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Nutritional diseases

Lavilla, Celia R.

Aquaculture Department, Southeast Asian Fisheries Development Center

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CHAPTER SEVEN

Nutritional diseases

Celia R. Lavilla

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

TYPES OF FEEDS

Two types of diets are in use to nourish fish in aquaculture. Diets designed to add nutrients to food obtained from the pond or aquarium environment are called supplemental feeds. Feeds for intensively reared fishes that do not get nutrients supplied from the environment are called complete feeds. Complete feed formulas are based on the nutrient requirements of fishes, availability of the essential components, and digestibility of the ingredients used to prepare them. Various stages of fish require various sizes of feeds, thus, according to size, feeds are classified as: (1) larval feeds, (2) starter feeds, (3) grower feeds, (4) finisher feeds, (5) broodstock feeds, and (6) maintenance diets.

Supplemental or complete feeds may be prepared in moist, semi-moist or dry form. The type of ingredients used to prepare fish diets determines the type of diet. Moist rations are prepared from ingredients with high moisture content such as raw fish, meat products, wet vegetable products and similar ingredients. The moisture content of most moist rations is 70%. Semi-moist rations are prepared from dry products (dried fish products, cereal grains and other dry animal and vegetable products) that are added to ingredients with high moisture content. The final moisture content of semi-moist diets is approximately 35%. Dry rations are prepared from dry animal and vegetable products with a final moisture content of about 10%.

Larval fish must ingest and digest feed particles, and then absorb and utilize the released nutrients. Most research has focused on larval feed ingestion. Only now are researchers beginning to look at digestion, absorption, and utilization. It has been suggested that live food is needed because the prey contains digestive enzymes which help the larvae in digestion. They also contain relatively high levels of free amino acids that the larvae can easily absorb.

COMPONENTS OF FEEDS

Proteins and amino acids

Table 7-1 shows the types and biologic functions of proteins. The required concentration of protein in the feed will vary with species, size, age, water temperature, energy levels in the diet, and the quality of protein (digestibility and amino acid content) incorporated in the diet. Fish demand a balanced composition of amino acids in amounts that are adequate to sustain growth. Diets that are insufficient in essential amino acids may give rise to well-defined deficiency diseases. Table 7-2 lists the essential and non-essential amino acids. When used as the major protein source, processed fishmeal meets the dietary amino acid requirement. The use of mainly vegetable protein in a diet may make amino acid supplementation necessary. Protein is the most expensive ingredient in fish feed, and it may be tempting to economize on this ingredient. Too little protein will inhibit or retard growth, and may reduce resistance to disease.

Table 7-1. Types and biological functions of proteins

Type of Protein	Function
Enzymes	
DNA polymerase	repairs and replicates DNA
Lipase	digests lipids
Protease	digests proteins
Transport proteins	
Hemoglobin	transports oxygen in vertebrate blood
Hemocyanin	transports oxygen in invertebrate blood
Contractile proteins	
myosin and actin	contractile proteins of muscles
Protective blood proteins	
Antibodies	binds with foreign proteins
Fibrinogen and thrombin	blood clotting proteins
Hormones	
Insulin	regulates glucose metabolism
Structural proteins	
Keratin	components of skin, scales, etc.
Collagen	components of connective tissues

Table 7-2. Essential and non-essential amino acids for fish

Essential amino acids	
Arginine	Methionine
Histidine	Phenylalanine
Isoleucine	Threonine
Leucine	Tryptophan
Lysine	Valine
Non-essential amino acids	
Alanine	Glycine
Asparagine	Proline
Aspartic acid	Serine
Cysteine	Tyrosine
Glutamine	Glutamic acid

Carbohydrates

In crustaceans, carbohydrates either become stored as glycogen or form part of the structural make-up as chitin. Carbohydrates may be used as an energy supply by some species, but only up to a certain level. Most fish species have limited ability to metabolize carbohydrates, and have no nutritional demand for them. Since fish poorly utilizes carbohydrates, it eliminates the nutrient from the body in feces, and cause pollution concerns in fish farm sites. Some sources of carbohydrates for fish feeds are given in Table 7-3.

Table 7-3. Biological functions and sources of carbohydrates

Biological functions	
Storage polysaccharides :	Starch Glycogen
Structural polysaccharides :	Chitin Cellulose
Carbohydrate sources for fish feeds	
barley, grain	molasses, wood
bread flour	oats, grain
cassava meal	pea, seed
corn germ meal	rice bran
corn starch	rice grain
dextrin	rye, grain
linseed, flax	sorghum, gluten meal
molasses, beet	sorghum, grain
molasses, cane	wheat, grain
molasses, citrus	wheat, bran
molasses, corn	oats, hull

Fats and lipids

Lipids supply the fish with essential fatty acids that are necessary structural components of all cell membranes. Lipids are of great importance for physiological processes. They are also necessary for absorption of some vitamins, and constitute a relatively cheap energy source. The quality of lipids used in the diet should not be ignored. Polyunsaturated fatty acids are easily oxidized under storage especially under warm temperature. Rancid lipids are known to induce lipoid liver disease in fish.

Vitamins

Vitamins are complex organic substances that are essential to a wide variety of metabolic processes. They are only required in small amounts in the diet, but requirements for vitamins may increase during growth and spawning. Vitamins are divided into two classes: fat-soluble and water-soluble. Some vitamins like ascorbic acid are unstable in storage. This is why some feed manufacturers use more stabilized forms of ascorbic acid in their feed formulations. A listing of essential vitamins for fish as well as the forms of ascorbic acid used in fish feeds are given in Table 7-4.

Table 7-4. Essential vitamins for fish

Vitamins and their principal functions	
Vitamin A	Essential for normal vision
Vitamin D	Essential for bone formation
Vitamin E	Essential for membrane stability
Vitamin K	Essential for normal blood clotting
Thiamin	Co-factor in energy-yielding reactions
Riboflavin	Co-factor in metabolism
Pyridoxine	Co-factor in amino acid metabolism
Pantothenic acid	Co-factor in metabolism
Niacin	Co-factor in metabolism
Folic Acid	Essential for blood formation
Vitamin B 12	Essential for blood formation
Ascorbic Acid	Essential for collagen synthesis
Choline	Component of phospolipids, membranes
Myo-Inositol	Component of phospolipids, membranes
Forms of ascorbic acid used in fish feeds	
Crystalline	Very unstable
Fat-coated	Fat coating makes up 30% of weight Losses occur during extrusion; unstable during feed stage
Ascorbate-2-sulfate	Very stable; low availability for many species
Ascorbate-2-phosphate	Very stable; high availability

Minerals

Fish require minerals for maintenance of osmotic balance between body fluids and their environment. Seven major minerals are required in large amounts and constitute 60-80% of all inorganic materials in the body. The seven minerals are calcium, phosphorous, sulfur, sodium, chlorine, potassium, and magnesium. Trace minerals are just as essential as major minerals, but are needed only in small amounts. The nine essential trace minerals are iron, copper, manganese, cobalt, zinc, iodine, molybdenum, fluorine, and selenium.

Pigments

Pigments added to feeds result in an attractive red flesh color in salmonids. Quite large levels of shrimp meal in the diet can achieve good results as well. Feed manufacturers usually include synthetic analogues of the naturally occurring pigment asthaxanthin or cathaxanthin as additives.

Antioxidants, binder, and other feed components

Fish feeds contain high levels of unsaturated oils that easily oxidize resulting in breakdown of oils and other nutrients. This can be controlled by adding antioxidants such as butylhydroxytoluene (BHT), butylhydroxyanisole (BHA), ethoxyquin, and tocopherol (vitamin E). The levels of BHT, BHA, and ethoxyquin allowed in feeds by regulations often are not adequate to control oxidation of the high levels of unsaturated oils in fish feeds. When additional antioxidants are needed, feed formulators should supplement with vitamin E to the levels permitted to protect the oils in fish feeds.

Fiber is an indigestible dietary material derived from cell walls. In concentrations of less than 8% fiber may add structural integrity to pellet feeds. Larger amounts often impair pellet quality. Carbohydrates are important binders in commercial fish feed production. Taste or palatability of the ration for each of the fish species must also be considered. Complete feed rations include attractants to draw fish attention to the feed.

DETERMINING THE NUTRITIONAL STATUS OF FISH

Nutritional deficiency signs, like those listed in Table 7-5, are useful in diagnosing feed problems. However, the absence of visible signs does not necessarily mean that the fish is in optimal or desirable nutritional health. Increasing the dietary level of all essential nutrients result in a heightened physiological response up to the point at which the dietary requirements are met. Beyond a certain level of nutrient availability, concomitant increase in physiological response is not seen. This is the dietary level that supports optimal nutritional status. At some level below that requirement, visible deficiency signs appear in the fish. The range of dietary intake between that resulting in visible signs of deficiency and that resulting in no attendant increase in physiological function is an area referred to as subclinical deficiency. Fish receiving this level of an essential nutrient do not show signs of deficiency, yet they may be nutritionally compromised and less able to resist infectious disease or cope with suboptimal water quality conditions. These points are illustrated by Hardy (1991) in Figure 7-1.

Diagnosis of nutritional diseases requires knowledge of the following:

a. External and internal disease signs:
b. Quantitative clinical chemistry of blood or tissue of diseased fish;
c. Histopathological examination;
d. Histochemical analysis; and
e. Proximate analysis of the diet given.

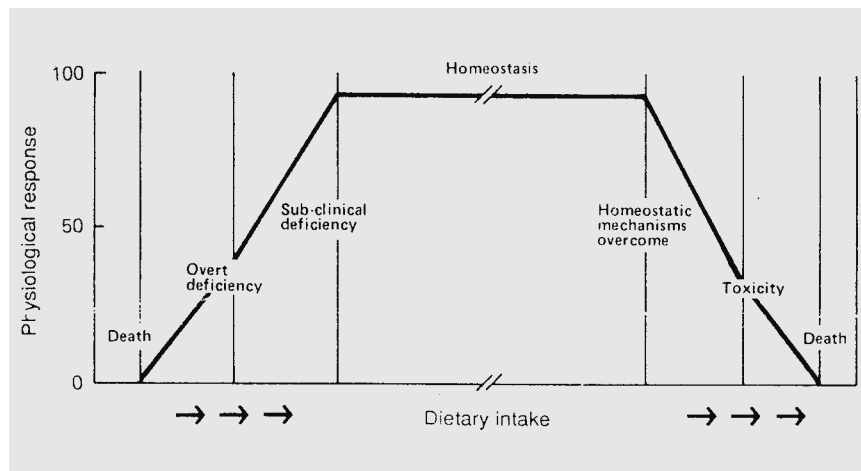

Figure 7-1. The relationship between the dietary intake of an essential nutrient and the physiological response of an animal or fish

Table 7-5. Summary of nutritional deficiency signs commonly observed in fish

Signs	Possible nutrient deficiencies
Anemia	Folic acid, inositol, niacin, pyridoxine Riboflavin, vitamins A, B_{12}, C, E, and K
Anorexia	Biotin, folic acid, inositol, niacin, pantothenic acid, pyridoxine, riboflavin, thiamine, vitamins A, B_{12} and C.
Ascites	Vitamins A, C, and E
Atrophy, gills	Pantothenic acid, vitamin A
Atrophy, lateral muscle	Biotin, thiamine, vitamin E
Cartilage abnormality	Vitamins C and A, tryptophan
Cataract	Methionine, riboflavin, thiamine, zinc
Cloudy lens	Methionine, riboflavin, zinc
Clubbed gills	Pantothenic acid
Decoloration, skin	Fatty acids, thiamine, vitamin A
Deformation, bone	Phosphorus, vitamin A
Deformation, lens	Vitamin A
Disease resistance, low	Protein, vitamin C
Dystrophy, muscular	Selenium, vitamin E
Edema	Niacin, pyridoxine, thiamin, vitamins A and E
Equilibrium loss	Pyridoxine, thiamine
Erosion, fin	Fatty acids, riboflavin, vitamin A, zinc
Exophthalmia	Pyridoxine, vitamin A, C, and E
Fatty liver	Biotin, choline, fatty acids, inositol, vitamin E
Feed conversion, poor	Vitamin E, biotin, calcium, choline, fat, folic acid, inositol, niacin, protein, riboflavin
Fragility, fin	Folic acid
Growth, poor	Biotin, calcium, choline, fat, folic acid, inositol, niacin, pantothenic acid, protein, pyridoxine, riboflavin, thiamin, vitamins A, B_{12}, C, D, and E
Hemoglobin, low	Iron, vitamins A, B_{12} and C
Hemorrhage, eye	Riboflavin, vitamin A
Hemorrhage, gill	Vitamin C
Hemorrhage, kidney	Choline, vitamins A and C
Hemorrhage, liver	Vitamin C
Hemorrhage, skin	Niacin, pantothenic acid, riboflavin, vitamins A and C
Lethargy	Folic acid, niacin, pantothenic acid, thiamine, vitamin C
Lipoid liver	Fatty acids, rancid fat
Lordosis	Vitamin C
Low glycogen, liver	Vitamin C
Myopathy, cardiac	Essential fatty acids
Necrosis, liver	Pantothenic acid
Pinhead	Starvation
Pigmentation, iris	Riboflavin
Scoliosis	Phosphorus, tryptophan, vitamins C and D
Swimming, erratic	Pyridoxine, pantothenic acid
Vacuolation, liver	Vitamin C

Information on the feeding practices, feeding rates, storage conditions of feeds, and feeding behavior of the fish are also important.

Diagnosis of dietary carbohydrate-related pathology can be made by blood glucose and liver glycogen analyses. Histopathological examination of stained tissue sections can be useful in the diagnosis of nutritional diseases. Diagnosis of vitamin deficiencies is difficult. Gross signs, blood chemistry, hematology, tissue vitamin assay, gross pathology, histopathology and ration vitamin assay may be necessary for making a diagnosis.

NUTRITIONAL DEFICIENCY DISEASES OF FISH

Deficiency diseases in fish occur when the tissue reserves have been depleted and the dietary supply of any necessary nutrient falls below the level that supports optimum nutritional status. Table 7-5 shows the common deficiency signs observed in fish. It is important to realize that by the time signs of nutritional deficiency are visible, fish have usually stopped eating, and feeding fortified diets will not completely reverse the condition in the fish population and prevent losses. Several pathological syndromes have been reported in fish.

Amino acid deficiency syndrome

Like other deficiency diseases, diagnosis of malnutrition involving protein or amino acid deficiencies is extremely difficult because many indications of deficiency are non-specific. Many of the same diagnostic findings noted in protein and amino acid deficiency are similar to physical and functional alterations found in the diagnosis of diseases caused by other etiological agents. Probably the most common sign of protein and/or amino acid deficiency in fishes is reduction or cessation of growth. Limiting even one indispensable amino acid will affect growth. Feeding a diet in which an essential amino acid has been removed will cause growth to cease until the amino acid is restored to the diet.

Fats and lipids deficiency

Among the clinical features manifested by fish with essential fatty acid deficiency are depigmentation, fin erosion, cardiac myopathy and fatty infiltration of the liver. Lipoid liver disease is usually seen in fish fed on trash fish or pelleted diets in which part of the lipid component has gone rancid. Rancid lipids react with protein to lower its biological value and have a deleterious effect on those vitamins which are not themselves antioxidants. Fish suffering from lipoid liver disease have extreme anemia (manifested by pallor of the gills), bronzed rounded heart and a swollen liver with rounded edges.

Vitamin imbalances

Depletion of the body storage of any single vitamin can be responsible for specific or general disease signs. Disease signs and gross pathology usually suggest which vitamin may be deficient, and other diagnostic procedures are then used to complete the diagnosis. Results of research in which specific vitamins have been reduced or eliminated from the diets of fishes have demonstrated the

usual signs, gross pathology and histopathology to be expected from specific vitamin deficiency (Table 7-5). Nutritional myopathy syndrome in cultured fishes in Japan is a disease characterized by necrosis and degeneration of the lateral musculature of fish. It has been associated with vitamin E deficiency and with lipid peroxides in the diet. Supplementation of vitamin E was confirmed to prevent myopathic changes in carp.

Mineral deficiencies

Mineral deficiency studies with fishes are difficult because all traces of each mineral to be studied must be removed from both food and water. Thus, only a limited number of mineral deficiencies are known or have been studied in fishes. The role of calcium, potassium, and magnesium in bone and joint metabolism is well recognized and there is evidence that imbalances or deficiencies of these can result in spinal abnormalities which leave the fish deformed thereafter.

NUTRITIONAL DISEASES OF SHRIMPS

Feeding farmed shrimps generally depends on the culture system employed. The primary goal is to provide balanced nutrients to the farmed population and this is influenced by the quality and quantity of the nutritional resource and biomass of shrimps that rely on this as a source of food. Thus, in extensive production systems, feed is often not applied or the quality, in terms of nutrient composition, is low because the farm population can derive sufficient levels of balanced nutrition from the pond system. However, at higher stocking and biomass, the ability of the pond environment to sustain shrimp growth becomes limiting. At this point balanced nutrition of the applied feed is essential for continued growth of the population. Hence, the importance of nutritional quality of feeds is a function of the type of culture system and the shrimp biomass within the system. For obvious reasons, nutritional deficiency disease is much more important in semi-intensive through super intensive systems than it is in extensive culture. The following nutritionally-related diseases or syndromes have been observed in cultured shrimps:

Vitamin C deficiency

Vitamin C deficiency or black death disease occurred in juvenile to subadult penaeid shrimp cultured intensively in tanks. All penaeid species are considered susceptible. The cause is low level of vitamin C in the diet, and lack or absence of algal growth or other vitamin C sources in the culture system. Presence of black lesions below a non-ulcerated cuticle provides a presumptive diagnosis of the disease. Diagnostic confirmation is done by histological demonstration of the characteristic melanized hemocytic nodules within tissues of high collagen content such as the connective tissues beneath the shell. Bacterial septicemia often accompanies the disease, suggesting an important role of vitamin C in crustacean defense to infection. Experimental results showed that vitamin C-deficient shrimps had slower wound-healing process than shrimps with adequate vitamin C in their diet.

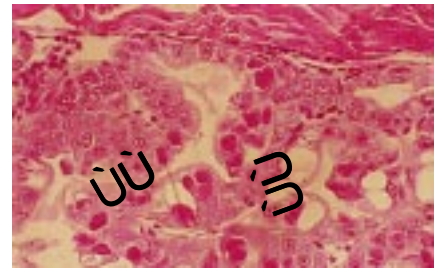

Figure 7-2a. Histological section of the hepatopancreas of shrimp juvenile at the start of the experiment showing numerous monodon baculovirus (MBV) occlusion bodies (arrows) (Hematoxylin and Eosin, 200x)

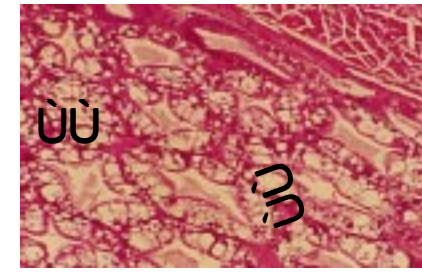

Figure 7-2b. Histological section of the hepatopancreas of shrimp juvenile fed a diet with 100 ppm phosphated ascorbic acid. Note the absence of MBV occlusion bodies and the presence of numerous storage vacuoles (arrows) (Hematoxylin and Eosin, 200x)

Shrimps in vitamin C-deficient populations had reduced growth and are prone to shell disease. Under experimental conditions, monodon baculovirus (MBV)-infected shrimps fed diets with 100 ppm phosphated ascorbic acid (MAP) or higher showed a marked absence of infection after three months (Fig. 7-2a, b) Shrimps fed vitamin C-deficient diets in the same experiment had MBV occlusion bodies still evident in the hepatopancreatic tubules after the same test period. The extent of vitamin C deficiency in cultured crustacean populations is unknown, but subclinical vitamin C deficiency may be widespread and common in intensive culture settings. Once identified as a problem, control is achieved through proper diet formulation to deliver sufficient vitamin C needed for shrimp growth. Highly stable forms of vitamin C are presently available for use, thus the problem of vitamin C degradation during feed manufacture and storage should no longer be a problem.

Chronic soft-shell syndrome

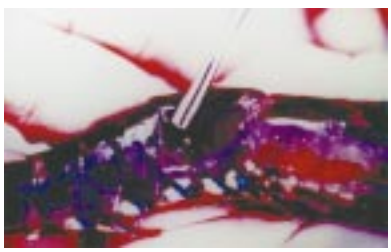

Figure 7-3. Pond-reared *Penaeus monodon* juvenile with chronic soft-shell syndrome

Soft-shell syndrome (Fig. 7-3) is a condition noted in farmed *Penaeus monodon* in the Philippines, India and probably other areas within the culture range of this species. In regions where it occurs, the syndrome is recognized as a significant problem that adversely affects production of grow-out farms. Soft-shelled shrimps are weak, prone to epibiotic fouling and cannibalism, and have reduced market value. The soft-shell condition results with the delay of several weeks in the normal hardening process of the cuticle following molting. Although the presence of the soft-shelled condition correlated well with the environmental conditions of high soil pH, low water phosphate, low organic matter, and insufficient water exchange, inadequate feeding practices or insufficient food supply are the primary causes of the soft-shell problem. The atrophied condition of the hepatopancreas of soft-shelled shrimps support the hypothesis of underfeeding as a cause for the syndrome. Infrequent water exchange would support this conclusion because lack of an adequate supply of water leads farmers to reduce feed inputs because of instability in water quality conditions and greater likelihood of losses due to oxygen problem.

Blue Disease or blue shell syndrome

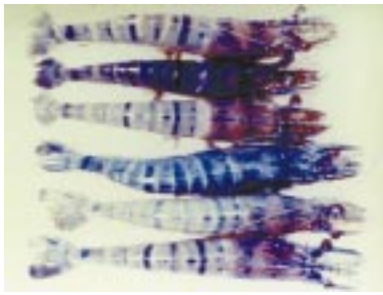

Figure 7-4. *Penaeus monodon* adults with blue shell syndrome (2nd and 4th from left

Shrimps with this disease/syndrome are light blue rather than grayish-brown in color (Fig. 7-4). Although the syndrome is not well understood until now, a deficiency in dietary carotenoids (i.e. asthaxanthin) is believed to be one of the causes. Prevention and control of blue disease is achieved through dietary manipulation. Future work is needed to provide practical guidelines to farmers on management of the syndrome. Basic research work to further understand the effect of carotenoid dietary deficiency on crustacean health is also important. The role of environmental factors in the development of the syndrome also needs to be investigated.

Body cramp or cramped tail syndrome

Body cramp refers to a syndrome of uncertain etiology. Nutritional factors, such as an imbalance in the Ca:Mg ratio and vitamin B deficiency, have been implicated as etiologic agents. Body cramp and muscle necrosis often occur together with the striated muscles as the target organ. Body cramp describes a functional lesion in which the muscle contracts but is unable to relax, hence, the abdomen or tail remains flexed for an extended period. The role of environmental factors in the development of this syndrome are presented in Chapter 6.

Underfeeding

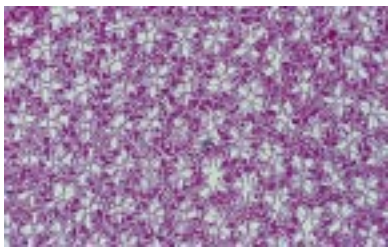

Figure 7-5a. Normal structure of the hepatopancreas of juvenile shrimp showing well-vacuolated cells lining the tubules (Hematoxylin and Eosin, 40x)

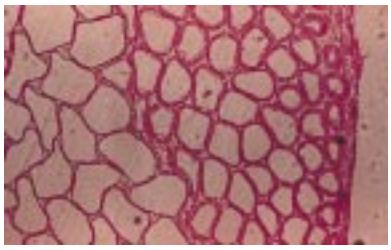

Figure 7-5b. Hepatopancreas of underfed shrimp juvenile showing severely atrophied tubules due to the absence of stored food (Hematoxylin and Eosin, 40x)

Underfed shrimps appear to have loose and oversized shells due to the reduction of the muscular mass in the abdominal region. The most prominent lesion associated with underfeeding appears in the hepatopancreas. There is a marked atrophy of the tubules due to the absence of stored food in the cells (Fig. 7-5a, b). Underfeeding usually results because of management inexperience. This happens when regular monitoring and estimation of shrimp biomass is not carried out, or the feeding rate is inappropriate because the animals are overstocked. Underfeeding could also be due to inadequate availability of energy to sustain growth of the shrimps in the pond, or a lack of specific, limiting nutrients necessary for growth. In either case the initial sign is reduction or lack of growth of the population in the production setting. A diagnosis of underfeeding requires careful review of production records, water quality monitoring data, and animal examination. Since the principal sign of reduced or no growth is non-specific, many factors must be considered and eliminated in the diagnostic process.

Prevention and control of an underfeeding problem is based on identification of the underlying contributing conditions and correction of these deficiencies. For example, if feeding rates are reduced to prevent water quality problems, then increased water exchange, reduction of anticipated harvest biomass, or both are needed. Improved growth of shrimps following a change in feed or feeding regimen supports the diagnosis of an underfeeding problem.

FEED QUALITY PROBLEMS WITH HEALTH IMPLICATIONS

Improper storage

Feed storage is of particular importance in the tropics where the warm humid weather promotes rapid deterioration of feeds. Feeds in bags should be stored in a cool, dry area and low humidity must be maintained because moisture enhances mold growth and attracts insects. If proper storage conditions are not maintained, several factors can cause spoilage of the feed and pose problems such as:

- loss of vitamins and nutrients
- rancidity
- contamination with microorganisms or their metabolic products

Artificial feeds contain ingredients that are prone to become rancid like fish meal, copra meal, rice bran and fish oils. Rancid oils can be toxic, may destroy other nutrients, and will cause off-flavor of the feed. Rancidity in feeds has a negative effect on its palatability, nutritional value, and it may also lead to toxicity problems. Fish that have prolonged exposure to rancid feeds may exhibit reduced appetite, slow growth and low feed efficiency.

Another feed-related problem with fish health implication is connected with the use of ingredients that are contaminated with microorganisms which may produce various toxins. This is primarily a problem with fish meal and other animal by-product meals, which are contaminated with *Salmonella*. Avoiding this problem involves the establishment of standards for fish feed ingredient quality. The feed standards could include maximum acceptable levels of microbial contamination.

Aflatoxin contamination

Molds like those belonging to genus *Aspergillus*, which grow on the feeds, cause spoilage. These may also produce toxins such as aflatoxin. While aflatoxin may be produced during feed storage, it may also be produced in feed ingredients before these are used to make feeds. So, the establishment of standards of feed ingredients must include examination of suspected materials for aflatoxin. In feeds that are susceptible to mold formation during storage, the addition of mold inhibitors is a recommended practice. The chosen inhibitor should not affect the palatability of feed to fish.

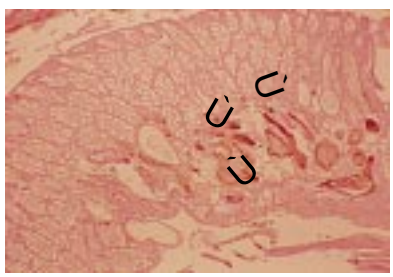

Figure 7-6. Hepatopancreas of shrimp fed diets with 200 ppb aflatoxin B_1 showing melanized tubules in the central region (arrows) (Hematoxylin and Eosin, 40x)

Aflatoxicosis in shrimp

Because of the rampant occurrence of red disease-like lesions in cultured shrimp in the early 1990s, a study on the possible role of aflatoxin B_1 ($AFLB_1$) on the development of the disease was conducted. Juvenile shrimps were given diets with various levels of $AFLB_1$, namely: 25, 50, 75, 100, and 200 parts per billion (ppb) for two months. Control shrimps were fed complete diets without $AFLB_1$. The results showed that shrimps fed diets containing $AFLB_1$ of 75 ppb and above had comparatively poor growth rates. In fact, shrimps given diets with 200 ppb $AFLB_1$ showed negative growth after two months. The primary organ that is affected in aflatoxicosis is the hepatopancreas. Melanized lesions develop primarily in the central region of the organ causing dysfunction (Fig.

7-6). As the hepatopancreas is the organ responsible in the digestion, absorption and storage of food, damage in its tissues leads to slow growth. Shrimps exposed to $AFLB_1$ were also prone to shell disease.

Studies also show that *P. monodon* juveniles can tolerate aflatoxin B_1 levels of up to 52.3 µg/kg feeds, but histopathological changes are usually evident in shrimp tissues at a level of 26.5 µg/kg. Growth retardation was observed at levels of more than or equal to 73.8 µg/kg feeds.

Toxic components of the diet

Naturally occurring organic compounds found in some feed ingredients produce toxic responses in fish. Tannic acid and cyclopropenoid fatty acids induce liver cancer in fish. Gossypol, a toxin present in untreated cottonseed meal, causes anorexia and ceroid accumulation in the liver. Phytic acid, which ties up with zinc in the feed, and growth inhibitors found in soybean meal can be destroyed by proper heating during processing. Chlorinated hydrocarbons occur as contaminants in fish meal and can cause mortality when present in fry feeds. Broodstock transfer these compounds from the feed to their eggs, resulting in low hatchability and high mortality of fry. The environment and feeds should be free from toxicants to maintain the health and efficient production of fish. Feed ingredients may also contain anti-nutritional factors. Most of these factors are found in ingredients of plant origin. The most common anti-nutritional factors in feed ingredients are trypsin inhibitors found in soybean meal.

The use of leaf meals as non-conventional protein sources for shrimp diets has been studied. *Leucaena leucocephala* (called "ipil-ipil" in the Philippines) leaves are one of the promising sources of plant protein for shrimp diets, however, the leaves contain a poisonous amino acid, mimosine, which causes pathological changes in the storage cells of the shrimp's digestive organ. The mimosine content of the *Leucaena* leaves could be reduced by 70% if the leaves are soaked in freshwater for 24 h prior to incorporation in the artificial feeds.

Transmission of diseases through trash fish

The use of fresh or raw trash fish as a food presents the possibility of transmitting specific fish pathogens directly. Viral and bacterial diseases, as well as parasites can be transmitted to healthy fish by feeding them with contaminated trash fish. This problem will be minimized if trash fish is stored frozen in single-ration packages. Microorganisms and parasites that do not survive freezing temperatures will be eliminated. Thawing and re-freezing trash fish should be avoided to prevent their spoilage. Trash fish that have gone through a heating step to destroy any fish pathogen that might be present is more appropriate than raw fish.

SUMMARY

Diagnosis of nutritional diseases is difficult because many signs exhibited by fish are non-specific and most nutritional deficiencies are hard to define. A compilation of data on feed composition and feeding management, as well as husbandry practices, are needed to define a case. Most of data on fish and

shrimp nutritional diseases were gathered under experimental conditions. Under farm conditions, most of that definition would be clouded with errors in husbandry practices or secondary infection. Therefore, attempts to diagnose nutritional diseases should be carefully done using every available technique to define the case.

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