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A Review of Some Viral, Neoplastic, Environmental and Nutritional Diseases of African Fish

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Abstract: A review of some viral, neoplastic, environmental and nutritional diseases of African fish was carried out to educate fish famers on some likely challenges faced and their management processes n culture fisheries. The virus disease: Infectious Pancreatic Necrosis (IPN), Virus Hemorrhagic Septicemia, Carp Spring Viraemia (CSV) and Lymphocystis virus; Neoplastic fish diseases: Malignant Tumor and Benign Tumors; Disease of unknown etiology: Broken Head Disease and Open belly disease; Naturally occurring cases; Environmental fish diseases: Depletion of dissolved oxygen, temperature, exteme pH, mechanical trauma. Nutritional fish diseases: Deficiency in Essential Amino Acid (EAA), Feed proteins known to contain toxic amino acids and remedies for anti nutritional factors are areas reviewed.

Key words: African fish diseases, environmental, neoplastic, nutritional, unknown etiology, virus

INTRODUCTION

Viruses occur in particles. Unlike bacterial cells, have no nucleus, neither organelle nor metabolic apparatus. A virus particle is referred to as virion. It depends on the synthesizing structure of the host cells for replication. Viruses are therefore obligate pathogens. Pathogenic viruses are either spherical or rod - shaped. All other groups are referred to as complex or binal virus. Viruses are classified on the basis of the type of nucleic acid in the virion (Bassey, 2011).

The methods of isolation and identification of fish virus pathogens are cell culture techniques. Cytopathic effects caused on cultured cells and serum neutralization techniques. No antibiotic or chemotherapeutic agent can treat or control virus disease in fish. Test and slaughter, quarantine, restriction of movement, sanitation and disinfections are effective control measures. Vaccines can be used to control a number of virus diseases in higher vertebrates, but seldom controls viral disease of fish (Bassey, 2011).

The absence of active virological research in Africa restricts our knowledge of viral infection to those detectable by externally visible, or microscopically recognizable virus-induced pathological changes. The only confirmed case of viral infection in African fish is Lymphocystis, in cichlids from the East African lakes (Paperna, 1973) which is manifested by readily detected, macroscopic dermal pustules (pox-like infection). A case of mortality, with characteristic abdominal dropsy of carp, in Kajansi farm, Uganda, could have been associated with viral (*Rhabdovirus carpio*) aetiology. The rhabdovirus-caused carp disease (SVC) occurs predominantly in coldwater regions, although, in cell culture the virus was found to grow in temperatures up to 31ºCFijan et al., 1971).

Recently, a whirling viral disease of tilapia fry has been reported by Avtalion and Shlapobersky (1994) to cause mortalities of laboratory reared (less than one monthold) Oreochromis aureus, O. niloticus, Sarotherodon galilaeus and tilapia hybrids including red tilapia. Affected fish were dark skinned, anorexic and showed characteristic whirling behaviour prior to death. Symptoms first occurred in 4-8 day old fish, death appeared first in 6-8 day old fish and subsequently, another wave in 14-18 day old fish. Hexagonal particles, iridovirus-like, of about 100 nm, were demonstrated within inclusions in the endoplasmic reticulum in cells from the brain tissue.

Herpes-like infections of nuclei and cytoplasm of haematopoietic kidney cells were demonstrated in pond-reared goldfish from Israel, which were also showing severe infection of the same tissue by gram negative bacteria (apparently *Aeromonas salmonicida atypica*).

Tissue cultures of cells from African cichlids (of *Oreochromis* spp. and their hybrids), used for detecting viruses, have been established in USA (endothelial cell line from *O. mossambicus* bulbus arteriosus [TmB] - Lewis and Marks, 1985) and in Taiwan (from 'Tilapia' - *O. niloticus x mossambicus* ovaries [TO-2] - Chen *et al*.,

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1983a and kidneys [TK-1] - Chen *et al.*, 1983b). A birnavirus (reminiscent of European eel virus [EVE], related to the AB strain of IPNV) was isolated (on tilapia derived cell lines and on CHSE-214 continuous salmonid derived cell line) from *O. mossambicus* (Hedrick *et al.*, 1983; Chen *et al.*, 1985). EVE viruses were incriminated in the aetiology of eel branchionephritis (Chen *et al.*, 1985).

The highly contagious herpes virus, causing disease in cultivated American catfish (CCV), in the Southern USA, proved to be transmissible and pathogenic to *Clarias batranchus* and therefore comprises a potential epizootic risk to catfish (siluroids) if introduced into Africa with its American catfish host. Cusack and Cone (1986) reviewed studies suggesting that parasitic crustaceans can be vectors of viral infections of fish. Ahne (1985) transmitted SVC virus to carp via *Argulus foliaceus* and leeches, acting as mechanical vectors.

Neoplasia and degenerative chronic conditions are often linked to anthropogenic stresses on the ecosystem, in particular due to release of xenobiotic materials to the environment (Edwards and Overstreet, 1976; Hawkins *et al*., 1988). These situations are less obvious in the less developed regions and, although in some places they may exist, relevant documentation is presently lacking. Certain fish diseases do not have any known causes for them. Environmental diseases result from inadequacies in the physical and chemical characteristics of the water. Nutritional pathologies can result from the ingestion of feed proteins containing toxic amino acids.

A review of the virus disease: Infectious Pancreatic Necrosis (IPN), Virus Hemorrhagic Septicemia, Carp Spring Viraemia (CSV) and Lymphocystis virus; Neoplastic fish diseases: Malignant Tumor and Benign Tumors; Disease of unknown etiology: Broken Head Disease and Open belly disease; Naturally occurring cases; Environmental fish diseases: Depletion of dissolved oxygen, temperature, exteme pH, mechanical trauma. Nutritional fish diseases: Deficiency in Essential Amino Acid (EAA), Feed proteins known to contain toxic amino acids and remedies for anti nutritional factors was reviewed to educate fish famers on some likely challenges faced and their management processes and culture fisheries.

Infectious Pancreatic Necrosis (IPN): This condition is caused by, RNA virus, a Revirus. It is an acute highly contagious disease of young salmon fishes, characterized by anorexia, gradual loss of equilibrium, violent body flexing, loss of weight, anemia and mortality. At autopsy, there can be hemorrhages of the viscera. The intestinal lumen is filled with bile-tinged mucus. There can be exophthalmia and dropsy. Electron microscopy, clinical signs of disease and isolation of virus on cultured cell lines are useful for the disease diagnosis. There is no known treatment. Destroy infected fish and maintain good hygienic conditions in the farm area.

Infectious Hematopoietic Necrosis (IHN): This condition is an acute to sub acute hemorrhagic disease of the spleen and anterior kidneys. RNA, a rhinovirus, causes it. Transmission can be through oral and by contact with an infected fish. Mortality can be as high as 100%. Early signs of the disease include lethargy, dark coloration, feeble swimming, vertical position in the water and some affected fish may roll in the water.

Exophthalmia and distended abdomen can be observed. Hemorrhages can be present at the base of the fins, the dorsal area of the head and the gills. At autopsy, serious fluid in the abdomen can be seen. The intestine is filled with bile-tinged mucus. The liver is pale. Pofectial hemorrhages are in the peritional lining, the adipose tissue, muscles and the mesenteries. Kidney becomes swollen and oedematous. The disease can be diagnosed through history of occurrences, presence of clinical signs, pathologic lesions, isolation and identification, test and slaughter method, disinfection of eggs with lodophor and good hygiene in the hatchery are better control measures, because there are no known treatments of the condition.

Virus hemorrhagic septicemia: This is an acute to chronic virus disease. It is caused by a Rhabdovirus. The virus particles are shed from infected fish in urine and faces. Hence, transmission of the disease is through contact with contaminated water. Incubation period of the disease is between ten and fifteen days. Signs and autopsy of the disease are similar to that of infectious hematopoietic necrosis except for the presence of dermal inflammation in virus hemorrhagic septicemia. Other noticeable similarities at autopsy include hemorrhages in muscle tissue, liver, viscera fat, gonads and other organs. There are ascites and exophthalmia. The liver turns pale or yellowish. Kidney and spleen become swollen when it is chronic. This condition can be diagnosed with the signs of the disease, histopathology, isolation and identification of the virus pathogen. There is no therapeutic procedure for the disease condition. Prevention is through quarantine and restriction of fish suspected to be infected, strict sanitation of hatchery facilities, test and slaughter method.

Carp spring viraemia (CSV): This disease is an acute to chronic virus disease of various carp species. Most epizootics of this disease occur during the rainy season when the pond water temperature begins to increase. The disease is also known to occur during the cold months of the year. Rhabdo virus causes the condition. Signs of the disease include hemorrhage on the skin and around the anus, abdomen swelling, and paleness of the gills and in

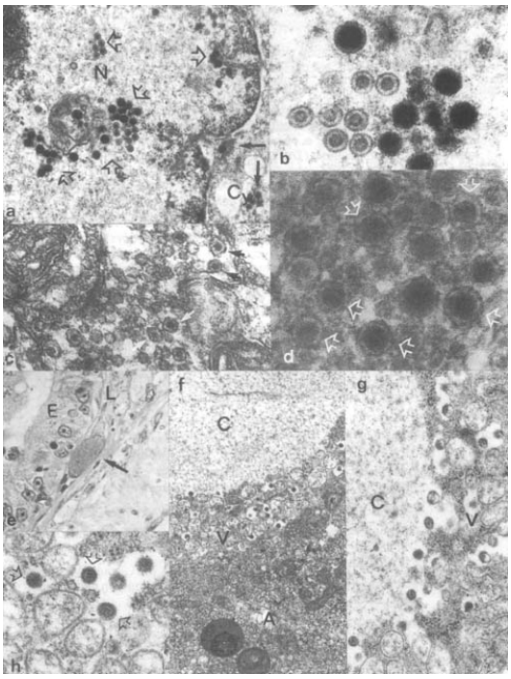

Plate 1: Viral infections

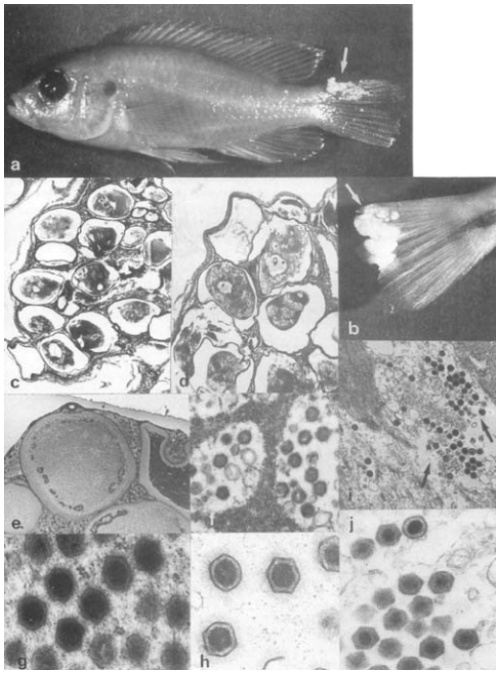

Plate 2: Lymphocystis and other iridovirus-like infections

chronic cases, ulcerative hemorrhagic dermatitis and extensive odema.

Internal signs include profuse quantities of serum or blood tinged fluid in the abdomen, pale or hemorrhagic liver, necrotic muscle tissues and hemorrhages on the surface of the swim bladder, the intestine, kidney and spleen. Diagnostic procedures include isolation and identification of the virus with histopathology and the use of clinical signs.

Treatment is not known but control can be effective with quarantine, restriction of movement of infected fish and effective sanitation.

Lymphocystis virus: A variety of marine and freshwater fish; in Africa known only from cichlids; including species of *Tilapia*, *Oreochromis* and *Haplochromis*. In cichlid fish in Lakes Victoria (Nyanza) (*Oreochromis variabilis* and *Haplochromis* spp.), in Lake George (*H.elegans*) and L. Kitangiri (*Tilapia amphimelas* and *O.esculentus*) in East Africa.

Description taxonomy and diagnosis: Infection manifested in one to numerous dermal clusters of rounded pustules or wart-like growths. Histological sections reveal aggregates of grossly hypertrophic cells (in cichlids 200-330 μm in diameter), enclosed within a thick hyaline (eosinophilic) wall and an extremely large nucleus and nucleolus. Cytoplasm contained basophilic (DNA) inclusions, (numerous, small-rounded in cichlids) and vacuoles (Paperna, 1973). Lymphocystis viruses are large (160-300 nm), icosahedral, DNA viruses (iridovirus-like) replicating within the cytoplasmic inclusions and are released into the cytoplasm to form regular arrays (Paperna *et al*., 1987).

Plate 1 Viral infections: Transmission electron microscopic (TEM) view of Herpes-like infection in gold fish, naked (80-100 nm in size) and coated viral particles (106-153 nm) formed in the nucleus (N) and spread into the cytoplasm (Cy) (Plate 1a, b). Viral particles (size about 100 nm) recovered from tilapia fry affected by whirling disease (by TEM) (Plate 1c, d). Hypertrophic virus infected endothelial cell in the lamina propria of gourami (*Trichogaster trichopterus*) gut (Histology by light microscopy [LM]) (Plate 1e). TEM view of virus infected endothelial cell from the gourami; the corona-like particles are 111-141 nm in size. A. host-cell peripheral cytoplasmic layer; C, debris filled interior space of the cell; V, virus forming layer (Plate 1f-h).

Plate 2 Lymphocystis and other iridovirus-like infections: *Tilapia amphimelas* (50 mm long) from L. Kitangiri, Kenya, infected with lymphocystis (arrow) (Plate 2a). Tail of *Haplochromis* sp. from L. Victoria (Entebbe), with cluster of lymphocystis cells (Plate 2b). Histological sections of a cluster of hypertrophic lymphocystis cells from L. Victoria *Haplochromis* sp. (cells' diam. 0.25-0.3 mm) (Plate 2c, d). Histological section (in LM) of an hypertrophic lymphocystic cell (size 0.13-0.16 mm) from gourami, virus particles are visible as

fine dots (Plate 2e). Lymphocystis virus particles (166-216 nm wide) from gourami, in active (Plate 2g, h) and aged (Plate 2f) cells. i,j. Iridovirus-like particles (size 141-193 nm) from viscera of gourami.

Life history and biology: Viral particles are released to the water when infected cells eventually degenerate, burst or are sloughed off (Paperna *et al.*, 1987). Infection was experimentally transmitted among fish of the same or closely related species through an inoculum of viral material, or immersion in water contaminated with the contents of lesions (Cook, 1972; Vaughan, 1979). The virus was grown in a cell line that had been established from fins and caudal tissue of the same host fish species (Walker and Hill, 1980).

Pathology: Even if it spreads throughout the fish skin (not in cichlids), infection is rarely fatal and eventually (within 20 days to 4 months) regresses (Cook, 1972; Paperna *et al.*, 1982). Tissue response around the nodules is very restricted. Gradual regression of infection was found to coincide with evident degeneration of lymphocystis infected cells, while fractionation of the hyaline capsule was followed by subsequent invasion of the lumen by macrophages and lymphocytes. The regression of infection and absence of recurrent infection suggests acquired resistance (Cook, 1972; Roberts, 1976; Paperna *et al.*, 1982, 1987).

Epizootiology: Natural infection is low, in 4 out of 265 (1.5%) *O. variabilis* and 8 of 98 (8%) *Haplochromis* sp. in Northern Lake Victoria, and in 1 out of 95 (1%) *H.elegans* in Lake George (Paperna, 1973). In culture installations infection may become epizootic (Paperna *et al*., 1982).

Neoplastic fish diseases: Neoplastic fish disease refers to abnormal growth in any of the organs with resultant loss of structural and functional ability of the affected organ. The resultant tumor growth may be lethal or mildly pathologic. In cases of malignant tumor or cancer, the increase in the cellular elements of the body cannot be controlled. It can spread from one organ to the other through the circulatory system, when a fraction detaches from the primary site. This is when tumor is lethal. A few cases of tumor are known is fishes. However, fish culturist should not ignore their importance in fishes and public health.

All data are on individual fish picked up from collected samples or commercial catches due to their obvious malformation, anorexia or distinct tumour-like growths. Additionally there is a fair number of documented descriptions of neoplasia in aquarium reared African fish. Some of these conditions are worth elaboration, others will be briefly mentioned. Malformations occurring in cultured fish or induced following hybridisations are not included due to their epizootiological irrelevance. The same applies to congenital anomalies reported from cultured stock (Roberts and Sommerville, 1982).

Haller and Roberts (1980) report a case of dual neoplasia in a specimen of *Sarotherodon spilurus* from brood stock held in a brackish water farm near Mombasa, Kenya. A large tumour on the flank consisted entirely of lymphocytes-a lymphoma, and the second in the kidney was identified as a renal tubular adenoma. A case of multiple fibroma has been reported from *Malapterurus electricus* and two cases of a spermatocytoma, from *Protopterus aethiopicus* in the USA and Japan; the former also with renal melanoma (Stolk, 1957a; Nigrelli and Jakowska, 1953; Prince Masahito *et al.*, 1984). Adenoma of the pharyngeal glands was described in *Haplochromis multicolor* (Stolk, 1957b) and Osteochondroma in *Hemichromis bimaculatus* (Nigrelli and Gordon, 1946).

The etiology of tumor is not clear. Several factors are responsible. Age, heredity, immunologic factors, presence of carcinogens (irritants of tissues) and oncogenic viruses are synergy of the disease. Any single factor cannot cause the disease.

Malignant tumor: This can be referred to as Hepatoma (Liver cell carcinoma). The condition is caused by the aflatoxin of *Aspergillus flavins*. The organism grows on moldy feedstuff and produce aflatoxins, which are responsible for this tumor in fish. Infection depends on fish species and age of the fish. Enlargement of liver, which may be visible through the body wall is a sign of the condition. Post mortem inspection reveals grossly enlarged and distorted liver.

Metastasis occurs in some cases by enlargement of several other organs with similar tumor tissues present. To control the condition, avoid storing feed and feedstuff in humid environment. Prevent feed from moulds. There is no known treatment. Aflatoxin can however be detoxified with the application of ammonia at a specific level to feedstuff under increased atmospheric pressure, with a combination of heat and moisture.

Benign tumors: This is also referred to as pappilomas. Pappilomas are wart like tumors found on the skin, lips, fins, opercula and other external parts of the fish body. It can be numerous among certain benthic fishes. The etiology of pappilomas of fishes is yet known. The chronic development of benign tumor of the fibrous connective tissue in the body organs such as liver, spleen, stomach, kidney and gill tissues is referred to as *Viscera granuloma*. It appears as small discrete white to grey lesions in any of these organs. The cause is not known, but seem to be associated with infectious agents such as

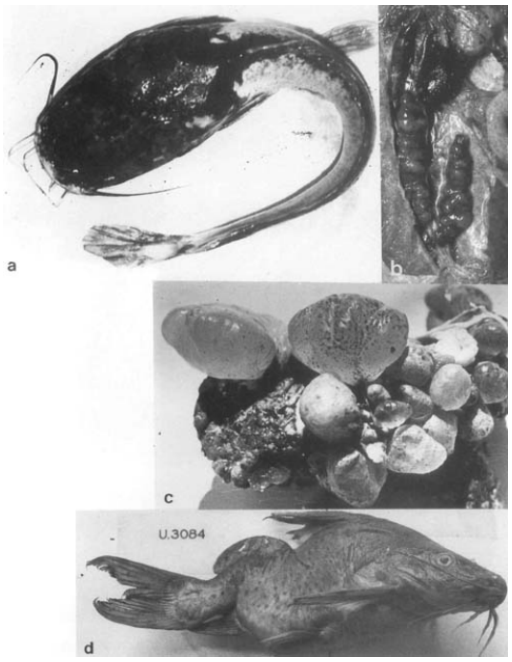

Plate 3: Deformations, degenerations and neoplasia

viruses; bacteria, fungi, parasite and antibiotics entering the tissue. Cases of neoplasm should be reported to a pathologist, who can identify the type of tumor. Pappilomas and fibrosis can be controlled in animals surgically or with the application of certain therapeutics. The effectiveness of these control measures in fish is not certain.

Disease of unknown etiology: Certain fish diseases do not have any known causes for them. Two examples of such diseases of unknown etiology are:

Broken head disease: The symptoms of this condition are pop eyes, soft skull and sometimes deformed caudal fins. In the later stage of the disease there is a gradual destruction of the arboresent organs. This can cause an exudative inflammation of the skull with gas production. The skull finally breaks laterally, parallel to the joints of the skull plates. The disease is particularly prevalent in catfish larger than 10 cm. Healed fish have thickened and curved skulls. Avoid adverse conditions such as contaminated water and poor food quality to prevent the disease.

Change pond water and increase water flow rate when first signs of the disease are observed. The supply of food can be stopped for a few days and replaced by fresh food rich in minerals and vitamins until the fish recover again. The duration ranges from 3-6 weeks.

Open belly disease: In this condition, fry or fingerling stay in vertical positions on the water surface. Fish can also be seen swimming actively with swollen bellies. The intestine becomes necrotic. Bacteria from the intestine invade the abdominal cavity and cause lyses of the abdominal wall with gas and fluid production. The belly expands and breaks open. The disease occurs especially when fry and fingerlings are raised in feeds containing 40% digestible proteins. Within one or two days, high mortality can occur. Reduction of feeding regime and elimination of all the fish bearing the first symptoms of the disease are therapic measures.

Naturally occurring cases: Anorexia with metaplasia and Neoplasia in *Clarias gariepinus* in Lake Victoria (Plate 3). Emaciated male and female specimens, both over 70 cm long. Necropsy of the female revealed subdermal myxofibroma (or myxofibrosarcoma), grossly enlarged spleen due to haemangioma (or haemangioendothelioma) and the ovary comprised of several large (up to 4 cm diameter) cysts full of serous liquid. In the male fish only the testes were slightly enlarged with seminiferous tissues replaced intermittently with zones of fibrosis and necrosis (Tumour identification by Dr J.C. Harshbarger, Registry of Tumor in Lower Animals, Smithsonian Museum, US Nat. Mus.- accession no. RTLA 759A & B).

Emaciated *Protopterus aethiopicus* from L. Victoria, with no otherwise detectable gross pathology.

Epithelioma in two full sized *Malapterurus electricus* from L. Albert. (Plate 4) Tumours occurred on the lips, head and flanks. Proliferative tissue was of two types, one involving predominantly regular skin epithelial cells (malpigi cells) and the other consisting mainly of the glandular (albuminoid) "fear cells" (RTLA 760; Paperna, 1975). *Haplochromis* sp. from l. victoria with subcutaneous lipoma (RTLA 761) Plate 4. Mature *Synodontis afrofisheri* with curved spine from L. Victoria.

Plate 3 Deformations, degenerations and neoplasia: a. Emaciated *Clarias gariepinus*, from L. Victoria. Necropsy of such fish revealed neoplastic processes as well as degenerative changes in the gonads (b, c, f) (Plate 3a). Degenerative testes with parts of the seminiferous tissue being replaced by connective tissue and necrotic deposits (Plate 3b). Cystic ovary of specimen a. d. Spinal curvature in adult *Synodontis afrofisheri* from northern L. Victoria (x 0.6) (Plate 3c).

Plate 4 Neoplasia and other abnormalities; Epithelioma in *Malapterurus electricus* from northern L.Victoria. Gross pathology. Histology of proliferative epithelium from the mouth region, where only malpigi cells are involved (Plate 4a, b). Skin area where glandular, albuminoid cells are also involved (Plate 4c, d, e). Spleen with large tumour identified as haemangioendothelioma (Plate 4f). *Haplochromis* sp. with large dermal lipoma (Plate 4g).

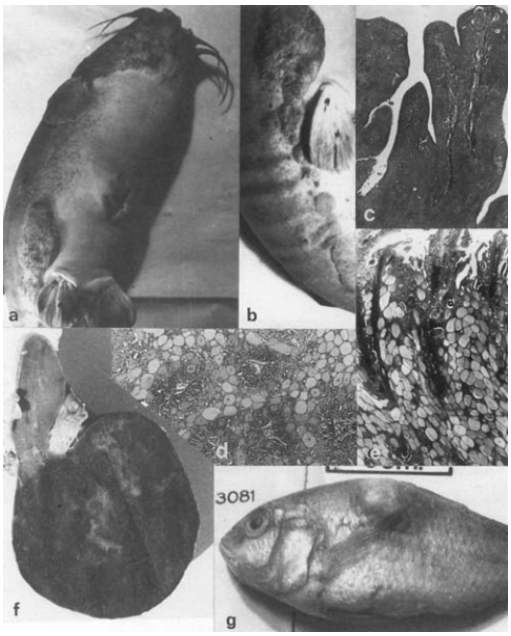

Plate 4: Neoplasia and other abnormalities

Environmental fish diseases: These are disease resulting from inadequacies in the physical and chemical characteristics of the water. Diseases caused by environmental factors are.

Depletion of dissolved oxygen: Oxygen depletion in fishpond can result in fish kill. Pond water can dissolve atmospheric gases. Accidental or intentional addition of domestic waste, decaying algae, plant and animal tissues, slaughter house effluent, animal waste and run off from animal feed, can cause severe oxygen depletion in fish ponds.

Low levels of dissolved oxygen cause fish to gulp atmospheric air frequently, migrate to shallow water areas, reduce its activity, and have lethargy and restlessness. This needs to be confirmed through water analysis. Biochemical and chemical oxygen demand may aid the diagnosis. Flush out the pond water and remove any visible oxygen depleting substance. Refill pond with fresh water. In addition, avoid over fertilization.

Temperature: Fishes can be grouped based on the survival of optimal temperature into coldwater fishes (pscychrophilic), cool water fishes (mesophilic) and warm water fishes (thermophilic). The temperature range, for the respective groups are 4ºC, 0-32ºC and 22-33ºC. Anything other than the optimal temperature range can result to anemia and stunted growth in the fish species. Rapid environmental change beyond the normal survival temperature range can cause thermal trauma. A sudden drop in temperature produces hemolysis afrigore, with vacuolar degeneration of red blood cells (escape of hemoglobin with a subsequent reduction in blood uptake of oxygen). Fish death can occur in about 3 days. However, temperature fluctuation of 12ºC can be tolerated.

Extremes in pH: Fish living in different pond environments adapt to different pH range, they can survive. The pH range from 6.2 to 9.2 is acceptable for most fish species. However, more tolerant fish species are capable of surviving outside this range without any physiological defect. Extremes of acidic or alkaline pH occur, if ponds are constructed on acid sulphate soil or areas with high level of lime.

Fish dies in cases of extremes high or low pH. Non-optimal pH can stress fish and subsequently die of secondary causes. The signs that are common with extremely in pH include skin and gill irritation observed as erythema of the skin and increased opercula vibration. The surface capillaries in the fish may rupture. Impairment in maintenance of the normal pH of body fluids, alteration in the acid - base balance and inability to excrete bicarbonate ion or oxygen absorption are pathophysiological features.

The condition can be diagnosed with the signs such as hemorrhages in external body surfaces of fish and analysis of pond water for pH. Diagnosis are better done in late afternoons for the most alkaline pH, the fish are subjected to. The pH of pond can therefore be closely monitored. Addition of lime and removal of aquatic plants can adjust extremely acidic pH in the pond. Flooding the pond adjusts extremely alkaline pH.

Mechanical trauma: The process of seining and sorting fishes (Brood stock) prior to or during and after spawning can result in injury. This can be entry points for fish pathogens. Overcrowding of fishes in culture facilities can cause fighting, biting and cannibalism which can also result in injuries. Low water levels and unfavorable environment expose fish to predators. Predation can also cause injury that can result in infection and fish mortality. Rough handled incubated fish eggs can be subjected to mechanical injury and physical shocks beyond the blastoderm stage. Therefore, avoid mechanical injury and handle incubated eggs carefully. Disinfect wounded fish promptly with suitable disinfectants at recommended dosage.

Nutritional fish diseases: Nutritional disease can be referred to as the manifestation of excess or deficiency in various important feed stuff including carbohydrate, proteins, lipids, mineral salts and vitamins. Most nutritional diseases are difficult to diagnose because of their chronic nature, with the condition only manifesting over a long period of time.

Table 1: Major dietary induced nutritional pathologies

Pathology condition	Dietary	Imbalance
Scoliosis/Lordosis	Deficiency of:	Tryptophan, Magnesium Phosphorus, Vitamine C
	Toxicity of:	Lead, Vitamin A. Oxidized fish oil
Cataract	Deficiency of	Methione Ttryptophan Zinc Magnesium, copper selenium, Manganese, vitamin a. riboflavin
	Toxicity of:	Mercury, choline oxidized fishoil
Fin erosion	Deficiency of:	Lysine, Tryptophan zinc, Riboflavin Inositol, Niacin, Vitamin C
	Toxicity of:	Lead, Vitamin A
Fatty liver	Deficiency of:	Choline, essential fatty acidst oxicity of:Oxidized fish oil
Fish skin	Deficiency of:	Riboflavin, Panthonic acid, Niacin, Thiamine, Inositol, Vitamin C Vitamin A, Vitamin K
Hemorrhages	Toxicity of:	oxidized fish oil

Onouha and Deekae (1987)

Signs of nutritional disease can also be marked by secondary disease condition due to pathogens. Accurate diagnosis of nutritional disease therefore requires critical observation for external and internal signs, analysis of the fish feed for various nutrient components and comparison with the requirement for fish. Iodine deficiency can cause goiter observed as a swelling between the base of the tongue and the last gill arch. Insufficient iron can result in anemia, while deficiency in calcium and phosphorus can cause reduced skull growth and skeletal deformities

Overfeeding can also cause disease and death. For instance, Lipoid hepatic degeneration, Enteritis and Haptoma can result from overfeeding. Fish maintained under intensive culture systems rely totally on the provision of a nutritionally complete diet throughout their cycle. Specific nutrient deficiencies can therefore cause certain nutritional pathologies in culture fish.

- A. Deficiency in Essential Amino Acid (EAA) such as lysine, methioine, tryptophan, arginine and leucine can cause fin erosion, increased mortality rate, cataract (blindness) and renal calcinosis of the fish. Poor feed formulation can cause these deficiencies. Fishmeal is rich in balanced essential amino acid profile. Majority of other protein sources include splant protein, yeast, meat, bone meal, blood meal, oil seeds, and algae. These lack one essential amino acid or the other. Silage is deficient in tryptophan. It is therefore clear that during feed formulation, care must be given to the choice of feedstuff used to obtain the desired dietary essential amino acid profile. Dietary essential amino acid deficiency can come from excessive heat treatment of feed proteins during feed/diet manufacture. Essential amino acid deficiencies can occur due to the disproportionate levels of specific amino acids or from the chemical treatment of feed protein with acids or alkalis due to the loss of free tryptophan and lysine/cystine respectively.
- B.Feed proteins known to contain toxic amino acids. Include alkali treated soybean (toxic amino acid-lysinoalanine), the legume, *Leuceana lecocephala* (toxic amino and mimosine) and the fava bean, *vicia faba (*toxic amino acid-dihydroxphenylalanine. Table 1 summarizes the dietary imbalances resulting in the manifestation of six major nutritional pathology conditions.

Anti nutritional factors in feedstuffs: These are toxic substances associated with fish feed ingredients that affect them by altering their nutritional value. Hence, affect the health of the fish. They can be of external or exogenous origin, in which case they are regarded as contaminants. They may also be inherent in the feedstuffs, and are referred to as anti-nutritional factors. The contaminants are herbicides, pesticides and microbial toxins (Mycotoxins). The anti-nutritional factors inherent in feedstuffs include protease inhibitors, phytates, cycloporpenoid fatty acid, gossypol and thiaminases.

Dietary Essential Fatty Acid (EFA) deficiency causes reduced growth and poor food conversion efficiency. Dietary essential mineral deficiency can also cause reduced growth, poor food conversion efficiency, bone demineralization, skeletal deformity and abnormal calcification of ribs. When these substances are excess in the diet, they exact negative effects on fish growth and feed efficiency. It is also believed that nutritional pathology can come from anti-nutritional factors present in plant feedstuff and certain additive (binders and stabilizer) added during the processing of feedstuff. Their presence may result in reduced growth and poor food conversion efficiency.

Protease inhibitors: These are substances that interfere with the digestion of protein by inhibiting protease enzymes. They also inhibit the activities of enzymes that digest other food nutrients such as lipids and carbohydrates. This inhibitor includes, lectins. The enzymes they inhibit include: trypsin, chemotrypsin, lipases and amylases. The protease inhibitor isfound in raw soybean, groundnuts (in the tannins), and navy or kidney beans. These inhibitors influence the digestion and utilization of many nutrients. They depress and alter pancreatic enzymes secretion in various simple stomached fish. They decrease lipid absorption and metabolizable energy of fish diet. Gall-bladder contraction, increased excretion of bile and low intestinal proteolyses activity are caused by them. They also affect methionine metabolism. Lectins (Hemoglutinins) cause agglutination of red blood cells (Erythrocytes) *in vitro.*

Gossypol: This is a yellow pigment located in the pigment glands of cottonseeds. Gossypol is mainly found in cottonseeds. There is also free gossypol acetate in nature. Gossypol is detrimental to growth and

development of various monogastric animals. It binds with lysine thereby decreasing its availability. It also accumulates in the muscles, kidney and live tissues causing some disorders.

Phytates: Phytic acid is the hexaphosphoric acid ester of inositol. It forms strong complexes called phytates with protein, phosphorus, zinc, calcium and magnesium. All plant protein sources including soybean and cottonseeds contain phytic acid. Phytic acids interfere with both mineral and protein availability due to the formation of phytates. For instance, the phosphorus contained in oilseed meals and soybean meal is largely available to monogastric animals due to the formation of the complex. These animals lack the enzyme phytase, which release phosphorus from phytic acid complexes.

Thiaminases: These are enzymes that destroy thiamin, which is one of the essential amino acids found in animal proteins. Thiaminases are found in uncooked fish flesh such as the head, skin and viscera. The destruction of thiamin by thiaminases, lead to poor growth and feed conversion in the fish. This means poor utilization of proteins in the diet.

Cyclopropenoid acids: These are cyclopropene-ringed fatty acids such as malvalic acid and sterculic acid, which cause nutritional difficulties. The fatty acids are mainly found in cottonseed oil. These fatty acids cause depressed growth and stimulated aflatoxin induced hapotoma formation in fish.

Other anti-nutritional factors: Linseed meal contains an antipyridoxine factors and a cyanogenic glycoside. Rape seed meal contains glycosides, which when hydrolyzed can yield goitrogen. However, some vitamins such as vitamin B_6 pyridoxine and vitamin B_{12} (Cyanocobalamin) can limit the effect of anti-nutritional factors.

Remedies for anti nutritional factors: One of the best ways to reduce the effects of these anti nutritional factors is by heating. But extensive heating or prolonged storage should be avoided. This can result in impairment of protein quality by millard or browning reaction. Cooking or proper heat treatments destroy the trypsin inhibitor contained in the tannins of groundnut skin. Ruminants are not affected by these anti-nutritional factors because the rumen fermentation renders the factors ineffective. fermentation of feedstuffs containing them can be a type of remedy. Increasing the protein level on the diet can diminish the effects of these inhibitors.

In case of gossypol, addition of iron salts to cottonseed meal and the production of glandless varieties of cotton can reduce or eliminate its effects. However, the addition of iron salts causes discoloration of the meal and may render it unacceptable. So, the production of glandless cottonseed is preferable. This can be done through selecting cultivation of cottonseeds, which contain very low levels of gossypol. The use of defatted cottonseed meal is recommended in feed formulation to reduce the levels of cyclic fatty acids.

The use of diets containing uncooked fish flesh within several hours after mixing also reduces or eliminates the effect of thiaminases. Supplementation of the essential diet nutrients with mineral is recommended in feed formulation. However, it should be noted that trypsin inhibitors are not completely bad. The reason is that colostrums contain trpysin, which aids immune globulin absorption in fish fry/fingerlings by suppressing proteolysis actively in the small intestine. Any proteolysis can alter the protein structure and destroy the effectiveness of the immune globulins.

CONCLUSION

Adequate knowledge some viral, neoplastic, environmental and nutritional diseases of African fish: Infectious Pancreatic Necrosis (IPN), Virus Hemorrhagic Septicemia, Carp Spring Viraemia (CSV) and Lymphocystis virus; Neoplastic fish diseases: Malignant Tumor and Benign Tumors; Disease of unknown etiology: Broken Head Disease and Open belly disease; Naturally occurring cases; Environmental fish diseases: Depletion of dissolved oxygen, temperature, exteme pH, mechanical trauma. Nutritional fish diseases: Deficiency in essential amino acid (EAA), Feed proteins known to contain toxic amino acids and remedies for anti nutritional factors is very essential in culture fisheries management.

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