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Nutritional Myopathy Syndrome in Cultured Fishes

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Nutritional Myopathy Syndrome is characterized by degeneration and necrosis in striated muscle fibers in the lateral musculature, and deposition of ceroid pigment in macrophages, hepatic cells, adipose cells and the other tissues. This disease sometimes occurs in cultured fishes as the yellowtail (*Seriola quinqueradiata*), red sea bream (*Pagrus major*), tiger puffer (*Takifugu rubripes*), Japanese flounder(*Paralichthys olivaceus*), rainbow trout (*Oncorhynchus mykiss*), carp (*Cyprinus carpio*) and the other fishes, which fed diets containing rancid fat and lipids. I studied natural incidents of this disease in the yellowtail, red sea bream, tiger puffer, japanese flounder and rainbow trout, and performed feeding experiments to prove this disease being caused by oxidized fatty acids and vitamin E deficiency in carp and channel catfish.

1. Red sea bream

In red sea bream, a name of "Yellow fat disease" is common. This disease occurs in fish fed rancid fish mince. Diseased fish display the brown-colored adipose tissues around visceral organs. In such lesions, adipose cells are destroyed, fused, lipo-protein deposited in, and infiltrated by many macrophages (Fig. 1). Infiltrated macrophages engorged lipids resulting in the deposition of ceroid pigments the cytoplasms (Fig. 2). Such ceroid-laden macrophages markedly appear in the liver in which hepatic cells show atrophy and ceroid deposition, hematopoietic tissue, heart and digestive tracts. In the spleen ceroid and hemosiderin-laden macrophages appear (Fig. 3,4). The lateral musculature shows myopathy as atrophy, hyalinization and necrosis of muscle fibers. The chemical analysis in the brown-colored adipose tissues of diseased fish resulted in high TBA values (1270-1760 micro-mol/g), high peroxide values (111-136) and low levels of vitamin E (1.3-7.3mg/100g) (Table. 1). On the other hands, the analysis in the normal vitamin E showed lower TBA values (605-1320 micro-mol/g) and low peroxide values (0-2.2) (Table. 1). These data indicate auto-oxidation occurring in neutral fat of adipose cells under the vitamin E deficient condition, resultting in adipocyte destruction and deposition of lipo-protein. The auto-oxidation would also occur in lipids of hepatic cells, striated muscle fibers and erythrocytes, resulted in hepatocytic ceroidosis, myopathy and hemolysis.

2. Yellowtail

Nutritional myopathy syndrome occurred in fish fed rancid fish mince. Diseased fish are markedly thin across the back and show the jellied lateral musculature and the atrophic liver. The lateral musculature displays severe myopathy in the red and white musculature. Infiltrations of ceroid-laden macrophage extensively occur around the blood vessels in the myopathic musculature, the liver in which hepatic cells are severely atrophic and the hematopoietic tissue. In the spleen, ceroid and hemosiderin-laden macrophages appear (Fig. 5).

Table 1. Chemical analysis in the visceral adipose tissue of red sea bream.

Fish	Yellow Fat Diseased			Normal*1		
	1	2	3	1	2	3
Vitamin E	7.3	1.3	4.3	18.5	6.5	8.5
α-Tocopherol	7.3	1.3	4.3	18.5	6.5	8.5
TBA value	1570	1760	1270	605	1320	884
Peroxide value	123	111	136	0	2.2	0

*1 : Fed on fish mince with vith vitamin mixture
Vitamin E, α-tocopherol : mg/100g
TBA value : μmol/g

3. Tiger puffer

The fry and young fish that fed rancid fish mince and moist pellets containing rancid fish or rancid brown meal usually undergo nutritional myopathy syndrome showing the severely atrophic body in the fry and the thin back in young fish. Myopathy occurs more severely in red muscle fibers than white muscle fibers accompanying melanophore proliferation and infiltrations of ceroid-laden macrophages in the connective tissue of the musclature. Lipo-protein and ceroid deposit in hepatic cells, and the intestinal mucouse and submucosa. Feeding studies revealed that the supplement of vitamin mixture containing much vitamin E (250-500 mg/100g) was effective on prevention of myopathy and ceroidosis.

4. Japanese flounder

The japanese flounder also undergo nutritional myopathy syndrome when they fed rancid fish mince. The diseased fish are thin. Myopathic changes occure in the lateral white musulature. Hepatic cells are usually atrophic. Renal tubules and glomeruli are necrotic showing formation of cellular casts and urinary calculi and being replaced by granulation tissue. Ceroidosis is usually slight.

5. Rainbow trout

Ceroidosis with myopathy occurred in fish commercial pellet including rancid fish meal. Diseased fish show the dark body coloration, thinner across the back and brown-colored liver. The hepatic cells display heavy deposition of ceroid accompaying extensive infiltration of ceroid-laden macrophages. Ceroid deposition occurs in the cardiac muscle fibers. Ceroid-laden macrophages infiltrate in the intestine, gills and hematopoietic tissue (Fig. 6). Ceroid and hemosiderin-laden macrophages appear in the spleen. Feeding studies revealed supplement of vitamin mixture being effective on treatment of the diseased fish. And, feeding tests with brown meal of either a poor or a high quality in amago salmon and carp resulted in the occurrence of ceroid deposition in the hepatic cells of fish fed the meal of a poor quality as well as in the experimental carp.

6. Carp fed rancid pupae of silkworm

Carps are usually reared with diets containing pupae of silkworm and diseased fish with thin across the back sometimes occur. I performed a feeding study with rancid pupae in carps. After 6 month feeding, diseased fish showing thin across the back appeared.

The diseased fish display extensive myopathic changes in the lateral white musculature (Fig. 7). Ceroid and hemosiderin deposit in hepatic cells. Ceroid and hemosiderin-laden macrophages infiltrate in the spleen. The kidney is replaced by ceroid-laden macrophages accompanying the degenerating glomeruli and renal tubules. B-cells in the Langerhans' s islets look normal.

7. Carps fed rancid oils of pupae, anchovy and sandlance

Because carps were revealed to undergo myopathy and cerodosis as well as the yellowtail, tiger puffer, japanese flounder and rainbow trout, I performed feeding studies in carps to prove rancid oils causing ceroidosis and myopathy. The diets were made of white fish meal(crude protein : 45.6%), 10% oil(peroxide value : 19.1 in pupa oil, 78.3 in anchovy oil or 28.0 in sandlance oil), vitamin E free vitamin mixtures and mineral mixture. The control diet was supplemented with vitamin E (25 mg/100g).

After 6 month feeding on pupa oil and 5 month feeding on anchovy oil, all experimental fish showed thin across the back, extensive myopathy in the lateral musculature, ceroidosis in the visceral organs. After 3 month feeding on sandlance oil, 50% fish underwent myopathy and ceroidosis. On the other hand, no sign of myopathy occurred in the control fish fed vitamin E supplemented diets. These results indicate that myopathy and ceroidosis are caused by feeding on oxidized oils. Oxidized oils, contained in fish mince of anchovy and sandlance, would cause the incidents of yellowtail, tiger puffer and japanese flounder as well as in the brown meal in the case of rainbow trout and in pupae in the case of carp.

8. Carp fed oxidized methyl linolic acid

This experiment was performed in carp to reveal that oxidized unsaturated fatty acid causes myopathy and ceroidosis in the vitamin E free condition. Carps fed the casein diets containing oxidized methyl linolic acid (peroxide values : 675-1140) and vitamin E free vitamin mixture displayed severe myopathy and ceroidosis after 5 months. On the other hand, the control fish fed on the diets supplemented with witamin E (50mg/100g) showed no sign of myopathy.

9. Channel catfish fed the vitamin E free-diet

This experiment was performed in channel catfish to prove the vitamin E deficiency causing myopathy. Channel catfish fed on the vitamin E free-casein diet including only saturated fatty acids displayed extensive myopathy in the lateral musculature and very slight ceroidosis. On the other hand, the control fish fed a dist supplemented with vitamin E (7.5mg/100g) displayed no sign of myopathy. These results indicated that vitamin E deficiency would induce auto-oxidation in lipids of the striated muscle fibers and free-radical from the hydro-peroxide would damage muscle fibers resulting in myopathy.

Discussion

Nutritional myopathy syndrome in the yellowtail, tiger puffer, japanese flounder, rainbow trout and carp, and yellow fat disease in the red sea bream are characterized by myopathic changes in the lateral musculature and ceroidosis in various tissues and organs. The feeding studies indicate that the feeding rancid oils would cause vitamin E deficiency, and lipids, and then, free-radical from the hydro-peroxide would damage cells, resulting in either myopathy or yellow fat disease. The freeradical might damage erythrocytes resulting in hemolysis and hemosiderin deposition in phagocytic macrophages. The absorbed oxidized fat and auto-oxidized fat would be phagocytized and treated to ceroid in macrophages. The auto-oxidized fat also would be treated to ceroid in hepatic cells resul-

ting in hepatocytic ceroidosis. These myopathy and ceroidosis could be effectively protected by the supplement with vitamin E.

Legend

Ashley L. M. (1972) : in Fish Nutrition. (edi. by Halver J. E.).

Lovell R. T., Miyazaki T., Rasegnator S. (1984) : Requirement for *a*-tocopherol by channel catfish fed diets low in polyunsaturated tryglycerides. J. Nutri., 114, 894-901.

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Watanabe T., Takashima F, Ogino C., Hibiya T. (1970) : Requirement of young carp for α-tocopherol. B. Japan. Soc. Sci. Fish., 36, 972-976.

Explanation of Figures

Fig. 1 The early lesion of visceral adipose tissue of yellow fat diseased fish of red sea bream. The adipose cells are destroyed, fused and followed by deposition of lipoprotein that is stained black with Sudan black B stain. Sudan black B Stain, X160.

Fig. 2 The developed lesion of visceral adipose tissue of yellow fat-diseased fish of red sea bream. The cellular fusion and lipoprotein deposition become to extensive and are infiltrated by many macrophages. Macrophages engorge with lipoprotein resulting in ceroid deposition in. Sudan black B stain, X80.

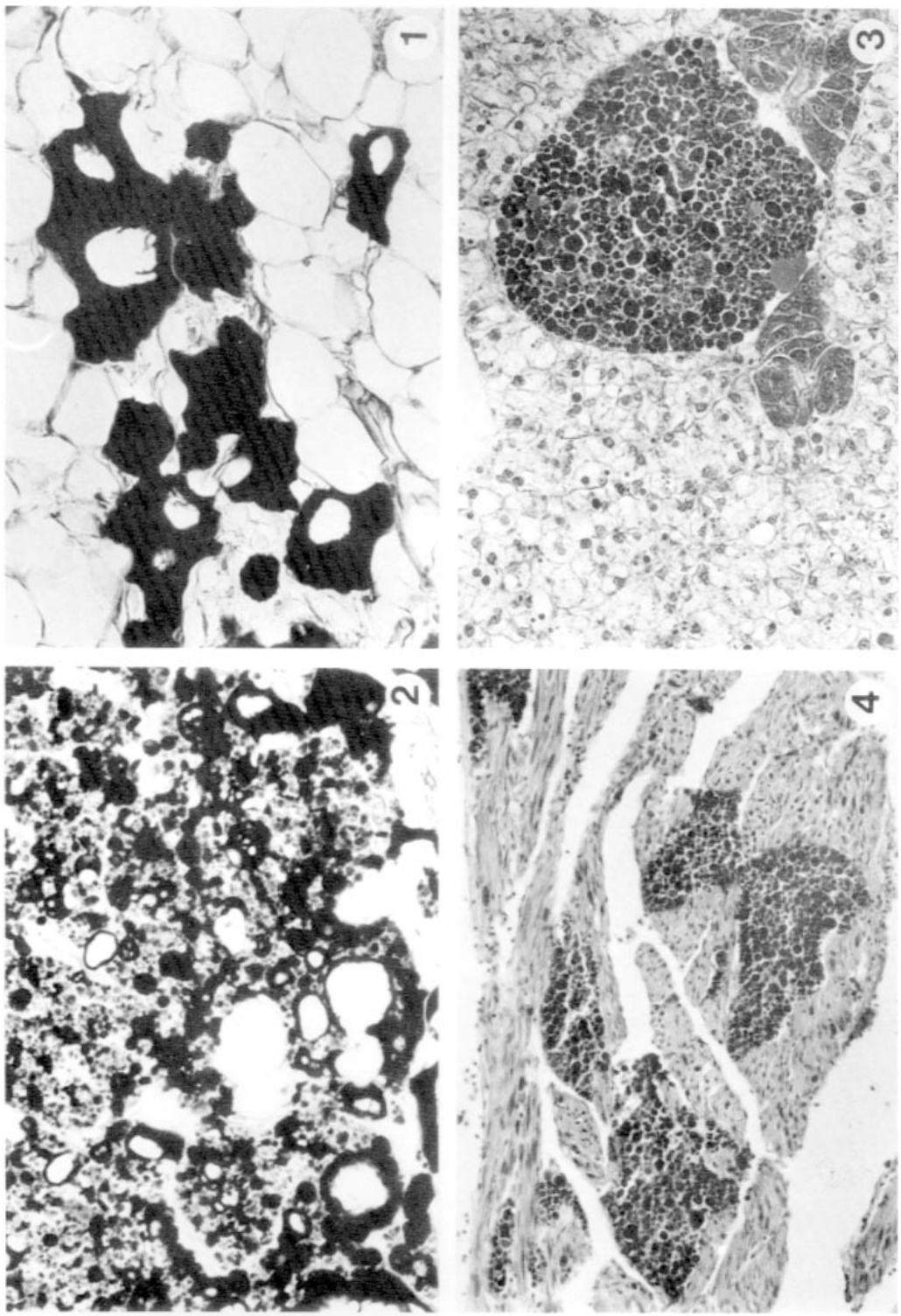
Fig. 3 The liver of diseased, red sea bream showing infiltration of ceroid-laden macrophages and ceroid deposition in the hepatic cells. PAS reaction, X160.

Fig. 4 The ventricle of heart of diseased, red sea bream. Many ecroid-laden macrophages infiltrate into cardiac muscle PAS reaction, X200.

Fig. 5 A cross section of the lateral white musculature of yellowtail with nutritional myopathy syndrome. Muscle fibers are extensively atrophic and necrotic. Azan stain, X50.

Fig. 6 Marked ceroidosis in hepatic cells of rainbow trout. Ceroid is stained and revealed with Sudan black B stain. Sudan black B stain, X320.

Fig. 7 Extensive myopathy in the lateral white musculature of carp fed rancid pupae. Muscle fibers display atrophy, splitting, hyalinization and necrosis. Azan stain, X160.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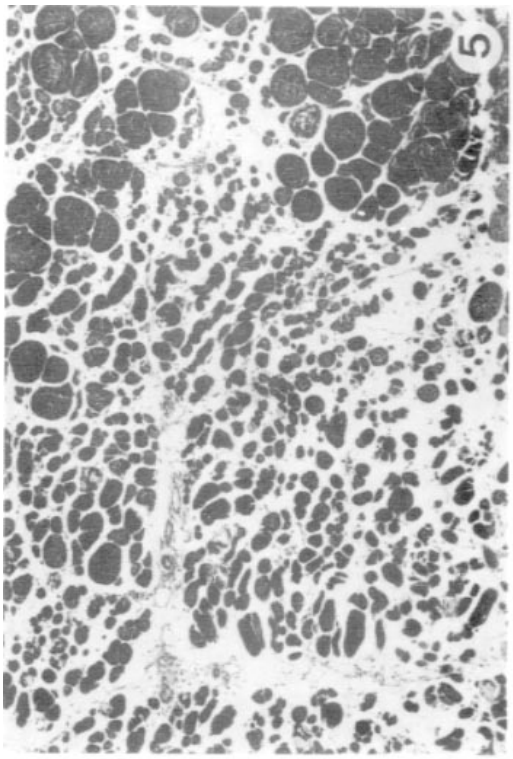
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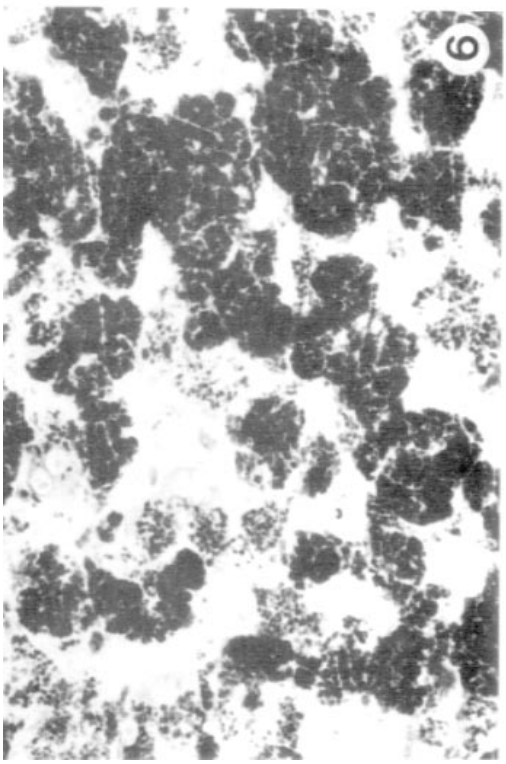
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