NUTRITIONAL DEFICIENCIES PRESIDING TO DISEASE CONDITIONS IN CULTURED FISH

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Abstract

It is when fish are confined and fed supplemental feeds that nutritional deficiency symptoms occur. Only complete understanding requirement of cultured organisms will allow the fed manufacturer or cultist to provide adequately for the species under culture. The nutritional deficiencies are reviewed as absolute and non-absolute terms. The review concluded that deficit are interwoven and usually not limited to inadequacy or non inclusion of a certain feed ingredient especially for aquatic animals.

Key words: Nutrient, deficiencies, Cultured Fish, Diseases

Introduction

Wild fish seldom show signs of nutritional diseases because natural aquatic food are fairly nutritious especially in the essential growth factors like vitamins and minerals occurring abundantly in plant organisms (Lagler *et al., 1972)* It is when fish are confined under artificial conditions that nutritional deficiencies occur (Love,1977). Only complete understanding of the nutritional requirements of cultured organisms will allow the feed manufacturer of culturist to adequately provide for the species under culture. Generally, nutritional diseases are not recognized as problem in the warm water pond fish culture unless stocking rates approach or exceed 4,000kg per hectare (Olufemi, 1998). This is believed to be due to availability of natural foods in the pond environment in variance to concrete tanks and the likes, It is also well known that prolonged storage of feed may result in reduction of feed quality particularly for vitamin C and essential fatty acids. Contamination of feeds by Aflatoxins producing strains of Aspergillus has also been reported as causing problems in Tilapia farms in Africa and Southeast Asia (Olufemi *et al.*, 1983)

Nutritional diseases are notoriously hard to define in absolute terms, since it is rare for a single deficiencies to exist. Single deficiency conditions were often described based on feeding test diets with one particular component omitted. A general syndrome is often in appearance or poor growth being the usual sign of ill health.

The nutritional deficiencies are described under absolute and non-absolute nutritional deficiencies.

ABSOLUTE NUTRITIONAL DEFICIENCY: STARVATION

Reasons for starvation vary in according to circumstances. Complete deprivation may occur when fish are accidentally left in a facility, o in attempt to rear wild fish they may refuse to accept artificial foods they do not recognize leading to starvation and unfavorable condition.

Features of starved fish – they are usually darker than normal and the flesh is softer. Starved larvae are referred to as pin heads because of their apparently enlarged head and slender body. Gills may be pale and starving fish often have heavy parasite burdens. An necropsy there is lack of abdominal fat, often a distented gall bladder and general loss of bloom on all visceral organs. Histopathological features are marked reduction in sarcoplasmic content of individual myofibrils, with vacuolation and

central migration of sarcolemmal nuclei. There is an apparent increase in fibroblast and collagen content of the digestive tract and other organs darkening and shrinkage of exocrine pancreatic tissue. The melano macrophage centres are prominent, possible as a manifestation of the extension catabolism which has taken place (Cowey and Roberts, 1978)

NON ABSOLUTE DIETARY DEFICIENCIES AND IMBALANCES

Protein deficiency:

It is in the interest oi food manufacturer to economize protein components in fish diet, because it is the most expensive. However, protein is essential from maintenance, growth, reproduction and repletion of depleted tissues during migration and may also be used for energy metabolism. Nutritionally, satisfactory diets for cultured fish are those which have sufficient essential and non-essential amino-acids. The protein and amino acids requirements for some selected fish species in Nigeria were researched into and published by several authors including Eyo (2003) Tagbenro, *et al.*, (2000) Faturoti (et al 1986) Dificiency symptoms may sometimes occure even in the presence of apparent luxus of amino aicds. Such situation may arise either because the protein is not completely digested or because certain amino acids have been rendered biologically unavailable during processing. A liberal supply of good protein must be available to fish throughout life (Eyo, 2003)

Deficiency symptoms: Reduced growth rate is the general symptom for protein deficiency and soliosis (curvature of the spine) Occurs as related trypotophan deficiency. In salmon it was found out that scoliosis is related to the depletion of serotonin (5-HT) one of the Trp metabolites and a known neuro-transmitter in the central nervous system and scolisis included tryptophan is however reversible (Akiyama, 1992)

Carbohydrate deficiency:

Fish has limited capacity for carbohydrate metabolism, excessive CHO levels may results in liver degeneration and excess glycogen deposition. For instance salmonids may store high level of glycogen in the liver and also exhibit symptoms of diabetes when fed too much CHO (Halver, 1972; Snesko, 1972). Diseases like sekoke occur in carp fed with high level of starch rations (Yokote, 1974)

Lipids deficiency:

Fish are capable of synthesizing fatty acids of the ?7 and ? series but not ?6 and ? 3 series therefore the ?6 and ?3 series are essential for fish unless these series are present up to 1% in diet, deficiency symptoms may result. Among dificiency features are depigmentation, fin erosion, cardial myopathy and fatty infiltration of the liver in Salmonids (Cowey and Roberts, 1978). Turbot a wild marine fish ha shown a similar requirement for ?3 series fatty acids as fresh water species, while deficiency symptoms include ceroid deposition in the liver and thickening of the cell walls of lipid storage tissue. In fish species where liver is not a major storage organ a pathological syndrome – lipoid live disease may occur especially farmed fish fed trash fish or pelleted diets in which part of the lipid component has gone rancid. Lipids are tox when rancid and react with protein to lover its illogical value with a deleterious effect on vitamins that are not anti-oxidants. (Dupree and Hunner, 1984).

Vitamin Deficiencies

Vitamins are of two groups-the fat soluble and water soluble vitamins. Fat soluble vitamins are complex organic substances found in different forms. The water soluble vitamins are coenzymes of many essential enzymes catalyzing carbohydrate metabolism. The fatty soluble vitamins can be stored within the body and metabolized only slowly, so that cumulative hypervitaminoses resulting from

relatively massive intakes are possible, they are namely vitamins A,D,E and K

Vitamin A has 2 active forms, A1, and 2 (Amalcher, 1972) Hypovtaminoses of Vitamin A in fish results in poor growth, keratomalacia, odemena, exphotalmia, ascites, blindess and hemorrhages at the base of the fins and kidneys (Gupta and Gupta, 2005)

Vitamin D referred to as calciferol may not be an absolute requirement for fish though reduced bone ash and poor food conversion occur in deficiencies cases.

Vitamin E inclusion in feeds prevents rancidity of unsatureated fats and Vitamin A degeneration. Deficiency results in myopathy, steatites, ceroid in liver, spleen and kidney, clubbed, gills, exphthalmia, pericardial oedema, red blood, cell fragility, depigmentatio, microcytic, anaemia, exudaive diathesis, hemorrhages poor growth and mortalities.

Vitamin K's primary function is as a component of the blood clotting mechanism, it is also bacteriostatic with co-enzyme properties. Deficiency results in prolonged clotting time and hemorrhages into muscles and viscera coupled anemia in chronic cases. The signs are similar to those of Viral Hemorrhagic Septicemia infection. When dicoumarols (rat poison) contaminates fish food it antagonizes vitamin K metabolism. Use of poison in storage must be handled with great care: (Gupta and Gupta, 2006).

Vitamin B1 (Thiamin) is water soluble, it is essential for digestion, reproduction and central/peripheral nerve functions. Thiamin deficiency results in change of colour, hemorrhages at base of fin, hyper excitability with paralysis or aberrant swimming, anorexia, ataxia, muscle atrophy, oedema, poor growth, vascular degeneration, melanosis in older fish, cornea opacities and death. (Deupree and Hunner, 1984).

Vitamin B2 (Riboflavin): is important in respiration within poorly vascularised tissues such as the cornea of the eye. Deficiency causes corneal vascularisation, cloudiness, haemorrhagic eye, photophobia, dim vision, poor appetite, darkened skin and xerophthalmia (Halver, 1972).

VitaminB6 (Pyridoxine): Deficiency causes nervous disorder, epilepsies, anemia, growth reduction anorexia, ascites flexing of opercles, hyper-irritability, indifference to light death and rapid onset of rigor mortis.

Pantothentic acid: Deficiency causes clubbed gills, loss of appetite, lethargy necrosis of jaw, barbells, scarring cellular atrophy, sluggishness gills covered with exudates, general mumpy appearance prostration, channel catfish exhibited reduced growth with nutritional gill disease with insufficient levels of dietary Pantothenic acid (Stickney, 1979).

Nicotinic acid: Deficiency causes loss of appetite, erratic hemoglobin and low erythrocytes impaired muscus production "blume slime disease" convulsion, anorexia, anemia colonic lesions, edema of stomach and colon, uncoordinating jerky movements, muscle spasm·lethargy photophobia, swollen gills, and high mortality.

Folic acid: Deficiency symptoms are anemia, malformation and incomplete development of blood cells resulting in poor growth hemorrhagic kidney and intestine, anorexia, ascites, dark coloration, erythropenia, exphthalmia and fragility of caudal fin.

Ascobic acid: Defiency symptoms are growth retardation, lordiosis, and in erosion (Moore *et al* 1984); Ibiyo et al 2007. Impaired would healing, focal hemorrhage, twisted/deformed hyaline cartilage in gill filaments and sclera of the eyes.

Inositol: Deficiency results to anemia, bloated stomach, poor growth, anorexia and skin lesions.

B12 (Cobalamin): Deficiency symptoms are anorexia, erratic hemoglobin and erythrocyte counts fragmentation.

P-Amino benzoic acid: Non inclusion in feed does not show any significant change in growth, appetite or survival.

Biotin: Deficicy symptoms are anemia anorexia, blume slim disease colonic lesions contracted caudal fins, dark colouration erythrocyte fragmentation, moralities muscle atrophy poor growth spatic convulsion.

Choline: Deficiency causes anemia, poor food conversion, poor growth, vascular stasis and hemorrhage in kidney and intestine.

MINERAL DEFICIENCIES

Calcium deficiencies causes poor growth, poor feed efficiency and high mortality. Phosphorus insufficiency portrays skeletal abnormalities poor growth and feed efficiency and bone mineralsation, while Magnesium deficiencies causes renal calcinosis, loss of appetite, poor growth and high mortality, Deficiency symptoms of iron are hypo chronic microcytic anemia, copper deficiency is only evident in poor growth. Manganese deficiencies is evident in short and compact body and abnormal tail growth, iodine deficiency is thyroid hyperplasia and zinc deficiencies are cataract, caudal fin and skin erosion coupled with growth depression.

Conclusion

Deficiency symptoms are seen to be interwoven and nutritional disorder in any cultured organism may not be limited to just a particular deficiency or non inclusion of certain feed components. It is therefore advisable culturist and feed manufacturers to include adequate amount of essential nutrients in feed at all times irrespective of the animal's stage of growth. PREVENTION IS BETTER THAN CURE

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