Dietary essentiality of trace minerals in aquaculture-A Review

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ABSTRACT
An element is considered as dietary essential when its absence or insufficiency in diet causes deficiency syndrome and supplementation in normal level brings back normal health. An organism can neither grow nor remain healthy without the element in question. The element should have a direct influence on the organism and involved in the metabolism. Effects of the essential elements cannot be wholly or partly replaced by any other elements. All forms of aquatic inhabitants require some inorganic elements in little or trace amounts for their normal growth and metabolism. Trace minerals do not exist only by themselves but in combination with others. Therefore too much of one element may lead to imbalances in others resulting in disease or adverse effect in metabolism. Factors such as diet, absorption ability, toxicities and drug-nutrient interactions may play a role in maintaining a balance of trace minerals in the animal body. In comparison to the farmed animals, the knowledge on dietary essentiality of minerals in fish is scarce being mainly restricted to iron, copper, manganese, zinc, iodine, selenium, cobalt and chromium as components of body fluids, co-factors in enzymatic reactions, structural units of non-enzymatic macromolecules, etc. Investigations in fish are comparatively complicated as both dietary intake and its uptake from availability in water have to be taken in account for determining the total mineral budgets. The importance of trace minerals as essential ingredients in diets, although in small quantities, is also evident in fish. Though requirements of trace minerals have been studied in some species, still research work needs to be intensified for other freshwater fish species.

Key words: Copper, Fish nutrition, Iron, Manganese, Requirement, Selenium, Trace minerals, Zinc.

INTRODUCTION
All terrestrial and aquatic organisms require inorganic elements or minerals for their normal life processes. Fishes have the ability to absorb inorganic elements from diets as well as from their external environment in both freshwater and marine water. Minerals, which comprise the ash of biological materials remaining after the organic substances have been completely burnt or oxidized in the body of all animals including fish, are known to be essential for cellular metabolism. Freiden, (1984) described that an element is considered essential when its deficient intake produces an impairment of function and when restoration of physiological levels of the element prevents or relieves the deficiency. Organisms can neither grow nor complete its life cycle without those inorganic elements. They should have a direct influence on the organisms and involved in the metabolism. Minerals, which are present in fairly large quantities in biological materials, are those of calcium, phosphorus, potassium, sodium, magnesium, etc. The principle minerals present in microquantities (or trace levels) are iron, manganese, zinc, copper, cobalt, selenium, chromium and iodine (Lall, 1979). There are approx fifteen trace elements considered to be essential in animals. Among these the physiological role of a deficiency of chromium, cobalt, copper, fluorine, iodine, iron, manganese, molybdenum, selenium and zinc is well recognized (Lall, 1989). The main function of those essential elements in the body is formation of skeletal structure, colloidal systems maintenance (osmotic pressure, viscosity and diffusion) and regulation of acid-base equilibrium. They serve as an important component of hormones, enzymes and enzyme activators. Calcium and phosphorus are required for the formation of the skeletal structures of the body and scales. Sodium, potassium and chloride along with phosphates and bicarbonates maintain homeostasis and acid-base balance equilibrium. A fixed number of specific trace minerals viz., iron, manganese, copper and zinc are firmly associated with a specific protein in metalloenzymes, which produce a unique catalytic function.

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Non-metal iodine is necessary for the biosynthesis of thyroid hormones, which in turn greatly affects development and metabolism in all vertebrates (Lall, 1979, 1989).

The concentration of minerals in the body of an aquatic organism depends on the food source, environment, species and stages of development and physiological status of the animal. Most of the organisms accumulate and retain minerals from the environment; however, their incorporation is highly selective. In marine food chains, a unique transport of trace minerals has been reported (Bernhard and Andreea, 1984). Body utilization efficiency of dietary mineral elements depends on the availability of the element from a feed ingredient or complete diet. Many mineral bioavailability crisis are being increasingly recognized in humans and animal nutrition. Several factors influencing bioavailability include the level and form of the nutrient, particle size and digestibility of the diet, physiological and pathological conditions of the fish, waterborne mineral concentration and the species under consideration. Among these factors, those related to the chemical state are important because the element may assume different molecular forms, valence state and ligands when ingested from different diets (Forbes and Erdman, 1983). Some biologically important compounds contain minerals as an inherent part of their structure, e.g., hemoglobin and vitamin B12. Fish maintains a delicate balance of the body levels of the trace minerals by integrating the various parameters of uptake, storage and excretion. Information on nutritional requirements of freshwater fish for trace elements is scarce particularly because many are needed in extremely small amounts and these pose difficulty in analysis. The soluble and non-absorbable substances which are thereby formed in the gastrointestinal tract of the animal may either prevent or aid the uptake, transport and metabolism of the element. During these processes certain other inorganic elements compete with the element under consideration for favorable binding sites. In addition to all these diet-related mechanisms, several aspects linked to the environment influence mineral bioavailability (Watnabe et al., 1980). Although the trace minerals are required by the animal in very small quantities (usually less than 100 mg kg$^{-1}$ dry diet), they are absolutely required for normal growth. If excess amounts of the elements are ingested and assimilated, toxicity may develop. Their ability to regulate abnormal concentrations varies with the species. Some finfish and crustaceans are capable to excrete high proportion of excessive metal intake and can consequently regulate the concentration in the body at relatively normal levels. These take place for essential elements like iron, copper and zinc (Bryan, 1976). Sublethal effects of several metals on aquatic organisms have been well demonstrated by Bryan (1979). Most of the sublethal toxicity appears to be of a biochemical origin and causes morphological, physiological (growth, swimming performance, respiration and reproduction) and behavioral changes (Bryan, 1976; Albaster and Lloyd, 1980). The toxicity mechanisms of metal ions include blocking of essential biological functional groups of enzymes, displacing the essential metal ion in the biomolecule (enzyme or protein). The trace minerals play immense role in cellular metabolism so their requirement in diet plays crucial role in Aquaculture Nutrition.

**Iron**: Iron (Fe) has an active part in oxidation and reduction reactions and electron transport associated with cellular respiration. It is found in complexes bound to proteins such as haem, in enzymes such as microsomal cytochromes, catalase, etc. and in non-haem compounds such as transferrin, ferritin and flavin iron enzymes. Iron in form of haemoglobin occurs in erythrocytes while transferrin form is found in plasma (Watnabe et al., 1997). The haem molecule has ferrous or ferric ions in the center of a porphyrin ring. Haemoglobin contains 4 porphyrin groups per molecule. Myoglobin, a similar type of compound present in the skeletal or heart muscle, contains one ferrous porphyrin group per molecule (Lall, 1979). The iron content of fish is very low compared to that of mammals (Van Dijk et al., 1975). Though limited information on absorption and metabolism of iron in fish is available, but the process is generally the same as in other vertebrates. Although the gill membrane absorbs iron to a certain extent, the intestinal mucosa is considered to be the major site (Watnabe et al., 1997). Addition of iron (Ferrous sulphate) improved the growth of sword tail and platy fish indicating a nutritional benefit from dissolved iron in water (Roeder and Roeder, 1966). Among the different forms of iron, on comparing the effectiveness in preventing anaemia it was seen that ferrous and ferric chlorides were equally active (Sakamoto and Yone, 1979). It is reported elsewhere that addition of ferrous sulphate to trout diet shows significant increase in oxidation of that diet (Desjardins et al., 1987). As in other animals, ascorbic acid is involved in the metabolism of iron and vice-versa in fish (Desjardins, 1985). A recent study on the effects of dietary iron supplementation on growth performances, fatty acid metabolism and composition has been reported in rainbow trout (Senadheera et al., 2012). It shows that graded level of feed supplemented dietary iron can have a positive effect on LC-PUFA (Long Chain Polyunsaturated Fatty Acid) biosynthesis in fish. It is also observed that dietary iron level have a positive effect on n-3 LC-PUFA content and can also modulate the activity of fatty acid desaturase enzymes (Table 2).
It is reported elsewhere that deficiency of iron induces anemia in brook trout (Kawatsu, 1972), yellow tail (Ikeda et al., 1973), red sea bream (Sakamoto and Yone, 1978a) and also carp (Sakamoto and Yone, 1978b) (Table 1). Iron deficiency is also known to cause yellowish white liver condition in carp (Sakamoto and Yone, 1978b). In channel catfish poor feed utilization and lowering of plasma iron level and transferrin saturation is noted (Gatling and Wilson, 1986). The hatching rate of rainbow trout eggs was poor when the iron content was low in feed (Desjardins 1985). Dietary iron toxicity signs develop in rainbow trout fed more than 1,380 mg Fe/kg (Desjardins et al., 1987). The dietary iron requirement is presented in Table 3.

The major factors influencing iron absorption are the proportion of organic and inorganic components of the diet, the amount ingested and the conditions of the digestive

<table>
<thead>
<tr>
<th>Trace Minerals</th>
<th>Deficiency syndrome</th>
<th>Fish species</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>Iron (Fe)</td>
<td>Anaemic condition</td>
<td>Brook trout</td>
<td>Kawatsu, 1972</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yellow tail</td>
<td>Ikeda et al., 1973</td>
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<td></td>
<td></td>
<td>Red Sea Bream</td>
<td>Sakamoto and Yone, 1978a</td>
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<td></td>
<td></td>
<td>Carp</td>
<td>Sakamoto and Yone, 1978b</td>
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<td></td>
<td></td>
<td>Eels</td>
<td>Nose and Arai, 1979</td>
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<td></td>
<td>Haemoglobin suppression with transferrin saturation</td>
<td>Channel Catfishes</td>
<td>Gatlin and Wilson, 1986</td>
</tr>
<tr>
<td>Copper (Cu)</td>
<td>Poor hatching rate</td>
<td>Rainbow trout</td>
<td>Desjardins, 1985</td>
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<tr>
<td></td>
<td>Reduced heart cytochrome c oxidase</td>
<td>Channel catfish</td>
<td>Gatlin and Wilson, 1986</td>
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<tr>
<td></td>
<td>Reduced liver Cu-Zn superoxide dismutase</td>
<td>Channel catfish</td>
<td>Gatlin and Wilson, 1986</td>
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<td></td>
<td>Reduced growth and cataract formation</td>
<td>Common Carp</td>
<td>Satoh et al., 1983b</td>
</tr>
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<td></td>
<td>Hitra disease (Coldwater bacterial disease caused by <em>Vibrio salmonicida</em>)</td>
<td>Atlantic salmon</td>
<td>Poppe et al., 1986</td>
</tr>
<tr>
<td>Manganese (Mn)</td>
<td>Reduced growth</td>
<td>Rainbow trout, Carp, Tilapia</td>
<td>Ishac and Dollar, 1968; Ogino and Yang, 1980</td>
</tr>
<tr>
<td></td>
<td>Skeletal abnormalities</td>
<td>Rainbow trout and Carp</td>
<td>Satoh et al., 1983a,b.</td>
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<td></td>
<td>Cataracts</td>
<td>Rainbow trout</td>
<td>Satoh et al., 1983a,b.</td>
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<td></td>
<td>Dwarfism</td>
<td>Rainbow trout and Carp</td>
<td>Satoh et al., 1983a,b.</td>
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<td></td>
<td>Disturbance in bone metabolism</td>
<td>Rainbow trout and Carp</td>
<td>Satoh et al., 1983a,b.</td>
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<tr>
<td></td>
<td>Low activity of Cu-Zn superoxide dismutase and Mn superoxide dismutase in cardiac muscle and liver</td>
<td>Rainbow trout</td>
<td>Knox et al., 1982</td>
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<tr>
<td></td>
<td>Poor hatchability and low Mn level of eggs</td>
<td>Rainbow trout and Brook trout</td>
<td>Takeuchi et al, 1981; Lall, 1989</td>
</tr>
<tr>
<td></td>
<td>Reduced growth, high mortality, eroded fins and skins, Short body dwarfism</td>
<td>Rainbow trout</td>
<td>Ogino and Yang, 1978</td>
</tr>
<tr>
<td></td>
<td>Dwarfism, low appetite, Zn – Ca levels, and serum Zn concentrations</td>
<td>Channel catfish</td>
<td>Satoh et al., 1983a</td>
</tr>
<tr>
<td></td>
<td>Reduced egg production and hatchability</td>
<td>Common carp and Rainbow trout</td>
<td>Takeuchi et al., 1981</td>
</tr>
<tr>
<td>Iodine (I)</td>
<td>Thyroid hyperplasia</td>
<td>Brook trout</td>
<td>Gaylord et al., 1914</td>
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<td></td>
<td>Growth depression</td>
<td>Salmonids</td>
<td>Higgs and Eales, 1982</td>
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<tr>
<td>Selenium (Se)</td>
<td>Growth depression</td>
<td>Rainbow trout</td>
<td>Hilton et al., 1980</td>
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<td></td>
<td>Muscular dystrophy</td>
<td>Rainbow trout</td>
<td>Satoh et al., 1983b</td>
</tr>
<tr>
<td></td>
<td>Exudative diathesis</td>
<td>Channel catfish</td>
<td>Gatlin and Wilson, 1983</td>
</tr>
<tr>
<td>Cobalt (Co)</td>
<td>Reduces intestinal synthesis of Vitamin B_{12}</td>
<td>Atlantic salmon</td>
<td>Poston et al., 1976</td>
</tr>
<tr>
<td></td>
<td>Exudative diathesis</td>
<td>Rainbow trout</td>
<td>Bell et al., 1985</td>
</tr>
<tr>
<td>Chromium (Cr)</td>
<td>Improper Glucose utilization</td>
<td>Common Carp</td>
<td>Limsuwan and Lovell, 1981</td>
</tr>
</tbody>
</table>

TABLE 1: Deficiency syndrome of trace minerals in some fishes
tract. The inorganic iron is reduced to the ferrous state and freed from the conjugate for absorption. The iron from animal sources may occur as porphyrin, myoglobin and haemoglobin. It may be in a complex form with phytin in cereals. The availability of iron in the various feed stuffs for fish depends on its form (organic and inorganic) (Watnabe et al., 1997). Feeds of animal origin other than milk by-products are good source of iron. Common fish feed ingredients viz., fish meal and meat meal contain 150 to 800 mg Fe/kg (approx.), cereals grain ranges from 30 to 60 mg/kg and oil seed protein contain iron ranges from 100 to 200 mg/kg approximately (Lall, 1989).

**Copper:** Copper (Cu) is an essential trace element for all animals including fish (O’Dell, 1984; Mertz, 1986; Lall, 1989). It plays a fundamental role in the activity of enzymes such as cytochrome oxidase, superoxide dismutase, lysyl oxidase, dopamine hydroxylase and tyrosinase. In addition, copper-proteins and chelates also have metabolic roles (Watnabe et al., 1997). Copper metabolism revealed similarities to mammals in the distribution of copper and copper dependent enzymes (Syed and Coombs, 1982). Copper levels are high in eyes (iris and choroid), liver, brain and heart. A copper-protein complex ceruloplasmin exhibiting oxidative activity occurs in blood plasma (Watnabe et al., 1997). Speruloplasmin appears to be enzymatic in function though its specific character is unknown. Apart from the enzymatic function, Cu proteins and chelates are involved in various pivotal metabolic roles (Table 2). Marine invertebrates, especially mollusks, possess a blue coloured Cu containing complex in the haemolymph called the

<table>
<thead>
<tr>
<th>Trace minerals</th>
<th>Chemical form used/Source</th>
<th>Model fish</th>
<th>Importance/Essentiality</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron (Fe)</td>
<td>Ferrous sulphate (FeSO₄)</td>
<td>Sword, platy fish, trout, rainbow trout</td>
<td>Improved growth, increased diet oxidation, proper fatty acid metabolism, and biosynthesis of LC-PUFA</td>
<td>Roeder and Roeder, 1966; Desjardins et al., 1987; Senadheera et al., 2012</td>
</tr>
<tr>
<td>Copper (Cu)</td>
<td>Copper sulphate pentahydrate (CuSO₄)</td>
<td>All fish species, rainbow trout, carp, channel catfish, marine invertebrates</td>
<td>Anaemia prevention, proper enzymatic activity, metabolism, proper growth</td>
<td>Sakamoto and Yone, 1979; Ogino and Yang, 1980; Murai et al., 1981; Lanno et al., 1985b; Watnabe et al., 1997</td>
</tr>
<tr>
<td>Manganese (Mn)</td>
<td>Manganese sulphate (MnSO₄·4H₂O) and manganese chloride (MnCl₂)</td>
<td>All species, channel catfish, rainbow trout and brook trout</td>
<td>Good growth, increased protein synthesis, prevented fat synthesis in liver, proper growth, proper hatchability of eggs</td>
<td>Satoh et al., 1987b</td>
</tr>
<tr>
<td>Zinc (Zn)</td>
<td>Zinc sulphate (ZnSO₄), zinc nitrate (ZnNO₃), zinc chloride (ZnCl₂)</td>
<td>Rainbow trout, rainbow trout and brook trout, rainbow trout</td>
<td>Improve growth and reduce cataract problem, feed efficiency</td>
<td>Satoh et al., 1987a and 1987b, Ogino and Yang, 1980; Murai et al., 1981; Lanno et al., 1985b; Watnabe et al., 1997</td>
</tr>
<tr>
<td>Selenium (Se)</td>
<td>Selenite, Selenate, selenomethionine, selenocystene</td>
<td>Salmon fry, atlantic salmon, selenite and tocopherol, rainbow trout</td>
<td>Mortality noted in deficiency and prevented when administered, prevention of oxidative cellular injury, prevention of deficiency signs</td>
<td>Poston et al., 1976; Bell et al., 1987; Bell et al., 1985</td>
</tr>
<tr>
<td>Cobalt (Co)</td>
<td>Cobalt chloride and cobalt nitrite</td>
<td>Carp, tilapia</td>
<td>Enhances growth and haemoglobin production, higher growth and good protein efficiency ratio, improved growth, energy retention and liver glycogen deposition</td>
<td>Castell et al., 1986; Anadu et al., 1990; Shiau and Lin, 1993</td>
</tr>
<tr>
<td>Chromium (Cr)</td>
<td>Chromic oxide, Chromic chloride, High Cr-yeast, Chromium nicotinate, Chromium picolinate</td>
<td>Tilapia, carp</td>
<td>Improved growth, energy retention and liver glycogen deposition, improve glucose utilization</td>
<td>Shiau and Lin, 1993; Hertz et al., 1989</td>
</tr>
</tbody>
</table>

**TABLE 2:** Summary of research work done on these trace minerals

- **Iron (Fe):** Iron is an essential trace element for all animals including fish. It plays a fundamental role in the activity of enzymes such as cytochrome oxidase, superoxide dismutase, lysyl oxidase, dopamine hydroxylase and tyrosinase. Feeds of animal origin other than milk by-products are good sources of iron. Common fish feed ingredients viz., fish meal and meat meal contain 150 to 800 mg Fe/kg (approx.), cereals grain ranges from 30 to 60 mg/kg and oil seed protein contain iron ranges from 100 to 200 mg/kg approximately (Lall, 1989).

- **Copper:** Copper (Cu) is an essential trace element for all animals including fish (O’Dell, 1984; Mertz, 1986; Lall, 1989). It plays a fundamental role in the activity of enzymes such as cytochrome oxidase, superoxide dismutase, lysyl oxidase, dopamine hydroxylase and tyrosinase. In addition, copper-proteins and chelates also have metabolic roles (Watnabe et al., 1997). Copper metabolism revealed similarities to mammals in the distribution of copper and copper dependent enzymes (Syed and Coombs, 1982). Copper levels are high in eyes (iris and choroid), liver, brain and heart. A copper-protein complex ceruloplasmin exhibiting oxidative activity occurs in blood plasma (Watnabe et al., 1997). Speruloplasmin appears to be enzymatic in function though its specific character is unknown. Apart from the enzymatic function, Cu proteins and chelates are involved in various pivotal metabolic roles (Table 2). Marine invertebrates, especially mollusks, possess a blue coloured Cu containing complex in the haemolymph called the...
haemocyanin. Copper also serves as an oxygen carrier in haemolymph of these organisms (Lall, 1989). The requirement for copper depends on physiological state of animal, copper content of the water, and the levels of zinc, iron, calcium and molybdenum, which are metabolic antagonists of copper. Hence, those elements compete for the same binding sites on proteins responsible for mineral absorption and synthesis of metalloenzymes. However, the details of such mechanisms in fish are little known. Afterwards it was reported that in rainbow trout for absorption in the intestinal tract, copper and zinc do not compete for the same binding site (Knox et al., 1982; Lanno et al., 1985b).

The copper content of tissue declines when the diets of carp and rainbow trout are devoid of copper (Ogino and Yang, 1980). However, in the same fish copper deficiency affected the activities of cytochrome oxidase in heart and copper zinc superoxide dismutase in liver. The copper requirement in different fish ranges from 3.5 mg/kg dry diet as reported (Watnabe et al., 1997). Higher Cu levels in diet about 600 mg/kg shows no adverse effect in Rainbow trout. But it is reported elsewhere that, 730 mg/kg Cu in diet reduced growth and induces an aversion to food (Lanno et al., 1985b). Copper toxicity cause damage to gills and necrosis to liver and kidney (Table 1). Higher copper levels depress growth and impaired feed conversion in channel catfish (Murai et al., 1981). A differential effect of ascorbic acid on dietary and waterborne copper has been noted which was unexplained to some extent. The toxicity and tissue retention of copper contained in water was affected by dietary ascorbic acid in carp and rainbow trout (Yamamoto et al., 1977, 1981). Later it was found that ascorbic acid had little effect on either the uptake and/or the metabolism of dietary copper in rainbow trout (Lanno et al., 1985b).

Most feeds and the aquatic medium generally contain copper in amounts adequate for fish. Plant and animal protein feed ingredients contain 5-30 mg/kg of copper, whey products and fish solubles are relatively rich sources of copper. Moreover during processing of feed copper content can be increased by using protein enriched sources. In processed feed ingredients wide variation can occurs in copper content due to metal contamination (Watnabe et al., 1997).

Manganese: Manganese is one the important elements required in fish. It is widely distributed in fish and other animal tissue as well. Mainly manganese is act as co-factor for the enzymes peptidase, arginase, succinic decarboxylase and also activates specific enzymes such as glycosyltransferase and non-specific enzymes such as kinases, transferases, hydrolases and decarboxylases. The manganese also acts as an integral part of metalloenzymes (Table 1). This element has a great implication in performing oxidative phosphorylation. Manganese content in mitochondria is higher than cytoplasm.

<table>
<thead>
<tr>
<th>Trace minerals</th>
<th>Model fish</th>
<th>Requirement level (mg/kg diet)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron (Fe)</td>
<td>Atlantic salmon</td>
<td>33 – 100</td>
<td>Bjornvic and Maage, 1993</td>
</tr>
<tr>
<td>Copper (Cu)</td>
<td>Rainbow trout and Carp</td>
<td>3</td>
<td>Ogino and Yang, 1980</td>
</tr>
<tr>
<td>Manganese (Mn)</td>
<td>Channel catfish</td>
<td>2.4</td>
<td>Gatlin and Wilson, 1984</td>
</tr>
<tr>
<td>Iodine (I)</td>
<td>Atlantic salmon</td>
<td>4.5</td>
<td>Lall et al., 1985</td>
</tr>
<tr>
<td>Selenium (Se)</td>
<td>Rainbow trout</td>
<td>0.15 – 0.38</td>
<td>Hilton et al., 1980</td>
</tr>
<tr>
<td>Cobalt (Co)</td>
<td>All general fishes</td>
<td>0.05 – 1.0</td>
<td>Watnabe et al., 1997</td>
</tr>
</tbody>
</table>
It is responsible for normal functioning of brain and proper lipid and carbohydrate metabolism. It has been further demonstrated that manganese has an active part in the activation of leucine aminopeptidase (Clark et al., 1987). Mechanism of manganese uptake from water as well as gastrointestinal tract was not well documented. Later on Srivastava and Agrawal (1983) reported the mechanism of water dissolved manganese uptake, but it is better absorbed through diet intake. Manganese has very important role in brood stock nutrition. The manganese content of the diet influences its level and that of other trace elements in gonads (Satoh et al., 1987a).

Manganese deficient supply in fish usually results in retard growth. It has been reported that rainbow trout and carp show poor growth when a diet with inadequate manganese is fed (Ogino and Yang, 1980). Satoh et al., (1983 a,b) further reported that due to manganese deficiency, dwarfism linked disturbances in bone formation and cataract in eye lens was observed in rainbow trout and carp and again a reduction in skeletal manganese content has been noted corresponding to insufficient dietary supply of manganese.

In tilapia deficiency syndrome included reduction in feed intake, loss of equilibrium, poor growth and increased mortality rate (Ishac and Dollar, 1968). Fish meal diet devoid of manganese significantly influences the mineral composition in common carp gonads. It was reported elsewhere that eggs of brook trout and rainbow trout subsequently shows poor hatchability when fed manganese deficient fish meal diets (Takeuchi et al., 1981 and Lall, 1989). In carp manganous sulfate and manganous chloride and better sources. Good growth was recorded in carp provided with manganous sulfate in the diet and it also increased protein synthesis and prevented fat synthesis in liver. The requirement of manganese in fish is 2-20 mg kg / dry diet. It is reported elsewhere that a supplement of 10 mg/kg manganese required for proper growth without deficiency symptoms (Satoh et al., 1987b). It was reported elsewhere that about 2.5 mg/kg manganese required for proper growth in young channel catfish (Gatlin and Wilson, 1984). The content of manganese in fishmeal ranged from 4-38 mg/kg, depending on the fish. Herring meal contains only 4-12 mg/kg. Among the plant sources, cereals contain 8-50 mg/kg and corn 4-11 mg/kg, rice bran, wheat middling and corn distillers dried soluble are good source of manganese (Wattab et al., 1997). On the basis of growth, appearance of dwarfism and the manganese content in vertebrae, it can conclude that manganese appendage was indispensable in diets formulated using brown fish meal and sardine meal, as noted for white fish meal based diets. However, the availability of manganese in different fish meals to carp was very high and not affected by tricalcium phosphate present in the diet (Satoh et al., 1989).

**Zinc:** Zinc is an important trace mineral in fish nutrition as it plays key role in various metabolic pathways like prostaglandin metabolism and structural role in nucleoproteins. Zinc is an integral part of about 20 metalloenzymes such as alkaline phosphatase, alcohol dehydrogenase and carbonic anhydrase. It also serves as a specific cofactor of several enzymes and act as a catalyst for regulating the activity of several Zn-dependent enzymes. Recent research of zinc-gene interactions has revealed the basic role of zinc in controlling growth (Chesters, 1991). Zinc has a structural role in nucleoproteins and involved in prostaglandin metabolism (Lall, 1979). Though a little data is documented related to this but it is evident that some clinical features of zinc deficiency may arise from disturbances in metabolism of protein and nucleic acid. Intake of zinc in fish occurs from both feed and water, but dietary supplement is more efficiently utilized. Gills and gastrointestinal tract are the main sites of zinc intake in fish (Pentreath, 1973; Lovegrove and Eddy, 1982). In winter flounder fish entire digestive tract is capable of absorbing zinc, but the uppermost position of intestine has the highest capacity and the stomach has the lowest (Shears and Fletcher, 1983). However Hardy et al. (1987) reported that in rainbow trout gills play a major role in excretion of dietary zinc. It is also documented that generally zinc is excreted through kidney and by chloride cells of gills (Bryan, 1976). It is reported elsewhere that even when an optimum dietary zinc is supplemented, uptake of water dissolved zinc is also occurs (Spry et al., 1988). Deficiency syndrome of zinc in fish is summarized in Table 1.

Deficiency of zinc results in poor growth, lowered digestibility of protein and carbohydrate, probably due to reduced carboxypeptidase activity (Ogino and Yang., 1978). Zinc deficiency also recorded eye lens cataract and erosion of fins and skin, and sign of short-body dwarfism (Ogino and Yang, 1979; Hughes, 1985). In catfish, diets low in zinc resulted reduced appetite, low growth, low bone zinc and calcium levels and serum zinc concentration (Gatlin and Wilson, 1983). The mineral composition of common carp gonads was significantly affected by zinc depletion from the mineral supplementation in fishmeal diet (Satoh et al., 1987a). Fortification of brood stock diets of salmon with zinc increased the content of this mineral in the ovaries of female Salmon (Hardy et al., 1984). However it is reported by Knox et al., (1982) that in rainbow trout amplified levels of zinc in diet (500 to 1000 mg/kg) cause reduction in haemoglobin, haematocrit and hepatic copper concentration. In rainbow trout zinc deficiency impairs immunological response (Kiron et al., 1987).
et al., 1993). Deficiency syndrome of zinc is summarized in Table 1.

Zinc availability differs in plant and animal source. Fish meal contains about 80 – 100 mg/kg of zinc. However, zinc concentration in vegetables and proteins ranges from 40 – 80 mg/kg and in cereal grains is 15 – 30 mg/kg. (Watnabe et al., 1997). However, Eisler (1980) have documented the zinc sources of various marine invertebrates, vertebrates and plants from several geographic locations throughout the world. The requirement of zinc in fish is presented in Table 3.

**Iodine:** Iodine metabolism is related to thyroid hormones which help in the regulation of the level of metabolic activity in fish. The hormones have wide influence on cellular oxidation, neuromuscular control, circulatory dynamics, nutrient metabolism and growth. Triiodothyronine (T3) is the major hormone of the thyroid gland and also an active precursor of thyroxine (T4) (Watnabe et al., 1997). Fish differ from mammals in iodine utilization and in the extrathyroidal metabolism of T3 and T4 (Higgs et al., 1982). T3 binds more strongly to plasma proteins than T4, and its turnover rate in trout is low compared to T4. Excretion of T3 and T4 occurs mainly through bile, but the gills and kidney also involved (Watnabe et al., 1997). A report of NRC (1983) noted that thyroid hyperplasia (goitre) induced by iodine deficiency in salmonid fish. Agrawal and Mahajan (1981) reported that iodine uptake by thyroid tissue was reduced in ascorbic acid deficient catfish. Iodine can be taken up from the surrounding water via the gills, and the uptake rate is inversely dependent on the calcium content of the water (Hunn and Fromm, 1966). The plasma iodine level of freshwater fish ranges from 0.5 to over 2000 µg/l (Gregory and Eales, 1975). It is dependent on the iodine content of the diet and water, on the iodine level of tissue and on the iodine-binding capacity of plasma proteins. Freshwater fish depend more on a dietary source for iodine. The iodine from the diet is easily absorbed in the digestive tract (Gregory and Eales, 1975).

As seawater contains more iodine than freshwater, iodine deficiency signs appear more in freshwater fish. Age, physiological state and stress factors considerably influence the requirement for this mineral. Gaylord et al., (1914) noted thyroid carcinoma results from the deficiency of iodine in brook trout.

Marine plants and animals are rich sources of iodine. Some seaweed contain up to 0.1% iodine. There is a wide variation in the iodine content of feedstuffs; animal proteins, excluding fish meals, contain only negligible amounts. Normal herring meal and capelin meal have only 5-10 mg I kg⁻¹. On the other hand, Atlantic white fish meal may contain up to 60-90 mg I kg⁻¹. The iodine content in plant protein concentrates is very low (Watnabe et al., 1997).

**Selenium:** Selenium is an integral component of glutathione peroxidase (Rostruck et al., 1973). The active level of this enzyme in liver or plasma is indicative of Selenium supply to the organism. Selenium along with vitamin E is essential to prevent nutritional muscular dystrophy. Selenium is implicated in the metabolism of tocopherol compounds. Both act as an antioxidant substance in fish. Selenium compounds are also capable of protecting heavy metal toxicity like cadmium and mercury (Watnabe et al., 1997). A correlation between selenium and copper has been observed in rainbow trout and Atlantic salmon (Hilton, 1989). Selenium acts as the principal factor in the protective mechanism against oxidative cellular injury. In the liver the glutathione peroxidase activity is lowered, while glutathione transferase activity and pyruvate kinase activity in plasma were increased. Poston et al., (1976) reported that salmon fry shows mortality when diet with selenium deficient is supply and again recovered when administered with selenium. In a study reported by Bell et al., (1987) on Atlantic salmon it was highlighted that selenium has a protective mechanism against oxidative cellular injury. It was reported elsewhere that maximum glutathione peroxidase activity noticed in the plasma of rainbow trout when fed with diet fortified by selenium in the range of 0.15 – 0.38 mg/kg (Hilton, 1980). In an another report by Bell et al., (1985) it was showed that a diet with a combination of selenium and tocopherol at 0.9 mg/kg and 41 mg/kg respectively can prevent deficiency signs in rainbow trout.

Selenium deficiency results growth depression, loss of appetite, lethargy, reduced muscle tone and mortality in Atlantic salmon (Poston and Combs, 1979). Deficiency of selenium and vitamin E are the probable factors responsible for Hitra disease in farmed salmon (Fjølstad and Heyeraas, 1985; Poppe et al., 1986). Apart from the deficiency symptoms, it is observed that a reduction in tissue concentration of vitamin E and selenium, low haematocrit values and increased haemolytic rates (Watnabe et al., 1997) and some deficiency syndromes are presented in Table 1.

Fish derive selenium from both diet and water. High levels of Selenium (40 – 130 µg/l) in water are toxic to fish. The usual concentration of selenium in water is less than 0.1 µg/l (Watnabe et al., 1997). Han et al., in 2011 reported that the dietary selenium requirement is 1.18 mg/kg in case of Gilbel carp (Carassius auratus gibelio). Selenium is stored in various tissues, except liver, in its inorganic form. Selenium as selenite is effectively taken up through the gills. Selenium

**Table 1.**
and vitamin E are complement to each other activity, and protect biological membranes against lipid oxidation.

Fishmeal and marine by-products are the rich sources of Selenium to fish. Plant materials vary widely in their Selenium content. Among the source of selenium selenomethionine was the most digestible (92%), and fishmeal (47%) the least digestible sources of selenium. The glutathione peroxidase selenium ration indicated that selenium supplied as selenite or selenocystine was a better source for plasma glutathione peroxidase than was selenium from selenomethionine. Though the comparative availability of Selenium is poor, still fishmeal-based diets generally provide sufficient selenium to satisfy the nutritional requirement of fish (Watnabe et al., 1997).

**Cobalt:** Cobalt is a component of cyanocobalamin (vitamin B\(\text{_{12}}\)), constituting nearly 4.5% of its molecular weight. (Watnabe et al., 1997). Most animals need the element for the synthesis of the vitamin B\(\text{_{12}}\) by intestinal microflora, and such bacteria have also been isolated from the intestinal tract of fish (Kashiwada et al., 1970). Cobalt as part of vitamin B\(\text{_{12}}\) is associated with nitrogen assimilation, and synthesis of haemoglobin and muscle protein. In addition, cobalt influences certain enzymes. Cobalt binds to insulin (Cunningham et al., 1955) and also reduces plasma glucose levels (Roginski and Mertz, 1977). Dietary cobalt has been found to be beneficial for haematology and growth of fish; the erythrocyte count of young carp increased and rainbow trout grew better (Frolova, 1960 and Sabalina, 1964; Steffens, 1989) and gold-spot mullet exhibited improved survival and growth (Ghosh, 1975). Cobalt in carp diets resulted in better growth (Sukhoverkov, 1967) survival of hatchlings (Khan and Mukhopadhy, 1971) and improved synthesis of protein (Bhanot and Gopalakrishnan, 1973). The uptake of cobalt has been demonstrated in rainbow trout eggs at the embryonic development stage (Kuenze et al., 1978). Hertz et al. (1989) examined the effects of cobalt on glucose metabolism of carp. Based on glucose utilization, they suggested an increase in insulin effectiveness caused by cobalt. They also mentioned that the mineral may be associated with a protein-sparing effect based on increased incorporation of labeled leucine into white muscle proteins, and improved growth and protein efficiency in cobalt fed fish. Very high doses of cobalt (0.1-5 g Co/kg body weight) were toxic to rainbow trout, resulting in haemorrhages in the digestive tract and alterations in white blood cells (Sabalina, 1968; Steffens, 1989). Cobalt deprivation reduced the intestinal synthesis of vitamin B\(\text{_{12}}\) in catfish (Limsuwan and Lovell, 1981).

Cobalt can be absorbed from the surrounding water through the gills as well as from the diet. Several studies with various trout by Phillips and coworkers (Phillips et al., 1956, 1957, 1958, 1960) examined both modes of cobalt uptake. The uptake of waterborne cobalt increased with a rise in temperature and decrease in waterborne calcium. It was found in brook trout that a large proportion of the mineral absorbed from the diet was at first retained in the digestive tract. The initial metabolism after absorption of cobalt occurs in the pyloric caecum and intestine. The dietary cobalt demand of fish has been put at 0.05 mg kg\(^{-1}\) diet (Lovell, 1979). Generally, the cobalt content in fish feeds is in the range of 1 – 6 mg kg\(^{-1}\) diet (Tacon and De Silva, 1983). The mineral is supplied as cobalt chloride. Cobalt chloride and cobalt nitrate from feed and cobalt chloride from ambient water improved growth and enhanced haemoglobin production in carp (Castell et al., 1986). Cobalt chloride supplementation also resulted in higher growth and protein efficiency ratio in tilapia (Anadu et al., 1990). The feed composition has an appreciable effect on cobalt supply as the demand for the trace element is very low and can probably be derived from numerous feedstuffs (Steffens, 1989).

**Chromium:** Chromium (Cr) shows essentiality for both animals and humans. It is mostly occurs in oxidative states viz., Cr (II), Cr (III) and Cr (VI). Cr (III) is required for proper carbohydrate and lipid metabolism. The ability of Cr to form co-ordination compounds and chelates, is an important chemical characteristic that makes this essential element available to living organisms. In food Cr is available in inorganic form Cr (III) as a part of biologically active molecule. Though the biological structure of the active form of Cr is not fully characterized, it appears to be a dinicotinatochromium (III) complex, stabilized with glutathione or its constituent amino acids (Toepfer et al., 1977). Biologically the function of Cr is closely alike with that of insulin. Most of the Cr-potentiated reactions are also insulin dependent as well. In humans Cr potentiates the action of insulin in vitro and in vivo; maximal in vitro activity requires a chemical form termed the glucose tolerance factor and tentatively identified as Cr-nicotinic acid complex (Mertz, 1993). It was reported elsewhere that supplementation of Cr with carp diets improves the glucose utilization by modulation of endogenous insulin activity (Hertz et al., 1989). Again Shiau and Lin, (1993) reported that Tilapia diet containing glucose with chronic oxide showed improved growth with better energy retention and liver glycogen deposition. But Nag and Wilson in 1997 reported that there is no effect in glucose utilization in Channel catfish supplemented with Cr containing diets. No adverse effect is noticed in rainbow trout fed with low Cr containing purified diet as reported by Tacon and Beveridge (1982).
The dietary toxicity of Cr (III) is not well established, though reports have been made on the toxicological effects of Cr (VI) in brook trout by Benoit (1976). The commonly used Cr feed supplements are chromic chloride, high Cr-yeast, Cr-nicotinate, Cr-picolinate.

CONCLUSION

Trace minerals are essential for fish and involved in the normal metabolism and life processes. The trace minerals requirement in fish are; Iron 30-170, Copper 3-5, Manganese 2-30, Zinc 15-80, Iodine 4-5, Selenium 0.15 to 8 and Cobalt 0.05 to 1.0 (mg/kg diet). The minerals are required in extremely small quantity in the diet but excess supplementation through a continuous process can cause severe toxicity. In deficient condition, growth is retarded with abnormal metabolism in particular. It is also a matter of concern about the potential causes of conditioned trace element deficiencies such as food processing methods, dietary interactions, disease conditions and genetic disorders. Fish can absorb part of the required minerals directly from the water through gills or even through their entire body surface. The minerals absorbed from water do not meet the total requirement and a certain supplementation through the diet is required whether in the natural food or supplementary feed. Definitive data on requirements should involve systematic tests on the metabolic functions of the mineral. Precise analytical procedure is essential for detection of ultra trace element at tissue level, when present in minute quantity. There is also insufficiency activity regarding trace minerals effect on fatty acid metabolism specially PUFA. It should be taken care of so that specific diet could be composed with supplemented trace minerals to meet up the need. These approaches should highlight future research and potential strategies to maximize the activity on the requirement of trace mineral nutrition in fish. Once this requirement is met, the normal growth, survival and essential cellular metabolism may be ensured.

REFERENCE


