- Several fresh water dips are required to remove other stages of *Amyloodinium*.
- The most common treatment is the use of copper in water.
- Water that has free copper at the level of 0.2 mg/l is used in treating fish that are affected by Amyloodinium.
- Formalin dip 200ppm for 15-20 min daily until the fish is free of parasites
- Transfer the fish to a separate disinfected tank.

ii) Parasitic copepods infestation

Mainly infests the broodstock and subadults kept for long time in the cement tank for breeding. The failure of water filtration system, stagnated water and high salinity are predisposing factors in which the *Parapetalus occidentalis* are multiply and infect the host.

Cause: Maxillopoda | Siphonostomatoida | Caligidae

Subclass of copepod species of Caliginae

Species : *Parapetalus occidentalis* Wilson, 1908

Clinical signs

- Rubbing, darkened body colour, extensive abrasions on the body surface and altered swimming behavior.
- Loss of appetite was noted in debilitated fishes.
- Acute mortality due to hypoxia and anoxia

Lesions

- Heavily infested fish are emaciated and suffer from severe respiratory disturbance accompanied by massive mucous secretions.
- Parasites from cobia skin - Fig. 7 & 8

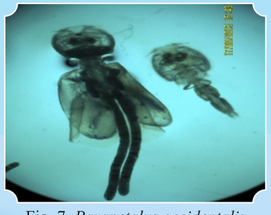

Fig. 7. *Parapetalus occidentalis* Wilson, 1908 female and male

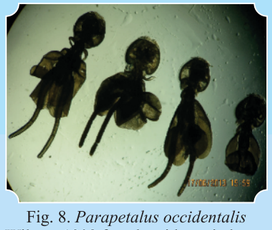

Fig. 8. *Parapetalus occidentalis* Wilson, 1908 female with genital core

Treatment

- Formalin dip 200ppm for 5-15 min daily or until the parasite control.
- Fresh water dipping, reverese osmosis (RO) water dipping (2-5min), and reduce the salinity to detach the parasite.
- Ulcerated skin treated with Povidone Iodine solution dip for 1-2 min as antiseptic to enhance the healing process and detach the parasite.
- Keep the fishes in low salinity (5ppt)
- Immersion for 5-10 mins in irritants like Euginol, Formalin (200ppm), Hydrogen Peroxide (3%) and Potassium Permanganate facilitates detachment of *Parapetalus occidentalis*

D. SKELETAL DEFORMITIES

- Skeletal deformities are commonly encountered in both cultured and wild fish, with a higher frequency in hatchery populations. Undeveloped maxillary process, pug headed, mandibular prognathism (Fig. 9), and scoliosis (Fig. 10) are some of the anomalies reported in cultured cobia fingerlings. Such anomalies can cause economic loss to the fish farmers.
- Evidence suggest that such abnormalities are induced during the embryonic and post-embryonic periods and it has been proposed that these conditions have a multifactorial etiology which include genetic, nutritional and environmental factors.
- Anomalies due to nutritional factors may be prevented by supplementing mineral mixtures and probiotics along with the feed to the brood stock and fingerlings.

Fig. 9. Pug head-mandibular prognathism

Fig. 10. Scoliosis1

Treatment

- Selective culling may be practiced in the hatchery to avoid the economic loss.

E. NONSPECIFIC MORTALITY

- Mortality occurs during the transfer of fingerlings from hatchery to cage once after completion of nursery phase. Supplementing immunostimulant and mineral mixture to improves the immune status of the fingerlings, reducing the mortality.
- Mortality may occurs due to higher density (Recommended 750 to 800/6m dia cage) in cages
- Less depth with more transparent light and increased temperature may leads to acquired vertebral abnormalities. A depth of 4-5m depth is advisable.
- Mortality may occur due to feeding of contaminated or spoiled trash fish. Hence, feed with trash fish as fresh as possible in the cages.

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Diseases of Cobia in Hatchery and Grow-out Cages

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Introduction

Mariculture of marine finfish has been growing rapidly on a global basis especially with the development and expansion of sea cage farming. Cobia farming would be presumably become an emerging aquaculture industry in India near future due to its high market demand, amenable for breeding and farming in captivity. However, the industry faces various threats including viral, bacterial and parasitic diseases, when it is cultured in hatchery and in open seawater systems. Sustainable aquaculture production can only occur when fish are healthy and free from diseases.

- Fish cultured in floating cages or in ponds become particularly susceptible to diseases when various environmental parameters such as temperature, salinity, dissolved oxygen and suspended particles fluctuate suddenly or widely, or by rough handling operations.

In this brochure, common diseases of cobia their symptoms, diagnosis, therapeutic and prophylactic methods are described below;

DISEASES IN CAGE CULTURE

A. Bacterial diseases

Bacterial diseases are mostly caused by opportunistic bacteria due to sudden environmental changes. Among the bacterial pathogens, bacteria belonging to the genus, Vibrios are primarily responsible for mortality and economic loss in the cobia farming. In recent days infection caused by *Photobacterium* spp. and *Pseudomonad* spp. also gains prominence especially in grow-out farming.

1) Vibriosis

Vibriosis commonly affecting all stages of the fish cultured in the cage system. *Vibriosis*, a disease caused by numerous species of *Vibrio*.

Cause: *Vibrio alginolyticus, Vibrio harveyi* and *V. parahaemolyticus*

Clinical signs

- Loss of appetite
- Surfacing and abnormal swimming behavior
- Corneal opacity
- Exoptholmia
- Acute mortality

Lesions

- Haemorrhage on the external surface of fish
- Haemorrhagic gastritis & Ascites (Fig. 1)
- Haemorragic and fibrinous pericarditis (Fig. 2)
- Pale gills with profuse mucous secretion

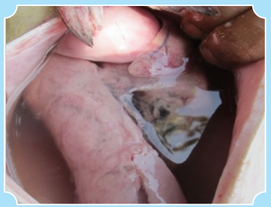

Fig. 1. Distended abdomen with white to serosanguinous fluid

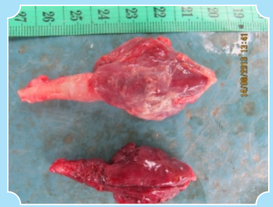

Fig.2. Haemorrhagic and sticky chalky white fibrinous adhesion in pericardium and myocardium

2) Photobacteriosis, or pseudotuberculosis

Cause: *Photobacterium damselae* sub sp. *piscicida.*

It is the causative agent of the fish disease photobacteriosis, also known as pasteurellosis or pseudotuberculosis. The mortality of cobia fingerlings with clinical signs lasted for 2 weeks with total mortality of 40%.

Clinical signs

- Loss of appetite
- Surfacing and abdominal swelling
- Acute mortality

Lesions

- Abdominal cavity filled with yellow gelatinous fluid
- Liver and kidney haemorrhagic

3) Septicaemia due to *Pseudomonas aeruginosa*

The *Pseudomonas* spp are common and emerging bacterial pathogen in fishes, this species is highly flexible opportunistic pathogen, capable of existing in a variety of environment, including aquaculture environment. The etiological agents commonly found are *P. fluorescens*, *P. diminuta*, *P. aeruginosa* and *P. putida* with different degrees of virulence, which are responsible for high mortality and disease outbreaks in various fish species including cobia.

Clinical signs

- Loss of appetite
- Surfacing and circling movement
- Acute mass mortality

Lesions

- The external skin surface showed severe haemorrhage and redness in the lower abdomen
- Abdominal cavity revealed presence of clear transparent peritoneal fluid 5 to 8 ml
- Liver focal congestion at the caudal lobe

Treatment and control

- Approved antibiotics, after antibiotic sensitivity testing based on farming system
- Supplement the immuno-modulator and mineral mixture along with the feed for Fingerlings and within the empty capsules for subadults and broodstock.
- Reduce stress and overcrowding.
- Reduce feeding frequency for few days
- Replace the fouling net.
- Relocate the cages once in three months and during an outbreak in order to control the cross infection and to facilitate better water exchange.
- Regularly monitor the water quality parameters at the cage site or in the ponds.Monitor the vibrio load of the water and sediment periodically

B. Viral Nervous Necrosis (VNN)

Cause : *Betanodavirus*

Clinical signs

Abnormal swimming behaviour, change in skin pigmentation.

Lesions

- Brain (Fig. 3) and Liver (Fig. 4) congestion
- Brain - vacuolation (“holes” or “swiss cheese”) appearance in histological sections

Control and prevention

- Procuring and culturing specific pathogen free (SPF) seeds from the certified hatchery
- Supplement of immunostimulants to enhance the fish immunity.
- Avoid stresses to the cultured fish

Fig. 3. Brain congestion

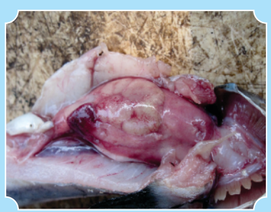

Fig. 4. Liver sever congestion at caudal lobe

DISEASES IN HATCHERY

Commonly affecting the nursery phase or fingerlings stages those reared in the hatchery system.

C. PARASITIC DISEASES

i) Disease : ***Amyloodiniosis*** or Marine velvet disease.

Cause : The dinoflagellate *Amyloodinium ocellatum*

Life cycle

The feeding stage of *A. ocellatum* is known as trophont. The trophont attaches to the fish by anchor-like roots or rhizoids and infests the gills, fins and body of the host fish .It feeds on the epithelial tissues of the skin and gills for several days before detaching from the host. The encysted infective stage is known as tomont. That subdivides internally to for more than 200 numbers of infective dinospores. Trophont and tomonts resist against the treatment and disinfectants. The excysted dinospores are freely swimming in the water which will attach the host again and develop as trophont.

Clinical signs

- Loss of appetite
- Abnormal swimming and rubbing the body against the tank surface
- Sluggish swimming and surfacing
- Acute mortality

Lesions

- Darkening of the skin and fin
- No specific gross lesions
- Swabs from gill surface shows trophonts (Fig. 5 & 6)

Fig. 5. Trophonts attached to the secondary gill lamellae

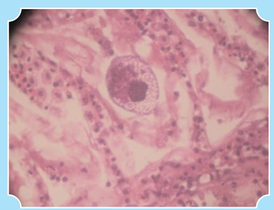

Fig. 6. Trophonts in the gill

Treatment and control

- Only the free-swimming dinoflagellate form (dinospore) is susceptible to treatment. The encysted form is not susceptible to any treatment.
- Fresh water dip for 2-3min may remove the dinospores from the fishes.