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Research Article

Effects of Dietary Taurine on Growth, Body Composition, Blood Parameters, and Enzyme Activities of Juvenile Sterlet (Acipenser ruthenus)

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To evaluate the effects of dietary taurine on *Acipenser ruthenus*, a total of 180 fish with an initial weight of 14.13 ± 0.10 g, were stocked into 15 (300 liters) tanks (12 fish/replicate) for 70 days. Five experimental diets (38.60% crude protein and 18.78% crude lipid) supplemented with 0 (T0), 5 (T5), 15 (T15), 30 (T30), and 50 (T50) g/kg taurine were used. The experimental fish were fed to visual satiation four times daily. At the end of the experimental period, weight gain, thermal growth coefficient, and survival in fish fed T30 diet was higher ($P < 0.05$) than other experimental fish. Food conversion ratio in all treatments containing taurine was significantly lower than T0 treatment ($P < 0.05$). The highest and the lowest total essential amino acids of muscle were found in T50 and T0 treatments, respectively. The highest serum cholesterol and low-density lipoprotein levels were observed in the T30 treatment ($P < 0.05$). An increasing trend was observed in the values of alanine aminotransferase, aspartate aminotransferase, and lactate dehydrogenase activities of serum from T0 to T30 treatments and the highest level was recorded in T30 ($P < 0.05$). The highest lysozyme, trypsin, chymotrypsin, alkaline phosphatase, lipase, and amylase activities were also observed in T30. Catalase activity in T15 was higher than other treatments ($P < 0.05$). Malondialdehyde activity in T0 was significantly higher than other experimental treatments ($P < 0.05$). The highest total numbers of bacteria and lactic acid bacteria of the gut was recorded in T30 and T5, respectively. The overall results indicate that the T30 experimental diet induced the improvement of growth performance, hepatic, digestive, and antioxidant enzyme activities in *A. ruthenus*.

1. Introduction

Fish meal is generally considered to be an excellent source of protein for inclusion in feeds for farmed fish, but increase in demand and rising prices for this commodity have resulted in increased use of protein sources of terrestrial plant origin in aquafeeds [1]. Plant protein sources are often deficient in some essential amino acids, and they contain little or no taurine [1]. Although there is a long history of the reporting of advantageous physiological effects of taurine in mammals [2, 3], the study of the effects of taurine on fish physiology is more recent [1, 4, 5].

Taurine is the final product of the metabolism of sulfur-containing amino acids. Taurine can ionize both its amine and sulfonic groups [4]. The main pathway for the synthesis of taurine varies among species. It depends on the tissue type, pathway, and synthesis quantity [3]. The cystein sulfinate decarboxylase enzyme (CSD) is responsible for the decarboxylation of cysteine sulfonate to hypotaurine. Finally, hypotaurine is converted to taurine by changing the metabolic pathway and combine with oxygen (Ma et al., 2021). Liver and brain tissues in various mammalian species are responsible for the endogenous taurine synthesis during enzymatic oxidation. In fish, taurine is as effective in many physiological activities as mammals like osmoregulation, oxidation, and appetite stimulation [5].

Taurine biosynthesis varies among species, and differences in size, age, feeding habits, and activity of CSD enzyme can lead to variation in taurine requirement [7–9]. Freshwater finfish have more ability to synthesize taurine than marine

finfish. Therefore, when the diet is a plant-based protein source, taurine must be added to the diet as an exogenous taurine source. Freshwater finfish, including common carp (*Cyprinus carpio*), rainbow trout (*Oncorhynchus mykiss*), and Atlantic salmon (*Salmo salar*) can produce endogenous taurine in the *trans*-sulfurization pathway [7, 10–12]. Although in bluegill (*Lepomis macrochirus*), CSD enzyme activity was lower in fish fed high levels of taurine [13]; when the fish fed with diets containing soybean protein concentrate, they showed high plasma taurine levels. The conversion of dietary methionine hydroxy analogue to taurine may have stimulated growth in *O. mykiss* [14]. In other words, dietary taurine supplementation could improve growth performance, digestibility, feed efficiency, digestive enzyme activity, body amino acid profiles, and serum biochemical compositions in freshwater finfish fed a plant-based diet with insufficient levels of dietary cysteine and methionine [15–17].

Nevertheless, in the *chondrostean*, this pathway for taurine synthesis has not been reported clearly. Most sturgeon communities are declining significantly due to overexploration, habitat destruction, and pollution. Sterlet is the only sturgeon species that lives in both Asia and Europe [18]. Sterlet is an important commercial species; however, many issues related to its management and protection remain unresolved. In order to improve the current situation, better understanding of the sterlet nutritional requirements is a significant step in the aquaculture development of this specie. Because the cost of sturgeon production is higher than other farmed fish due to the long culturing period, any possible problems in supplying fish meal will cause losses for the aquaculture industries. On the other hand, pelagic fish harvesting to feed farmed fish is not sustainable for a long time and cannot be relied on. Therefore, considering the current situation, one of the ways to reduce the dependence on the fish meal in aquatic diets is to use cheaper alternatives. This replacement can reduce reliance on the fish meal. A significant issue in this regard is the issue of eliminating possible problems with alternative nutrients and how dietary supplements affect growth and production factors. Therefore, taking into consideration the importance of taurine supplementation and inadequate related data, the present study was designed to investigate the effects of dietary taurine on growth performance, muscle amino acid profile, serum biochemical indices, digestion, hepatic, and antioxidant enzyme activity and gut bacteria of *Acipenser ruthenus*.

2. Materials and Methods

2.1. Experimental Fish and Culture System Design. *A. ruthenus* were obtained from the Hantoshzadeh commercial sturgeon-culture company in Dezful, Khuzestan province, Iran. Prior to start of the experiment, the experimental fish were acclimatized for 10 days. After the acclimatization period, 180 healthy fish (based on appearance), with an average initial weight of 14.13 ± 0.10 g, were stocked into 15 (300 liters, 39.5 cm radius × 61 cm height) polyethylene tanks (12 fish per replicate). To maintain water quality during the experimental period, the flow rate of water was maintained at 4.4 liters per minute. The tanks were equipped with an air stone for aeration. Temperature, dissolved oxygen, and pH were recorded daily in the morning and evening using a multimeter (Extech EC 400, USA). During the experimental period, the water temperature was maintained at 25.1 ± 0.1°C. The dissolved oxygen and pH levels remained within 5.9-6.1 ppm and 7.3-7.8 range, respectively. Water total alkalinity (194 ± 30.1 mg/l, HI8453102, Hanna Instrument Ltd., South Africa), total hardness (205 ± 21.8 mg/l), N-ammonia (0.01 ± 0.002 mg/l, Hanna HI97715, South Africa), and N-nitrite (0.01 ± 0.003 mg/l, Hanna HI96728, South Africa) were measured and recorded weekly. The photoperiod was based on natural environmental fluctuations (from late-September to mid-November, 12 h light : 12 h dark approximately).

2.2. Experimental Diets and Feeding Trials. Five isonitrogenous (38.60% crude protein) and isolipidic (18.78% crude lipid) diets were produced. The nonsupplemented diet (0% taurine) used as a control experimental diet only contained the existing taurine in the fish meal (designated as T0). The other four experimental diets were supplemented with graded taurine levels of 5, 15, 30, and 50g/kg dry weight of the diet (designated as T5, T15, T30, and T50, respectively). The cellulose was used in diets as filler. The taurine contents of the different experimental diets (T0, T5, T15, T30, and T50) were measured and recorded as 0.53 ± 0.1, 5.61 ± 0.1, 14.73 ± 0.7, 31.01 ± 1.1, and 49.38 ± 1.2 g/kg dry weight diet, respectively, which were significantly different ($P < 0.05$). Formulation and composition analysis [19] of experimental diets are shown in Table 1. The fish were fed with these dietary treatments by hand to visual satiation four times daily (at 06:00, 10:00, 14:00, and 18:00 hours) for 70 days. Fish meal, soybean meal, wheat gluten, wheat flour, soybean oil, fish oil, mineral and vitamin supplements (Hashtgerd supplement company), gelatin (as a binder, Beyza feed mill company), and cellulose used for preparation of experimental feeds were purchased from the local market. Taurine (Sigma-Aldrich 808616, CAS 107-35-7, EC number 203-483-8, 2-aminoethanesulfonic acid ≥ 99.0%) was obtained from Merck KGaA, Germany. To produce the experimental diets, feed ingredients were mixed, and then, water was added to the mixture. The resultant dough was pelleted using a meat grinder, and the pellets were dried and crushed into the appropriate size (2.5 mm). The dried diets were packed and stored at -20°C until use.

2.3. Sample Collection and Analytical Procedures

2.3.1. Growth Performances, Feed Utilization, and Somatic Indices. All the fish in each tank were weighed and counted individually at the beginning and end of the feeding period to calculate the survival rate (SR), weight gain (WG), specific growth rate (SGR), thermal growth coefficient (TGC), food conversion ratio (FCR), and protein efficiency ratio (PER). The length of the fish was recorded at the end of the feeding trial for computation of condition factor (CF). Somatic indices were obtained by sacrificing six fish from each treatment (2 fish per replicate) to calculate hepatosomatic index (HSI), viscero somatic index (VSI), and intraperitoneal fat ratio

Table 1: Ingredients, formulation, and analyzed compositions of the experimental diets (g/kg dry weight diet).

Ingredients (g/kg DW diet)	Diets				
	T0	T5	T15	T30	T50
Fish meal†	180	180	180	180	180
Soybean meal†	350	350	350	350	350
Wheat gluten†	170	170	170	170	170
Wheat flour†	100	100	100	100	100
Soybean oil	60	60	60	60	60
Fish oil	40	40	40	40	40
Mineral premix††	20	20	20	20	20
Vitamin premix†††	10	10	10	10	10
Binder (gelatin)	20	20	20	20	20
Taurine	0	5	15	30	50
Cellulose	50	45	35	20	0
Proximate composition ($n = 3$)					
Crude protein	383.1 ± 28	383.3 ± 25	385.0 ± 30	389.1 ± 49	389.7 ± 32
Crude lipid	188.5 ± 11	190.1 ± 25	189.4 ± 18	184.0 ± 17	187.3 ± 21
Ash	118.4 ± 17	129.8 ± 19	125.0 ± 20	122.8 ± 13	118.1 ± 14
Moisture	64.6 ± 10	59.1 ± 11	61.6 ± 15	64.6 ± 18	69.1 ± 16
Taurine content	0.53 ± 0.1	5.61 ± 0.1	14.73 ± 0.7	31.01 ± 1.1	49.38 ± 1.2
Carbohydrate §	245.4	237.7	239.0	239.5	235.8
GE (MJ kg^{-1}) §§	20.75	20.69	20.73	20.62	20.70
Essential amino acid (EAA, mg/g dry weight)					
Arginine (Arg)	22.98	22.85	22.97	23.14	23.51
Histidine (His)	7.83	7.74	7.72	7.92	8.08
Leucine (Leu)	22.84	22.77	23.21	23.07	23.59
Lysine (Lys)	16.73	17.09	16.96	17.30	18.01
Methionine (Met)	3.08	3.51	3.33	3.40	3.39
Phenylalanine (Phe)	13.21	13.27	13.95	13.91	13.36
Threonine (Thr)	13.39	13.06	13.72	14.15	14.05
Isoleucine (Ile)	12.23	12.56	12.88	12.20	12.86
Valine (Val)	14.87	14.47	14.81	15.07	14.67
Total EAA	127.16	127.31	129.55	130.16	131.52
Cysteine (Cys)	0.23	0.32	0.35	0.39	0.41
Total nonessential amino acid (NEAA)	168.10	167.51	170.76	171.03	172.06
Total amino acid	295.76	294.82	300.31	301.19	303.58
TEAA/TNEAA	0.756	0.760	0.759	0.761	0.764

†Proximate composition as g/kg dry-weight basis (fish meal (64.1% crude protein, 3.8% crude lipid), soybean meal (41.6% crude protein, 2.1% crude lipid), wheat gluten (42.8% crude protein, 1.8% crude lipid), wheat flour (11.3% crude protein, 1.1% crude lipid), gelatin (97.0% crude protein)). ††Per kilogram: Mg: 700 mg; Fe: 25 mg; Co: 4 mg; Cu: 5 mg; Zn: 80 mg; NaCl: 10 g; dicalcium phosphate: 26 g. †††Per kilogram: vit. A: 1,000 IU; D3: 5,000 IU; E: 20 mg; B5: 100 mg; B2: 20 mg; B6: 20 mg; B1: 20 mg; H: 1 mg; B9: 6 mg; B12: 1 mg; B4: 600 mg; C: 50 mg. ‡2-Aminoethanesulfonic acid ≥ 99.0%, Sigma-Aldrich 808616, Merck KGaA, Germany. §Carbohydrate content obtained by subtracting the sum of protein, lipid, ash, and moisture content from 100. §§Gross energy, calculated based on 0.017, 0.0398, and 0.0237 MJ g^{-1} for carbohydrate, lipid, and protein, respectively.

(IPF) at the end of the study period. The equations used for the calculation of growth performance, feed utilization, and other parameters were as below:

Weight gain (WG) = Final wet weight (g) − initial wet weight(g),

Specific growth rate (SGR) = [ln final weight (g) − ln initial weight (g)] × 100/experiment duration (day),

Condition factor (CF) = [Fish mass (g) /Fish total length^3 (cm)] × 100,

$$\text{Thermal growth coefficient (TGC)} = \left[\left(\sqrt[3]{\text{final weight (g)}} - \sqrt[3]{\text{initial weight(g)}}\right) / (\text{temperature (°C)} \times \text{experiment duration (day)})\right] \times 1000,$$

Feed conversion ratio (FCR) = Feed intake (dry weight, g)/total wet weight gain (g),

Protein efficiency ratio (PER) = Total wet weight gain (g) /protein intake (dry weight, g),

Hepatosomatic index (HSI) = [Liver weight (g)/fish wet weight (g)] × 100,

Viscero somatic index (VSI) = [Viscera weight (g)/fish wet weight (g)] × 100,

Intraperitoneal fat ratio (IPF) = [Intraperitoneal fat weight (g)/fish wet weight (g)] × 10,

Survival rate (%) = (Final fish number/initial fish number) × 100.

2.3.2. Biochemical Composition. The proximate composition of the experimental diets, muscle (right side), and liver tissues (2 samples per replicate) was analyzed by following the standard official method of AOAC [19]. Moisture was determined by drying the samples at 105°C to a constant weight (protocol number 934.01). Ash content was determined by combustion in a muffle furnace at 550°C for 2 h (protocol number 942.05). Total nitrogen was measured by micro-Kjeldahl technique (Auto Kjeldahl Buchi, K370, Switzerland), and crude protein was calculated by multiplying total nitrogen by 6.25 (protocol number 2001.11). Total lipid (protocol number 920.39) was determined gravimetrically following chloroform-methanol (2 : 1) extraction in a Soxtec System (SoxtecTM 2050 system, Sweden) using the method suggested by Folch, Lees, & Sloane Stanley [20]. Amino acid concentrations in diets and muscle tissues were analyzed using the Waters Pico-Tag method and high-performance liquid chromatography (HPLC, Agilent 1090 system, Palo Alto, CA, USA). The samples were prepared before analysis by the method described by White, Hart & Fry [21]. The HPLC system used for the analysis of amino acids (Knauer, Germany) was measured by column 18 and fluorescence detector (RF5300 Knauer, Germany) at a wavelength of 570 nm.

2.3.3. Serum Biochemical Parameters, Lysozyme Activity Assay, and Antioxidant Enzyme Activity. Blood was randomly collected from the caudal vein of three fish per tank (3 fish per replicate). Serum samples were separated by centrifuging at 3000 g for 10 min at 4°C. For blood biochemical analyses, the sera were transferred to Eppendorf tubes (5415R) and stored at -80°C until analysis. Serum biochemical parameters were analyzed using a biochemical autoanalyzer (Mindray BS-200, China) with commercial diagnostic kits (Pars Azmoon kit, Tehran, Iran). Serum biochemical measurements were conducted to determine serum levels of total protein (TP), albumin (ALB), glucose (GLU), triglyceride (TG), cholesterol (CHO), high-density lipoprotein (HDL), low-density lipoprotein (LDL), calcium (Ca), inorganic phosphorus (P), and magnesium (Mg). Globulin content (GLO) was calculated by subtracting the albumin values from the total serum protein. Serum levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were measured by the IFCC method at 340 nm [22]. The procedure adopted for ALT activity was the same as for AST activity except the substrate comprised of 0.2 M DL-alanine instead of L-aspartic acid. The enzyme assay of alkaline phosphatase (ALP) was made according to the DGKC method of Moss & Henderson [22]. Lactate dehydrogenase (LDH) activity was evaluated by the rate of NADH oxidation at 430 nm in the presence of pyruvate [23]. The lysozyme activity in the serum was determined using a turbidimetric assay according to the method of Ellis [24] by measuring the lytic activity of the *A. ruthenus* serum against lyophilized *Micrococcus lysodeikticus* (Sigma, St Louis, MO, USA). A unit of lysozyme activity was defined as the amount of serum causing a reduction of absorbance of 0.001 per minute at 530 nm at 25°C.

The antioxidant enzyme activity and lipid peroxidation in serum samples were measured to illustrate the effects of dietary taurine on antioxidant capacity. Serum samples (3 fish per replicate) were centrifuged at 13000 g for 15 min at 4°C, and the resultant supernatants were used directly for the antioxidant activity analysis [25]. Superoxide dismutase (SOD; BXC0531A kit, Biorex®, UK), glutathione peroxidase (GPX; BXC0551A kits, Biorex®, UK), and catalase (CAT; ZB-CAT96 Kit, ZellBio® GmbH) activities were measured by a spectrophotometer (Specord 250, Germany; wavelength 550 nm, 340 nm, and 405 nm, respectively). Lipid peroxidation levels were determined based on the malondialdehyde (MDA) content, also known as thiobarbituric acid reactive substances (TBARS), which was measured colorimetrically at 535 nm using the method of Buege and Aust [26].

2.3.4. Digestive Enzyme Activity. At the end of the feeding trials, fish were dissected (3 samples per replicate) on an ice tray to remove the gastrointestinal tract (GI) to determine the activity of the digestive enzymes. The intestine of three fish from each tank was quickly removed, weighed, and frozen in liquid nitrogen, and then stored at -80°C until the determination of digestive enzyme activity. Enzyme extraction was performed by adding homogeneous buffer solution (Tris-HCl 50 mM, KCl 50 mM, and CaCl 20 mM) into the digestive tracts at 9 : 1 (*v*/*w*) ratio at pH 7.8, before homogenization (IKA, Ultra-turrax®, USA). Homogenized samples were poured into 1.5 ml vials. The homogenates were centrifuged in a refrigerated centrifuge (K System Centurion Model, UK) at 30000 g for 10 min at 4°C. Then, the resulting supernatant was dispensed into 1.5 vials and used for enzyme assays. The protein content of homogenates was measured following Bradford [27]. Trypsin activity (E.C.3.4.21.4) was assayed with N-benzoyl-L-arginine ethyl ester (BAEE). Crude extracts were incubated for 2 min (25°C) in 2 ml of Tris/$CaCl_2$ buffer, pH 8.1. The trypsin activity was recorded at 253 nm [28]. Chymotrypsin activity (E.C.3.4.21.1) was assayed with benzoyl tyrosine ethyl ester (BTEE). Crude extracts were incubated for 2 min in 2 ml of Tris/$CaCl_2$ buffer, pH 7.8. The chymotrypsin enzyme activity was detected at 256 nm. The enzyme activity unit (U/mg protein) was defined as the amount of enzyme needed to hydrolyze 1 mg of the substrate (BAEE or BTEE) per minute per milligram of protein solution. Lipase (at wavelength 580 nm, [29]), ALP (at wavelength 405 nm, [30]), and amylase (at wavelength 405 nm, [31]) activities were measured (using Pars Azmoon Biochemical Photometric kit, Iran) by Mindray BS-200 biochemical autoanalyzer, China.

2.3.5. Analysis of Gut Bacteria. The bacterial analysis was carried out at the end of the experiment. The fish (9 fish per treatment) were sterilized, and the gut of each fish was aseptically removed. The gut contents (1 g sample) were

homogenized. The homogenized gut samples of three fish of each replicate were used to avoid individual variations in the gut bacteria ([32]; a single sample from each tank). The homogenates were serially diluted into 10^{-7} with sterile normal saline (0.85%). Then, 0.1 ml of the appropriate dilutions spread on agar plates. Gut bacteria were quantified as colony-forming units (CFU). The total numbers of bacteria (TNB) were analyzed using trypticase soy agar (TSA, SP290, HiMedia laboratories Pvt. Ltd., India) and were incubated at 31°C for 72 hr. Lactic acid bacteria (LAB) were analyzed by using MRS agar (M641, HiMedia laboratories Pvt. Ltd., India) and were incubated at 37°C for 48 hr in a BOD incubator [33, 34]. After the incubation period, the plates were assayed and CFU was recorded as average of triplicate plates.

2.4. Statistical Analysis. The results are presented as mean ± SE (standard error) of the mean calculated from triplicates. One-way ANOVA was used to evaluate the effects of different dietary taurine levels ($P < 0.05$). Tukey's HSD test was used to compare the results. Data were analyzed using SPSS version 16 (SPSS Inc., Chicago, IL, USA). The broken-line regression method considering data on fish WG was used to quantify the minimum dietary taurine supplementation in *A. ruthenus*.

3. Results

3.1. Growth Performances, Feed Utilization, and Somatic Indices. Except in the case of VSI, results showed that different levels of dietary taurine significantly ($P < 0.05$) affected growth, feeding, somatic, and survival indices of experimental fish (Table 2). Weight gain, SGR, TGC, and survival rate in sterlet fed 30 g/kg of taurine supplement (T30) was higher than fish in other experimental treatments. CF in treatments containing 5 and 15 g/kg of dietary taurine was higher than other experimental treatments. FCR in all treatments containing taurine was significantly lower than T0 (control treatment). Sterlet fed 30 g/kg taurine supplementation showed lower FCR than fish fed other dietary taurine treatments ($P < 0.05$). Also, PER in T30 treatment was significantly higher than other treatments. In somatic indices, increasing levels of taurine supplementation led to an increase in HSI of experimental fish in different dietary treatments. However, in the case of the IPF index, the T0 treatment was significantly higher than other experimental treatments. According to break-point regression method analysis, the optimum inclusion of dietary taurine in *A. ruthenus* was estimated to be 30.55 g/kg, when WG was plotted against dietary taurine levels (Figure 1).

3.2. Proximate Composition and Muscle Amino Acid Profile. The proximate analysis results of muscle and liver samples and analysis of amino acid compositions of experimental *A. ruthenus* are presented in Tables 3 and 4, respectively. All indices of proximate analysis of muscle and liver samples were not affected by dietary taurine levels except muscle lipid content. The highest lipid content of muscle was recorded in T50 treatment ($P < 0.05$). However, there was no significant difference in muscle lipid content between T50 and T30 treatments.

Based on the results, the quantity of the amino acid profile of the *A. ruthenus* muscle was significantly influenced by the taurine treatments except in the cases of histidine, valine, glutamic acid, alanine, proline, and tyrosine ($P < 0.05$, Table 4). Among the essential amino acids (EAA) of the *A. ruthenus*, arginine, lysine, methionine, threonine, and isoleucine showed the highest value in the T50 treatment ($P < 0.05$). However, there was no significant difference in the arginine content between T50 and T30 treatments. Also, leucine and phenylalanine significantly showed the lowest value in the control treatment (T0). The maximum and minimum rates of EAA were observed in the *A. ruthenus* which were fed T50 and T0 dietary taurine, respectively. The amount of total EAA in the experimental fish muscle increased with increasing dietary taurine supplements. The highest value for nonessential amino acids (NEAA), aspartic acid, cysteine, and serine were recorded in the T50 treatment. The highest quantity of glycine was obtained in the T30 dietary taurine treatment. The maximum and minimum contents of total NEAA were observed in the *A. ruthenus* of the T0 and T50 experimental treatments, respectively. The total amino acid content of the experimental fish muscle was significantly influenced by taurine dietary supplements ($P < 0.05$). The lowest total amino acid value was recorded in the T0 treatment and the highest in the T50 treatment. Also, the results demonstrated that the total EAA to NEAA ratio of *A. ruthenus* was significantly influenced by taurine supplementation. The maximum ratio was observed in the T50 treatment. However, there was no significant difference in this ratio between T50 and T30 treatments (Table 4).

3.3. Serum Biochemical Parameters, Hepatic Enzyme Activity, and Lysozyme Activity. Examination of serum biochemical parameters of experimental fish at the end of the study period displayed no significant change in total protein, albumin, globulin, glucose, triglyceride, and magnesium among various treatments (Table 5). The highest cholesterol was found in 30 g/kg dietary taurine treatment ($P < 0.05$). HDL and LDL values were affected by experimental treatments and showed a significant difference with the T0 treatment. The maximum amount of HDL was observed in the T50 treatment. However, there was no significant difference in this parameter between T50 and T30 treatments. Also, the highest amount of LDL was observed in the T30 treatment. The serum calcium and phosphorus content of fish were affected by different dietary treatments. While the phosphorus content did not follow a specific trend under the influence of different taurine supplementation levels, an increase in the levels of dietary taurine led to an increase in serum calcium levels. Hepatic enzyme activities were significantly affected by various dietary taurine treatments (Table 6). The activity of ALT, AST, and LDH enzymes increased up to 30 g/kg dietary taurine treatment ($P < 0.05$). However, ALP activity did not follow a similar pattern. However, the highest ALP activity was recorded in the T30 treatment. The highest levels of lysozyme activity were

Table 2: growth performances, feed utilization, and somatic indices of *Acipenser ruthenus* juvenile fed diets with different levels of taurine (mean ± SE values of triplicates ($n = 3$)).

	Experimental diets					P value
	T0	T5	T15	T30	T50	
IBW (g fish^{-1})	14.06 ± 0.26	14.19 ± 0.27	14.20 ± 0.23	14.04 ± 0.26	14.16 ± 0.24	>0.05
FBW (g fish^{-1})	33.19 ± 1.88^{c}	35.20 ± 1.79^{b}	36.91 ± 1.55^{b}	41.61 ± 2.38^{a}	38.42 ± 2.26^{ab}	0.01
WG (g fish^{-1})	19.13 ± 1.23^{c}	21.01 ± 1.32^{b}	22.71 ± 1.31^{b}	27.57 ± 1.93^{a}	24.26 ± 1.82^{ab}	0.01
SGR (%)	1.22 ± 0.02^{c}	1.29 ± 0.04^{b}	1.36 ± 0.04^{b}	1.55 ± 0.03^{a}	1.42 ± 0.03^{ab}	0.01
CF (%)	0.29 ± 0.01^{b}	0.33 ± 0.01^{a}	0.32 ± 0.01^{a}	0.29 ± 0.01^{b}	0.28 ± 0.02^{b}	0.04
TGC	0.42 ± 0.02^{b}	0.43 ± 0.01^{b}	0.49 ± 0.08^{ab}	0.53 ± 0.09^{a}	0.49 ± 0.16^{ab}	0.03
FCR	2.39 ± 0.15^{d}	1.73 ± 0.38^{c}	1.85 ± 0.12^{c}	1.03 ± 0.08^{a}	1.29 ± 0.05^{b}	0.01
PER	1.09 ± 0.11^{d}	1.51 ± 0.23^{c}	1.40 ± 0.16^{c}	2.49 ± 0.11^{a}	1.99 ± 0.11^{b}	0.01
FI (g fish^{-1})	45.72 ± 2.24^{a}	36.33 ± 2.29^{b}	41.99 ± 1.98^{a}	28.39 ± 2.07^{c}	31.29 ± 2.09^{bc}	0.02
HSI (%)	1.08 ± 0.09^{b}	1.23 ± 0.08^{ab}	1.27 ± 0.10^{ab}	1.34 ± 0.10^{ab}	1.41 ± 0.12^{a}	0.04
VSI (%)	12.28 ± 1.27	12.35 ± 1.35	14.96 ± 1.25	14.66 ± 1.14	14.65 ± 1.20	>0.05
IPF (%)	1.36 ± 0.24^{a}	0.41 ± 0.07^{b}	0.71 ± 0.10^{b}	0.56 ± 0.10^{b}	0.72 ± 0.16^{b}	0.04
Survival rate (%)	88.89 ± 1.05^{c}	91.66 ± 1.83^{b}	91.67 ± 1.82^{b}	97.23 ± 1.16^{a}	94.45 ± 1.08^{ab}	0.03

IBW: initial body weight; FBW: final body weight; WG: weight gain; SGR: specific growth rate; CF: condition factor; TGC: thermal growth coefficient; FCR: feed conversion ratio; PER: protein efficiency ratio; FI: feed intake; HIS: hepatosomatic index; VSI: viscero somatic index; IPF: intraperitoneal fat ratio. A different superscript in the same row denotes statistically significant differences ($P < 0.05$).

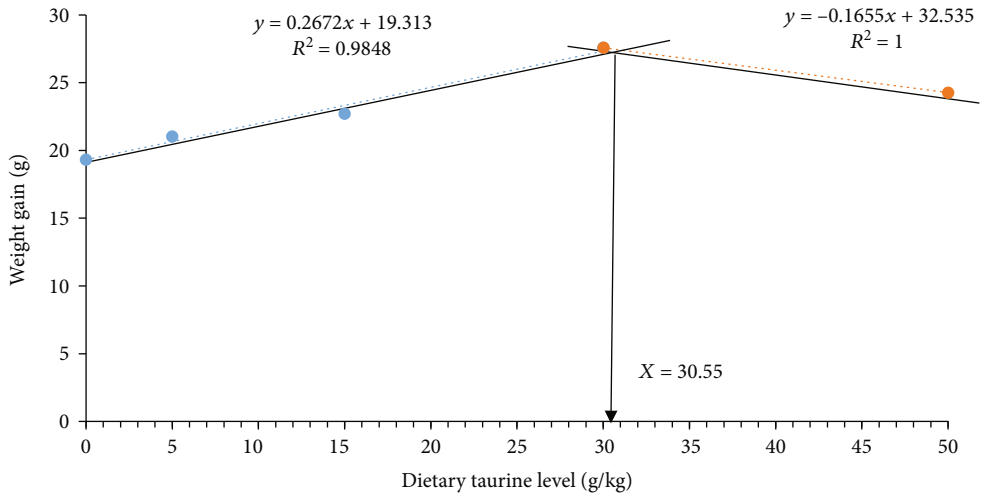

Figure 1: The broken-line regression method considering data on fish weight gain to quantify the minimum dietary taurine level in *Acipenser ruthenus*.

observed in 30 g/kg dietary taurine treatment, which significantly varied from other treatments ($P < 0.05$).

3.4. Digestive Enzyme Activity. The results of the effect of different levels of taurine in the diet on the digestive enzyme activity of *A. ruthenus* are presented in Table 7. Trypsin activity increased in fish-fed dietary taurine compared to the T0 treatment. The highest trypsin activity was observed in the T30 treatment ($P < 0.05$). Also, alkaline phosphatase and α-amylase activities had a similar pattern as trypsin activity. In other words, these enzyme activities increased significantly with the rise in the levels of dietary taurine up to T30 treatment, although the activity of these enzymes decreased in T50 treatment. The chymotrypsin activity had an irregular trend. The highest activity of this enzyme was observed in the T30 treatment ($P < 0.05$). Also, the highest lipase activity was shown in the treatment containing 30 g/kg of the dietary supplement. However, there was no significant difference in the activity of this enzyme between T30 and T15 treatments ($P > 0.05$).

3.5. Antioxidant Enzyme Activity and Lipid Peroxidation. Different levels of taurine in the experimental sterlet diet did not show any significant changes on the activity of SOD and GPX among various experimental treatments ($P > 0.05$). The treatments containing values of 15, 30, and 50 g/kg of taurine showed higher catalase activity compared to the other treatments ($P < 0.05$). The MDA level in the T0 treatment was higher than the other treatments containing different dietary taurine levels ($P < 0.05$, Table 8).

Table 3: Proximate composition of *Acipenser ruthenus* juvenile fed diets with different levels of taurine (mean ± SE values of triplicates ($n = 3$)).

	Experimental diets					P value
	T0	T5	T15	T30	T50	
Muscle composition (% wet weight)						
Moisture	70.82 ± 2.11	69.47 ± 2.54	70.21 ± 2.09	68.50 ± 3.10	67.51 ± 2.97	>0.05
Crude protein	19.12 ± 1.02	19.90 ± 1.10	19.71 ± 0.96	20.98 ± 1.06	20.86 ± 1.18	>0.05
Crude lipid	5.11 ± 0.56b	5.28 ± 0.55b	5.29 ± 0.65b	6.28 ± 0.44a	6.42 ± 0.056a	0.03
Ash	1.41 ± 0.23	1.64 ± 0.21	1.66 ± 0.20	1.92 ± 0.18	1.92 ± 0.24	>0.05
Liver composition (% wet weight)						
Moisture	68.12 ± 2.65	67.41 ± 5.12	66.48 ± 2.98	68.05 ± 3.43	69.21 ± 2.65	>0.05
Crude protein	6.19 ± 0.56	6.32 ± 0.65	6.71 ± 0.55	6.28 ± 0.70	6.10 ± 0.55	>0.05
Crude lipid	17.40 ± 0.99	17.61 ± 0.90	18.44 ± 0.89	18.66 ± 1.00	18.76 ± 1.10	>0.05

A different superscript in the same row denotes statistically significant differences ($P < 0.05$).

Table 4: Muscle amino acids profile (mg/g dry weight) of Acipenser *ruthenus* juvenile fed diets with different levels of taurine (mean ± SE values of triplicates ($n = 3$)).

	Experimental diets					P value
	T0	T5	T15	T30	T50	
Essential amino acid (EAA)						
Arg	8.61 ± 0.91^{c}	11.62 ± 1.01^{b}	11.46 ± 1.12^{b}	13.61 ± 1.21^{a}	14.66 ± 1.11^{a}	0.02
His	3.32 ± 0.05	3.21 ± 0.04	3.37 ± 0.04	3.18 ± 0.07	4.21 ± 0.07	>0.05
Leu	8.05 ± 0.58^{b}	9.26 ± 0.94^{a}	9.36 ± 0.98^{a}	9.55 ± 0.99^{a}	10.77 ± 1.00^{a}	0.04
Lys	9.21 ± 1.01^{c}	10.61 ± 1.00^{b}	10.34 ± 0.99^{b}	10.74 ± 0.98^{b}	13.77 ± 0.99^{a}	0.02
Met	3.03 ± 0.04^{d}	3.11 ± 0.05^{c}	3.34 ± 0.04^{bc}	4.01 ± 0.06^{b}	4.74 ± 0.06^{a}	0.01
Phe	4.20 ± 0.07^{b}	4.95 ± 0.06^{a}	4.89 ± 0.08^{a}	5.29 ± 0.08^{a}	5.24 ± 0.07^{a}	0.04
Thr	4.46 ± 0.04^{c}	6.40 ± 0.05^{ab}	6.65 ± 0.05^{ab}	6.31 ± 0.03^{b}	7.01 ± 0.06^{a}	0.03
Ile	4.64 ± 0.07^{c}	5.35 ± 0.04^{b}	5.36 ± 0.04^{b}	5.53 ± 0.07^{ab}	6.93 ± 0.09^{a}	0.02
Val	4.89 ± 0.11	5.47 ± 0.14	5.46 ± 0.15	5.63 ± 0.14	6.92 ± 0.17	>0.05
Total EAA	50.41 ± 3.21^{c}	59.98 ± 2.12^{b}	60.22 ± 2.77^{b}	63.85 ± 1.84^{b}	74.25 ± 4.01^{a}	0.03
Nonessential amino acid (NEAA)						
Asp	9.72 ± 1.10^{c}	11.29 ± 1.01^{bc}	11.79 ± 1.14^{b}	12.01 ± 1.00^{b}	15.58 ± 0.94^{a}	0.03
Glu	20.44 ± 2.27	24.60 ± 2.47	22.79 ± 2.34	22.70 ± 3.45	25.22 ± 3.29	>0.05
Cys	0.20 ± 0.01^{c}	0.21 ± 0.01^{c}	0.42 ± 0.02^{b}	0.57 ± 0.07^{a}	0.66 ± 0.06^{a}	0.02
Ala	6.62 ± 0.45	7.56 ± 0.34	6.82 ± 0.22	7.71 ± 0.31	7.94 ± 0.40	>0.05
Gly	6.18 ± 0.10^{d}	7.35 ± 0.11^{c}	8.50 ± 0.11^{b}	9.04 ± 0.14^{a}	7.80 ± 0.11^{c}	0.01
Pro	5.06 ± 0.09	5.31 ± 0.10	6.22 ± 0.11	6.36 ± 0.09	6.03 ± 0.10	>0.05
Tyr	3.66 ± 0.09	3.41 ± 0.08	3.71 ± 0.10	3.62 ± 0.11	4.65 ± 0.10	>0.05
Ser	5.12 ± 0.07^{c}	5.44 ± 0.08^{bc}	5.85 ± 0.04^{b}	5.81 ± 0.09^{b}	6.84 ± 0.10^{a}	0.02
Total NEAA	57.00 ± 2.47^{c}	65.17 ± 3.36^{b}	66.10 ± 4.01^{b}	67.81 ± 3.78^{b}	74.72 ± 4.11^{a}	0.01
Total amino acid	107.41 ± 6.54^{c}	125.15 ± 4.32^{b}	126.32 ± 4.87^{b}	131.67 ± 2.85^{ab}	148.97 ± 4.91^{a}	0.01
TEAA/TNEAA	0.88 ± 0.01^{c}	0.92 ± 0.01^{b}	0.91 ± 0.01^{b}	0.94 ± 0.02^{ab}	0.99 ± 0.02^{a}	0.02

Arg: arginine; His: histidine; Leu: leucine; Lys: lysine; Met: methionine; Phe: phenylalanine; Thr: threonine; Ile: isoleucine; Val: valine; Asp: aspartic acid; Glu: glutamic acid; Cys: cysteine; Ala: alanine; Gly: glycine; Pro: proline; Tyr: tyrosine; Ser: serine. A different superscript in the same row denotes statistically significant differences ($P < 0.05$).

3.6. Gut Bacteria Analysis. A comparison of the effects of different taurine supplementations on TNB and LAB is presented in Figure 2. Examination of the experimental results displayed a significant effect of different levels of taurine on the TNB and LAB counts. The highest number of TNB was recorded in the T30 treatment ($5.86 \times 10^9 \pm 1.21 \times 10^9$ CFU/g, $P < 0.05$). However, the maximum amount of LAB was observed in the treatment containing 5 g/kg of the

Table 5: Serum biochemical parameters of *Acipenser ruthenus* juvenile fed diets with different levels of taurine (mean ± SE values of triplicates ($n = 3$)).

	Experimental diets					P value
	T0	T5	T15	T30	T50	
TP (g/dl)	1.24 ± 0.16	1.33 ± 0.22	1.11 ± 0.25	1.55 ± 0.36	1.11 ± 0.21	>0.05
ALB (g/dl)	1.03 ± 0.13	1.07 ± 0.12	0.87 ± 0.24	1.28 ± 0.25	0.80 ± 0.12	>0.05
GLO (g/dl)	0.20 ± 0.15	0.26 ± 0.10	0.24 ± 0.05	0.27 ± 0.07	0.30 ± 0.10	>0.05
GLU (mg/dl)	59.66 ± 1.76	56.00 ± 1.01	57.33 ± 1.85	61.66 ± 5.19	55.66 ± 1.45	>0.05
TG (mg/dl)	219.66 ± 39.73	242.33 ± 35.70	274.66 ± 47.89	305.33 ± 42.55	204.33 ± 26.84	>0.05
CHO (mg/dl)	54.00 ± 5.15^{b}	73.33 ± 8.45^{ab}	65.33 ± 8.19^{ab}	77.66 ± 6.35^{a}	68.66 ± 7.40^{ab}	0.04
HDL (U/l)	9.00 ± 0.57^{b}	8.66 ± 0.88^{b}	10.66 ± 0.33^{ab}	12.33 ± 0.33^{a}	12.66 ± 0.88^{a}	0.03
LDL (U/l)	19.66 ± 0.88^{b}	22.00 ± 1.00^{b}	30.20 ± 0.57^{a}	30.33 ± 2.33^{a}	27.33 ± 1.76^{a}	0.04
Ca (mg/dl)	7.06 ± 0.13^{c}	7.23 ± 0.14^{bc}	7.03 ± 0.08^{c}	7.50 ± 0.15^{b}	7.73 ± 0.08^{a}	0.02
P (mg/dl)	9.93 ± 0.83^{b}	12.30 ± 0.55^{a}	7.80 ± 0.86^{c}	11.8 ± 0.30^{ab}	9.16 ± 0.37^{bc}	0.02
Mg (mg/dl)	1.9 ± 0.15	1.73 ± 0.03	1.70 ± 0.05	1.73 ± 0.03	1.70 ± 0.00	>0.05

TP: total protein; ALB: albumin; GLO: globulin; GLU: glucose; TG: triglyceride; CHO: cholesterol; HDL: high-density lipoprotein; LDL: low-density lipoprotein; Ca: calcium; P: inorganic phosphorous; Mg: magnesium. A different superscript in the same row denotes statistically significant differences ($P < 0.05$).

Table 6: Hepatic enzymes activity (U/l) and lysozyme activity (U/mg protein) of *Acipenser ruthenus* juvenile fed diets with different levels of taurine (mean ± SE values of triplicates ($n = 3$)).

	Experimental diets					P value
	T0	T5	T15	T30	T50	
ALT	209.00 ± 3.88^{d}	278.66 ± 2.96^{c}	274.00 ± 5.51^{c}	410.00 ± 2.88^{a}	312.66 ± 2.66^{b}	0.01
AST	2.33 ± 0.31^{b}	3.33 ± 0.33^{ab}	3.66 ± 0.33^{ab}	5.00 ± 0.21^{a}	2.56 ± 0.66^{b}	0.03
ALP	290.02 ± 5.77^{b}	278.10 ± 5.13^{c}	204.00 ± 2.30^{d}	306.09 ± 2.08^{a}	200.10 ± 1.51^{d}	0.00
LDH	948.60 ± 22.40^{b}	1057.60 ± 80.70^{ab}	1055.60 ± 90.70^{ab}	1248.00 ± 21.50^{a}	1057.00 ± 25.60^{ab}	0.03
Lysozyme	6.61 ± 0.77^{c}	8.81 ± 0.38^{c}	12.49 ± 0.96^{b}	17.49 ± 0.71^{a}	9.57 ± 0.55^{bc}	0.02

ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase; LDH: lactate dehydrogenase. A different superscript in the same row denotes statistically significant differences ($P < 0.05$).

Table 7: Digestive enzymes activity (U/mg protein) of *Acipenser ruthenus* juvenile fed diets with different levels of taurine (mean ± SE values of triplicates ($n = 3$)).

	Experimental diets					P value
	T0	T5	T15	T30	T50	
Trypsin	5.09 ± 0.10^{b}	5.91 ± 0.09^{ab}	5.91 ± 0.19^{ab}	6.35 ± 0.22^{a}	5.97 ± 0.26^{ab}	0.03
Chymotrypsin	15.97 ± 0.91^{d}	14.73 ± 1.04^{d}	19.42 ± 0.86^{c}	42.44 ± 1.14^{a}	29.35 ± 1.06^{b}	0.00
Alkaline phosphatase	1480.4 ± 95.0^{b}	1543.5 ± 146.2^{b}	2097.2 ± 128.3^{ab}	2422.8 ± 170.2^{a}	2109.4 ± 229.3^{ab}	0.03
Lipase	16.28 ± 0.94^{c}	29.33 ± 0.83^{b}	32.88 ± 1.09^{a}	33.27 ± 1.10^{a}	14.61 ± 0.79^{c}	0.01
Amylase	1.85 ± 0.01^{c}	2.21 ± 0.05^{b}	2.26 ± 0.03^{b}	3.46 ± 0.01^{a}	2.31 ± 0.05^{b}	0.02

A different superscript in the same row denotes statistically significant differences ($P < 0.05$).

dietary taurine supplement ($1.6 \times 10^{9} \pm 0.46 \times 10^{9}$ CFU/g, $P < 0.05$).

4. Discussion

The results of this research displayed an improvement in growth performances in the experimental treatments containing high amounts of dietary taurine, and on other hand, the lower contents of dietary taurine lowered the growth performances of the experimental fish. Most of the growth indices, feed utilization, and survival rate in the treatments containing taurine supplementation had a significant improvement compared to the control treatment. Maximum growth was observed in the T30 treatment. The studied species tend to consume animal protein sources in the diet. Therefore, reducing the amount of fish meal in the diet affects the efficiency of feed and thus growth indices and survival rate. In this study, taurine supplementation improved FCR in plant-based protein diets. Growth suppression is one of the most commonly reported symptoms of a taurine

Table 8: Antioxidant enzymes activity (U/ml) and MDA content (μmol/l) of *Acipenser ruthenus* juvenile fed diets with different levels of taurine (mean ± SE values of triplicates ($n = 3$)).

	Experimental diets					P value
	T0	T5	T15	T30	T50	
SOD	109.86 ± 0.38	111.33 ± 0.66	110.83 ± 0.79	111.88 ± 0.98	109.26 ± 0.55	>0.05
CAT	65.54 ± 1.04^{b}	66.70 ± 0.41^{b}	71.06 ± 1.37^{a}	70.53 ± 1.12^{a}	70.78 ± 0.75^{a}	0.03
GPX	68.82 ± 1.87	68.01 ± 1.26	70.88 ± 1.18	69.95 ± 1.92	68.85 ± 1.82	>0.05
MDA	40.01 ± 0.96^{a}	31.28 ± 0.56^{b}	32.02 ± 0.63^{b}	30.92 ± 0.86^{b}	31.00 ± 0.51^{b}	0.04

SOD: superoxide dismutase; CAT: catalase; GPX: glutathione peroxidase; MDA: malondialdehyde. A different superscript in the same row denotes statistically significant differences ($P < 0.05$).

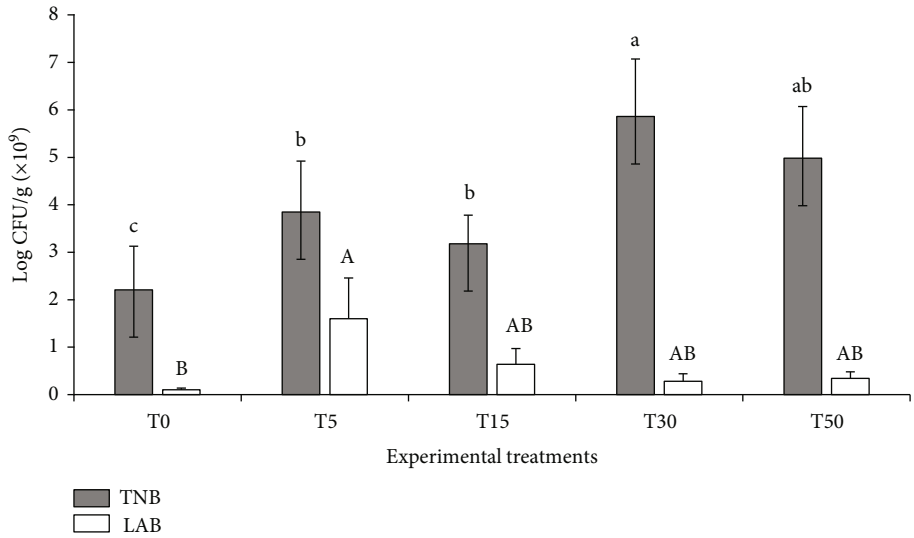

Figure 2: Total numbers of bacteria (TNB, gray bar) and lactic acid bacteria counts (LAB, white bar, $\times 10^9$ CFU/g) of *Acipenser ruthenus* juvenile fed diets with different levels of taurine (mean ± SE values of triplicates ($n = 3$)). A different superscript in the same-colored bars denotes statistically significant difference ($P < 0.05$).

deficiency. Growth improvement has also been reported in other species that used taurine supplementation in basal diets. Jirsa et al. [35] reported an improvement of White seabass (*Atractoscion nobilis*) weight when taurine was added up to 18 g/kg to soy-based diets. Similar results were obtained for Florida pompano (*Trachinotus carolinus*, [36]), Turbot (*Scophtalmus maximus*, [37]), and Korean rockfish (*Sebastes schlegeli*, [38]). Takagi et al. [39] stated that dietary taurine above 34 g/kg suppresses growth indices in yellowtail (*Seriola quinqueradiata*). Inadequate dietary taurine could reduce growth indices and survival rates. Growth performances were significantly affected by dietary taurine [5]. No significant decrease was recorded in growth indices of sterlet-fed T50 and T30 diets. Few studies have reported the side effects of extra taurine in diets [37, 40]. Growth performance alone is not adequate for the study of the taurine effects in the diet. Growth is the result of the nutrient deposition in tissues after metabolic consumption, which could be affected by some amino acids due to appetite stimulation. Dietary taurine may be affected by feeding behavior [41]. Dietary taurine content in carp also changed the motivational characteristics of the diet [3]. Therefore, dietary taurine supplementation could be effective on feed utilization. As a result, a significant improvement in FCR and PER indices was found in the sterlet-fed T30 diet compared to the T50 diet. Also, dietary taurine could affect growth performance through osmotic regulation [42], regulating digestive enzyme activity [43], osteoclast formation [44], increasing antioxidant enzymes and amino acid catabolism, and glycogenesis [45]. Taurine could also act as a signaling molecule. Dietary taurine in the gastrointestinal tract of rats stimulated gastric acid secretion by attaching to receptors of gamma-aminobutyric acid (GABA) [46]. However, complete compensation for growth indices under the influence of taurine supplementation in freshwater fish has not yet been determined.

A reduction in the muscle lipid content was displayed in the sterlet fed low levels of dietary taurine. Other proximate composition parameters of muscle and liver had no significant relation with dietary taurine levels. Few studies have reported changes in the body's proximate composition in response to different levels of the taurine supplement. In these studies, there was a reducing trend in body lipid content in response to decreased dietary taurine levels ([37]

(used dietary taurine 0, 5, 10, and 15 g/kg); [47] (used dietary taurine 0 and 10 g/kg); [48] (used dietary taurine 0 and 7.5 g/kg)). On the other hand, contradictory results have been reported by Espe et al. [11] in *S. salar*. However, differences in species, diet, storage conditions, culture management, and fish size should be considered while comparing different study results. The mechanism of the effect of taurine on body lipid content has not yet been determined. Improving the essential amino acids ratio and intermediate metabolic patterns may potentially affect lipid deposition in muscle. The results indicate that there was a significant effect of taurine on the quantities of muscle amino acids. Total EAA and NEAA content in the muscle increased with increasing dietary taurine levels. The correlations between protein absorption and enzymatic-catabolic activity of amino acids have been reported in European sea bass (*Dicentrarchus labrax*; [49]). An inadequate amino acid profile of the diet may affect the activity of these enzymes [50]. Several studies have reported the positive effects of taurine modification on a plant-based diet and its effect on improving growth characteristics in fish [9, 51, 52]. Improving protein efficiency is achieved when a soybean-based diet is supplemented with taurine. This improvement may be due to the ability of taurine to suppress intestinal mucosal damage. These damages could be produced by soybean nutrients. Therefore, the absorption of the nutrients from the intestine is improved [45]. Taurine is not used for protein synthesis or energy production. However, taurine is involved in many physiological processes. High levels of taurine in the diet may be involved in other amino acid functions. Therefore, dietary taurine could indirectly affect protein or energy production. Diets supplemented with taurine could stimulate the storage of amino acids involved in the osmotic balance of cells, including glycine and arginine [5]. Thus, an increase in the amino acid content of muscle in the sterlet, which is probably used as stored amino acids in protein synthesis, is due to the high levels of dietary taurine. However, the muscle protein content of the experimental fish was not significantly affected by taurine supplementation despite its increasing trend (Tables 3 and 4).

In this study, increasing dietary taurine levels affected cholesterol, HDL, and LDL parameters. The highest content of serum lipid parameters was recorded in experimental fish fed high levels of dietary taurine. The biological activity of taurine in the bile salts production which is vital for intestinal digestion and lipid absorption, has been well documented [2, 53]. Bile salts are formed by the combination of bile alcohols and bile acids. Both of which are derived from cholesterol [54, 55]. Amphipathic properties are essential for miscellaneous solubilization of dietary lipids, which are obtained by esterification or by N-acylamidation with glycine and taurine in bile acids [56]. As a result, taurine increases the absorption of dietary lipids. Most cultured species secrete only bile acids such as C24 cholic acid and chenodeoxycholic acid. C27 bile alcohol has been found in sturgeon and paddlefish, and the 5α-cyprinol sulfate was reported as the major secreted bile salt in carp [56]. Dietary taurine supplementation in Japanese flounder (*Paralichthys olivaceus*) and *S. schlegeli* increased the bile salts [38, 57]. Also, it has been shown that such an increase depends on dietary protein sources [58] and dietary lipid levels [7]. In other words, dietary taurine has a positive effect on lipid digestion and thereby could improve growth. Similar results were reported by Maita et al. [59]. In their study, on *S. quinqueradiata* fed by soybean-based diet, plasma cholesterol levels enhanced when combined with dietary cholesterol and taurine supplements.

An increase in lysozyme activity was observed by an increase in the dietary taurine supplementation level up to 30 g/kg. Taurine is associated with several health indicators, including tissue regeneration [59], reduction of metal toxicity [60], and hepatotoxicity of bacterial lipopolysaccharide [61]. Taurine is an influential regulator for proinflammatory and immune responses. Taurine is effective in neutralizing oxidative stress resulting from inflammatory regulation and expression of leukocyte genes [62]. The combination of taurine with hypochlorous acid (HOCl, a cytotoxic oxidant) produces the stable oxidant taurine chloramine. Therefore, it reduces oxidative stress (Table 8). Taurine could also be involved in regulating inflammatory responses by inhibiting TNF-α, PGE2, and nitric oxide production. This mechanism is accomplished by regulating the production of lymphocytes, the activity of macrophages, granulocytes, monocytes, and the interleukins 6 and 8 productions (Wang et al., [63, 64]). Therefore, it could be stated that dietary taurine increases the survival rate by improving the level of immunity (Table 2).

Despite the growing interest in using taurine as a supplement to diets with low fishmeal levels, its effect on hepatic enzyme activity remains unclear for many aquaculture species. The liver and brain were the primary tissue of taurine synthesis [12, 65]. Therefore, a taurine deficiency could have the most impact on the liver tissue. In the present study, hepatic enzyme activity increased with increasing dietary taurine content up to the T30 treatment. Taurine could stimulate the synthesis of antioxidant enzymes. Taurine acts as an antioxidant through osmoregulation and membrane stability [8]. Antioxidant analysis of fish fed with the dietary taurine showed that the activity of catalase increased in the experimental treatments containing 15, 30, and 50 g/kg dietary taurine. The MDA value in the T0 treatment was significantly higher than the other treatment containing dietary taurine. As a result, taurine reduced the levels of lipid peroxidation in the experimental fish. Bañuelos-Vargas et al. [45] stated that taurine supplementation improves catalase activity and reduces lipid peroxidation levels in *Totoaba macdonaldi*. Taurine supplementation also reduced lipid peroxidation in zebrafish (*Danio rerio*) exposed to ethanol by improving SOD and CAT activities [65]. Despite the existence of various mechanisms in reducing the level of lipid peroxidation in fish-fed diets containing taurine supplementation, the antioxidant function of taurine is important in this regard. However, taurine is a weak scavenger for oxygen superoxide, hydrogen peroxide, and peroxynitrite [66].

Results of the present study indicate that taurine could improve the digestion and absorption of nutrients in plant protein-based diets (about 30 g/kg) in *A. ruthenus* by affecting digestive enzyme activity. The addition of taurine to the common dentex (*Dentex dentex*) diet showed a significant

increase in lipase activity. Chatzifotis, Polemitou, Divanach & Antonopoulou [67] stated that the maximum activity of this enzyme was found in pyloric caeca in the fish-fed diet containing taurine and 250 g/kg soybean protein source. Nguyen, Khaoian, Fukada, Suzuki & Masumoto [68] reported that lipase activity increased in the gastrointestinal tract of *S. quinqueradiata* fed with a fermented soybean meal diet supplemented with taurine. Positive effects of the taurine supplement on the digestive enzyme activity have been observed in Cobia (*Rachycentron canadum*; [69]). Adding 1.0 to 15.0 g/kg taurine to the diet of *Paralichthys olivaceus* has increased taurocholic acid by 2 and 4 times, respectively [57]. Dietary taurine content in fish is valuable for digestion and absorption. Improving the digestion and absorption of dietary nutrients improved feed efficiency and consequently increased growth performance in the T30 treatment (Table 2). Taurine may affect the gut bacteria by enhancing digestion and absorption. Vegetable protein sources may have antinutrient factors that may affect the bioavailability of minerals and other nutrients, leading to reduced growth [70]. Soybean protein has a low content of nonstarch polysaccharides, fibers, and allergic compounds, especially saponins. However, in some species, these contents induce intestinal and hepatic morphometric changes. The result may be a reduction in the digestibility of diet [71, 72]. Taurine increased the absorption of nutrients, especially lipid components, through the morphological normalization of the intestine. Therefore, it provided a suitable substrate for gut bacteria and improved feed efficiency and growth indices in the experimental sterlet.

Based on the results, it could be stated that increased levels of taurine supplementation had beneficial effects on the growth and survival indices of *A. ruthenus* and that 30 g/kg of dietary taurine improved the growth performance. Also, 30 g/kg taurine supplementation level improved hepatic, digestive, and antioxidant enzyme activity in *A. ruthenus*.

Data Availability

The data sets generated and/or analyzed during the current study are available from the corresponding author on request.

Conflicts of Interest

The authors declare that they have no conflict of interest.

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