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# Effects of Dietary Selenium Level on Growth Performance, Antioxidant Capacity, and Lipid Metabolism Gene Expression in Juvenile Yellow Catfish (Pelteobagrus fulvidraco) Postprint

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## Abstract

This experiment aimed to investigate the effects of dietary selenium content on growth performance, antioxidant capacity, and lipid metabolism gene expression in juvenile yellow catfish (Pelteobagrus fulvidraco). A total of 450 juvenile yellow catfish with initial body weight of (2.12$±$0.01) g were selected and randomly divided into 6 groups with 3 replicates per group and 25 fish per replicate. They were fed six isonitrogenous and isolipidic experimental diets with selenium contents of <0.05 (control group), 0.15, 0.23, 0.35, 0.47, and 0.96 mg/kg for 8 weeks. The results showed that the weight gain rate of yellow catfish exhibited a trend of increasing first and then decreasing with increasing dietary selenium content, reaching the maximum at a dietary selenium content of 0.23 mg/kg, which was significantly higher than that of the control group (P<0.05). The feed conversion ratio showed an opposite trend to the weight gain rate, reaching the minimum at a dietary selenium content of 0.23 mg/kg, which was significantly lower than that of the control group (P<0.05). The survival rate was 100% in all groups. The whole-body crude protein content was highest in the group with dietary selenium content of 0.35 mg/kg, significantly higher than that in the control group and the group with dietary selenium content of 0.23 mg/kg (P<0.05). The whole-body crude lipid content in the control group was significantly lower than that in the groups with dietary selenium contents of 0.15, 0.47, and 0.96 mg/kg (P<0.05), while the whole-body moisture content in the control group was significantly higher than that in the groups with dietary selenium contents of 0.15, 0.35, 0.47, and 0.96 mg/kg (P<0.05). The plasma cholesterol content was lowest in the group with dietary selenium content of 0.23 mg/kg, significantly lower than that in the control group (P<0.05). The whole-body selenium content and hepatic glutathione peroxidase (GPx) activity increased

with increasing dietary selenium content, showing a positive dose-response relationship with dietary selenium content. Hepatic superoxide dismutase (SOD) activity exhibited an initial increase followed by a decrease as dietary selenium content increased from <0.05 mg/kg to 0.47 mg/kg, and then increased again at a dietary selenium content of 0.96 mg/kg. The relative mRNA expression levels of hepatic lipoprotein lipase (LPL) and fatty acid synthase (FAS) in the control group were significantly lower than those in the group with dietary selenium content of 0.23 mg/kg (P<0.05). Using weight gain rate as the evaluation criterion, the optimal dietary selenium content for juvenile yellow catfish was calculated to be 0.20 mg/kg through nonlinear regression analysis.

## Full Text

## Abstract

This experiment was conducted to investigate the effects of dietary selenium content on growth performance, antioxidant capacity, and lipid metabolism gene expression in juvenile yellow catfish (*Pelteobagrus fulvidraco*). A total of 450 juvenile yellow catfish with an initial body weight of (2.12$±$0.01) g were randomly divided into 6 groups with 3 replicates per group and 25 fish per replicate. The fish were fed six isonitrogenous and isolipid experimental diets containing <0.05 (control), 0.15, 0.23, 0.35, 0.47, and 0.96 mg/kg selenium for 8 weeks. The results showed that the weight gain rate (WGR) of yellow catfish increased initially and then decreased with increasing dietary selenium content, reaching its maximum at 0.23 mg/kg, which was significantly higher than the control group (P<0.05). The feed conversion ratio (FCR) showed the opposite trend, reaching its minimum at 0.23 mg/kg, which was significantly lower than the control group (P<0.05). Survival rate was 100% in all groups. Whole-body crude protein content was highest in the 0.35 mg/kg group, significantly higher than the control and 0.23 mg/kg groups (P<0.05). Whole-body crude lipid content in the control group was significantly lower than in the 0.15, 0.47, and 0.96 mg/kg groups (P<0.05), while whole-body moisture content in the control group was significantly higher than in the 0.15, 0.35, 0.47, and 0.96 mg/kg groups (P<0.05). Plasma cholesterol content was lowest in the 0.23 mg/kg group, significantly lower than the control group (P<0.05). Whole-body selenium content and liver glutathione peroxidase (GPx) activity increased with dietary selenium content, showing a positive dose-response relationship. Liver superoxide dismutase (SOD) activity increased initially, then decreased as dietary selenium content rose from <0.05 mg/kg to 0.47 mg/kg, and increased again at 0.96 mg/kg. The relative expression levels of lipoprotein lipase (LPL) and fatty acid synthase (FAS) mRNA in the control group were significantly lower than in the 0.23 mg/kg group (P<0.05). Using WGR as the evaluation index, nonlinear regression analysis estimated the optimal dietary selenium content for juvenile yellow catfish to be 0.20 mg/kg.

**Keywords:** selenium; juvenile yellow catfish; growth performance; antioxidant

capacity; lipid metabolism gene expression

## Introduction

Selenium is an essential trace element for animals that functions in the form of selenoproteins. Glutathione peroxidase (GPx) and deiodinase (DIO) are two well-characterized classes of selenoproteins. GPx can catalyze the reduction of hydrogen peroxide and organic hydroperoxides such as cholesterol and long-chain fatty acid peroxides, thereby protecting cells from oxidative damage. DIO is a class of membrane proteins that catalyzes the conversion of thyroxine (T4) to triiodothyronine (T3), which is 5–8 times more biologically active than T4 and plays a crucial role in the synthesis and regulation of active thyroid hormones. Selenium is vital for maintaining normal animal growth, regulating lipid metabolism, energy metabolism, neuroendocrine function, and enhancing antioxidant capacity. Currently, the selenium requirements and antioxidant effects have been reported for various aquatic animals including rainbow trout (*Oncorhynchus mykiss*) [1], channel catfish (*Ictalurus punctatus*) [2], large yellow croaker (*Larimichthys croceus*) [3], grouper (*Epinephelus malabaricus*) [4], cobia (*Rachycentron canadum* L.) [5], Japanese seabass (*Lateolabrax japonicus*) [6], common carp (*Cyprinus carpio*) juveniles [7], abalone (*Haliotis discus hannai* Ino) [8], and Chinese mitten crab (*Eriocheir sinensis*) juveniles [9]. However, studies on selenium' s effects on body fat, liver fat, and serum lipid metabolism have only been conducted in African catfish (*Clarias gariepinus*) [10], largemouth bass (*Micropterus salmoide*) [11], white sturgeon (*Acipenser transmontanus*) and green sturgeon (*Acipenser medirostris*) juveniles [12], and common carp [13].

Yellow catfish belongs to the family Bagridae, order Siluriformes, and is widely distributed in major inland water bodies in China, particularly in lakes and reservoirs in the middle and lower reaches of the Yangtze River. It is an important small bottom-dwelling economic fish species with delicious meat and rich nutritional value. In recent years, yellow catfish aquaculture has developed rapidly in China, especially in southern regions, with enormous potential in domestic and international markets. Currently, some scholars have studied the effects of copper, iron, zinc, and manganese on growth and physiological functions of yellow catfish, but research on selenium has not been reported. Therefore, this study investigated the effects of dietary selenium supplementation at different levels on growth performance, antioxidant capacity, and lipid metabolism gene expression in juvenile yellow catfish to provide a theoretical basis for the rational application of selenium in formulated feeds for this species.

## Materials and Methods

### 1.1 Experimental Diets

A basal diet was formulated using casein as the protein source, fish oil and lecithin oil (4:1) as the lipid source, and wheat flour as the carbohydrate source,

containing 39.96% crude protein and 9.93% crude lipid. The composition and nutrient levels are shown in . Sodium selenite (Tianjin Guangfu Fine Chemical Industry Co., Ltd., analytical grade) was used as the selenium source and added to the basal diet at different levels to formulate six experimental diets. Selenium supplementation levels were determined based on reported requirements for rainbow trout (0.38 mg/kg) [1], channel catfish (0.25 mg/kg) [2], and grouper (0.70 mg/kg) [4]. All feed ingredients were provided by Guangzhou Feixite Aquatic Technology Co., Ltd., and were ground to pass through a 60-mesh sieve. Sodium selenite, vitamin premix, and mineral premix were mixed using the progressive enlargement method, followed by addition of fish oil, lecithin oil, and water. The mixture was extruded into 2.5 mm pellets using an SLX-80 twin-screw extruder, dried at 50 °C, cooled naturally, sealed in bags, and stored at -20 °C. The actual selenium concentrations in the six experimental diets, measured by hydride generation-atomic absorption spectrometry, were <0.05 (control), 0.15, 0.23, 0.35, 0.47, and 0.96 mg/kg.

### 1.2 Experimental Fish and Culture Management

Juvenile yellow catfish were purchased from Qingyuan Huangsha Fisheries Base in Guangdong Province and temporarily reared in an indoor recirculating aquaculture system at the Aquaculture Research Laboratory of the Institute of Animal Science, Guangdong Academy of Agricultural Sciences. During the 2-week acclimation period, fish were fed a commercial diet (42.24% crude protein, 7.06% crude lipid, 10.06% ash, 9.42% moisture, 0.46 mg/kg selenium) to satiation twice daily (09:00 and 16:00). The system consisted of 18 cylindrical fiberglass tanks (350 L capacity, 80 cm diameter, 70 cm height) with an actual water volume of 300–320 L. At the start of the experiment, 450 healthy juvenile yellow catfish with uniform size (average weight (2.12$±$0.01) g) were selected and stocked into the 18 tanks at 25 fish per tank. The tanks were randomly assigned to 6 groups (3 replicates per group) and fed the corresponding experimental diets twice daily (09:00 and 16:00) at 4–6% of body weight, adjusted according to feeding and growth. Feed intake and mortality were recorded daily, and water temperature was monitored. Aeration was provided continuously 24 h/day with natural light. Water temperature was maintained at 26.0–32.0 °C, pH at 7.0–7.5, ammonia nitrogen <0.2 mg/L, and no selenium was detected in the water. The feeding trial lasted 8 weeks.

### 1.3 Sample Collection and Analysis

**1.3.1 Sample Collection** At the end of the feeding trial, fish were fasted for 24 h before counting and weighing. Six fish per tank were randomly selected and stored at -20 °C for determination of whole-body composition and selenium content. Another 10 fish per tank were anesthetized with MS-222 (60 mg/L, Suzhou Xinyong Biomedical Technology Co., Ltd.), and blood was collected from the caudal vein into anticoagulant tubes (BD), mixed, and centrifuged at 4,000 r/min for 10 min at 4 °C to prepare plasma samples, which were stored at -

78 °C for plasma biochemical analysis. After blood collection, fish were dissected on ice to obtain liver samples. Livers from three fish were pooled as one sample, snap-frozen in liquid nitrogen, and stored at -78 °C for analysis of LPL and FAS mRNA expression. Livers from the remaining fish were pooled and stored at -78 °C for determination of GPx, SOD activities, and malondialdehyde (MDA) content.

**1.3.2 Sample Analysis** Moisture content in feed and whole-body samples was determined by oven drying at 105 °C, crude protein by the Kjeldahl method, crude lipid by ether extraction, and ash by combustion at 550 °C. Whole-body selenium content was measured by hydride generation-atomic absorption spectrometry. Plasma glucose (GLU), total protein (TP), cholesterol (CHO), and triglyceride (TG) concentrations were determined using a Hitachi 7170A automatic biochemical analyzer. Liver MDA content, GPx activity, and SOD activity were measured using commercial kits from Nanjing Jiancheng Bioengineering Institute according to the manufacturer' s instructions.

For gene expression analysis, liver tissue stored at -78 °C was ground into powder in liquid nitrogen. Lysis buffer was added according to the kit instructions, and total RNA was extracted using the TaKaRa RNA Mini Kit. Real-time quantitative PCR was used to determine the relative expression levels of LPL and FAS mRNA in juvenile yellow catfish liver, with $\beta$-actin as the internal reference gene. Specific primers for LPL (EU882966.1), FAS (JN579124.1), and $\beta$-actin (EU161066.1) were designed using Primer 5.0 software based on sequences from yellow catfish cDNA (Table 2 ). The main instruments used were a quantitative PCR system (Biorad CFX connect) and a spectrophotometer (Thermo Scientific NanoDrop ND2000). The 20  L reaction mixture contained 2  L template cDNA, 4  L primer mix, 4  L ddH2O, and 10  L SYBR Green qPCR Kit (All-in-One™ miRNA qRT-PCR Detection Kit). Each sample was run in triplicate, and a no-template PCR reaction served as the negative control. Cycling parameters were: 95 °C for 12 min; 40 cycles of 95 °C for 10 s, 58 °C for 10 s, and 72 °C for 10 s. Melting curves were analyzed using Option Monitor Software 2.03 version (MJ Research, Cambridge, MA), and expression differences were calculated using the 2-ΔΔCt method.

### 1.4 Index Calculation

Weight gain rate (WGR, %) = 100 × (final body weight + dead body weight - initial body weight) / initial body weight.
Feeding rate (FR, %) = 100 × total feed intake / {[(initial total body weight + final body weight) / 2] × feeding days}.
Feed conversion ratio (FCR) = total feed intake / (final body weight + dead body weight - initial body weight).
Survival rate (SR, %) = 100 × final fish number / initial fish number.

### 1.5 Statistical Analysis

Experimental data (except survival rate) are expressed as mean ± standard error. SPSS 11.5 software was used for statistical analysis. Data were first tested for homogeneity of variance. If variance was homogeneous, one-way ANOVA was used, followed by LSD test for multiple comparisons. If variance was not homogeneous, Dunnett' s T3 test was used. $P<0.05$ was considered statistically significant.

## Results

### 2.1 Effects of Dietary Selenium Content on Growth Performance of Juvenile Yellow Catfish

As shown in , after 8 weeks of feeding, the WGR of juvenile yellow catfish was highest in the 0.23 mg/kg selenium group, significantly higher than the control group ($P<0.05$), while the FCR was lowest in this group, significantly lower than the control group ($P<0.05$). The FR was highest in the control group, significantly higher than the 0.15 and 0.23 mg/kg groups ($P<0.05$). Survival rate was not affected by dietary selenium content ($P>0.05$), with 100% survival in all groups. Nonlinear regression analysis of WGR [14] indicated that the optimal dietary selenium content for juvenile yellow catfish was 0.20 mg/kg ([Figure 1: see original paper]).

### 2.2 Effects of Dietary Selenium Content on Body Composition of Juvenile Yellow Catfish

As shown in , after 8 weeks of feeding, whole-body crude protein content was highest in the 0.35 mg/kg selenium group, significantly higher than the control and 0.23 mg/kg groups ($P<0.05$). Whole-body crude lipid content in the control group was significantly lower than in the 0.15, 0.47, and 0.96 mg/kg groups ($P<0.05$). Conversely, whole-body moisture content in the control group was higher than in other groups, with significant differences compared to the 0.15, 0.35, 0.47, and 0.96 mg/kg groups ($P<0.05$). Whole-body selenium content was highest in the 0.96 mg/kg group, significantly higher than all other groups ($P<0.05$), and lowest in the control group, significantly lower than all groups except the 0.15 mg/kg group ($P<0.05$).

### 2.3 Effects of Dietary Selenium Content on Plasma Biochemical Indices of Juvenile Yellow Catfish

As shown in , dietary selenium content had no significant effect on plasma TP, GLU, or TG concentrations ($P>0.05$) after 8 weeks. However, plasma CHO concentration was lowest in the 0.23 mg/kg selenium group, significantly lower than the control group ($P<0.05$).

### 2.4 Effects of Dietary Selenium Content on Liver Antioxidant Indices of Juvenile Yellow Catfish

As shown in , liver GPx activity increased with dietary selenium content and was significantly higher in the 0.96 mg/kg group than in all other groups ($P<0.05$). Liver SOD activity increased initially, then decreased, and increased again at 0.96 mg/kg, with the highest value in this group, significantly higher than all other groups ($P<0.05$). There were no significant differences in liver MDA content among groups ($P>0.05$).

### 2.5 Effects of Dietary Selenium Content on Liver LPL and FAS mRNA Expression in Juvenile Yellow Catfish

As shown in , after 8 weeks of feeding, the relative expression level of liver LPL mRNA was highest in the 0.23 mg/kg selenium group, significantly different from the control group ($P<0.05$), then decreased with further increases in selenium content, but increased again at 0.96 mg/kg. The relative expression level of liver FAS mRNA showed a similar trend, with the lowest value in the control group, significantly lower than all groups except the 0.47 mg/kg group ($P<0.05$).

## Discussion

### 3.1 Effects of Dietary Selenium Content on Growth Performance of Juvenile Yellow Catfish

In this study, the highest WGR was observed at 0.23 mg/kg dietary selenium, indicating that appropriate selenium supplementation is essential for maintaining normal growth and improving WGR in juvenile yellow catfish, while both deficiency and excess can inhibit growth. Using WGR as the evaluation index, nonlinear regression analysis estimated the optimal dietary selenium content to be 0.20 mg/kg. This result is similar to findings in channel catfish [2] (0.25 mg/kg) but lower than those reported for rainbow trout (0.38 mg/kg) [1], cobia (0.788 mg/kg) [5], grouper (0.7 mg/kg) [4], Japanese seabass (0.40–0.63 mg/kg) [6], common carp juveniles (0.434–0.517 mg/kg) [7], grass carp juveniles (0.438–0.631 mg/kg) [15], abalone juveniles (1.408 mg/kg) [8], Chinese mitten crab juveniles (0.4–0.6 mg/kg) [9], and Chinese shrimp (0.44 mg/kg) [16], but higher than that for large yellow croaker juveniles (0.178 mg/kg) [3]. These differences may be attributed to variations in feeding habits, growth stages, physiological characteristics, culture environments, culture modes, and feed processing techniques among aquatic species. Except for large yellow croaker, carnivorous marine species such as rainbow trout, cobia, grouper, Japanese seabass, and abalone generally have higher selenium requirements. The relatively high requirements in Chinese mitten crab and Chinese shrimp may be related to their molting process, which may increase trace element demands, though this hypothesis requires further investigation. Yellow catfish and channel catfish belong to the same order (Siluriformes) and share close genetic relationships and

similar physiological characteristics and living habits, which may explain their comparable selenium requirements.

### 3.2 Effects of Dietary Selenium Content on Body Composition of Juvenile Yellow Catfish

Whole-body crude protein, crude lipid, ash, and moisture are important parameters for evaluating nutritional status in fish nutrition research [16]. There is a negative correlation between moisture content and crude protein, crude lipid, and energy content, and moisture content can effectively reflect relative changes in crude protein and crude lipid [17]. The results indicate that dietary selenium content affected body composition in juvenile yellow catfish. The control group had the lowest whole-body crude lipid content, significantly lower than the 0.15, 0.47, and 0.96 mg/kg groups, and crude lipid content generally increased with dietary selenium content. Conversely, whole-body moisture content decreased with increasing selenium content, consistent with trends reported by Jonsson et al. [17]. Therefore, selenium deficiency reduced body fat deposition, while high selenium increased it, suggesting that selenium significantly influences lipid metabolism in juvenile yellow catfish. Similarly, studies on largemouth bass found that high dietary selenium increased liver fat content and affected hepatic lipid metabolism [11]. In mammals, selenium increased body fat content in rats, and selenium deficiency decreased 5’ -deiodinase (5’ -DIO) activity, impairing non-shivering thermogenesis in brown adipose tissue [18]. However, in white sturgeon [12] and green sturgeon [19] juveniles, increasing dietary selenium significantly decreased body crude lipid and energy content while increasing moisture content. In African catfish, high dietary selenium also reduced body crude lipid content [10]. De Riu et al. [19] suggested that selenium toxicity in sturgeon caused consumption of fat and protein to meet energy demands. Additionally, species differences and initial growth rates may affect fat deposition. The mechanism underlying reduced body fat content at high selenium levels remains unclear, but it is evident that selenium plays an important role in regulating lipid metabolism, and both deficiency and excess affect fat deposition.

### 3.3 Effects of Dietary Selenium Content on Plasma Biochemical Indices of Juvenile Yellow Catfish

Environmental factors such as temperature, light, stocking density, and salinity, as well as physiological factors including reproductive cycle, age, sex, and nutrition, and social factors like hierarchy can affect blood indices in fish. Blood biochemical indices provide a sensitive method for monitoring stress induced by culture conditions [20]. Plasma concentrations of total protein, glucose, cholesterol, and triglycerides effectively reflect nutritional health status. Abnormalities in protein, carbohydrate, and lipid metabolism typically alter plasma total protein and cholesterol concentrations [21], making cholesterol a useful indicator for detecting lipid metabolism disorders [22]. Studies have shown that selenium

deficiency decreases DIO activity in rats [23], leading to abnormal elevation of plasma low-density lipoprotein cholesterol (LDL-C), consistent with our finding that plasma cholesterol was higher in the control (selenium-deficient) group. Apolipoprotein (Apo)B mediates LDL binding to LDL receptors (LDL-R), facilitating LDL-C clearance from circulation. T3 promotes expression of LDL-R [24] and ApoB [25], but T3 requires DIO-catalyzed deiodination of T4. Selenium deficiency reduces DIO activity [23], ultimately causing elevated plasma LDL-C. Other studies suggest that increased plasma cholesterol due to selenium deficiency may be related to reduced antioxidant capacity and oxidative attack by free radicals, which increase plasma glucose and cholesterol [26].

### 3.4 Effects of Dietary Selenium Content on Liver Antioxidant Indices of Juvenile Yellow Catfish

This study found a dose-response relationship between whole-body selenium content and dietary selenium content in juvenile yellow catfish, with selenium content increasing as dietary selenium increased, consistent with findings in abalone [8] and grouper [4]. Liver and kidney are tissues with high selenium accumulation [27]. Liver GPx and SOD activities and MDA content are important indicators of selenium' s antioxidant capacity [28-29]. Selenium is the active center of GPx, and liver GPx activity effectively reflects selenium status. Studies in rainbow trout [1], channel catfish [2], grouper [4], and abalone [8] found that GPx activity increased significantly with selenium deposition, consistent with our results. GPx, SOD, and catalase (CAT) constitute the first line of cellular defense, and the balance between GPx and SOD activities is crucial for protection against oxidative damage [30]. SOD primarily captures superoxide anions, minimizing oxidative damage in host immune defense [31]. Copper serves as the structural center of SOD and is essential for enzyme stability. The relationship between copper and selenium has been reported, with studies in Atlantic salmon showing a positive dose-response relationship between liver selenium and copper accumulation [32]. However, other studies indicate that high doses of selenium and copper can mutually modulate toxicity by reducing bioactivity [33]. In this study, liver SOD activity increased initially with dietary selenium, decreased when selenium exceeded 0.23 mg/kg, and increased again at 0.96 mg/kg. We hypothesize that appropriate selenium levels exert a positive synergistic effect on SOD activity, while excessive selenium inhibits SOD activity, and very high selenium levels reactivate SOD to resist oxidative stress induced by high selenium, though this requires further verification. MDA is a product of lipid peroxidation and a key indicator of oxidative damage [34]. Studies in rainbow trout found that appropriate sodium selenite or organic selenium supplementation reduced serum and muscle MDA content [35], but this study found no significant differences in liver MDA content among groups, similar to findings in Siberian sturgeon (*Acipenser baeri*) [28] and rainbow trout [29]. Pacini et al. [28] suggested that when selenium is adequate, high GPx activity effectively prevents hepatic lipid peroxidation. However, we speculate that the lack of significant differences in liver MDA content may be related to

the detection method used, as Küçükbay et al. [36] used high-performance liquid chromatography, which is more sensitive than the kit method used in this study.

### 3.5 Effects of Dietary Selenium Content on Liver LPL and FAS mRNA Expression in Juvenile Yellow Catfish

LPL is a key enzyme for fat deposition in animal tissues and the rate-limiting enzyme for triglyceride hydrolysis to glycerol and free fatty acids (FFA), playing an important role in lipid metabolism and transport. LPL controls the distribution of lipid substrates between adipose tissue and other organs, thereby determining the metabolic fate of dietary lipids—whether stored as body fat or consumed as energy substrates—and ultimately determining body fat accumulation [37]. FAS catalyzes the synthesis of palmitic acid (C16:0) from acetyl-CoA and malonyl-CoA, and its abundance and activity are crucial for body fat deposition. To date, few studies have reported on selenium regulation of lipid metabolism-related gene expression, with only Liang Yang [38] investigating this in chicken adipose tissue, finding that selenium deficiency promoted LPL mRNA expression, thereby enhancing fatty acid uptake and adipose tissue formation. In contrast, our study found lower relative expression levels of liver LPL and FAS mRNA in the control (selenium-deficient) group, consistent with the lower whole-body crude lipid content observed in this group. Combined with the elevated plasma cholesterol in selenium-deficient fish, we propose that selenium deficiency may affect the activation status of insulin signaling cascade factors, thereby mimicking, enhancing, or interfering with insulin regulation of carbohydrate and lipid metabolism [38], ultimately affecting LPL and FAS mRNA expression and body fat deposition. Currently, selenium regulation of lipid metabolism is a novel research area with limited data, and the underlying mechanisms require further investigation.

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