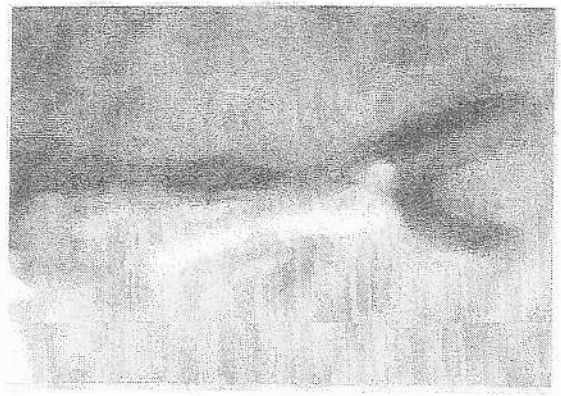

Fig3.3. Moribund catfish with the extensive hemorrage in the caudal fin and anal fin (x1).

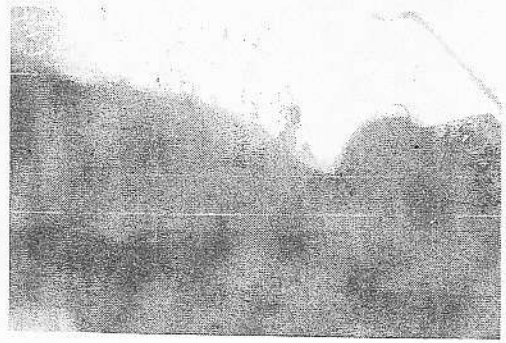

Fig 3.4. The inflamed kidney of the catfish (Arius bilinatus(x1)

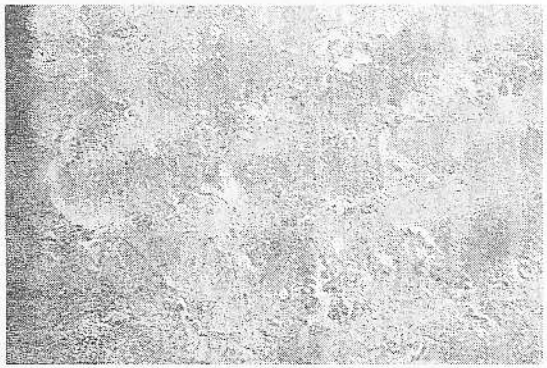

Fig 3.5. The extensive necrosis of heamopoetic cells in kidney of catfish (Arius bilinatus) (x100)

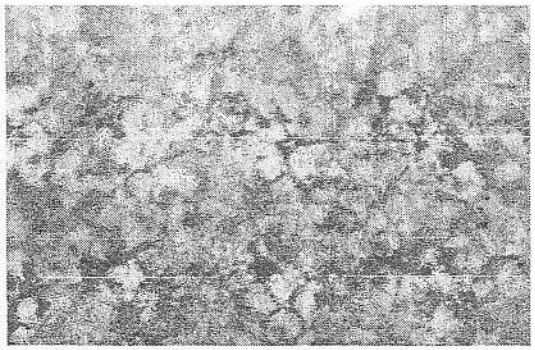

Fig.3.6. The extensive vacuolarised hepatic cells with fat droplets . Focal necrotic cells also are seen in the affected liver of moribund cat fish. (x100)

Fig 3.7. Cellular infiltration, Focal necrosis in heart muscle of cat fish (x100)

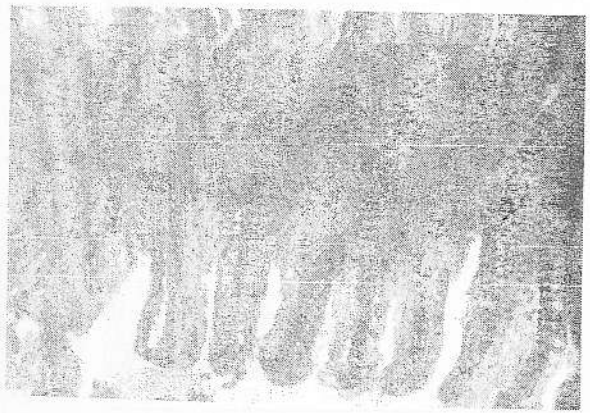

Fig 3.8. Lamellar fusion and extensive necrosis and hemorrage in gill lamella of cat fish (x100)

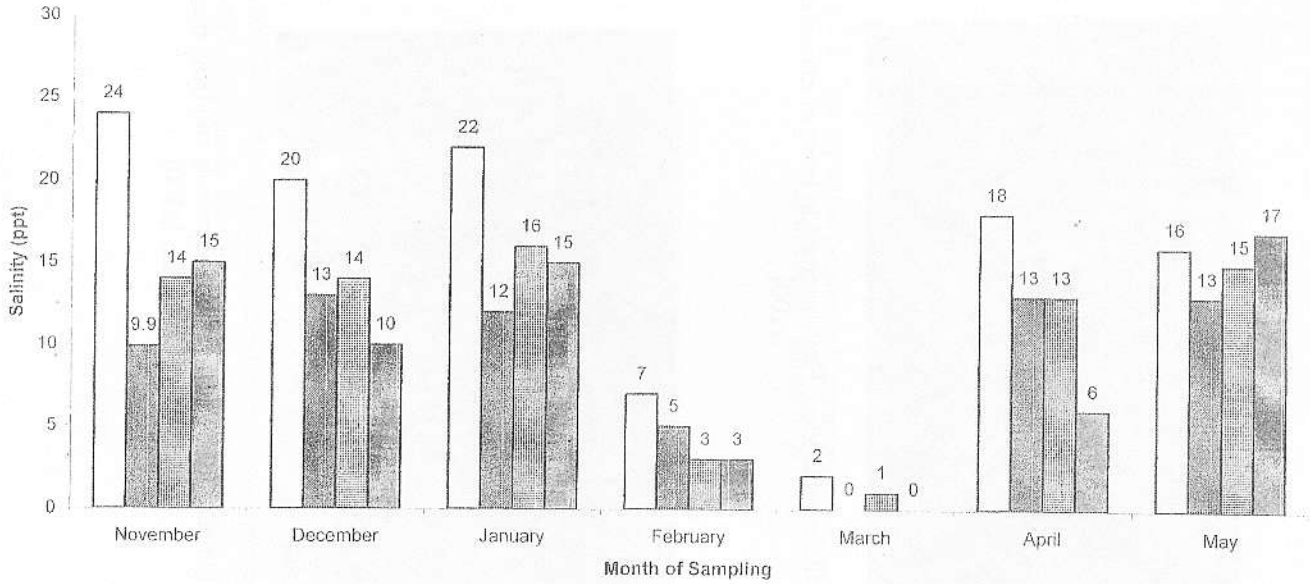

Fig 3.9: Salinity (ppt) Variation in Different Sampling Places in Batticaloa Lagoon

Station	November	December	January	February	March	April
Urani	16.2×10^{-6}	11.6×10^{-6}	4×10^{-6}	5×10^{-6}	8.6×10^{-6}	1.6×10^{-6}
Bridge	0.2×10^{-6}	12×10^{-6}	5.2×10^{-6}	7.5×10^{-6}	4×10^{-6}	4×10^{-6}
Barmouth	17×10^{-6}	17.2×10^{-6}	8.6×10^{-6}	10.1×10^{-6}	4×10^{-6}	6×10^{-6}
Koddamunai	12.4×10^{-6}	10.6×10^{-6}	6×10^{-6}	7×10^{-6}	1.6×10^{-6}	6×10^{-6}

Fig 3.10: Dissolved Oxygen Concentration (ppm) in four different sampling places in Batticaloa Lagoon