Health Management in Marine Cage Culture of Finfishes

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Background

Presently, the aquaculture development is towards increased intensification and commercialization of fish production with an intention to maximise profits in low volume-high density mode. Global food fish production from aquaculture reached 52.5 million tons in 2008, compared to 32.4 million tons in 2000 (Saravanan *et al.*, 2012). Like any other farming sectors, the likelihood of occurrence of diseases increases as culture operations intensify and expand involving diverse species and eco-system. Thus, the aquaculture industry has been overwhelmed with its share of diseases caused by viruses, bacteria, parasites, etc. Along with feed and seed, diseases are also being considered as one of the factors influencing the profitability of the farming since diseases are now a primary constraint to the development and expansion of culture of many aquatic species. Several factors can be attributed to the present situation of disease problems in aquaculture: 1. increased globalization of trade of live aquatic animals and their products; 2. the intensification of aquaculture mediated through the translocation of brood stock, larvae, fry and fingerlings; 3. the development and intensification of ornamental fish trade; 4. interactions between cultured and wild fish populations, especially in cage farming in natural waters; 5. inadequate biosecurity measures; 6. Absence of vaccines for major bacterial and viral diseases.

A unique feature of farming in natural aquatic environment is that once a pathogen is introduced into the farming system and becomes, treatment of the infected animals is not possible due to the limitation in using drugs to avoid environmental contamination. Since large numbers of animals are kept at high density in close proximity, aquaculture systems can create a conducive environment for infectious disease to establish and spread. Compared to terrestrial farming system, aquaculture poses a continuous threat for the emergence of new diseases due to new fish species being cultivated.

The nature and severity of diseases are mostly influenced by the species of fish being cultured, conditions in which the fish are reared and farm management. Fish cultured in floating cages in natural waters are particularly susceptible to diseases when

environmental variables such as temperature, salinity, dissolved oxygen and suspended particles suddenly or widely alter.

Generally, compared to temperate conditions, progression to disease once the pathogens are established in warm water environments is rapid. Like terrestrial animal diseases, fish diseases are also classified into infectious and noninfectious diseases. Infectious diseases of are caused by various infectious agents like viruses, bacteria, parasites, or fungi. Non-infectious diseases are generally due to environmental stress, chemical contaminants or nutritional deficiencies.

Economic Significance of Aquatic Animal Diseases

Aquatic animal diseases have profound social and economic impacts on the public, businesses and economies that depend directly or indirectly on aquatic animal production. Considerable importance is being given for the study of economic importance of aquatic animal diseases at the farm and national level, while information on many of the diseases of maricultured species is currently not available. Understanding the economic importance of aquatic animal diseases at the national level necessitates its understanding at the farm level, so that measures in can be taken up for the effective allocation of resources to develop required control and prevention strategies for sustaining the aquatic production and enhancing profitability of farming.

Infectious Diseases of Maricultured Finfish Caused by Viruses

Viruses are microorganisms that can replicate only inside the living cells of other organisms. Viruses can infect all types of life forms including animals, plants and microorganisms, including bacteria and archaea. Virus particles or virions normally consist of three components: i) genetic material made up of either DNA or RNA which carries the genetic information; ii) a coat protein that protects these genetic materials; iii) a lipid envelope that surrounds the protein coat which may be absent in some viruses. The shape of viruses may be helical or icosahedral or more complex structures.

Fish viruses have been the subject of research interest in the past two decades. Compared to diseases caused by freshwater fish viruses, there have not been extensive studies on marine fish viruses. Establishment of various fish cell lines lead to path breaking research in fish virology in the recent years. Major viral groups under which fish viruses can be classified include herpes virus, iridovirus, rhabdo virus, reo virus, noda virus and calci virus. However, the most lethal viral disease-causing enormous loss to finfish farming is the disease caused by betanodavirus.

Viral nervous necrosis: Betanodavirus is one of the genera making up the family Nodaviridae which is the etiological agent of Viral Nervous Necrosis (VNN) also known as encephalomyelitis and vacuolating encephalopathy and retinopathy. This virus has remained as a major threat for the establishment and expansion of Asian seabass (*Lates calcarifer*) and striped jack (*Pseudocaranx dentex*). The disease was first documented in 1990 in hatchery-reared Japanese parrotfish (*Oplegnathus fasciatus*) in Japan and Asian sea bass in Australia. Later, it was reported in turbot (*Scophthalmus maximus*), European sea bass (*Dicentrarchus labrax*), redspotted grouper (*Epinephelus akaara*), striped jack (*Pseudocaranx dentex*) and more recently in cultured warm-water and coldwater marine fish species throughout the world. An Indian strain of betanodavirus belonging to RGNNV group was isolated from Asian seabass juveniles reared in a brackish water farm in Bhimavaram in Andhra Pradesh in 2012. Outbreak of mortality due to nodavirus infection in Asian seabass juveniles cultured in freshwater cages in the southwest coast of India has also been reported. Mortality of Asian seabass juveniles cultured in indoor cement tanks as well as open sea cages and in cobia cultured in cages in India associated with RGNNV was also recorded.

Betanodaviruses can infect fish species belonging to tropical, sub-tropical, or temperate waters. These viruses can multiply at an optimum temperature depending on the strain of the virus. For RGNNV, the optimum temperature requirement is 25–30°C while for SJNNV, it is 20–25°C. Mostly, betanodaviruses are a concern in marine fish species. The species susceptible cobia, sea bass, seabream, bluefin tuna, grouper, halibut, surgeonfish, lined surgeonfish and tiger puffer. The freshwater fish species susceptible to betanodaviruses include tilapia and the guppy.

In farming system stress factors like high density, transportation, and high temperature can act as predisposing factors making the fish susceptible to VNN. Although young fishes are more susceptible, older fishes may also get infected especially when water temperature is high.

During the acute stage of the disease, when the mortality is very high, especially in juveniles, there would be no gross lesions on the body surface or gills. However, affected juveniles and older fish show an abnormal swimming behaviour such as spiral, whirling, floating with inflation of swim bladder, or lying down at rest, circling on their own axis. This erratic swimming behaviour may not be noticed in infected fish larvae. Grossly, the brain is oedematous and, in many cases, severely congested.

Microscopically, lesions are characterized by severe vacuolation and necrosis of the central nervous system. In general, the anterior brain is more severely affected when

compared to the posterior part of the brain and spinal cord. Larvae of the fish are more severely affected by betanodaviruses than juveniles. The most characteristic lesion in the fish larvae is the presence of vacuoles in the grey matter of the brain which are intracytoplasmic. Basophilic, intra cytoplasmic inclusions have been reported in brain cells of Asian seabass. Lesions in the retina of the infected fish have also been described in all Species. The lesions include vacuolation of the cellular components of the retina especially the bipolar and ganglionic nuclear layers. Under transmission electron microscopy, fish betanodaviruses appear icosahedral, non-enveloped with a mean diameter of about 25 nm. The virions may be membrane bound by endoplasmic reticulum or are free in the cytoplasm and may present as paracrystalline arrays. Cells containing virions normally include neurones, astrocytes, oligodendrocytes and microglia cells.

VNN can be diagnosed by 1. Demonstration of characteristic vacuolar lesions in the brain or retina by light microscopy; 2. Detection of virions and viral antigens by electron microscopy and serology; 3. Detection of viral nucleotides by molecular techniques including RT PCR, RT Nested PCR, cloning and sequencing by designing the primers for the strain of interest; 4. Tissue culture of virus in a suitable cell line.

Betanodaviruses are highly resistant to various environmental conditions, and they can survive for a long time in sea water. The disease can also be reproduced by simple cohabitation of the healthy fish with infected fish. Control measures are including imposing strict bio- security to exclude the virus from the farm premises. The broodstocks should be tested for the presence of viruses in the gonadal tissues and VNN specific antibodies in serum and any positive fish should be culled at first.

Red sea bream Irodivirus Infection: Red Sea bream iridovirus disease is caused by Red sea bream iridovirus (RSIV) of Megalocytivirus. This disease is an emerging epizootic disease of fish having a significant impact on marine and brackish aquaculture systems. It is caused severe mortality in many cultured and wild fish species of several countries. So far, red sea bream iridovirus disease has been reported from more than 30 cultured marine fish species of many east and Southeast Asian countries. Recognizing its potential impact on the fisheries sector, World Organisation for Animal Health has listed this disease as a reportable disease.

The iridovirus infected fish are lethargic, swim aimlessly and exhibit anaemia, petechiae of the gills and enlargement of the spleen. The mortality ranges from 20–60%. Histopathological findings of the disease include eosinophilic degenerated cells and basophilic enlarged cells in the spleen, heart, kidney, liver and gills. Appearance of

inclusion body-bearing cells is reported to be the pathognomonic lesion in the infected fish. The presence of the virus can also be confirmed by transmission electron microscopy examinations which show viral particles in all the vital organs like spleen, liver, kidney and brain. The viral particles are found to have a central spherical highly electron-dense nucleoid, surrounded by an electron translucent peripheral zone.

Although the disease can be diagnosed based on histopathology and electron microscopy to some extent, these tools do not provide sufficient and precise information for identification of different Megalocytivirus species. The recommended method by World Organization of Animal Health is the use of molecular diagnostics to differentiate RSIV and ISKNV employing two primer sets as a confirmation assay.

Megalocytivirus has a wide host range with mostly similar clinical signs. The steps towards disease prevention include achieving alkaline pH around of the system by disinfecting the reservoirs such as ponds and tanks with sodium hypochlorite or potassium permanganate at 100–200 mg/l or higher.

Infectious Diseases of Maricultured Finfish Caused by Bacteria

Diseases of marine finfish caused by pathogenic bacteria have been one of the limiting factors for the sustainability of aquaculture and among infectious diseases, those caused by bacterial pathogens remain to be the largest single cause of economic losses in aquaculture world over (Colorni *et al.*, 1981). It is noteworthy to mention that a large number of aquatic marine bacteria are opportunistic as they are normally present in seawater, sediments and fish gut. They generally do not cause infection in fish under normal environmental conditions. It is evident that pathogenic bacteria ubiquitous to marine and estuarine ecosystems are the significant drivers of mortality due to bacterial diseases in marine fish culture (Pruzzo *et al.*, 2005).

Vibriosis: The most common Gram-negative bacteria associated with marine fish belong to the family Vibrionaceae. Vibriosis, disease caused by the bacteria of the species Vibrio, is among the most common diseases leading to considerable mortality and economic loss to cultured fish and shellfish in the tropics. Juveniles are more susceptible to Vibriosis than adult fish, hence mortality may reach upto 80% in case of juveniles. It has been reported that a few species of Vibrio including *V. parahaemolyticus, V. alginolyticus, V. harveyi, V. owensii*and *V. campbelli* are recognized as the most common vibrio species causing vibriosis farmed aquatic animals. In case of vibriosis, the pathogen may enter the host orally, through skin lesion and gill surface consequent to wound caused by ectoparasites and protozoa.

Fish affected by classical vibriosis show typical signs of a generalized haemorrhagic septicaemia with the presence of haemorrhagic lesions at the base of fins, ulcerations on the body surface, especially in chronic cases, exophthalmia and corneal opacity. Ailing fish are often anorexic with pale gills due to anaemia arising from haemorrhages. Microscopic lesions in case of vibriosis also reflect the haemorragic nature of the disease.

Histologically, bacteria invading the dermis, subcutaneous adipose tissue, and the underlying musculature are evident. Affected tissues are necrotic and heavily infiltrated by granulocytes. Gill filaments and lamellae are also infiltrated by neutrophills with haemorrhage. Liver shows hypertrophy of the bile ducts, necrosis, haemorrhage and congestion. In myocardium, loss of cross striations and infiltration of polymorphoneuclear cells into the endocardium is noticed. Kidneys reveal characteristic lesions of acute glomerulonephritis with increased expression of melanomacrophage centres. Gastric mucosa contains engorged capillaries and loss of tubular glands. Extensive tissue lesions in vibriosis are primarily due to the release of proteinases and other extra-cellular enzymes produced by the bacteria.

Vibrios are gram negative rods characteristically curved, or comma shaped. This morphological appearance may not be always observed when organisms are selected for gram staining from solid media. Specific media like Thiosulfate-citrate-bilesaltssucrose agar (TCBS) agar may be used for selective growth of Vibrios. Species level identification can be done by biochemical tests, PCR using specific primers and 16S rDNA amplification using universal primers and sequencing.

Even though vibrios are susceptible to majority of broad-spectrum antibiotics, limitations exist based on the farming system. Since vibrios are opportunistic pathogens, vibriosis can be best managed by proper husbandry practices. Handling, transportation, overcrowding, low dissolved oxygen and increased water temperature make the farmed fish susceptible to vibriosis. Periodical enumeration of the bacterial load of water and sediment would help in preventing outbreaks. Although species specific vaccines have shown effective protection against the specific bacterial strain, the antigenic diversity of Vibrio strains and their serotypes have made the vaccines unable to elicit protection against multiple Vibrio infection s resulting in slow progress of vaccine development. Due to diversity of V*ibrios* and their serovars, the advancement in vaccine development against vibriosis has been dawdling, and commercial vaccine is not currently available. However, attempts have been made to vaccinate fish against different *Vibrio spp* using oral, killed and sub-unit vaccines.

In case of oral vaccination, the vaccine is either mixed with the feed, top dressed on the feed, or bio-encapsulated. Bio-encapsulation is used when fish fry are to be vaccinated. In case of bio-encapsulation, live feed, such as artemia nauplii, copepods or rotifers incubated in a suspension of vaccine are fed to the fish fry. Oral vaccination causes no stress to the fish. However, they have a very short term stability once mixed with the feed. More recently, the outer membrane proteins molecules are used for development of subunit vaccines due to the exposed epitopes on the bacterial surface and conserved nature in different serovars.

It has also been demonstrated that outer membrane proteins molecules like OMP -K acts as protective antigen against fish vibriosis caused by *V. alginolyticus*. *Photobacteriosis*: Photobacteriosis, also known as fish pasteurellosis, is caused by the halophilic bacteria *Photobacterium damselae* subsp. *Piscicida* Gauthier *et al.* (1995) included the fish pathogen *Pasteurella piscicida* in the species P. damselae according to phylogenetic analyses of 16S rDNA sequences and DNA/DNA relatedness and named as *Photobacterium damselae* subsp. *piscicida*. Pasteurellosis has been a serious disease in Japan affecting the aquaculture production considerably. The disease is characterized by the presence of whitish nodules on liver, spleen and kidney. Severe mortalities occur in pasteurellosis when water temperature is above 18– 20 °C. Below this temperature, fish can harbour the pathogen for prolonged periods without causing clinical infection. The disease affects various species of fishes like yellow tail juveniles (*Seriola quinqueradiata*), ayu (*Plecoglossus altivelis*), black seabream (*Mylio macrocephalus*), red seabream (*Acanthopagrus schlegeli*), oval file fish (*Navodan modestus*) and red grouper (*Epinephelus okaara*).

Fish pasteurellosis is a septicaemic disease and manifests as an acute or chronic form. Pale gills, dark pigmentation and presence of petechial haemorrhages on the body surface and fin base are normally observed in acute form. Enlarged spleen and mottled liver are seen internally. In case of chronic form, nodules resembling tubercles are seen in spleen and kidney.

Another disease caused by *Photobactrium damselae subsp. damselae* is also responsible for mortality in many cultured marine fish species. This disease has been reported from India in cage farmed cobia. The lesions are haemorrhagic in nature resembling the lesions found in vibriosis. The pathological manifestations in both the infections are primarily due to the extra cellular products (ECP) secreted by the bacteria. Both pathogens are normal inhabitants of marine environment. Disease transmission in case of photobacteriosis occurs through direct contact and ingestion. The bacteria are

unable to survive in fresh or brackish water. Predisposing factors for the outbreak normally include rise in water temperature. The pathogen can be isolated and cultured on marine agar and ordinary media supplemented with sodium chloride. The organism can be confirmed by biochemical tests, 16S rDNA sequencing and slide agglutination. *Photobacterium damselae subsp. piscicida* has to be differentiated from *Photobactrium damselae* subsp. damselae using a multiplex PCR that combines specific primers for 16S rRNA and urease genes.

Several commercial vaccines against *P.damselae* subsp. *piscicida* are available, wherever the disease is more prevalent. Efficacy of these vaccines depends on the species of fish, fish size, etc. Since outbreak of pasteurellosis normally occurs during larval stages to fingerling stage, a vaccination programme involving dip immunisation during the larval stage with a booster dose when fish reaches a size of 1–2 g is advocated.

Streptococcosis: This is a re-emerging disease of both freshwater and marine fish caused by gram positive bacteria characterized by central nervous system damage followed by exophthalmia and meningoencephalitis. Streptococcosis is also known as "pop-eye", since one of the most characteristic clinical signs found in this disease is the accumulation of muco-purulent exudates around the eyes. Streptococcosis, a problem in both farmed and wild marine fish, has been reported from USA, Japan, and Spain. In Japan and Spain, this disease forms a major limiting factor for the marine fish production of yellowtail turbot. Fish of all the size are susceptible. The sea water and sediment harbour the pathogen which can be isolated from these sources round the year. It has been reported that this pathogen can survive in frozen products for at least 6 months. Warm water streptococcosis which occurs when water temperatures are above 15 °C is normally caused by *L. garvieae*, *S. iniae*, *S. agalactiae* and *S. parauberis*. Cold water streptococcosis seen when water temperatures are below 15°C is generally caused by *L. piscium* and *V. salmoninarum*. Pathogens responsible for warm water streptococcosis are of zoonotic importance since they can cause disease in humans. Horizontal transmission can occur through injuries and abrasions.

Streptococci capable of causing disease in marine fish falls into three categories: alphahaemolytic, beta-haemolytic, and non-haemolytic. The majority of disease epizootics are generally caused by streptococci belonging to alpha-haemolytic group. Among these fish streptococci, *L. garvieae, S. iniae* and *S. parauberis* can be regarded as the main aetiological agents causing diseases in marine aquaculture. *L. garvieae* infects marine fish like yellowtail in Japan. *S. Iniae* is an important fish pathogen causing

disease and mortality in many cultured fish species in both tropical and sub- tropical environments. *S. iniae* is the main aetiological agent of streptococcosis in tilapia in USA and rainbow trout in Israel. However, *S. iniae* was also isolated from marine fish including yellowtail, flounder, European seabass, and Asian seabass. There have been no reports from India. *S. parauberis* is reported to be endemic to cultured turbot.

The lesions caused by different streptococcal species in diverse host species are similar. The lesions are of general septecemic in nature affecting liver, spleen, eye, brain and kidney. The eyes show severe exophthalmia with granulametous inflammation. Granulomas with the presence of bacteria may also be seen in pericardium, alimentary tract, peritoneum, brain, ovary, testes and spleen. The most significant clinical signs are exophthalmia, distended abdomen, haemorrhages in the eyes, opercula, fin base, ulceration of the body surface and darkening of the skin. Internally, the abdominal cavity is filled with variable amounts of purulent exudate. A yellowish exudate often covering the peritoneum and the pericardium may be seen. Yellowish exudates may also be seen in cranial cavity. Haemorrhages are found on all visceral organs.

Fish streptococci can be generally isolated from internal like spleen, liver and brain using brain heart infusion agar or tryptone soya agar supplemented with 1% yeast extract or 0.5% glucose, and growth is enhanced on blood agar. The incubation period is 24 days at 25-30°C. The isolates are then characterized either biochemically or serologically. Significant characteristics are spherical or ovoid colony morphology and formation of pairs or chains. All strains of streptococci are Gram positive, oxidase and catalase negative, non-motile and non-sporulating. Slide agglutination and immunofluorescent techniques are widely used for diagnosis of streptococcosis. PCR based diagnosis including 16S rRNA amplification and sequencing can also be employed.

Good husbandry practises including avoiding over-crowding, excess feeding, handling and the timely removal of diseased or dead fish would help to minimise the economic losses due to streptococcosis. Apart from this, vaccination, use of chemotherapeutics and immunostimulants has also been tried. Many of the isolated strains were sensitive to antibiotics like erythromycin, tetracycline, ampicillin and doxycycline. Attempts have been made to develop vaccines against streptococcosis in fish. Intra-peritoneal route was found to be most effective. β-1-3-glucans used as immunostimulants is also found to be effective.

Parasitic Diseases Maricultured Finfish

There is less scope for control over water quality and flow as well as the presence of other marine life, including planktonic or net fouling organisms in marine cage farming. The advancement in cage aquaculture has been associated with emergence of parasitic diseases. Parasitic diseases can have a serious economic impact on marine cage culture because anti-parasitic treatment of fish in cages is often practically not possible or highly expensive. Many parasitic diseases affecting cage cultured fishes are caused by ectoparasites. An overall reduction in parasite diversity and a shift from endoparasites to ectoparasites is at least partly due to the use of manufactured feed since trophic transmission is likely when fishes are fed with trash fish.

Monogeneans: Monogeneans or monogenetic trematodes are flatworms attached to body, fin & gills of fish. Monogeneans infecting marine fish have a direct life cycle and are considered dangerous due to their high rate of transmission among fish in culture systems. Innate susceptibility and stressful environmental conditions can cause massive infections. Capsalid monogeneans have a flattened, leaf-like body with attachment organs, inhabit the skin and occasionally on the gills and even nostrils. They feed on epithelial cells and mucus causing excessive mucus production, skin lesions, opaque eyes, anorexia and mortality. Many of them have low host- specificity making them dangerous pathogens in fish culture facilities. Benedenia, Neobenedenia and Megalocotyloides are some of the genera that have high pathogenic potential. Dactylogyrids are common gill parasites of teleost fishes and are characterized by the presence of two pairs of large hooks or hamuli that helps parasite attachment. The attachment and feeding cause severe irritation, excess mucous production, epithelial hyperplasia, erosions, ulcerations, haemorrhage and hyperemia with leukocytic infiltrations. Heavily affected fish may die due to asphyxia as a result of gill pathology and interference with gaseous and ionic exchange mechanisms. Easy multiplication & dispersion coupled with its direct life cycle make them more dangerous in culture systems. Other important monogenean parasites of cultured fish include Diclidophorids which include Heterobothrium and Neoheterobothrium and Microcotylids, capable of causing serious mortalities in cultured fish.

Copepods: Copepods are considered the most important disease-causing parasites in both wild and cultured fish populations. They are small, dioecious crustaceans, with female carrying eggs in egg sacs attached to the genital segment. The egg hatches and develops into nauplius that further develops into copepodids and eventually adults in a rather shortened life cycle. Typical examples include the sea lice

(*Lepeophtheirus salmonis*), and the cymothoid isopods, which are notorious parasites of cage reared fish. Caligids are generally known as sea lice and are common in tropical and warm temperate waters. They are dorso-ventrally flattened and attach to their hosts by a combination of claws and suction. Two important genera parasitizing fishes are Caligus and Lepeophtheirus. Sea lice infestation is the biggest problem faced by salmonid farming industry, where the cost of treatments alone could be around 6% of the fish production. The presence of sea lice on non-salmonid production systems has also been associated with mortality and diseases. Caligids are considered the most important parasites in marine fish aquaculture.

Isopods: Isopods are marine/brackish parasitic crustaceans inhabiting warm waters. Majority of the isopods are cymothoids parasitizing marine teleosts. Massive attack by the juveniles of the parasite can kill fry and fingerlings. The parasites that enter the gill chamber cause anemia and loss of gill filaments due to their movements and feeding habits. The genus Nerocila has been found in groupers, seabass and snappers in southeast Asian countries. Isopod menace is considered an emerging problem in Mediterranean Sea cages causing reduced growth and mortality. The first record of serious mortalities in cage cultured fishes in India was caused by the isopod *Cirolana fluviatilis* in cage cultured seabass in Cochin backwaters. Mortalities appeared one month after stocking and fish were found dead in cages with their flesh eaten away, leaving the remnants of skeleton. *C. fuviatilis* is a voracious, carrion-feeding bottom dwelling isopod, but sometimes colonize the fouled net surrounding the cage and attack the stressed fish causing heavy mortalities. This is an example where parasites/pests that have not been previously considered pathogenic can cause serious mortalities under certain circumstances.

Co-infection in Aquaculture Practices

Coinfections or concurrent infections are infections of fish caused by two or more genetically different pathogens where each pathogen causes damage to the host in concurrence with other pathogens. Coinfections may involve bacteria, fungi or viruses or their combination. Coinfections caused by homologous bacterial pathogens have been reported in many fish species. During co-infection, interactions between the infectious agents may lead to increase of the load of one or both pathogens or one or both may be suppressed or one may be increased and the other suppressed. Such interactions during coinfection can have a significant impact on the pathogenesis and severity of the diseases and should be considered during the planning of treatment using therapeutics and vaccination. Moreover, concurrent infections in marine cage

culture should gain greater emphasis as the farming is done in natural water bodies which harbour many infectious organisms

Nutritional Pathology of Cultured Marine Fishes

Nutritional diseases of cultured marine fish may develop consequent to deficiency, excess, or imbalance of nutrients like protein, lipid and fat present in the feed. Nutritional diseases do not generally develop all of a sudden instead they develop gradually since the fish have body reserves of nutrients that make up for nutritional deficiency up to a certain extent. Clinical signs of nutritional deficiency develop only when supply of any diet component falls below critical level. Further, when fish consumes excess food, whatever in excess would be converted to fat which will be deposited in body tissues and organs. The deposited fat affects the physiological functions of the fish.