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Trace minerals in fish nutrition

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Abstract

The role of trace elements in biological systems has been described in several animals. However, the knowledge in fish is mainly limited to iron, copper, manganese, zinc and selenium as components of body fluids, cofactors in enzymatic reactions, structural units of non-enzymatic macromolecules, etc. Investigations in fish are comparatively complicated as both dietary intake and waterborne mineral uptake have to be considered in determining the mineral budgets. The importance of trace minerals as essential ingredients in diets, although in small quantities, is also evident in fish. © 1997 Elsevier Science B.V.

Keywords: Fish nutrition; Minerals; Iron; Copper; Manganese; Zinc; Selenium

1. Introduction

Minerals are required for the normal life processes, and all animals, including fish, need these inorganic elements. Fish may derive these minerals from the diet and also from ambient water. Characteristic concentrations and functional forms of the minerals need to be maintained within narrow ranges for ordinary metabolic activities in cells and tissues. This is facilitated by the homeostatic mechanisms operating in the animal, catering for the fluctuations in dietary intake. The minerals are responsible for skeletal formation, maintenance of colloidal systems, regulation of acid–base equilibrium and for biologically important compounds such as hormones and enzymes. Mineral deficiencies can cause biochemical, structural and functional pathologies which depend on several factors, including the duration and degree of mineral deprivation.

The physiological importance of minerals is well documented for humans and some animals. However, many aspects of intake, function and bioavailability of trace minerals are still unclear. Not all of the trace elements essential for higher animals (Table 1) have

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Table 1
Trace minerals important for mammals, birds and fish

Cobalt	Arsenic [a]
Copper	Molybdenum [a]
Iron	Fluorine [a]
Manganese	Lead [a]
Selenium	Nickel [a]
Zinc	Silicon [a]
Chromium	Vanadium [a]
Iodine	Lithium [a]

[a] Limited information on fish.

been described in fish. Information on nutritional requirements of fish for trace elements is also fragmentary, particularly because many are needed only in very small amounts (Table 2). Although the trace metals 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. Therefore, the animal maintains a delicate balance of the body levels of the trace metals by integrating the various parameters of uptake, storage and excretion.

The biological availability of a mineral from the diet is marked by the efficiency with which the body utilizes the dietary mineral. It varies depending on the feedstuffs and the composition of a diet. Several factors influencing bioavailability include the level and form of the nutrient, particle size and digestibility of the diet, nutrient interactions which may be either synergistic or antagonistic, 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 states and ligands when ingested from different diets. The insoluble 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 favourable binding sites. In addition to all these diet-related mechanisms, several aspects linked to the environment influence mineral bioavailability.

Table 2
The trace mineral requirement ranges for fish

Mineral	Requirement [a]
Iron	30–170
Copper	1–5
Manganese	2–20
Zinc	15–40
Cobalt	0.05–1.0
Selenium	0.15–0.5
Iodine	1–4

[a] Expressed as mg mineral kg^{-1} dry diet.

The mineral nutrition of fish has been fairly well investigated and some of the achievements have been highlighted in reviews by Watanabe et al. (1988), Hilton (1989), Lall (1989) and Steffens (1989). The present article collates information on several trace elements in nutrition of fish, excluding shellfish, with emphasis on zinc and manganese.

2. Iron

Iron has an active part in oxidation/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. Haemoglobin occurs in erythrocytes while transferrin is found in plasma; the latter is the principal carrier of iron in blood.

The iron content of fish is very low compared to that of mammals (Van Dijk et al., 1975). Information on absorption and metabolism of iron in fish is very limited, 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. Deficiency of iron induces anaemia in brook trout (Kawatsu, 1972), yellowtail (Ikeda et al., 1973a), red seabream (Sakamoto and Yone, 1978a) and carp (Sakamoto and Yone, 1978b). Microcytic, hypochromic anaemia in iron-deficient brook trout was characterized by a drop in haemoglobin content and erythrocyte count and deteriorated growth rate. In carp, the anaemic condition was the same, but there was no growth retardation. Gatlin and Wilson (1986a) additionally noted poor feed utilization and lowering of plasma iron level and transferrin saturation in channel catfish. Sakamoto and Yone (1978b) identified a yellowish white liver condition in carp caused by iron deficiency. The hatching rate of rainbow trout eggs was poor when the iron content was low (Hirao et al., 1955).

2.1. Availability

It has been amply demonstrated that fish can take up iron via the gills. The iron content of blood plasma and of the water medium has been found to be positively correlated in salmonids (Merk, 1987). Addition of iron in water as ferrous sulphate improved the growth of sword tail and platy fish, indicating a nutritional benefit from iron dissolved in water (Roeder and Roeder, 1966). An adequate supply of iron from the diet reduced the dependence on gill absorption of iron in guppies (Segner and Storch, 1985).

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 tract. The inorganic iron is reduced to the ferrous state and freed from the conjugate for absorption. Studies on different iron salts in diets of red seabream demonstrated differences in absorption (Sakamoto and Yone, 1979). On comparing the effectiveness in preventing anaemia, it was shown that ferrous and ferric chloride were more highly available than ferric citrate.

Iron is one of the primary metals involved in lipid oxidation. Ferrous iron, which is more potent than ferric iron, catalyses the formation of hydroperoxides and free radical peroxides by providing a free radical initiator in the presence of unsaturated fatty acids and oxygen (Chvapil et al., 1974; Lee et al., 1981; Fujimoto et al., 1982). The supplementation of ferrous iron in fish diets having high levels of lipid and/or polyunsaturated fats may affect the stability of the diet. Desjardins et al. (1987) demonstrated that addition of ferrous sulphate to a trout diet significantly increased the oxidation of that diet.

As in other domestic animals, ascorbic acid is involved in the metabolism of iron and vice versa in fish (Hilton et al., 1978; Desjardins, 1985). The apparent interaction between iron and ascorbic acid metabolism may be due to the effects of iron supplementation on diet rancidity and dietary ascorbic acid stability (Desjardins, 1985). Although ascorbic acid deficiency affected iron-related blood parameters in many fish (Hilton et al., 1978; Lim and Lovell, 1978; Agrawal and Mahajan, 1980), it is surprising that increasing levels of ascorbic acid did not affect the absorption of iron in fish (Lanno et al., 1985a).

Feeds of animal origin such as fish meal and meat meal are rich sources of iron, containing about 400–800 mg kg^{-1}. Oil seeds contain 100–200 mg Fe kg^{-1}, while cereals contain 30–60 mg Fe kg^{-1}. The iron from animal sources may occur as porphyrin, myoglobin and haemoglobin. It may be in a complexed form with phytin in cereals. The availability of iron in the various feedstuffs for fish depends on its form.

3. Copper

Copper is important for animals as it is involved 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. One of the few investigations on copper metabolism in fish by Syed and Coombs (1982) revealed similarities to mammals in the distribution of copper and copper-dependent enzymes. Copper levels are high in the eyes where it is found along with melanins, bound to protein. Organs such as liver, brain and heart also contain comparatively large amounts of copper. A copper–protein complex, ceruloplasmin, exhibiting oxidative activity, occurs in blood plasma.

When the diets of carp and rainbow trout had low levels of copper, there was a decline in the tissue copper content (Ogino and Yang, 1980). Considerably higher dietary copper levels (about 600 mg kg^{-1}) did not adversely affect rainbow trout. However, over 730 mg Cu kg^{-1} diet reduced growth and induced an aversion to food (Lanno et al., 1985b). Copper toxicity may cause damage to gills and necrosis in liver and kidney. High levels of copper depressed growth and impaired feed conversion in channel catfish (Murai et al., 1981). In the same fish, Gatlin and Wilson (1986b) showed that a deficiency of copper affected the activities of cytochrome *c* oxidase in heart and copper–zinc superoxide dismutase in liver. The optimal level of copper in diet as determined for several fish ranges from 3 to 5 mg Cu kg^{-1} diet.

3.1. Availability

Most feeds and the aquatic medium generally contain copper in amounts adequate for fish. The requirement for this mineral depends to a great extent on the physiological state of the animal, the copper content of the water, and the levels of zinc, iron, cadmium and molybdenum, which are metabolic antagonists of copper. Copper and zinc may be antagonistic because of the similar nature of the valence shell hybrids (Hill and Matrone, 1970). Therefore they compete for binding sites on proteins responsible for mineral absorption and/or synthesis of metalloenzymes. However, the details of such mechanisms in fish are little known. It has been suggested that, when zinc absorption is not sufficient in trout, zinc–copper antagonism may come into play (Spinelli et al., 1979). Later studies (Knox et al., 1982; Lanno et al., 1985b) in rainbow trout indicate that copper and zinc do not compete for the same binding site for absorption in the intestinal tract.

An unexplained differential effect of ascorbic acid on dietary and waterborne copper has been noted. 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).

Plant and animal proteins feed ingredients usually contain 5–30 mg Cu kg^{-1}. Whey products and fish solubles are relatively rich sources of copper, providing over 85 mg kg^{-1}. More copper is taken up by protein-rich components under favourable moisture conditions during processing. Metal contaminants can cause wide variations in the copper content of processed feed ingredients.

4. Manganese

Manganese is important for fish and is widely distributed in fish and animal tissue. The mitochondria have a greater concentration of manganese than cytoplasm or other cell organelles. Manganese is necessary for the normal functioning of brain and for proper lipid and carbohydrate metabolism. The mineral has two roles: first as a cofactor for enzymes which form metal–enzyme complexes; and second as an integral part of metalloenzymes. Manganese activates specific enzymes such as glycosyltransferase and non-specific enzymes such as kinases, transferases, hydrolases and decarboxylases. The activation of leucine aminopeptidase by manganese has been demonstrated in sole (Clark et al., 1987).

An inadequate supply of manganese usually results in retardation of growth. Ogino and Yang (1980) obtained poor growth in rainbow trout and carp when the diets were low in manganese. When young tilapia were deprived of manganese, Ishac and Dollar (1968) observed reduced intake of food, loss of equilibrium, depressed growth and increased mortality. Other deficiency signs such as dwarfism linked to disturbances in bone formation and cataract of eye lens have been observed in rainbow trout and carp. A reduction in skeletal manganese content has been noted corresponding to insufficient dietary supply of that mineral (Satoh et al., 1983a,b,c). Knox et al. (1981) found that,

when the intake of manganese was low in rainbow trout, the copper–zinc superoxide dismutase and manganese superoxide dismutase activities in cardiac muscle and liver were reduced. However, Gatlin and Wilson (1984b) observed in catfish that the liver manganese superoxide dismutase activity was not affected, even at a low level of dietary manganese. The importance of manganese has been recognized in broodstock nutrition. The manganese content of the diet influences its level and that of other trace elements in gonads (Satoh et al., 1987c). The absence of manganese in a fish meal diet significantly influenced the mineral composition of common carp gonads. The eggs produced by broodstock of brook trout and rainbow trout fed fish meal diets lacking manganese contained only low levels of this trace metal and subsequently hatchability was poor (Takeuchi et al., 1981; Lall, 1989).

4.1. Availability

The mechanism of manganese uptake from the water medium and the gastrointestinal tract is not clear. Srivastava and Agrawal (1983) have demonstrated the uptake of waterborne manganese, but the mineral is better absorbed via the dietary mode. High levels of dietary calcium and phosphorus considerably reduce the absorption of manganese.

The availability of manganese differs in various inorganic salt supplements. Utilization of manganese in the form of manganous oxide is poor in Atlantic salmon (Lall, 1989). Manganous sulphate and manganous chloride are ranked as superior manganese sources for carp over manganous carbonate (Satoh et al., 1987e). Good growth was recorded in carp provided manganous sulphate in the diet and it was found that manganese supply increased protein synthesis and prevented fat synthesis in the liver (Romanenko, 1984).

In a study by Yamamoto et al. (1983), it was shown that the manganese contained in white fish meal did not satisfy the requirement of rainbow trout. In carp, a diet containing 55% white fish meal, providing 2–3 mg Mn kg^{-1}, fell short of a normal manganese supply. A supplement of 10 mg Mn kg^{-1} was necessary for good growth without deficiency signs (Satoh et al., 1987e). Another study compared the availability of manganese contained in white fish meal to carp with other types, such as brown fish meal and sardine meal with or without solubles (Satoh et al., 1989a). Growth and feed efficiency were poor in fish receiving various fish meal diets without supplementary manganese, but when the diet groups received additional manganese there was an effective improvement (Table 3). Judging from growth, appearance of dwarfism and the manganese content in vertebrae, it appears that manganese supplementation was necessary in diets formulated with brown fish meal and sardine meal, as noted for white fish meal based diets. Nevertheless, 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., 1989a). As the carp lacks a stomach, it cannot dissolve tricalcium phosphate and the chances of interaction between manganese and calcium or phosphate are few. However, later it was shown that supplementation of tricalcium phosphate to semipurified diets caused a reduction in manganese absorption leading to lower levels of the mineral in vertebrae of carp (Satoh et al., 1992a; Table 4). In a different experiment it was found

Table 3
Effect of supplementary manganese levels in four types of fish meal diets on growth, feed efficiency, appearance of dwarfism and manganese content in vertebrae of carp after feeding for 11 weeks

Fish meal diets indicating Mn status	Growth rate (%)	Feed efficiency	Dwarfism (%)	Vertebrae Mn ($\mu g\ g^{-1}$)
White fish meal				
Control [a]	857	1.11	0	1.9
No Mn [b]	691	0.92	21	0.8
Min. Mn [c]	853	1.00	0	1.9
Brown fish meal				
Control	796	1.00	0	1.8
No Mn	608	0.93	11	1.0
Min. Mn	778	1.05	0	1.4
Sardine meal without soluble				
Control	682	0.87	0	1.9
No Mn	585	0.83	11	0.9
Min. Mn	682	0.93	0	1.7
Sardine meal with soluble				
Control	765	1.04	0	1.9
No Mn	605	0.94	14	1.1
Min. Mn	720	0.98	0	1.4

[a] Diet with complete mineral mixture.
[b] Diet without manganese supplement.
[c] Diet with manganese supplement which, along with manganese derived from the fish meal, satisfies the manganese requirement of carp.
Source: Satoh et al., 1989a.

that the availability of manganese contained in white fish meal to rainbow trout also was very low, probably due to calcium and/or phosphorus in white fish meal, which may be the limiting factors (Satoh et al., 1991). It also was determined that more than 15 mg Mn

Table 4
Effect of supplementary tricalcium phosphate at various dietary manganese levels on mineral absorption in carp after feeding for 15 weeks

Dietary supplements		Mineral absorption (%)	
Mn ($mg\ kg^{-1}$)	$Ca_3(PO_4)_2$(%)	Mn	Zn
0	0	0.0	82.9
5	0	64.4	84.6
10	0	69.7	90.6
15	0	73.9	89.2
20	0	80.6	89.2
0	7	0.0	45.1
5	7	10.7	49.9
10	7	18.4	55.8
15	7	18.5	56.3
20	7	18.7	56.2

Source: Satoh et al., 1992a.

Table 5
Effect of various supplementary manganese levels on growth, feed efficiency and appearance of cataract in rainbow trout after 26 weeks of feeding

Supplementary Mn (mg kg^{-1})	Total Mn in diet (mg kg^{-1})	Growth rate (%)	Feed efficiency	Cataract (%)
0	5.1	3817	1.05	7
5	9.7	4021	1.07	7
10	14.4	4104	1.11	3
15	19.1	4296	1.05	0
20	23.9	4136	1.06	0

Source: Satoh et al., 1991.

kg^{-1} diet was necessary to sustain normal growth in rainbow trout (Table 5). Young channel catfish required around 2.4 mg Mn kg^{-1} to support good growth (Gatlin and Wilson, 1984b).

The content of manganese in fish meal ranges from 4 to 38 mg kg^{-1}, depending on the fish. Capelin meal and herring meal contain only 4–12 mg Mn kg^{-1}. Among the plant sources, cereals contain 8–50 mg Mn kg^{-1} and corn 4–11 mg Mn kg^{-1}. Rice bran, wheat middlings and corn distillers' dried soluble are good sources of manganese.

5. Zinc

Zinc is an important trace element in fish nutrition as it is involved in various metabolic pathways. It serves as a specific cofactor of several enzymes. In addition, zinc is an integral part of about 20 metalloenzymes such as alkaline phosphatase, alcohol dehydrogenase and carbonic anhydrase. Zinc is connected with prostaglandin metabolism and also may have a structural role in nucleoproteins. Recent research on zinc–gene interactions has assigned a basic role for this element in controlling growth (Chesters, 1991).

The zinc requirement of rainbow trout is normally met by dietary levels of 15–30 mg kg^{-1} diet (Ogino and Yang, 1978). The average range of zinc in salmon diets is 80–118 mg kg^{-1} (Tacon and De Silva, 1983). Supplementation of 15–600 mg Zn kg^{-1} diet did not produce any difference in growth or feed utilization, or any adverse effects on health of young rainbow trout. Wekell et al. (1983) also did not record any adverse effects to rainbow trout when several hundred milligrams of zinc were included per kilogram of diet. However, deficiency of zinc leads to growth retardation; when the diet contained only 1 mg Zn kg^{-1}, cessation of growth occurred (Ogino and Yang, 1978). Satoh et al. (1987c) demonstrated in a long duration experiment that supplementation of 40 mg Zn kg^{-1} to a white fish meal based diet improved growth in both rainbow trout and carp (Fig. 1). Deficiency of zinc also lowered the digestibility of protein and carbohydrate, probably due to the reduced carboxypeptidase activity (Ogino and Yang, 1978). Additional signs of zinc deficiency are eye lens cataract and erosion of fins and skin (Ogino and Yang, 1979; Hughes, 1985). Later, Satoh et al. (1983a) identified the characteristic

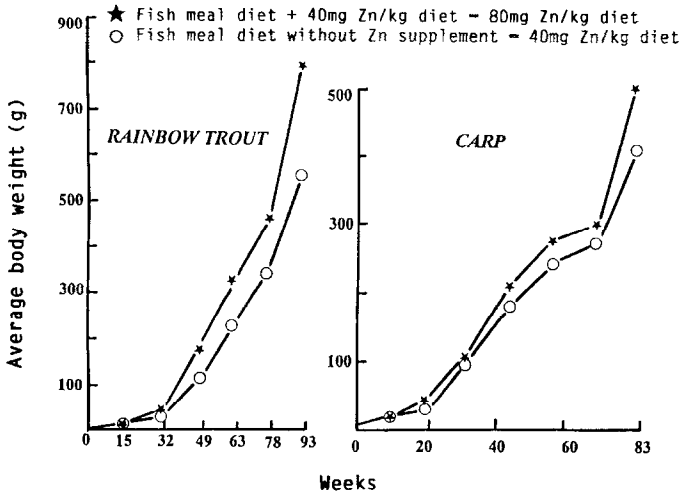

Fig. 1. Growth of rainbow trout and carp fed fish meal diets with or without supplementary zinc.

sign of short-body dwarfism. When catfish diets were low in zinc, appetite was reduced, resulting in low growth, low bone zinc and calcium levels and serum zinc concentration (Gatlin and Wilson, 1983). Zinc should be an essential component in broodstock diets as observed in rainbow trout (Takeuchi et al., 1981). Satoh et al. (1987c) reported that the mineral composition of common carp gonads was significantly affected by zinc deletion from the mineral supplement in a fish meal diet. Fortification of broodstock diets with zinc increased the content of this mineral in the ovaries of female salmon (Hardy et al., 1984). Zinc deficiency also has been found to impair immunological responses in rainbow trout (Kiron et al., 1993).

5.1. Availability

Fish can derive zinc from dietary sources as well as from water. The gills and gastrointestinal tract are involved in the uptake of this element. Relatively low levels of waterborne zinc, less than 1 mg Zn l^{-1} are toxic to fish. On comparing the contributions of dietary and waterborne zinc in rainbow trout, Spray et al. (1988) noted that uptake of zinc from water took place mainly through the gills, irrespective of dietary intake. It was possible to limit dietary zinc deficiency to a certain extent by increasing the zinc concentration in water. Shears and Fletcher (1983) concluded in winter flounder that the portion of the digestive tract with the highest capacity for absorbing zinc is the upper region of the intestine while the stomach has the least capacity. Zinc homeostasis is maintained in fish by regulating the excretory mechanisms and controlling gastrointestinal uptake.

The absorption and utilization of zinc may be affected by the chemical form of zinc in the diet, the source of protein and the presence of other dietary components such as calcium, phosphorus and phytic acid. In an early experiment, Ketola (1979) detected

zinc deficiency signs when white fish meal replaced herring meal in a conventional trout feed formulation. The inadequacy of zinc in white fish meal diets was again observed by Yamamoto et al. (1983) in a long-term experiment. Later, it was determined that diets containing 55% white fish meal provided only 40 mg Zn kg^{-1}, and an identical amount had to be supplemented as zinc sulphate or zinc nitrate for normal development in trout (Satoh et al., 1987d). When growth was compared among rainbow trout fed diets supplemented with different zinc compounds at a level of 20 mg Zn kg^{-1} diet, it was highest with zinc sulphate and lowest in fish fed zinc chloride (Table 6).

The poor absorption of zinc from white fish meal diets was due to high amounts of hydroxyapatite (mainly as tricalcium phosphate) in white fish meal derived from hard tissues such as bone (Satoh et al., 1987a,b). The reduction in availability necessitates the addition of an optimum zinc level in fish meal diets. Gatlin and Wilson (1984a) also made similar observations and had to supplement 150 mg Zn kg^{-1} in a practical catfish diet containing 42% soybean meal and 11% menhaden fish meal. Satoh et al. (1987b) examined the availability of zinc contained in white fish meal, brown fish meal, and sardine meal with or without solubles to rainbow trout. The deficiency of zinc was easily manifested in the white fish meal diet, which contained more tricalcium phosphate compared to the other three (Table 7). Hardy and Shearer (1985) and Satoh et al. (1987a) investigated the zinc antagonistic effect of tricalcium phosphate employing semipurified diets containing it at different levels. The high digestibilities noted for the mineral in the tricalcium phosphate free diets decreased markedly with the elevation of tricalcium phosphate in the diet (Table 8). Supplementation of 7% tricalcium phosphate to an egg albumin diet greatly reduced growth rate and feed efficiency of rainbow trout. A zinc supplement of 80 mg kg^{-1} diet was necessary in the above diet to obtain performance resembling a tricalcium phosphate free diet containing 40 mg Zn kg^{-1} (Table 9). In a related study with carp, it also was found that the availability of zinc was affected by dietary tricalcium phosphate, but the ill-effect on mineral utilization was less pronounced than in rainbow trout. This was because the tricalcium phosphate was not

Table 6
Effect of supplementary zinc in various forms and levels to fish meal diets on growth, feed efficiency and appearance of dwarfism and cataract in rainbow trout after feeding for 40 weeks

Supplementary Zn		Total Zn in diet (mg kg^{-1})	Growth rate (%)	Feed efficiency	Dwarfism (%)	Cataract (%)
Form	Quantity (mg kg^{-1})					
$ZnSO_4$	10	49.1	492	1.04	50	36
$ZnSO_4$	20	60.4	575	1.07	56	33
$ZnSO_4$	40	80.2	550	1.06	0	0
$ZnSO_4$	80	117.0	506	1.12	0	0
$ZnNO_3$	10	50.0	494	1.02	40	30
$ZnNO_3$	20	58.6	482	1.06	30	20
$ZnNO_3$	40	81.1	496	1.12	0	0
$ZnCl_2$	20	58.8	479	1.10	30	30
$5ZnO \cdot 2CO_3$	40	79.3	507	1.06	16	0

Source: Satoh et al., 1987d.

Table 7
Effect of deletion of zinc from four types of fish meal diets on growth, feed efficiency and appearance of cataract and dwarfism in rainbow trout after feeding for 27 weeks

Fish meal diets indicating Zn status	Growth rate (%)	Feed efficiency	Cataract (%)	Dwarfism (%)
White fish meal				
Control [a]	6959	0.92	0	0
No Zn [b]	4914	0.87	50	25
Brown fish meal				
Control	6432	0.97	0	0
No Zn	5650	0.98	37	16
Sardine meal without soluble				
Control	5226	0.98	0	0
No Zn	4756	0.96	10	10
Sardine meal with soluble				
Control	6418	0.95	0	0
No Zn	5001	0.91	16	21

[a] Diet with a complete mineral mixture.
[b] Diet with a zinc-deleted mineral mixture.
Source: Satoh et al., 1987b.

Table 8
Effect of supplementary tricalcium phosphate at two different dietary zinc levels on apparent digestibility of trace minerals in rainbow trout after feeding for 24 weeks

Dietary supplements		Apparent digestibility (%)	
Zn (mg kg^{-1})	$Ca_3(PO_4)_2$ (%)	Zn	Mn
20	0	100	81.4
40	0	88.9	75.2
20	4	44.4	54.8
40	4	36.6	60.6
20	7	0.0	50.1
40	7	12.0	29.2

Source: Satoh et al., 1987a.

Table 9
Effect of supplementary tricalcium phosphate at varying dietary zinc levels on growth and feed efficiency of rainbow trout after feeding for 12 weeks

Dietary supplements		Growth rate (%)	Feed efficiency
Zn (mg kg^{-1})	$Ca_3(PO_4)_2$ (%)		
40	0	404	0.83
20	7	206	0.51
40	7	294	0.71
60	7	335	0.70
80	7	407	0.79

Source: Satoh et al., 1987a.

dissolved in the intestine of carp as it has less gastric juice compared to rainbow trout (Satoh et al., 1992a).

Further experiments were performed on rainbow trout to clarify which portion of tricalcium phosphate, calcium or phosphorus, inhibits zinc availability (Porn-Ngam et al., 1993; Satoh et al., 1993). Mono-, di- and tri-basic phosphates of calcium, sodium and potassium incorporated in casein based diets were tested. The dicalcium phosphate containing diet performed better than the others because it had almost equal proportions of calcium and phosphorus. Excess dietary phosphorus unbalanced the calcium:phosphorus ratio, not only depressing growth, but also affecting the availability of zinc and manganese. There was a drop in the content of these minerals in the body and vertebrae. Satoh et al. (1993) suggested that phosphorus in tricalcium phosphate from white fish meal might primarily inhibit zinc availability, and that the existence of calcium in an appropriate proportion to phosphorus in the diet might improve the absorption and retention of zinc. Porn-Ngam et al. (1993) showed that a 1:1 ratio between calcium and phosphorus was best (Fig. 2). It was concluded that the available phosphorus content should be kept at less than 1.5% in rainbow trout diet to avoid reduction of zinc bioavailability. The diet formulation should maintain a calcium:phosphorus ratio close to unity when 1.8–2.4% phosphorus is derived from dietary ingredients such as fish meals.

Phytic acid present in plant protein sources like soybean meal or cotton seed meal strongly chelates divalent minerals, including zinc, to form insoluble phytates in the intestinal lumen, thereby lowering zinc availability. Therefore, commercial diets containing elevated amounts of fish meal and phytic acid require higher zinc contents. Richardson et al. (1985) fed semipurified diets containing various levels of calcium, phosphorus, zinc and sodium phytate to chinook salmon and noted that a high dietary phytic acid content (25.8 g kg^{-1}) depressed fish growth and feed performance. Increasing calcium and/or phosphorus also reduced zinc bioavailability. Satoh et al.

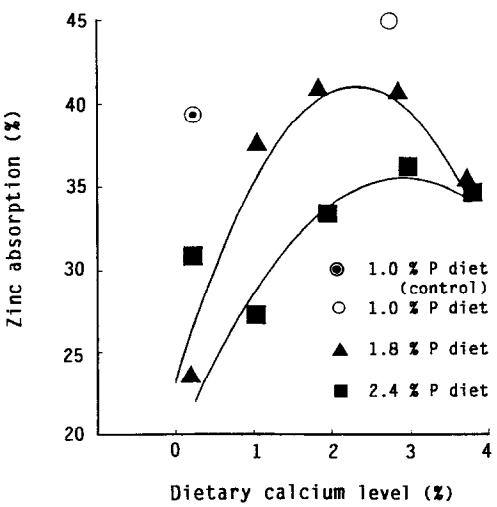

Fig. 2. Effect of phosphorus:calcium ratio in diet on absorption of zinc by rainbow trout.

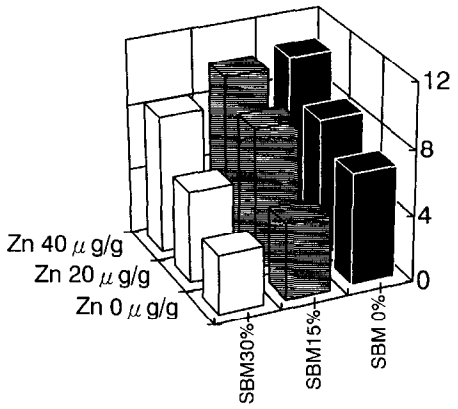

Fig. 3. Combined effect of dietary soybean meal (SBM) and zinc level on the whole-body zinc content of rainbow trout.

(1989b) observed that the elevation of phytic acid level from 1.1 to 2.2% in channel catfish diets containing 50 mg Zn kg^{-1} lowered weight gain and feed efficiency, apart from decreasing the zinc content in the vertebrae. Gatlin and Wilson (1984a) suggested that channel catfish required about 200 mg kg^{-1} when the diet contained about 1.1% phytic acid.

The influence of soybean meal on availability of dietary zinc to rainbow trout was examined in a series of experiments. With increases in dietary soybean meal the gain in fish body weight decreased. When the diet contained 30% soybean meal, body weight was comparatively low, even when zinc was supplemented at 40 mg kg^{-1} (Satoh et al., 1992b). The effect of dietary zinc deprivation was more pronounced at the higher dietary inclusion of soybean meal (among levels 0, 15 and 30%) as indicated by the comparatively low content of zinc in the whole body (Fig. 3). An interaction between zinc and phytic acid in soybean meal may be the reason for the low bioavailability of zinc. Later it was found that heat treatment of soybean meal by extrusion at 150°C improved growth performance of the diet. The original content of 1.3% phytic acid in the soybean meal was reduced to less than 1%, thereby enhancing zinc availability to fish.

Fish meal contains about 80–100 mg Zn kg^{-1}, whereas the range in vegetable protein concentrates may be 40–80 mg Zn kg^{-1}. Cereal grains contain 15–30 mg Zn kg^{-1}. Differences exist in the zinc availability from feedstuffs of plant and animal origin.

6. Cobalt

Cobalt is a component of cyanocobalamin (vitamin B_{12}), constituting nearly 4.5% of its molecular weight. Most animals need the element for the synthesis of the vitamin 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_{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 Šabalina, 1964; cited in 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 Mukhopadhay, 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 labelled 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^{-1}) were toxic to rainbow trout, resulting in haemorrhages in the digestive tract and alterations in white blood cells (Šabalina, 1968; cited in Steffens, 1989). Cobalt deprivation reduced the intestinal synthesis of vitamin B_{12} in catfish (Limsuwan and Lovell, 1981).

6.1. Availability

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).

7. Selenium

Selenium is essential for animals, including fish. It has been found to be an integral component of glutathione peroxidase. The activity level of this enzyme in liver or

plasma is indicative of selenium supply to the organism. Glutathione peroxide protects cells and membranes from oxidative damage by destroying hydrogen peroxide and hydroperoxides employing reducing equivalents from glutathione. Selenium in conjunction with vitamin E is essential for avoidance of nutritional muscular dystrophy. Selenium compounds also are capable of protecting from the toxicity of heavy metals such as cadmium and mercury.

Selenium deficiency generally results in growth depression. Mortality noted in salmon fry fed a selenium-deficient diet was prevented by administration of a diet containing 0.1 mg Se kg^{-1} and 500 IU vitamin E kg^{-1} (Poston et al., 1976). When Atlantic salmon were fed a selenium-deficient diet for 26 weeks, the deficiency signs recorded were lethargy, loss of appetite, reduced muscle tone and mortality. The best growth was achieved at a selenium level of 0.15 mg kg^{-1} (Poston and Combs, 1979). Selenium in combination with vitamin E was required to prevent muscular dystrophy in Atlantic salmon (Poston et al., 1976) and exudative diathesis in rainbow trout (Bell et al., 1985). Deficiency of selenium and vitamin E are the probable factors responsible for Hitra disease in farmed salmon (Fjoelstad and Heyeraas, 1985; Poppe et al., 1986). Apart from muscular dystrophy and myocardial degeneration, the disease manifests itself by anaemia, haemorrhages, and fluid in the peritoneal cavity and in the pericardium. In-depth studies on Atlantic salmon by Bell et al. (1987) have highlighted selenium as the principal factor in the protective mechanism against oxidative cellular injury. Apart from the deficiency signs mentioned earlier, they observed a reduction in tissue concentrations of vitamin E and selenium, low haematocrit values and increased haemolytic rates. The pancreatic tissue had swollen endoplasmic reticulum and areas of vacuolation. In the liver the glutathione peroxidase activity was notably lowered, while glutathione transferase activity and pyruvate kinase activity in plasma were increased.

Hilton et al. (1980) recorded maximum glutathione peroxidase activity in plasma of rainbow trout when the diet contained selenium in the range 0.15–0.38 mg kg^{-1}. Bell et al. (1985) reported that a dietary combination of selenium at 0.9 mg kg^{-1} and tocopherol at 41 mg kg^{-1} was sufficient to prevent deficiency signs in rainbow trout. They also recorded that the glutathione peroxidase activity in liver was reduced when selenium level was low in the diet (0.06 mg Se kg^{-1}) and that the effect was more pronounced when less vitamin E was provided. In channel catfish, Gatlin and Wilson (1984c) estimated that the requirement was 0.25 mg Se kg^{-1} diet at an adequate supply (30 mg kg^{-1}) of vitamin E, on the basis of growth and glutathione peroxidase activity. The development of deficiency signs in channel catfish involved interaction between vitamin E and selenium, but not when either of them alone was deficient (Gatlin et al., 1986). The exception to this was the activity of glutathione peroxidase and glutathione transferase.

High levels of selenium in the diet have toxic effects, resulting in reduced growth, feed efficiency and increased mortality. Levels above 13–15 mg Se kg^{-1} dry feed can be toxic (Hilton et al., 1980; Gatlin and Wilson, 1984c). Prolonged intake of 3 mg Se kg^{-1} diet also was detrimental (Hilton et al., 1980). Trout receiving over 10 mg Se kg^{-1} developed renal calcinosis (Hilton and Hodson, 1983). Hicks et al. (1984) also identified nephrocalcinosis in selenium-deficient rainbow trout. The renal tubules were degenerated, leading to inflammation.

7.1. Availability

Fish derive selenium from both water and diet. High levels of selenium (40–130 μg l^{-1}) in water are toxic to fish. Usually the concentration in water is less than 0.1 μg l^{-1}; hence the uptake through gills is very effective and the mineral is stored in various tissues, except liver, in its inorganic form. Selenium as selenite is taken up efficiently through the gills. For dietary selenium, which is stored in the organic form, the margin between the nutritive requirement at levels normally present in feed ingredients and the toxic threshold in the diet is narrow (Hodson and Hilton, 1983).

The synergism between vitamin E and selenium has already been mentioned. Selenium exerts its effect through the enzyme glutathione peroxidase, while vitamin E is a membrane-associated antioxidant and/or scavenger of free radicals. The two nutrients complement each other in their activity and protect biological membranes against lipid oxidation (Combs and Combs, 1986). The enhanced effectiveness of tocopherol is by inhibiting the formation of peroxides, whereas selenium has to degrade those peroxides already present (Oberbach and Hartfiel, 1988). It is also possible that dietary and environmental variables affect the vitamin E–selenium interaction in fish (Hilton, 1989). The minimum selenium requirement largely depends on the dietary level of vitamin E and is in the range 0.2–0.5 mg kg^{-1} diet.

In addition to functions mentioned earlier, selenium has been found to interact with several elements such as arsenic, sulphur, mercury, cadmium, etc. Hilton and Hodson (1983) demonstrated a strong positive correlation between liver selenium and copper levels in rainbow trout. This relationship also has been pointed out in farmed and wild salmon (Julshamm et al., 1985; Poppe et al., 1986). Hilton (1989) also indicated a definite metabolic interaction between dietary selenium and waterborne copper. Although the mechanism is unclear, both minerals are capable of modifying the toxicity of the other. The selenium and copper interaction reduces metabolically active or available selenium and copper in fish. The positive correlation of liver selenium and copper also suggests that these minerals bind together to form a selenium–copper complex by way of a selenoprotein complex (Hilton, 1989).

Selenium is available from various feed components and other selenium-containing compounds. Selenite and selenate are inorganic selenium compounds, whereas selenomethionine, selenium-methylselenomethionine, selenocystine and selenocysteine are organic complexes. Fish meal and marine by-products employed as feed ingredients provide adequate selenium to fish. Plant materials vary widely in their selenium content. Generally, the availability of selenium from fish meals has been considerably lower than from plant derived products. Bell and Cowey (1989) compared the digestibility and bioavailability of dietary selenium from fish meal, selenite, selenomethionine and selenocystine in Atlantic salmon. Selenomethionine was the most digestible (92%), and fish meal (47%) the least digestible, source of selenium. The glutathione peroxidase:selenium ratio indicated that selenium supplied as selenite or selenocystine was a better source for plasma glutathione peroxidase than was selenium from selenomethionine or fish meal. Even though the comparative availability of selenium is poor, fish meal based diets generally provide sufficient selenium to satisfy the nutritional requirements of fish.

8. Iodine

Iodine is related to thyroid hormones which regulate the level of metabolic activity in fish. The hormones have wide influence on cellular oxidation, neuromuscular control, circulatory dynamics, nutrient metabolism and growth. Triiodothyronine (T_3) is the major hormone of the thyroid gland and is supposed to be the active precursor for thyroxine (T_4). Fish differ from mammals in iodine utilization and in the extrathyroidal metabolism of T_3 and T_4 (Higgs et al., 1982). As T_3 binds more strongly to plasma proteins than T_4, its turnover rate in plasma of trout is low compared to T_4. Both T_3 and T_4 are excreted mainly in bile, but the gills and kidney may also be involved.

Thyroid hyperplasia (goitre) induced by iodine deficiency in salmonid fish was identified nearly a century ago (NRC, 1983). A survey indicated that carnivorous fish were more prone to goitre than were herbivorous or onmnivorous fish. Ikeda et al. (1973b) noted that growth was reduced in goldfish reared in aquaria containing water with a low concentration of iodine. Woodall and La Roche (1964) noted that chinook salmon fed a diet containing only 0.1 mg I kg^{-1} did not have growth depression and goitres, but had reduced iodine stores in the thyroid area. Agrawal and Mahajan (1981) found that iodine uptake by thyroid tissue was reduced in ascorbic acid deficient catfish.

8.1. Availability

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). As seawater contains more iodine than freshwater, iodine deficiency signs appear more in freshwater fish. The plasma iodine level of freshwater fish ranges from 0.5 to over 2000 $\mu g\ dl^{-1}$ (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). Although the iodine requirement of most fish has not been established, Lovell (1979) has recommended a minimum dietary level of 2.8 mg I kg^{-1}. Age, physiological state and stress factors considerably influence the requirement for this mineral.

Marine plants and animals are rich sources of iodine. Some seaweeds 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.

9. Chromium

Chromium is essential for normal carbohydrate and lipid metabolism. The role of chromium in glucose metabolism has been reported for poults and mammals. Chromium is considered to be a cofactor for insulin activity and part of an organic tolerance factor

(Anderson, 1981). Chromium chloride enhances glucose tolerance, increases lipogenesis rate and affects glycogen accumulation in the presence of insulin (Glinsmann and Mertz, 1966; Rosebrough and Steele, 1981; Steele and Rosebrough, 1981).

The influence of dietary chromium on glucose metabolism of carp was investigated by Hertz et al. (1989). Chromium salts improved glucose utilization and inhibited gluconeogenesis, probably by modulating the endogenous insulin activity. The effect of chromium on insulin action may be either by insulin stabilization and degradation or by increasing its affinity for its membrane receptors (Hertz et al., 1989). Supplemental dietary chromium increased the weight gain, energy deposition and liver glycogen content in tilapia fed glucose diets (Shiau and Lin, 1993).

The biological availability of this element depends on its characteristic ability to form coordination compounds and chelates. It occurs in food as part of a biologically active molecule and as inorganic trivalent chromium.

10. Other elements

Very little information related to fish is available for other trace elements such as molybdenum, fluorine, vanadium, etc. Deficiency signs and the requirements of these elements remain to be established.

11. Conclusions

The trace minerals are essential chemical elements involved in the normal metabolism of fish. The information currently available is very patchy, with the exception of zinc and manganese. The detailed mineral budgets are yet to be worked out and more research has to be done on the uptake, function and biological availability of many minerals. The hurdle lies in the fact that these minerals are required only in trace amounts and under experimental conditions it is difficult to maintain such minimal amounts in formulated diets, apart from keeping the medium devoid of the test element. Definitive data on requirements should involve systematic tests on the metabolic functions of the mineral. Refinement of the analytical procedures for ultra trace elements is warranted, particularly because of the wide variation in their tissue levels. In a broader sense, investigations also should consider the potential causes of conditioned trace element deficiencies such as food-processing methods, dietary interactions, disease conditions and genetic disorders. These approaches should guide future research on trace mineral nutrition in fish.

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