

Chapter 5. Nutritional Diseases

Edgar C. Amar and Celia R. Lavilla-Pitogo

Nutritional diseases of fish may develop as a result of deficiency (undernutrition), excess (overnutrition), or imbalance (malnutrition) of nutrients present in their food. The disease usually develops gradually because animals have body reserves that make up for nutritional deficiency up to a certain extent. Disease signs develop only when supply of any diet component falls below critical level. When there is too much food, the excess that is converted to fat and deposited in fish tissues and organs, may severely affect physiological functions of the fish.

LIPODOSIS

Lipodosis is a common nutritional disease among cultured food fish and various degrees of lipodosis have been observed in the liver of cage-cultured groupers including *Epinephelus coioides*, *E. malabaricus* and *Cromileptes altivelis* in Indonesia, Thailand and the Philippines.

Causative agent:

Feeding with rancid formulated feeds or with fatty or poorly stored trash fish can cause lipodosis.

Stages affected:

Fish in the grow-out stage are susceptible to lipodosis.

Gross clinical signs:

Affected fish grow poorly, lethargic, with opaque eyes, and shows slight distention of the abdomen. The liver is also abnormally pale (Fig. 5-1a). The normal color of liver is shown in Fig. 5-1b.

Effects on host:

Affected fish exhibit poor growth and low mortality, the liver has a pale appearance and histological sections show presence of large fat droplets (Fig. 5-2).

Transmission:

Lipodosis is non-infectious as the causative agent is not a pathogen. The presence of affected fish in a farm will not endanger the other healthy individuals.

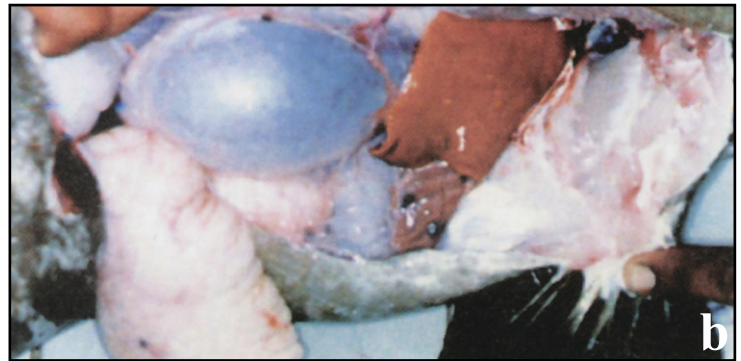


Fig. 5-1. Pale appearance of liver with lipodosis (a) and liver of normal grouper (b)(Photos from APEC/SEAFDEC, 2001).

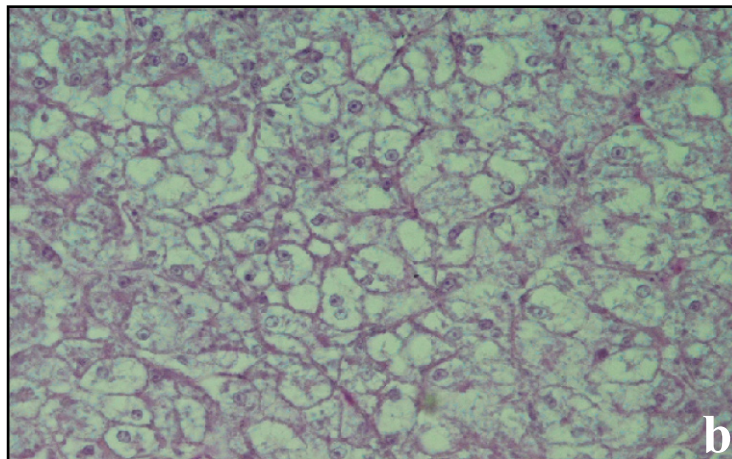
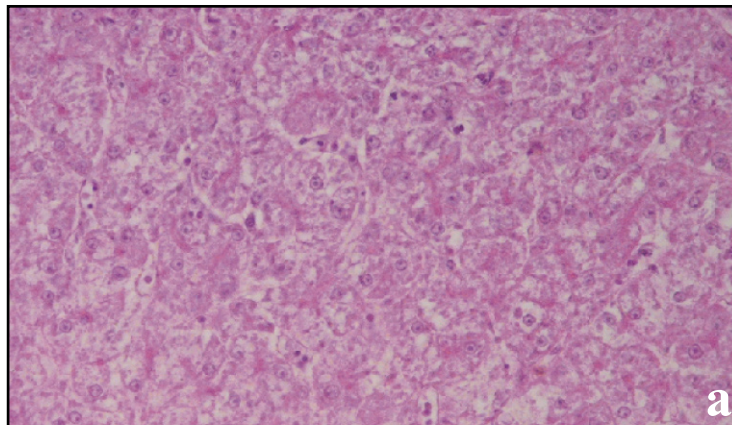


Fig. 5-2. Normal liver histopathology (a) and hepatocytes of *Epinephelus coioides* filled with clear vacuoles containing fat droplets (b). H & E stain.

Diagnosis:

The disease may be diagnosed through histopathology of liver and analysis of proximate analysis of artificial feeds. Since histopathology is required, this may have contributed to lack of reports.

Preventive and control methods:

Handle feeds properly, observe good storage practices and avoid feeding fish with spoiled and poorly stored trash fish (see Appendix 3). When lipodosis is confirmed, immediately discontinue use of remaining feeds and replace with a totally new batch of feeds.

FISH SCURVY

Nutrient deficiency rarely occurs naturally when diets have been formulated and prepared based on the species' requirement. However, some commercially-available diets for another species may sometimes be used in the absence of a suitable formulation, resulting in deficiencies. Spinal deformity associated with ascorbic acid deficiency has been reported to occur naturally in *Cromileptes altivelis* postlarvae in Indonesia. Natural occurrence in grow-out farms has also been reported in *Epinephelus tauvina* and *E. malabaricus* in Thailand.

Causative agent:

Ascorbic acid deficiency is the primary cause of scurvy.

Stages affected:

Fish in the grow-out stages are usually affected but spinal deformity may occur at the postlarval stages when inappropriate larval feeds are used. Spinal abnormality can also be experimentally induced in *C. altivelis* fingerlings when they are given diets devoid of vitamin C.

Gross clinical signs:

Affected fish exhibit gross signs such as anorexia, short snout, erosion of opercula and fins, hemorrhaging of eyes and fins, exophthalmia, swollen abdomen, abnormal skull, falling pharyngobranchials, severe emaciation, and spinal column abnormality such as scoliosis (Fig. 5-3) and lordosis.

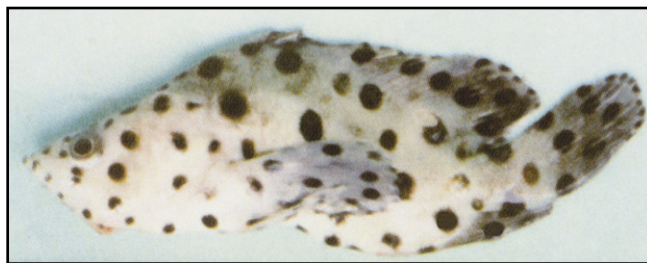


Fig. 5-3. Dorso-ventral curvature of the spinal column (scoliosis) in *Cromileptes altivelis* with ascorbic acid deficiency (Photo from Koesharyani et al., 2001).

Effects on host:

Affected fish exhibit poor growth or severe emaciation in prolonged deficiency. Histologically, hyperplasia and detachment of gill epithelium can be observed and the hepatocytes may contain large fat droplets.

Transmission:

Scurvy is a deficiency condition and is therefore non-infectious.

Diagnosis:

The deficient condition of fish can be confirmed by the curvature of the body with the hemorrhagic lesion at the broken vertebral column, histopathology of gills and liver. The formulation can also be examined as to the form and level of inclusion of ascorbic acid and further by analysis of tissue and feed samples for ascorbic acid content.

Preventive and control methods:

Use adequate amounts and stable forms of ascorbic acid (e.g. L-ascorbyl monophosphate or L-ascorbyl polyphosphate) in diet formulation. Provide the minimum requirement for the species if information is available (e.g., 30mg L-ascorbyl-2-phosphate mg/kg diet for *E. malabaricus*) allowing for losses during manufacture.

EFA DEFICIENCY

Marine fish larvae require essential fatty acids (EFA) for normal growth and development. Fatty acids are essential components of biomembranes and precursors of some physiological modulators such as the eicosanoids. Marine fish in general, unlike their freshwater counterpart, cannot effectively elongate and desaturate saturated fats to unsaturated ones and so require the presence of unsaturated fatty acids in their diets. Essential fatty acids such as docosahexaenoic acid [DHA, 22:5(n-3)] and eicosapentaenoic acid [EPA, 20:5(n-3)] are commonly found in live food such as microalgae (e.g., *Nannochloropsis*), copepods, rotifers and *Artemia*. Deficiency in these fatty acids is associated with larval mortality known as “shock syndrome” in which the larvae display unusual sensitivity to stress. Any handling or disturbance (e.g., sorting, transfer, strong aeration) of grouper larvae with this condition invariably results in mortality. This disease has been reported in *Epinephelus malabaricus* in Thailand and in *E. tauvina* and *E. fuscoguttatus* in Singapore.

Causative agent:

This condition is associated with low levels of essential fatty acids in live food.

Stages affected:

Day 21 larvae for *E. malabaricus*, stage 1 (about day 12) and stage 2 (day 23) for *E. tauvina* and *E. fuscoguttatus* are affected.

Gross clinical signs:

General body weakness and mortality is observed starting day 21 for *E. malabaricus*. High mortality occurs in stages 2 and 3 for *E. tauvina* and *E. fuscoguttatus*.

Effects on host:

Total mortality is observed after day 30 for *E. malabaricus*.

Transmission:

The disease is not transmitted to other healthy individuals by affected fish.

Diagnosis:

Visual observation of larval behavior (weak movement) is confirmed by fatty acid analysis of live food.

Preventive and control methods:

Feed 15 day-old larvae with brine shrimps enriched with fish oil at 25-50 ml/m³ of rearing water. While it is important to maintain a clean environment by sediment removal and water management, avoid unnecessary mechanical stress to the larvae by tender handling, using mild aeration and employing flow-through system of water management. The condition can be alleviated by enrichment of rotifers with n-3 HUFA or *Nannochloropsis* (syn. *Chlorella*). Bring the EFA levels of the live food to around 12% by enrichment. Supplementation with DHA is reported to slow down mortality in grouper while supplementation with EPA was less effective in other marine species.

NUTRITIONAL MYOPATHY ACCOMPANYING CEROIDOSIS

Lipid peroxidation can occur both in solution and in the cell because unsaturated fats are highly susceptible to oxidants. When peroxidation occurs in the cell membrane, cellular integrity is compromised that could lead to certain myopathies.

Causative agent:

This disease is associated with diets containing rancid fat or polyunsaturated fatty acids and low contents of vitamin E.

Stages affected:

Cromileptes altivelis fingerlings and broodstock are affected by this disease.

Gross clinical signs:

Affected fish show emaciation, darkening of body color (Fig. 5-4a), petechia at the base of the operculum (Fig. 5-4b) and occasional deformity of the spinal cord.

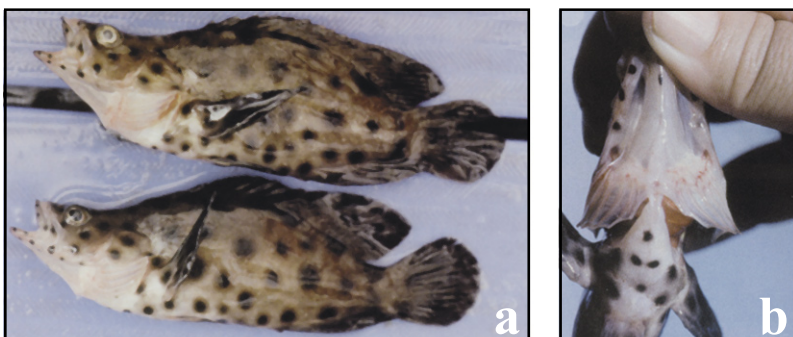


Fig. 5-4. Nutritional myopathy in *Cromileptes altivelis*: a) Darkening of body color and spinal deformity and b) petechia at the base of the operculum (Photos from Koesharyani et al., 2001).

Effects on host:

The disease can cause low but continuous mortality in *C. altivelis* fingerlings. It can also cause mass mortality in *C. altivelis* broodstock.

Transmission:

The disease is not caused by an infectious agent; it is non-transmissible.

Diagnosis:

Histopathologically, myofibril degeneration including extensive myolysis and macrophage invasion in degenerated fibers are observed in the skeletal muscles (Fig. 5-5a). Ceroid deposits, a kind of lipo-pigment which stains pink with PAS reaction in the hepatocytes, is typical (Fig. 5-5b).

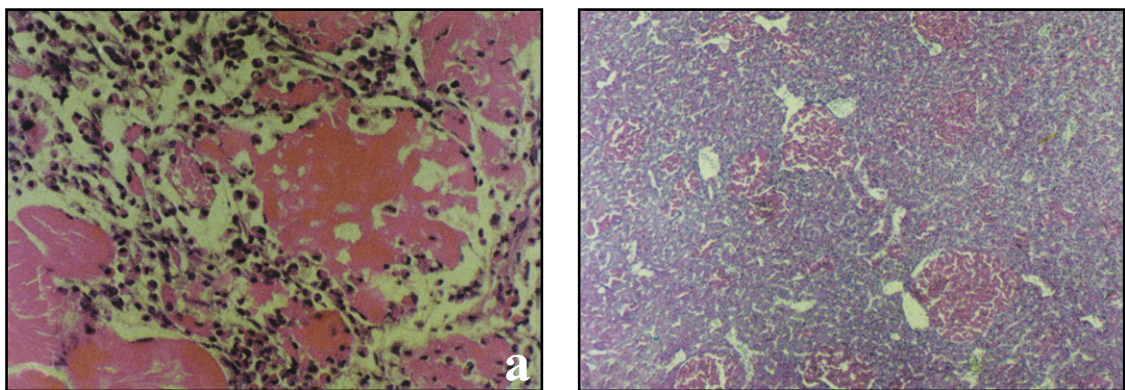


Fig. 5-5. Nutritional myopathy in *Cromileptes altivelis*: a) Macrophage invasion, H & E stain and b) ceroid deposits (pink in PAS reaction)(Photos from Koesharyani et al., 2001).

Preventive and control methods:

Proper food management can prevent this disease. To prevent rancidity of polyunsaturated fatty acids, food should be kept in freezers under -30°C and consumed as soon as possible. Antioxidants such as vitamin E are effective against this disease since they prevent peroxidative damage to cells. Enrichment of food with vitamin complex can stamp out mass mortality.

THIAMIN DEFICIENCY

Some species of fish belonging to the sardine and anchovy families contain enzymes that degrade thiamin contained in the trash fish itself. This is usually not a problem when trash fish are of mixed species or different species are fed alternately, but deficiency signs appear when single species are fed for extended periods.

Causative agent:

Deficiency of vitamin B₁ (thiamin) caused by thiaminase contained in sardine or anchovy as feed.

Stages affected:

This condition affects *Cromileptes altivelis* broodstock.

Gross clinical signs:

Affected fish exhibit whitish body color (Fig. 5-6a), erratic swimming behavior, mechanical injuries with hemorrhages on the body surface, especially around mouth, pectoral fins and abdomen (Fig. 5-6b).



Fig. 5-6. Thiamin deficiency in *Cromileptes altivelis*: a) Whitish body color and b) mechanical injuries around the mouth and pectoral fins associated with erratic swimming behavior (Photos from Koesharyani *et al.*, 2001).

Effects on host:

Thiamin is a co-enzyme of many enzymes catalyzing carbohydrate metabolism and is essential for normal nerve functions, digestion and reproduction. Thiamin deficiency affects the functions of the nervous system.

Transmission:

The disease is a deficiency condition and is not transmitted.

Diagnosis:

Histopathological lesions are mainly found in the brain where hemorrhages and degeneration of the nuclei of nervous cells occur.

Preventive and control methods:

Avoid prolonged feeding with sardine or anchovy only as feed. A mixture of different species of trash fish should be fed and regular supplementation (e.g., once a week) of a vitamin complex in the feed should be adopted. Excess administration of vitamin B₁ supplement should be undertaken if fish become deficient in thiamin.

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