Websites:
http://www.lifesciencesite.com
http://www.sciencepub.net

Emails:
editor@sciencepub.net
sciencepub@gmail.com

Life Science Journal

MARSLAND PRESS
Multidisciplinary Academic Journal Publisher

Ebinat Woreda Livestock Resource Development and Promotion Office

Abebe Mequanent

Manual For Fish Production Constraints And Fish Diseases in Ethiopia, Ebinat, Ethiopia
E-mail: abebemequanent@gmail.com

ABSTRACT: Fish is a limbless cold blooded vertebrae animal with gills and fins, living wholly in water. Fish farming is practiced in different parts of the World including Ethiopia. It is concentrated in Lake Tana and Rift valley lakes of the country. Even though Ethiopia has a beautiful water bodies with total surface area of 13,637 km^2 that have a potential to produce 94,541 tons annually, but the country only produce 45,610 tons of fish per year from both capture and Aquaculture fisheries, because of many constraints like: post harvest loss, poor infrastructure, less access to fishing materials, lack of market chain, over fishing, urbanization, agricultural expansion, wet land degradation, water hyacinth, climate change and fish disease, just like all other animals fish also suffer from various diseases and effects of pollutants. Fish disease such as:- viral, bacterial, fungal and parasitic fish diseases have a significant effect on fish production.
[Abebe M. **Manual for Fish production constraints and Fish diseases in ethiopia**. *Life Sci J* 2024;21(5):1-15]. ISSN 1097-8135 (print); ISSN 2372-613X (online). http://www.lifesciencesite.com. 01. doi:10.7537/marslsj210524.01.

Key words: *Constraints, diseases, Ethiopia, fish and production*

MANUAL FOR FISH PRODUCTION CONSTRAINTS AND FISH DISEASES IN ETHIOPIA

By
ABEBE MEQUANENT
(E-mail: abebemequanent@gmail.com)

Date: 4/25/2024
Ebinat, Ethiopia.

Table of Contents

1) INTRODUCTION

Fish farming has been practiced in different parts of the world including Ethiopia. In Ethiopia fishery production mostly concentrated in Lake Tana and great Rift-Valley Lakes. Still its production is under exploited with limited access and production status in food marketing system. The country has a number of beautiful water bodies with the total surface area of 13,637 km^2 that have a potential to produce 94,541 tons annually. But the country produces only 45,610 tons annually from both capture and Aquaculture fisheries. Fisheries are one of the important and renewable natural resource. The livelihood of many rural communities relies on the fishery sector. Fisheries are a key sector for reducing poverty, because it helps to diversify house hold income directly and indirectly. In the developing world, about 116 million people are benefited from the fishery sector and about 90% of them are working in the small-scale fisheries sector. Historically, Africa's fisheries output is dominated by capture fisheries, but today aquaculture is grown from time to time over the past decade. In Ethiopia fish production depend on the inland waters for the supply of fish as a cheap source of animal protein. It can also indirectly contribute by providing revenue for purchasing food for deficient areas and the major source of fish including river basins, major lakes, many swamps, flood plains and man-made reservoirs (Ackerman and Iwama 2001).

2) DEFINITION AND OVERVIEW ETHIOPIAN FISHERIES

Fishery is a part of the sea or rivers where fish are caught in large quantities. Fisheries refer to an organized effort by humans to catch fish or other aquatic species, an activity known as fishing. All fishing activities are categorized in capture fishery and Aquaculture. Capture fishery is the capture of usable aquatic organisms from the wild. Aquaculture is the farming of aquatic organisms such as fish, crustaceans, mollusks and aquatic plants.

Aquaculture is a food production technology where by fish or other aquatic organisms that produce greatly harvest than would naturally occur. Aquaculture involves cultivating fresh water and salt water populations under controlled conditions (Afonso *et al*., 2003).

Ethiopia is endowed with inland water for fish production as a cheap source of animal protein. Ethiopia has a number of lakes and rivers with substantial quantity of fish stocks. For example major lakes such as: Lake Tana, Ziway, Hawassa, Chamo, Abaya and reservoirs regularly Koka and Fincha and different rivers in the country (Ang *et al*., 2000).

Fish contain high quality protein (brilliant mind) and diversifying sources of income. Fauna is the animals of particular region, habitant or geological period. Flora is the plants of particular region, habitant or geological period. The fish fauna is dominated by 3 species: 1) The Nile tilapia (Oreochromis niloticus), 2) The African catfish (Clarias gariepinus) and a few cyprinids mostly Barbus species (Arias *et al*., 2012).

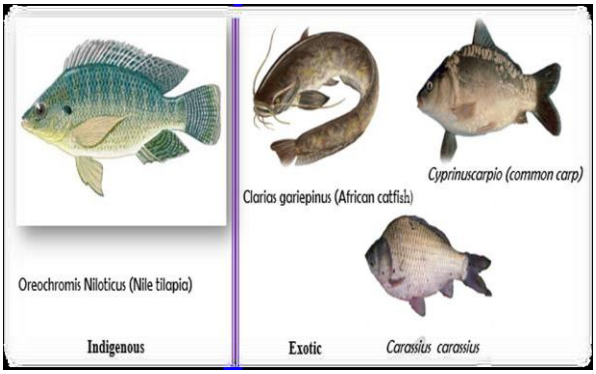

Fig 1: Most landing and preferred fish species in different water.

3) CURRENT FISHERY PRODUCTION SYSTEM IN ETHIOPIA

Ethiopia is known as the water tower of Eastern Africa which provides about 86% of the Nile water. The country has a number of beautiful lakes, reservoirs and small water bodies. The most fish product sources are fishery cooperatives from different: - Lakes, street traders, brokers and fish shop, hotels and also from restaurant (Astrofsky *et al*., 2000).

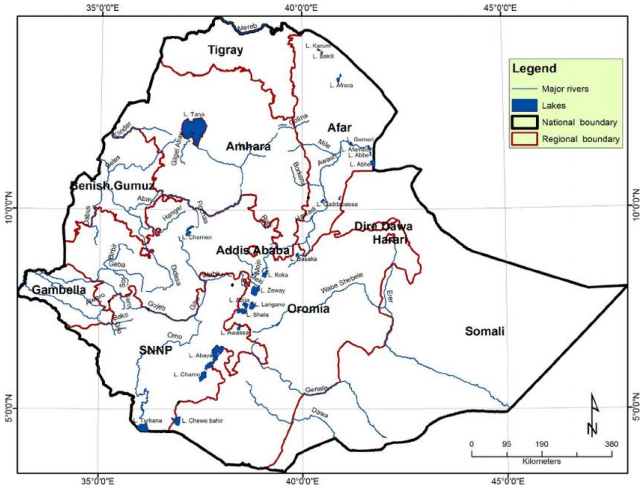

Fig 2: Map showing the major lakes and rivers in Ethiopia (source: Tesfaye and Wolff 2014)

Table 1: Ethiopian water bodies and their fish potential and production status.
Fish production potential of the country is estimated to be 94,541 tons annually for the main water bodies.

Water bodies Types	Area (km2)	Length (km)	Fishery potential (tone/year)
Major lakes	**7740**		**39,262**
Major reservoir	**1447**		**7879**
Small water bodies	**4450**		**25,996**
Rivers		**8065**	**21405**
Total	**13637**	**8065**	**94,541**

4) CHALLENGES OF FISHERY PRODUCTION SYSTEMS IN ETHIOPIA

Like other African country: Ethiopia is challenged with different constraints for overall fish development and fishery production system like:

4.1) Post-harvest losses: are caused by biochemical and microbiological spoilage changes that occur in fish after death. A live fish has natural defense mechanisms that help to prevent spoilage. Fishes are perishable products they spoil very quickly by with high temperatures, because temperature increase the activities of bacteria and enzymes in fish flesh and lastly resulted post-harvest fish losses. Globally fish lose due to spoilage is estimated to be 10 to 12 million tons per year which accounts 10% of total production of fish. The determinant factors for fish post-harvest losses include: Less market access, Size and species preference, inadequate infrastructure for fish handling, processing, storage, transportation and distance from the central market (Au, 2000).

4.2) Poor infrastructure, access to fishing materials and marketing constraints

Substantial potential fish marketing system exists in Ethiopia with ineffective marketing network. Fish marketing in Ethiopia is also influenced by: Poor transportation and Poor preservation facilities. Many researchers approve that there is serious problems in transportation and other necessary infrastructure. Fishermen in forced to transport their product for providing market by: using motorcycle which is too hefty and using donkey back. Due to lack of access to fishing equipment at different fishing areas the fishermen use a traditional gear (Boutin *et al*., 2012).

4.3) Overfishing: - In Ethiopia fishery is an open access and consequently there has been localized overfishing that bring risk for: Some commercially important species and overall aquatic resources. Most of time fishes are caught before reaching sexual maturity. The problems of Overfishing may rise due to: Poor awareness of fishermen on the length of first sexual maturity, the types and the mesh size of the fishing gears, uncontrolled and excess fishing practices, using narrow mesh sized nets, lack of government control over fishing and lack sense of ownership on the resource (Brock and Bullis 2001).

4.4) Urbanization, agricultural expansion and wetland degradation

In Ethiopia, wetlands covered about (22,600 km^2) surface area of the total land. These wetlands areas have contributed on protecting different: Pollutants, sediment, chemicals, fertilizer, and human sewage, animal waste, pesticides and heavy metals. Wetlands have provided habitat for fish breeding. Species of fish used in wetland are: Clarias gariepinus, Garadembecha, Labeobarbus intermedius and Labeo barbusnedgia (Cunningham *et al*., 2012).

4.5) Water hyacinth: - Water hyacinth (Eichhorni acrassipses) has been considered as the worst invasive weeds in relation to its negative impacts on: aquatic ecosystems, agriculture, fisheries, transportation, living conditions and social structures. Water hyacinth highly use and reduce the dissolved oxygen that led to fish kills caused by oxygen depletion. Now a day, these weeds are the main cause for declining fish production status in different lakes like Lake Tana (Darwish *et al*., 2012).

4.6) Climate change and Fish diseases

Ethiopia is facing a massive drought and food insecurity crisis as a result of: Shortage rains and droughts that have been resulted worse due to climate change by El Nino, Climate change seriously causes depletion of fishery activities; higher inland water temperatures decline the availability of fish stocks by: altering water quality and the trophic status of a given aquatic ecosystems. Sometimes due to rainfall vibration the highest runoff happened in different areas that bring the sediment load in the water bodies. In Lake Tana sediment load and siltation are current problems and the country of fish production also affected by diseases. Fish diseases are one of the problems of the fishery sector in the country. Parasites and disease associated conditions of the fish decreases fish production potential. It is the common and main problem for the entire world in both capture fishery and aquaculture. It may lead to

high mortality in a given water body or fishing site (Darwish *et al*., 2011).

5) FISH DISEASES AND THEIR CONTROL

Introduction:-Like all other animals fish also suffer from various diseases and effects of pollutants. In case of pollution death is rapid and fish of all sizes are affected. In case of disease, one individual or a group of individuals or in extreme case entire population is affected. Fish may die within few days, or several weeks or months. Diseases are more common and dangerous among fishes living in confined spaces as compared to wild (Deane and Woo 2006).

Most common signs of fish diseases are fish become sluggish, don't take food, rub its body with dikes or any other hard object in water, increase in breathing frequency, discoloration of body, rapid secretion of grey colored slime on the body, appearance of brown, black or white spots on the body, fin cuts and eaten out and whirling or tumbling movements.

6) VIRAL FISH DISEASES

6.1) Lymphocystis

Lymphocystis is a benign and self-limiting disease described in a broad range of freshwater and marine fish species.

Etiology: Lymphocystis is caused by an iridovirus. The disease is recognized worldwide and occurring in at least 125 species of teleosts belonging to 34 families and nine orders. The disease occurs in warm, cool and cold water fish species from freshwater, estuarine and marine environments (Decostere *et al*., 2004).

Clinical Signs: Lymphocystis is a chronic but seldom fatal disease, Develop macroscopic nodules (0.3–2.0 mm or more in diameter), this nodule occur primarily on the body surface but can also develop on the internal organs, the nodules appear cream colored to pink or gray, they take a week to a year or more to develop and the lesions eventually heal, leaving little **scar** tissue (Diamant *et al*., 2000).

Epizootiology and Transmission: The disease occurs with equal frequency in both sexes. It can occur in fish of any age, although the prevalence appears higher in young fish. Experimentally transmitted with relative ease within a genus level but difficulty between families of fish. Lymphocystis can be transmitted: experimentally by cohabitation, subcutaneous or intraperitoneal lesion implantation, exposure to water containing virus, feeding lesion and lesion homogenate and applying lesion homogenate to gills or scarified skin (Ferreira *et al*., 2006).

Necropsy finding: It causes fibroblast hypertrophy. Infected cells do not divide, but the cytoplasm and nucleus become very large. Chromatin condensation and fragmentation are evident and nucleoli are distorted or indistinct. Feulgen positive inclusion bodies can be seen in the cytoplasm. A thick hyaline capsule forms at the periphery of the cell. Plasma cells, lymphocytes, macrophages and polymorphonuclear leukocytes accumulate at the periphery of the cell (Ferreira *et al*., 2006).

Pathogenesis: The course of the disease is more rapid at warmer water temperatures. The course is usual at 25°C, and 11 developmental stages have been described. Viral inclusion bodies generally appear about 8 days after infection of a susceptible cell. The virus is detectable as early as 15 days post infection. The enlarged nucleus appears and reinfection is possible, but lesions in second and third infections are usually smaller. Cell-mediated and humeral responses to the infection have been demonstrated late in the course of infection. Regression of lesions begins when precipitin reactions between host serum and lesion homogenates has been seen.

Differential Diagnosis: Lymphocystis is usually differentiated from epitheliocystis on the basis of the cell type affected and the position of the nucleus of affected cells. The dermal fibroblasts affected by Lymphocystis usually display a central nucleus and where the epithelial cells affected by epitheliocystis show distinctly peripheral nuclear placement (Arias *et al.*, 2012).

Prevention: No vaccines are available. Careful screening of fish stock sources is the only known prevention. Reduction of fish trauma through appropriate social and behavioral management. Reduction of abrasions from harsh substrates or rough handling may help prevent the infection.

Control: Lymphocystis virus is remarkably stable under a variety of storage conditions. Significant levels of infectivity were recovered after 15 years from infected tissue dried over P_2O_5 at 4°C. Lymphocystis virus is inactivated when exposed to ether or chloroform, heat (56–60°C), or pH 3.0. The virus is stable to multiple cycles of freezing and thawing (Gauthier and Rhodes 2009).

Treatment: Lesion-bearing fish should be removed. The antineoplastic drug 6-**mercaptopurine** inhibits virus-specific synthesis and the appearance of virus-induced cytopathic effects in cell culture. And this drug has been used experimentally to control lymphocystis in fish. Surgical removal and cauterization of the wounds with dilute iodophor solution can be palliatively effective. Care should be taken to avoid burning the surrounding skin by prolonged exposure to the iodophor (Harmache *et al*., 2006).

6.2) Epidermal Papillomatosis

Definition: Epidermal papilloma is a neoplasm growing on the skin of fish. Epidermal papillomatosis occurs in many fish species, both in farmed and feral fish. Epidermal papillomatosis is a benign skin tumour, often also referred to as epidermal hyperplasia and skin neoplasia. Neoplasia is a common term used for abnormal hyperplasia of cells and is currently used as a synonym for tumours.

Etiology: Papillomatosis of the common carp (Cyprinus carpio) is known to be caused by the Cyprini herpes virus (CHV) and has been detected by in situ hybridization and Oncorhynchus masou virus (OMV). Both of these viruses are lethal, CHV mainly for juveniles. While OMV also causes mortality in adult fish (Hoeger *et al*., 2005).

Clinical sign: Grossly papillomas are growing on the skin and scales of fish. Depending on the presence of pigment cells in the affected area, papillomas can also be pigmented from pink to brownish. In the early stages, only a few, raised pale raised lesions are seen, but laterally numerous large papillomas and petechial hemorrhages on the papilloma. Best example of papilloma lesions is "cauliflower" like papillomas, which occur in and around the mouth. In certain species, some areas of skin are more commonly affected than others, such as lip papilloma in white sucker and papillomas in the fins of smelt (Hutchinson *et al*., 2006).

Epidemiology of epidermal Papillomatosis

Epidemiology of epidermal papillomatosis seems to be complex and is thought to be multifactorial in several fish species. Three major components are known to encompass and interact with the etiology and epidemiology of fish diseases include:- 1) pathogen 2) host and environment.

Pathogens in fish Papillomatosis

Even if there are a lot of etiology papillomatosis. Nevertheless, only two herpes viruses have been recognized to be oncogenic that is inducing neoplasia, for fish.

These oncogenic viruses are herpes virus Cyprini (CHV) and Oncorhynchus masou virus (OMV).

In the case of CHV, the viral genome has been found from several organs of fish in a latent infection: However, at this stage, no virus could be isolated.

Diagnosis: Epidermal papillomatosis is easy to diagnose by: Macroscopic inspection, and palpating by hand on the skin of the fish. The histopathology of papillomas is used for **confirmatory** diagnosis of the disease. Although viral antigens detected from the papillomas of fish, they may not always be evident. Molecular based techniques have only rarely been used for diagnostic purposes in fish papillomatosis.

6.3) Viral Hemorrhagic Septicemia (VHS)

Definition: VHS is a serious systemic disease of fish. VHSV is carried by at least 50 species of marine and freshwater fish. Is an emerging disease of freshwater fish in the Great Lakes region of North America?

Etiology: VHS is caused by the viral hemorrhagic septicemia virus (VHSV or Egtved virus). This virus is a member of the genus Novirhabdovirus, family Rhabdoviridae. VHSV contains a single serotype with three subtypes. VSHV strains differ in their virulence for fish species.

Transmission: VHSV is shed primarily in the urine and reproductive fluids (ovarian fluids, sperm). This virus has also been reported in the feces, but shedding is low. Reservoirs include clinically ill fish and asymptomatic carriers. Transmission can occur through the water or by contact. VHSV is thought to enter the body through the gills or possibly through wounds. Predation on infected fish is also thought to be a route of transmission. Fish-eating birds can introduce VHSV into areas by acting as mechanical vectors.

Clinical Signs: Affected rainbow trout are usually anorexic and may be either lethargic or hyperactive. Swimming behavior can also be abnormal. The coloring is usually darker than normal, but the gills are pale due to anemia and may have petechial hemorrhages. Hemorrhages can also be seen in the eyes and at the base of the fins. Bilateral or unilateral exophthalmia and ascites may be present. A neurologic form characterized only by abnormal swimming behavior. Limited information is available on the symptoms in other species (ICTV, 2007).

Morbidity and Mortality: VHSV infections appear to be particularly common in marine species. These infections are often subclinical. Clinical disease has been reported in freshwater fish and occasionally in marine species. Most epizootics occur on freshwater farms. Clinical disease can occur at any age, but younger fish appear to be most susceptible. Stress is a predisposing factor, and outbreaks can occur in subclinical carriers after a stressful event. The optimal temperature for active infection is 9-12°C (48-54°F).

Diagnosis: Susceptible species with hemorrhages, exophthalmia, nervous signs or other symptoms consistent with this disease. It is diagnosed by virus isolation in cell cultures. **Virus identity is confirmed by**: Virus neutralization, Immunofluorescence (IFA), an enzyme-linked immunosorbent assay or a polymerase chain reaction (PCR)–based assay.

Differential diagnosis include: Infectious hematopoietic necrosis, Enteric red mouth disease and Furunculosis.

Control: Viral hemorrhagic septicemia is a **highly contagious** disease. Quarantines are necessary to control outbreaks. There is evidence that VHSV is transferred from wild fish to farmed fish and vice versa. VHSV is sensitive to many common disinfectants including: Formalin, iodophor disinfectants, Sodium hydroxide and sodium hypochlorite.

Public Health: There is **no indication** that this disease is a threat to human health.

6.4) Infectious hematopoietic necrosis (IHN)

Definition: IHN is a serious viral disease of salmonid fish. This disease was first reported at fish hatcheries in Oregon and Washington in the 1950s.

Etiology: IHN is caused by infectious hematopoietic necrosis virus (IHNV). A member of the genus Novirhabdovirus and family Rhabdoviridae. Virus strains vary in their pathogenicity. IHNV isolates can be grouped into three genetic types, which are correlated mainly with geographic regions. The U genogroup includes isolates from Alaska, British Columbia, coastal Washington watersheds and the Columbia River basin. The L genogroup contains most of the viruses from California and the Oregon coast. The M genogroup contains isolates from Idaho, the Columbia River basin and Europe, as well as a virus from the Washington coast. The M genogroup has significantly higher genetic diversity than the L or U groups.

Transmission: IHNV is transmitted by clinically ill fish and asymptomatic carriers. This virus is shed in the feces, urine, sexual fluids and external mucus. Transmission is mainly from fish to fish, primarily by direct contact, but also through the water. IHNV can survive in water for at least one month, particularly if the water contains organic material. This virus can also be spread in contaminated feed. The gills or the digestive tract have been suggested as the major sites of virus entry. "Egg-associated" (vertical) transmission also occurs. The incubation period is 5 to 45 days (Arias *et al*., 2012).

Clinical Signs: this includes abdominal distension, exophthalmia, darkened skin and pale gills. Long, semi-transparent fecal casts often trail from the anus. Affected fish are typically lethargic, with bouts of hyper excitability and frenzied, abnormal activity. In sac fry, the yolk sac often swells with fluid. In fry less than two months old, there may be few clinical signs despite a high mortality rate. Surviving fish often have scoliosis.

Diagnosis: IHP should be suspected in salmonid fish with typical clinical symptoms and necropsy lesions, Virus identity is confirmed by: virus neutralization, immunofluorescence, enzyme-linked immunosorbent assay (ELISA), DNA probes or polymerase chain reaction (PCR) tests. Nucleic acids can also be identified directly in tissues by PCR (Karvonen *et al*., 2010).

Differential diagnosis: Includes infectious pancreatic necrosis, viral hemorrhagic septicemia and whirling disease.

Control: In areas where this disease is not endemic, **outbreaks** are controlled by: Culling, disinfection, Quarantines and other measures, Where IHNV is endemic, good biosecurity and sanitation decrease the risk of introducing the virus to a farm. Eggs should be disinfected with an iodophor solution and virus–free water should be used to incubate eggs and raise animals (Arias *et al*., 2012).

6.5) Infectious pancreatic necrosis (IPN)

Definition: IPN is a viral infection primarily of **trout and salmon**, but the virus isolated from other fish species.

Etiology: IPN is caused by **infectious pancreatic necrosis virus (IPNV),** A double stranded RNA virus classified within the genus *Aquabirnavirus* (family *Birnaviridae*). Several genogroups have been identified and are described by their different hosts and geographical origins. The most frequently highly virulent strain of IPNV is the **Sp serotype in genogroup 5.**

Signs of disease: Disease signs at the farm, tank or pond level are: Sudden and progressive increase in mortality particularly in faster growing individuals. Cumulative mortality rates from 10% to 90%. Low persistent mortality, Fish lying still on the bottom of tanks or ponds and fish swimming with a spiralling, corkscrew motion.

Gross pathological signs are: long, thin, whitish trailing fecal casts, swollen abdomen, darkening body color, typically pale gills, Exophthalmos (pop eye), lesions and ulcers in pancreas, esophagus and stomach, hemorrhages sometimes present in ventral areas, including the ventral fins, abnormally pale spleen, kidney, liver and heart of fry and intestines empty or filled with clear mucus (Lafrentz *et al*., 2012).

Microscopic pathological signs are: Extensive and/or severe necrosis of acinar pancreatic cells, Focal or generalized necrosis of live rand Sloughing of intestinal mucosa.

Host range: Both marine and freshwater fish species are susceptible to IPNV. The disease spread naturally by **mechanical vectors** such as: **Piscivorous birds** (including passage through the bird digestive system)

and blood feeding parasites and filter feeding mollusks.

Epidemiology: IPNV is highly contagious and fish that survive infection are presumed to become carriers. Asymptomatic carrier fish represent a risk for introduction of disease to healthy stocks. Viral transmission can occur horizontally and vertically. IPNV is shed in faces, urine, spawning fluids and external mucus. Spawning favors the transmission of IPN virus with increased levels of virus excreted in spawning fluids. Outbreaks of disease are most likely to occur when fish are stressed. IPNV can survive in both freshwater and saltwater environments (Arias *et al*., 2012).

Diagnosis: If young trouts suffer a rapidly increasing mortality. Exhibit some of the signs described. Confirmation of IPN requires: Isolation of the virus in cell culture and identification by a serum neutralization test using polyvalent, anti-IPN virus serum.

Differential diagnosis/ similar diseases: infection with salmonid alpha virus (SAV), infectious hematopoietic necrosis (IHN) and viral hemorrhagic septicemia (VHS).

Transmission: Most survivors of infection become lie-long virus carriers and thus shed varying quantities of virus over a long period. This results in a typical transmission of the disease from parents to progeny via the egg, and is probably one of the main factors for the geographical spread of IPN". Egg transmission is the normal means by which the virus is passed from one generation to another."

Prevention: Avoidance is the most effective control measure. This requires the incubation of virus-free eggs and the propagation of IPN-free stock in an uncontaminated water supply. This approach is the method of choice. Success depends on a rigorous fish health inspection program to prevent the introduction or inadvertant spread of IPN.

Treatment: There is no effective treatment for IPN but some success reported with povidone iodine treatments.

Key steps to remove the disease and/or agent from fish populations

Immediate: As there is no effective therapy, the only immediate control of the virus is to **eliminate infected stocks**.

Long term: Inspect all egg sources and hatchery fish populations **at least annually** for the presence of IPNV. Phase out all IPN virus infected brood stock and in the interim, do not transfer eggs from infected brood stocks to any hatcheries which are free of the disease.

7) FISH BACTERIAL DISEASES

7.1) Columnaris disease in fish

Definition: Fish bacterial disease which is available in large numbers of slender, motile bacteria present in the lesions and also called "**saddle-back** disease"

Etiology: - Flavobacterium columnare (F. columnare). It is belongs to the family Flavobacteriaceae. It has **column-like** structures. The pathogen also called **Bacillus columnaris.** The Organisms classified in the order **Myxobacteria.** It is long, thin, **Gram-negative** rods that are motile on agar media by a creeping or flexing motion.

The *F. columnare* cluster is subdivided in **three genomovars** based on: Differences in 16S rRNA sequences, Restriction fragment length polymorphism (RFLP) and DNA-DNA hybridization

Epidemiology: F. columnare is distributed worldwide in fresh water fish. The disease also assails many tropical freshwater aquarium fish. In fish industry, *F. columnare* is the **second most** prevalent bacterium, after *Edwardsiella ictaluri*, to cause disease and mortality. It brings losses estimated at 30 million dollars per year (Lammi, 2000).

Clinical signs: *F. columnare* causes acute to chronic infections and typically affects **the gills**, **the skin and fins**. Clinical sign depend on the virulence of the eliciting strain. Chronically small lesions start as areas of **pale discolorations of the skin. Fin deterioration** then occurs. **Yellowish-white degeneration is visible** in the ventral part of the first gill. **Respiratory distress** caused by damage to the gills (Darwish *et al*., 2011).

Figure 3: Gill lesions in a shubunkin

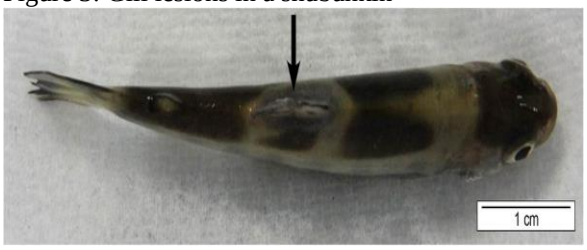

Figure 4: F. columnare induced a saddleback lesion.

Diagnosis: Timely detection of this pathogenic agent is important. The isolation of F. columnare is possible from external lesions and provided that the samples are taken from the edge of recent lesions. F. columnare requires low nutrient media. Depending on the strain, the bacterium grows between 15 and 37°C (optimum 25-30). F. columnare largely displays two colony types on solid media; smooth and rhizoid. Besides isolation, other methods may be used for detecting this pathogen.

This includes: ELISA, FAT, PCR and A loop-mediated isothermal amplification method (LAMP) for rapid detection of Flavobacterium columnare from infected fish organs.

Pathogenesis: The colonization of the fish tissue is to be regarded as a complex multistep process, which can be subdivided into the stages of **attraction**, **adhesion** and **aggregation**, requiring a step-by-step analysis. The exact factors mediating **colonization** have however not yet received the full attention they merit and to date remain largely unidentified. The location of fibrillar structures spanning the gap between the outer membrane and the **mucopeptide** layer might play a role in the **gliding motility** of the F. columnare bacterial cells. F. columnare produces **two types of mucus**. The first one is an **acidic polysaccharide and the 2nd polygalactosamine** (Cunningham et al., 2012).

Preventive measures: Management plays a key role in the prevention of the disease. Water treatment could aid in averting a bacterial outbreak. Salt and acidic bath treatments could be used to disinfect water contaminated by F. columnare. Vaccination and Bath immunization with bacteria was shown to protect. Prolonged feeding (over three months) of formalin-killed bacteria provided high levels of protection and genetic variation in resistance towards F. columnare (Li meng *et al*., 1996).

Treatment: External treatments are possible only in **early stages** of the disease. Drugs which have been used effectively in bath therapies are: chloramphenicol, nifurpirinol, nifurprazine and oxolinic acid. If the disease is in an ***advanced stage***, it is necessary to administer antimicrobials in the feed. Oxytetracycline given orally for up to 10 days proved effective in early as well as advanced outbreak. Nitrofuran can also be administered orally for 3 to 5 days (Cunningham et al., 2012).

7.2) Furunculosis

Definition: Furunculosis is characterized by a generalized bacteremia with focal necrotic swellings in the muscle tissue called **furuncles**. Furunculosis is the most commonly encountered bacterial pathogen in cultured salmonids.

Etiology: Furunculosis is caused by an ***Aeromonas salmonicida and it is*** gram-negative bacterium (Darwish *et al*., 2011).

Epidemiology: The disease occurs worldwide in freshwater and **it** has also been reported in the marine environment. It is known to occur in: North America, Europe, Asia and Africa (Watral and Kent 2007).

Susceptibility: All salmonid species are susceptible. Rainbow trout show some resistance. Young fish are the most susceptible, especially when the water temperatures are > 8º C. In hatcheries, pink and **chum** salmon are less likely to develop Furunculosis and since they are not reared long before being released to seawater (Cunningham *et al*., 2012).

Clinical Signs: In **acute** septicemia where rapid death may occur, Gross clinical signs may not develop. In **sub-acute and chronic** infections: **Body darkening**, lethargy, Loss of appetite is associated with: The typical necrosis in the muscle, visible as a swelling under the skin. These lesions ulcerate producing deep craters. **Erythema, petechiation and exophthalmia,** the abdomen of the fish may be **distended with internal ascitic** fluid and **Bloody fluid** may be discharged from the anal vent (Cunningham et al., 2012).

Transmission: Horizontal transmission to susceptible fish is via the water column or by the fecal-oral route, Diseased or carrier fish are point sources of infection, increasing water temperature **exacerbates** the incidence and intensity of infection and **No vertical** transmission of the bacteria been reported.

Diagnosis: **Presumptive diagnosis is made by:** Culture of a Gram-negative. **Oxidase** positive (an oxidase negative isolate has been described). **Non-motile** bacterial rod from blood, kidney or lesions on TSA or furunculosis agar with the production of a ***brown diffusible pigment,*** But not all bacteria produce pigment. **Diagnosis is confirmed by:** Biochemical tests, slide agglutination and FAT test specific for *A. salmonicida* (Arias *et al*., 2012).

Treatment: In nature, the disease usually results in mortality, In a hatchery, prognosis for the fish population is good. If the condition is caught early and antibiotic therapy is initiated. Public health: There are no human health concerns associated with A. salmonicida.

Figure 5: Typical furuncle lesion on adult sockeye salmon with furunculosis.

7.3) Vibriosis

Definition: Vibriosis is ubiquitous, typical **Gram-negative** acute septicemias with bacterial foci, necrosis, hemorrhaging and inflammation in most fish tissues.

Etiology: The genus ***Vibrio*** contains significant bacterial pathogens of marine fish that cause vibriosis. The primary pathogens include: *V.* (*Listonella*) *anguillarum*, *V. ordalii* and *V. salmonicida*. In addition, *Vibrio alginolyticus* may occur as a secondary invader. ***V. vulnificus*** is **generally restricted** to European and Japanese waters.

Epidemiology: Because vibriosis has occurred in an extensive number of fish species worldwide, most marine fish species are likely to be susceptible. All species of Pacific salmon and trout are susceptible to vibriosis that often involves *V. anguillarum*. **Coho salmon** seem to be more resistant while **chum and Chinook salmon** are very susceptible.

Clinical Signs: Characteristic clinical signs of vibriosis include: **inflammation and reddening** along the ventral and lateral areas of the fish, **petechial hemorrhaging** that develops at the base of fins, vent and within the mouth, Acute cases exhibit a darkened body with swollen, cutaneous lesions that ulcerate, releasing blood, there may also be **corneal opacity** and Internally, the intestine may be distended with a clear, viscous fluid.

Transmission: Horizontal transmission occurs from organisms in the water or contact between fish. **Outbreaks** have occurred in freshwater fish **fed carcasses** of marine fish. In Alaska, disease does not usually occur **until** seawater temperatures reach 8°C.

Diagnosis: Presumptive diagnosis is made by: Observing motile, curved Gram-negative bacterial rods in spleen squashes or peripheral blood smears, Bacteria can be isolated on tryptic soy agar (TSA) and sometimes requiring 1.5% NaCl. Confirmatory diagnosis is made using: biochemical test or slide agglutination tests (Noga, 2010).

Treatment: Epizootics of vibriosis in wild fish populations are rare but result in significant fish mortality. High mortality if not treated with antibiotics. Several **licensed vaccine preparations for aquaculture** have been effective in the control of vibriosis. **Public health**: **It is not considered to be human pathogens** (Cunningham *et al*., 2012).

7.4) Dropsy

Dropsy Is abdominal distention due to ascites, or the effusion and collection of fluid freely throughout the coelomic cavity.

Etiology: Dropsy may be caused by a **variety of potential etiological agents**, both infectious and noninfectious. *Rhabdovirus carpio* is the causative agent of dropsy. Parasitism of the coelomic cavity can be agent of dropsy (Yamamoto, 1975).

Clinical presentation: Clinically affected fish display coelomic distension of varying degrees of severity. Severely affected scaled fish may have protrusion of the scales. That causes them to stand erect from the body surface, causing **lepidorthosis** or a "**pine cone"** appearance.

Differential diagnosis: The differential diagnoses are numerous including: Not only the potential etiologies that lead to free coelomic fluid accumulation, but also other various causes of coelomic distension like: Neoplasia, organomegaly and Gastrointestinal obstruction, egg-binding (Karvonen *et al*., 2010).

Diagnosis: Diagnostic imaging may be useful for determining: If coelomic distention is related to fluid accumulation or some other cause, if free coelomic fluid is suspected or observed, coelio centesis. Evaluation of the coelomic fluid through cytology and Microbial (i.e., bacterial, viral and fungal) culture may often be diagnostically useful.

Treatment: Treatment and control options depend upon the underlying etiology.

Figure 6: Severe coelomic distention in a female tilapia.

7.5) Tuberculosis/Mycobacteriosis

Mycobacterial diseases of fish are common, particularly in intensive aquaculture systems and display aquaria. **All** fish are susceptible to Mycobacteriosis. Mycobacteriosis was formerly a problem in the salmon industry. Mycobacteriosis is a center important infectious disease in zebra fish colonies. Mycobacterium causes a **chronic** disease, usually characterized by wasting. Mycobacteriosis cause **granulomatous inflammation**.

Etiology: are M. marinum, M. fortuitum, and M. chelonea. The **first two** species of mycobacteria that are considered as widely distributed and important pathogens of fishes, **The differences between them are relatively small**, Mycobacterium marinum grows more **slowly** and Produces orange pigment if exposed to light; M. fortuitum grows **faster,** and produces creamy-colored growth. Both are capable of causing disease in all vertebrates, including humans. Mycobacterium marinum is usually found in tropical **saltwater** fishes and M. fortuitum in **freshwater** fishes.

Signs of the disease: Among the signs are: Emaciation, loss of appetite, sunken abdomens, Shallow grayish irregular ulcerations, Fin rot, deformities of the vertebral column Mandible, exophthalmos, and Loss of one or both eyes, Fish become listless and Show difficulties in maintaining balance and Small grayish tubercles or nodules in the liver (pathognomonic) (Arias *et al*., 2012).

Pathogenesis: Pathogenesis of mycobacteriosis in fishes is not known. It seems likely that considerable destruction of internal organs results in emaciation and death, when there are extensive open dermal lesions it seems likely that, as in corynebacterial kidney disease. Plasma proteins are lost. The mycobacteria in fish lesions produce toxins (Karvonen *et al*., 2010).

Diagnosis: Diagnosis of mycobacteriosis is relatively easy. Lesions caused by these bacteria contain rod-shaped, ***gram-positive*** bacilli. Differentiation from other gram-positive rods is made by the acid-fast (Ziehl-Neelsen) staining method. Establishing the presence of acid-fast bacteria in a lesion is sufficient for routine diagnosis. Mycobacteria may be isolated on special media that are commercially available. Separation of Mycobacterium fortuitum from M. marinum requires culturing on differential media and performance of numerous tests (Karvonen et al., 2010).

Transmission: infection is transmitted by the oral route and it can be infected through skin abrasions. **Incubation period**: The incubation period varies greatly and depends on: susceptibility, temperature, and the severity of exposure. It is difficult to specify the length of incubation (the time from infection to the appearance of the first signs of the disease).

Prevention: Prevention is thus far based solely on avoidance of infection. Therefore, suspected or infected fishes should never be introduced into a pond or aquarium. Quarantine is important in ornamental fishes. Since infection by the oral route is well documented, food containing unsterilized fish flesh should not be used. If water is re circulated among a number of aquariums. It should be continuously filtered and decontaminated by ultraviolet radiation.

Treatment: Tests performed with pure cultures of mycobacteria isolated from diseased fishes show that their resistance to drugs is high. The antibiotic **Kanamycin mixed with food** was effective in **curing** mycobacteriosis among ornamental fishes. The recommended dosage is **0.01% by weight, in food**. It has recently been reported that Kanamycin is absorbed from water by fishes. However, the significance of this absorption in the control of fish mycobacteriosis is as yet unknown.

7.6) Bacterial gill disease

Definition: Bacterial gill disease is a common external infection of hatchery reared salmonids and occasionally of warm water species reared under intensive conditions.

Etiology: Commonly caused by filamentous bacteria within the genus Flavobacterium (most often F. branchiophilum). Name changes of Flavobacterium: Previously known as members of the Myxobacteria. First placed in the genus Cytophaga, later changed to Flexibacter and now called Flavobacterium.

Susceptibility: All cultured salmonids are susceptible and the disease is found worldwide. In Alaska, sockeye, Chinook and Coho salmon appear to be most susceptible. Adults and yearlings are less susceptible than **fry** and fingerlings.

Clinical sign: Lethargic and a loss of appetite occurs, Large numbers of diseased fish **gather near the screen or outlet of the pond**, Exhibit exaggerated opercular movements, An increase in **mucus o**n the head and upper body. Gasping at the top of the water, and sluggish response to external stimuli such as: hitting the side of a tank Or waving a hand over the fish. The fish will swim high in the water column and align in a "**soldier-like**" fashion.

Transmission: Transmission occurs horizontally. Predisposing factors for **epizootic outbreaks are**: Sub-optimal environmental conditions and suspended solids or abrasive feeds. The incubation period can be as little as 24 hours or up to several weeks and the most commonly during periods of colder water temperatures below 5°C (Yamamoto, 1975).

Diagnosis: Fish with BGD have **pale, swollen gills, flared opercula**, are listless and do not feed well. Large numbers of filamentous bacteria are found attached to the gills causing: Epithelial hyperplasia and possibly fusion or clubbing of gill lamellae. **The causative organisms are:** Filamentous bacteria, Gram-negative, non-motile (or have gliding motility), grow on Cytophaga or TYES agars and Rod-shaped bacteria (Arias *et al*., 2012).

Treatment: Early intervention in the progression of the disease may reduce fish mortality and in a hatchery setting external chemical treatments with **hydrogen peroxide** to control the bacteria. A number of compounds are effective for the treatment of bacterial gill disease. Researches recommended **potassium permanganate** (KMnO4) with certain precautions at l-2 ppm (Arias et al., 2012).

8) FISH FUNGAL DISEASES

8.1) Dermatomycosis

Etiology: Various member of class Oomycete. And the **clinical sign**: Appearance of fine hair like tuffs hanging from the infected areas and the fin become eroded and hemorrhagic at the latter stage of disease. Infected eggs tend to stick together and finally die (Arias et al., 2012).

Treatment: Use copper sulphate solution (CuSO4*5H2O), at the dose of 1g per 10L of water for 30 minutes q 24hrs for 10 days, Potassium permanganate (KMnO3) at the dose of 1g per 10L of water for 30 minutes (treatment is repeated every 12 hours for 10 days) and Basic Violet K (it is only effective in the early stages of disease).

Prophylaxis: Do not take food from ponds where outbreaks of the disease and fish's death have occurred. The disease is best prevented by providing adequate aquarium maintenance routine and a balanced diet. Environmental management is essential: that is any uneaten food, dead mollusks and dead fish should be removed from the tank.

8.2) Branchiomycosis

Etiology: Branchiomyces demigrans and B. sanguinis (Phycomycetes Archiomycetes).

Clinical sign: This disease is seen in the **blood vessels of the gill tissue** and it **obstructs the circulation of blood through the gills**, which makes the gills lose their **bright red color**.

Treatment: Diseased fish can be treated with **malachite green** at: 0.1mg/l for extended periods of time or 0.3mg/l for 12 hours. Ponds with **enzootic branchiomycosis** should be: dried and treated with: Calcium oxide (quicklime) or 2 to 3 kg copper sulphate per **hectare**.

Prophylaxis: Strict sanitation and disinfection are essential for disease control. Dead fishes should be collected and daily and burned or deeply buried. Transportation of infected fish areas to non-infected areas must be prevents. Increase of water supply help in control of that disease.

8.3) Epizootic Ulcerative Syndrome (Red-spot Disease)

Definition: (EUS) or 'red-spot' is known colloquially, is an **ulcerative** syndrome of fish. It begins as a small area of reddening over a single scale, which subsequently spreads to involve a number of adjacent scales; this is the characteristic '**red spot**'. As the condition progresses, the '**red-spot**' expands: It results deepens, giving a deep ulcer and sometimes extends into the abdominal cavity (Arias *et al.*, 2012).

Etiology: A pathogenic **fungus, Aphanomyces invadans** causes EUS. Infection occurs when ***motile spores*** in the water are attracted to the skin of fish. The spores penetrate the skin and germinate, forming fungal filaments or hyphae. The hyphae invade widely into the surrounding skin and deeply into underlying muscle tissues: that is **resulting in extensive ulceration and destruction of tissues.**

Treatment: There are no specific control measures in fish for EUS in natural environments. In captive fish, early 'red-spot' lesions may respond to topical treatment with an antiseptic **iodophore** solution.

Prophylaxis: Increasing **salinity** of holding waters may prevent outbreaks of EUS in aquaculture ponds. Fish from infected waterways, especially those with lesions of EUS, should not be relocated to other waterways (Arias *et al*., 2012).

8.4) Saprolegniasis (Winter Fungus)

Definition: Saprolegniasis is the most common and economically important fungal disease of cultured **channel catfish** is winter saprolegniasis. Saprolegniasis describes any **cotton-like growth** adherent to skin or gills that include several genera of molds.

Etiology: is caused by water molds (**oomycetes**) mostly in the genus **Saprolegnia**. Genetic sequencing places oomycetes in the class **Oomycota**, phylum **Heterokontophyta**. Related to photosynthetic brown algae and saprolegnia (parasitica) are primary pathogens producing a systemic disease.

Epidemiology: All freshwater fish species and incubating eggs and other lower aquatic animals. Both vertebrates/invertebrates worldwide are susceptible to saprolegniasis.

Clinical sign: The mold produces **white/brown cotton-like foci** on the surface of the skin and/or gills. Early foci are **pale** with peripheral areas of **erythema** and central zones of lifted scales that frequently ulcerate, exposing underlying musculature. Systemic infections produce **mycelial**

masses in the gut and **viscera** causing: Peritonitis, Extensive hemorrhage and Necrosis and adhesions (Arias *et al*., 2012).

Transmission: Transmitted through ambient water by infectious biflagellated zoospores released from hyphal sporangia. Systemic infections in cultured fish occur by ingestion of uneaten food that has been colonized by mold hyphae. Environmental stress plays an important role in the etiology of the external disease. **Outbreaks** occur primarily after minor injury from handling or during crowded conditions.

Diagnosis: Diagnosis of winter fungus is based on typical gross clinical signs of white, cottony tufts of hyphae on the **skin, gills and other surfaces** of infected fish or eggs. **Wet mounts of mycelium from lesions show:** large, branching, non-septate hyphae. Terminal ends of older hyphae form club-shaped sporangia containing biflagellated zoospores. The mold can be isolated on cornmeal or potato agar.

Treatment: Maintaining **sufficient oxygen concentrations** (4 to 5 ppm) may also be important in avoiding winter Saprolegniasis. In the hatchery environment external fungus infections can be treated successfully with **1hr formalin drips.**

9) PROTOZOAN/PARASITIC FISH DISEASES

9.1) Costiasis

It is caused by: Costia necatrix. Symptom: It causes the skin of the infected fish to become cloudy and milky.

Treatment: Due to its inability to live in water above 28°C (82.4°F), treat as if it was Ich by using a commercial Ich treatment or technique. Other recommended treatments include: Malachite Green, Potassium Permanganate, Acriflavine and strong salt baths of 3%.

Prophylaxis: Newly bought fish are quarantined for 30 days. Before introducing new fish into the aquarium, give them three short-term therapeutic baths. It is not advisable to introduce pond plants without first disinfecting them. Nets, scrapers, feed boxes, thermometers, pulverize and other equipment should not be shared between several aquaria.

9.2) Whirling Disease

Caused by: A microscopic parasite called ***Myxobolus cerebralis*** and the **Symptoms** includes: Damaging cartilage, Whirling disease can kill young fish directly or cause infected fish to swim in an uncontrolled whirling motion.

Treatments: In watersheds where Myxobolus cerebralis is present in wild populations, there is currently no cure or means to eradicate it.

Prophylaxis: The key to preventing the spread of whirling disease is to prohibit the movement of the parasites from infected areas. Never transport live fish from one body of water to another. Do not dispose of fish heads, skeletons or **entrails** in any body of water.

9.3) Ichthyophthiriusis

It is caused by: *Ichthyophthirius multifilis* and its Symptoms includes: Small white spots resembling sand, Fish scratch against rocks and gravel, In advanced stages fish become lethargic and Redness or bloody streaks in advanced stages (Darwish et al., 2012).

Treatment: Raise water temperature, Medicate for 10-14 days, Reduce medication when treating scaleless fish, Discontinue carbon filtration during treatment and Perform water changes between treatments (Darwish et al., 2012).

Prophylaxis: Infection can be effectively controlled only by destruction or elimination of the free dividing tomonts or the tomites they release. In warm water systems (24–28°C), three to four daily transfers of fish to clean tanks will effectively reduce infection, while enabling the fish to develop tolerance to reinfections. Vacuum suction (infusion of fluid)

9.4) Diplostomosis

It is Caused by: Trematodes, Diplostomulum, and Symptoms: Small black spots on the body. Treatment: Black spot is generally easy to cure, there are a number of commercially available treatments and preventatives and it is fairly easy to treat with salt baths (Boutin *et al*., 2012).

9.5) Gut blocking

It is caused by: A cestodes, Eubothrium its symptoms and results mechanical damage to the gut (Boutin *et al*., 2012).

10) FISH CRUSTACEAN DISEASES

10.1) Argulosis disease

Argulus or fish louse is a large parasite that attaches to the external surface of host and can be easily seen with the help of our naked eye. Argulus is uncommon in fresh water aquarium fish but may occur if wild or pond raise fish are introduced into the tank. It is especially common in goldfish and koi.

Clinical sign: Debilitation or secondary bacterial or fungal infection may occur.

Prevention and treatment: Non drug treatment is removing the argulus manually by forceps. And drug treatment a 3% salt dip followed by 0.2% prolonged immersion to control it in goldfish and koi ponds or organophosphate compounds like dipterex, trichlorofon are used (Darwish et al., 2012).

10.2) Ergasilus disease

Ergasilus are often incidental findings on wild or pond raise fish and probably cause a few problems in small numbers. However their feeding activity causes severe focal damage and heavy infestation can be debilitating. Mostly affect the gill

of fresh water fish and commonly seen in warm weather.

Clinical sign: Debilitation or secondary bacterial or fungal infections can be seen.

Prevention and treatment: Drug treatment a 3% salt dip followed by 0.2% prolonged bath for 3 weeks (Boutin *et al*., 2012).

10.3) Lernaeasis disease

Lernaea also known as anchor worm is a common parasite of goldfish and koi. The copepod attaches to fish, mates and the male dies. The female then penetrates under skin of fish and differentiate into an adult.

Clinical sign: Debilitation or secondary bacterial or fungal infection may occur.

Prevention and treatment: Drug treatment a 3% salt dip followed by 0.2% prolonged immersion to control it in goldfish and koi ponds or organophosphate compounds like dipterex, trichlorofon are used. In permanent bath 0.25-0.5 ppm (0.1 ppm cold water ponds), 4 treatment at 4-7 days intervals (Darwish *et al*., 2012).

11) ACKNOWLEDGEMENTS

I would like to extend sincere acknowledgment to: Ebinat livestock resource development promotion office and all Ebinat animal health and animal production worker for their continual encouragement and motivation to develop this fish disease and fish production manual, other veterinarians who are living in and around Ebinat for their advice and technical support, plant cultivators and experts in the working districts, for their helps during constructing and development of this manual, for instance collecting of information. I also would like to thank gratefully for all respondents/ informants interviewed in this interesting manual developments.

12) REFERENCES

[1]. Ackerman, P.A. and Iwama, G.K. (2001). Physiological and cellular stress responses of juvenile rainbow trout to vibriosis. *Journal of Aquat. Anim. Health*, 13(2): 173-180.

[2]. Afonso, L.O., Basu, N., Nakano, K., Devlin, R.H. and Iwama, G.K. (2003). Sex related Differences in the organismal and cellular stress response in juvenile salmon exposed to treat bleached kraft mill effluent. *Fish Physiology, Biochem.* 29(2): 173-179.

[3]. Ang, P., Rattana-Apiromyakij, N. and Goh, C.L. (2000). Retrospective study of Mycobacterium marinum skin infections. *International Journal of Dermatology* 39:343−347.

[4]. Arias, C.R., Cai, W., Peatman, E. and Bullard, S.A.(2012): Catfish hybrid Ictalurus punctatus I. furcatus exhibits higher resistance to columnaris disease than the parental species. *Disease Aquat Organ*, 100:77–81.

[5]. Astrofsky, K.M., Schrenzel, M.D. Bullis, R.M. Smolowitz, R.A. and Fox, J.G. (2000). Diagnosis and management of atypical Mycobacterium spp. Infections in established laboratory zebrafish (Brachydanio rerio) facilities. *Comparative Medicine* 50(6):666−672.

[6]. Au, D. (2000). The application of histocytopathological biomarkers in marine pollution monitoring: *A review. Mar. Pollut*. Bull. **48**(910): 817834.

[7]. Boutin, S., Bernatchez, L., Audet, C. and Derôme, N. (2012): Antagonistic effect of indigenous skin bacteria of brook charr (Salvelinus fontinalis) against Flavobacterium columnare and F. psychrophilum. *Veterinary Microbiology*, 155:355–361.

[8]. Brock, J.A. and Bullis, R. (2001). Disease prevention and control for gametes and embryos of fish and marine shrimp. *Aquaculture* 197:137−159.

[9]. Cunningham, F.L., Jack, S.W., Hardin, D. and Wills, R.W. (2012): Pond-Level risk factors associated with columnaris disease on Mississippi commercial catfish farms. *Journal of Aquat Anim Health*, 24:178–184.

[10]. Darwish, A.M., Bebak, J.A. and Schrader, K.K. (2012): Assessment of Aquaflor, copper sulphate and potassium permanganate for control of Aeromonas hydrophila and Flavobacterium columnare infection in sunshine bass, Morone chrysops female x Morone saxatilis male. *Journal of Fish Disease*, 35:637–647.

[11]. Darwish, A.M., Mitchell, A. and Straus, D.L. (2011): Evaluation of a 4-h static copper sulphate treatment against experimental infection of Flavobacterium columnare in channel catfish (Ictalurus punctatus). *Aquaculture Res*, 43:688–695.

[12]. Deane, E. and Woo, N.S. (2006). Impact of heavy metals and organochlorines on histopathology 70 and histocytology 70 gene expression in black sea bream fibroblasts. *Aquat. Toxicol*. 79(1): 915.

[13]. Decostere, A., Hermans, K. and Haesebrouck, F. (2004). Piscine mycobacteriosis: a literature review covering the agent and the disease it causes in fish and humans. *Veterinary Microbiology* 99:159−166.

[14]. Diamant, A., Banet, M., Ucko, A., Colorni, W., Knibb, A. and Kvitt, H. (2000). Mycobacteriosis in wild rabbit fish Siganus

rivulatus associated with cage farming in the Gulf of Eilat, Red Sea. *Diseases of Aquatic Organisms* 39:211−219.

[15]. Ferreira, R., de Souza Fonseca, A.M., Afonso, M., Gomes da Silva, M.H., Saad, Z. and Lilenbaum, W. (2006). A report of mycobacteriosis caused by Mycobacterium marinum in bullfrogs (Rana catesbeiana). *The Veterinary Journal* 171:177−180.

[16]. Gauthier, D.T. and Rhodes, M.W. (2009). Mycobacteriosis in fishes: a review. The *Veterinary Journal* 180:33−47.

[17]. Harmache A, LeBerre M, Droineau S, Giovannini M. and Brémont M. (2006). Bioluminescence imaging of live infected salmonids reveals that the fin bases are the major portal of entry for Novirhabdovirus. *Journal of Virology*; 80:36, 55-9.

[18]. Hoeger, B., Hitzfeld, B., Kollner, B., Dietrich, D.R. and van den Heuvel, M.R. (2005). Sex and low level sampling stress modify the impacts of sewage effluent on the rainbow trout (Oncorhynchus mykiss) immune system. *Aquat. Toxicol*. 73(1): 7990.

[19]. Hutchinson, T.H., Ankley, G.T., Segner, H. and Tyler, C.R. (2006). Screening and testing for endocrine disruption in fish biomarkers as "sign posts," not "traffic lights," in risk assessment. Environ. *Health Perspect*. 114 (Supplement 1): 106114.

[20]. International Committee on Taxonomy of Viruses [ICTV]. (2007). Universal virus database, version 4. 00.061.1. 01.062.0.06.001. Infectious hematopoietic necrosis virus [online]. ICTV; 2006. Available at: http://www.ncbi.nlm.nih.gov/ICTVdb/ICTVdB*. Accessed 13 Jul 2007.

[21]. Karvonen, A., Rintamäki, P., Jokela, J. and Valtonen E.T. (2010). Increasing water temperature and disease risks in aquatic systems: climate change increases the risk of some, but not all, diseases. *International Journal of Parasitology*, **40**:1483–1488.

[22]. Lafrentz, B.R., Shoemaker, C.A., Booth, N.J., Peterson, B.C. and Ourth, D.D. (2012): Spleen index and mannose-binding lectin levels in four channel catfish families exhibiting different susceptibilities to Flavobacterium columnare and Edwardsiella ictaluri. *Journal of Aquatic Anim Health*, 24:141–147.

[23]. Lammi, R. (2000). Effects of primary and secondary treated bleached kraft mill effluents on the immune system and physiological parameters of roach. *Aquat. Toxicol*. 51(1): 55-67.

[24]. Li meng, H., David, J. Wise, H. and Edwin, H. and Robinson, J. (1996). Chemical Prevention and Treatment of Winter Saprolegniasis ("Winter Kill") in Channel Catfish Ictalurus punctatus. *Journal of the World Aquaculture Society* 27(1):1-6.

[25]. Noga, E. J. (2010). Problem 40. Lymphocystis. *In* Fish disease: diagnosis and treatment. 2nd edition. Wiley-Blackwell. Ames, Iowa. Pp. 171–173.

[26]. Watral, V. and Kent, M.L. (2007). Pathogenesis of Mycobacterium spp. in zebrafish (Danio rerio) from research facilities. *Comparative Biochemistry and Physiology, Part* C 145:55−60.

[27]. Yamamoto, T. (1975). Frequency of detection and survival of infectious pancreatic necrosis virus in a carrier population of brook trout (Saluelinusfontinali) in a lake. *Journal of Fish. Res. Boar2 Can*. 32: 568-570.

[28]. Yamamoto, T. (1979). Infectious pancreatic necrosis virus: Quantification of carriers in lake populations during a 6-year period. Journal of Fish. *Res Board Can*. 36:562.56.

4/25/2024