Effects of Starvation and Refeeding on Growth Performance, Blood Health, Antioxidant Capacity, and Immune Response of Juvenile Yellow Catfish (Pelteobagrus fulvidraco) under Ammonia Nitrogen Stress (Postprint)

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Abstract

To investigate the effects of starvation and refeeding on growth performance, blood health, antioxidant capacity, and immune response of juvenile yellow catfish (Pelteobagrus fulvidraco) under ammonia nitrogen stress, juvenile yellow catfish with an initial body weight of (14.36$±$0.21) g were randomly divided into a control group and an experimental group (3 replicates per group, 30 fish per replicate). The control group was fed to satiation manually for 42 days, while the experimental group was starved for 14 days and then refed to satiation for 28 days. Fish in both groups were exposed to 5.7 mg/L total ammonia nitrogen. The results showed that after 14 days of starvation under ammonia nitrogen stress, the body weight, head kidney macrophage phagocytic index, and serum lysozyme activity of juvenile yellow catfish in the experimental group were significantly lower than those in the control group ($P<0.05$), whereas serum alanine aminotransferase, aspartate aminotransferase activities, and uric acid content, as well as liver superoxide dismutase activity and malondialdehyde content were significantly higher than those in the control group ($P<0.05$). After 14 days of starvation followed by 28 days of refeeding under ammonia nitrogen stress, the final body weight of juvenile yellow catfish in the experimental group was significantly lower than that in the control group ($P<0.05$), but the specific growth rate was significantly higher than that in the control group ($P<0.05$). Serum alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase activities, and uric acid and triglyceride contents in the experimental group were significantly lower than those in the control group ($P<0.05$). Liver superoxide dismutase activity in the experimental group showed no significant difference compared with the control group ($P>0.05$).

Head kidney macrophage phagocytic index and serum total antibody content in the experimental group were significantly higher than those in the control group (P<0.05). Head kidney macrophage respiratory burst, serum total complement content, and lysozyme activity in the experimental group showed no significant differences compared with the control group (P>0.05). The results indicated that under ammonia nitrogen stress, starvation inhibited the growth and health of juvenile yellow catfish; after starvation and refeeding, juvenile yellow catfish exhibited partial growth compensation, and blood deterioration, antioxidant enzyme activity, and immune suppression were alleviated to varying degrees.

Full Text

Effects of Starvation and Refeeding on Growth Performance, Blood Health, Antioxidant Capacity and Immune Response of Juvenile Yellow Catfish under Ammonia Stress

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Abstract

This study investigated the effects of starvation and refeeding on growth performance, blood health, antioxidant capacity, and immune response of juvenile yellow catfish (*Pelteobagrus fulvidraco*) under ammonia stress. Juvenile yellow catfish with an initial body weight of (14.36$±$0.21) g were randomly divided into a control group and an experimental group, each with three replicates of 30 fish. The control group was fed to satiation for 42 days, while the experimental group was starved for 14 days and then refed to satiation for 28 days. All fish were exposed to 5.7 mg/L total ammonia nitrogen. After 14 days of starvation under ammonia stress, the experimental group exhibited significantly lower body weight, head-kidney macrophage phagocytic index, and serum lysozyme activity compared to the control group (P<0.05). Conversely, serum glutamic-pyruvic transaminase and glutamic oxalacetic transaminase activities and uric acid content, as well as hepatic superoxide dismutase activity and malondialdehyde content, were significantly higher in the experimental group (P<0.05). Following 14 days of starvation and subsequent 28 days of refeeding under ammonia stress, the experimental group showed significantly lower final body weight but higher specific growth rate than the control group (P<0.05). Serum glutamic-pyruvic transaminase, glutamic oxalacetic transaminase, and alkaline phosphatase activities, along with uric acid and triglyceride contents, were also significantly lower in the experimental group (P<0.05). Hepatic superoxide dismutase activity did not differ significantly between groups (P>0.05), while head-kidney macrophage phagocytic index and serum total antibody content were significantly higher in the experimental group (P<0.05). No significant differences were observed in head-kidney macrophage respiratory burst, serum total complement content, or lysozyme activity between groups ($P>0.05$). These results indicate that starvation inhibits growth and health in juvenile yellow catfish under ammonia stress, but refeeding can alleviate blood deterioration, antioxidant enzyme suppression, and immune inhibition, while inducing partial compensatory growth.

Keywords: juvenile yellow catfish (*Pelteobagrus fulvidraco*); ammonia; starvation and refeeding; growth; antioxidant enzyme; immune

Introduction

In intensive aquaculture systems, excessive ammonia nitrogen has become a chronic problem. While biofiltration and frequent water changes are commonly used to reduce ammonia levels, these methods are costly. Most cultured fish species are highly sensitive to ammonia, and excessive ammonia exposure causes growth retardation, gill tissue lesions, organ failure, and immune suppression, with behavioral manifestations including rapid breathing, hyperexcitability, fainting, convulsions, and even death. When ammonia concentrations spike or remain chronically elevated, aquaculture operations typically reduce daily feed rations, feeding frequency, or temporarily cease feeding to mitigate ammonia poisoning risks. Previous studies have shown that fish under starvation stress can mobilize stored body reserves to ensure minimum survival requirements, and often exhibit altered physiological characteristics after refeeding —a phenomenon known as compensatory growth. Compensatory growth is classified into three types: hyper-compensatory, full compensatory, and partial compensatory growth. However, research on physiological changes during starvation and subsequent compensatory growth in fish has typically been conducted under normal physiological conditions, with few studies examining whether compensatory growth occurs under ammonia stress.

Yellow catfish (*Pelteobagrus fulvidraco*) is an omnivorous species with carnivorous tendencies, widely distributed in the Yangtze and Pearl River basins and representing an important economic aquaculture species in China. According to the 2017 China Fisheries Statistical Yearbook, national yellow catfish production exceeded 410,000 tons by the end of 2016, with an annual growth rate of 17.32%, establishing it as a major freshwater aquaculture species. The fish is prized for its tender, delicious meat rich in essential amino acids and lack of intermuscular bones. However, high-density culture frequently leads to ammonia spikes, particularly during hot summer months. This study evaluated the effects of starvation and refeeding on growth performance, blood health, antioxidant capacity, and immune response of juvenile yellow catfish under ammonia stress to provide scientific guidance for developing mitigation strategies in yellow catfish aquaculture.

Materials and Methods

1.1 Culture Management

Juvenile yellow catfish were purchased from Jiaxing, Zhejiang, and acclimated for 30 days. One hundred eighty healthy, uniformly sized fish [(14.36$±0.21)g]wererandomlydistributedintosix300−Lplastictanksat30fishpertank.Basedonreportedsafea ionizedammonia0.10–0.11mg/L,pH6.6–7.0)usingapre−mixedammoniumchloride(NH_{4}$Cl) stock solution (10 g/L) adjusted every 7 hours. Total ammonia nitrogen was measured using the Nessler' s reagent method, with non-ionized ammonia concentration calculated accordingly. Fish were fed commercial yellow catfish feed. Culture water was dechlorinated tap water with daily water exchange of one-third total volume. Water temperature was maintained at 24–28 °C, dissolved oxygen $ 6.0mg/L,pH7.4±$0.2, nitrite <0.5 mg/L, under natural photoperiod.

1.2 Experimental Design and Sampling

The six tanks were randomly assigned to control and experimental groups (three tanks per group). The control group was hand-fed to satiation twice daily for 42 days. The experimental group was starved for 14 days, then refed to satiation for 28 days. Samples were collected on days 14 and 42. Prior to sampling, fish were fasted for 24 hours, weighed, and survival recorded. Three fish per tank were randomly selected, anesthetized with MS-222, and blood collected from the caudal vein. Serum was separated after incubation at 4 °C for 4 hours and centrifugation at 3000 r/min for 10 minutes, then stored at -80 °C. Livers were dissected and stored at -20 °C. An additional three fish per tank were dissected aseptically to obtain head-kidney for phagocytic index and respiratory burst assays.

1.3 Serum Biochemical Indices

Serum total protein (TP), albumin (ALB), globulin (GLOB), uric acid (URCA), total cholesterol (TC), triglycerides (TG), glucose (GLU), glutamic-pyruvic transaminase (GPT), glutamic oxalacetic transaminase (GOT), and alkaline phosphatase (AKP) were measured using an AU5800 automatic biochemical analyzer (Beckman Coulter, USA).

1.4 Hepatic Antioxidant Indices

Frozen liver samples were homogenized in ice-cold phosphate buffer (50 mmol/L, pH 7.4) at a tissue-to-buffer ratio of 1:9 (g/mL). The homogenate was centrifuged at 2000 r/min for 15 minutes at 4 °C, and the supernatant was collected for analysis. Hepatic total anti-oxidation capacity (T-AOC) was measured using the method of Miller et al., with one unit defined as the amount causing a 0.01 increase in absorbance per minute per mg protein. Superoxide dismutase (SOD) activity was determined using the method of Beauchamp et al., with one unit defined as the enzyme amount inhibiting NBT autoxidation by 50% per minute per mg protein. Catalase (CAT) activity was measured using the method of Aebi, with one unit defined as the enzyme amount causing a 0.1 decrease in absorbance per minute per mg protein. Malondialdehyde (MDA) content was determined by the thiobarbituric acid reaction using the method of Buege et al. All assays were performed using commercial kits (Nanjing Jiancheng Bioengineering Institute) following manufacturer protocols.

1.5 Serum Lysozyme Activity, Total Immunoglobulin and Total Complement

Serum lysozyme activity was measured using the method of Hultmark et al. with commercial kits (Nanjing Jiancheng Bioengineering Institute). Serum total immunoglobulin and total complement contents were determined using the method of Wu et al. with commercial kits (Shanghai Yuanye Bio-Technology Co., Ltd.), following manufacturer protocols.

1.6 Head-Kidney Macrophage Phagocytic Index and Respiratory Burst

Head-kidney was aseptically removed and placed in L-15 medium (Gibco) containing 100 IU/mL penicillin (Sigma), 100 g/mL streptomycin (Sigma), 10 IU/mL heparin (Sigma), and 2% fetal bovine serum (HyClone). Cell suspensions were prepared by squeezing tissue through a 100 m metal mesh. Macrophages were isolated by discontinuous density gradient centrifugation on 34%/51% Percoll (Sigma) solution (prepared by slowly layering equal volumes of 51% Percoll over 34% Percoll) at 600 r/min for 5 minutes at 4 °C. Isolated macrophages were washed several times with L-15 medium and adjusted to 1$×10^{7}$ cells/mL. Cell viability was assessed using 0.01% trypan blue, ensuring >95% viability. Phagocytic index was measured using the method of Pulsford et al., and respiratory burst was determined using the method of Stolen et al.

1.7 Calculation Formulas

Survival rate (SR, %) = 100 × (number of surviving fish / initial number of fish)
Specific growth rate (SGR, %/d) = 100 × (ln final body weight - ln initial body weight) / number of refeeding days
Feed efficiency ratio (FER) = (final body weight - initial body weight) / dry feed consumption

1.8 Statistical Analysis

Data were analyzed using t-tests and expressed as mean ± standard error (SE). Significance was set at $P<0.05$. All analyses were performed using SPSS 18.0.0 software on Windows.

Results

2.1 Effects of Starvation on Juvenile Yellow Catfish under Ammonia Stress

As shown in , after 14 days of starvation under ammonia stress, survival rate did not differ significantly between experimental and control groups ($P>0.05$). However, body weight was significantly lower in the experimental group ($P<0.05$). Serum total protein, albumin, globulin, total cholesterol, triglyceride contents, and alkaline phosphatase activity were significantly lower in the experimental group ($P<0.05$), while serum glutamic-pyruvic transaminase and glutamic oxalacetic transaminase activities and uric acid content were significantly higher ($P<0.05$). Hepatic superoxide dismutase activity and malondialdehyde content were significantly elevated in the experimental group ($P<0.05$). Head-kidney macrophage phagocytic index and serum lysozyme activity were significantly reduced ($P<0.05$).

2.2 Effects of Refeeding on Juvenile Yellow Catfish under Ammonia Stress

As shown in , after 14 days of starvation followed by 28 days of refeeding under ammonia stress, survival rate and feed efficiency ratio did not differ significantly between groups ($P>0.05$). Final body weight was significantly lower in the experimental group ($P<0.05$), while specific growth rate was significantly higher ($P<0.05$).

As shown in , serum glutamic-pyruvic transaminase, glutamic oxalacetic transaminase, and alkaline phosphatase activities, along with uric acid and triglyceride contents, were significantly lower in the experimental group ($P<0.05$). No significant differences were observed in serum total protein, albumin, globulin, total cholesterol, or glucose contents between groups ($P>0.05$).

As shown in , hepatic total anti-oxidation capacity and catalase activity were significantly lower in the experimental group ($P<0.05$), while malondialdehyde content was significantly higher ($P<0.05$). Hepatic superoxide dismutase activity did not differ significantly between groups ($P>0.05$).

As shown in , head-kidney macrophage phagocytic index and serum total immunoglobulin content were significantly higher in the experimental group ($P<0.05$). No significant differences were observed in head-kidney macrophage respiratory burst, serum total complement content, or lysozyme activity between groups ($P>0.05$).

Discussion

3.1 Effects of Starvation and Refeeding on Growth Performance

In intensive aquaculture, frequent starvation due to high stocking density and uneven feeding often results in zero or negative growth. In this study, 14 days of starvation significantly reduced growth performance and induced negative growth trends, though survival rate was unaffected. Most fish species can recover to pre-starvation physiological status after short-term starvation and refeeding, with some exhibiting hyper-compensatory growth, as reported in over 50 economic fish species including Amur sturgeon (*Acipenser schrenckii*), beluga (*Huso huso*), rainbow trout (*Oncorhynchus mykiss*), European sea bass (*Dicentrarchus labrax*), and Nile tilapia (*Oreochromis niloticus*). Yao et al. proposed that higher specific growth rate after starvation indicates partial compensatory growth. In this study, juvenile yellow catfish under ammonia stress exhibited significantly higher specific growth rate after 28 days of refeeding, despite lower final body weight, demonstrating partial compensatory growth. However, Yang and Yao reported hyper-compensatory growth in darkbarbel catfish (*Pelteobagrus vachelli*) under normal conditions, a discrepancy likely attributable to growth inhibition caused by ammonia toxicity.

3.2 Effects of Starvation and Refeeding on Serum Biochemical Indices

Serum biochemical indices are widely used to evaluate fish health status. Albumin participates in tissue repair and maintenance of plasma colloid osmotic pressure, while globulins are involved in specific immunity. Previous studies have shown that starvation reduces serum total protein, albumin, and globulin contents. In this study, these parameters were significantly lower after 14 days of starvation but recovered to control levels after 28 days of refeeding, suggesting that starvation impairs tissue repair, osmotic regulation, and immunity, while refeeding induces physiological compensation. Uric acid, the end product of purine metabolism, directly reflects protein catabolism. Serum uric acid increased significantly after starvation but decreased after refeeding, even falling below control levels, indicating reduced protein synthesis and enhanced catabolism during starvation, with refeeding decreasing energy demands. Serum triglycerides, total cholesterol, and glucose recovered partially after refeeding, likely due to energy substrates being transported to adipose tissue for lipid resynthesis. Stable blood glucose is vital for life activities; during starvation, glucose is maintained through glycogenolysis and gluconeogenesis. The absence of significant differences in serum glucose between groups after starvation and refeeding suggests that short-term starvation does not disrupt blood glucose stability. Serum transaminases play important roles in protein, lipid, and carbohydrate metabolism, while alkaline phosphatase regulates calcium and phosphorus absorption and maintains homeostasis. When tissues are damaged, these enzymes leak into blood. In this study, starvation significantly increased serum transaminase activities, which decreased below control levels after refeeding. Shi et al. reported similar transient increases in serum transaminases in banded blue sprat (*Oplegnathus fasciatus*) during starvation, with recovery to control levels after refeeding. In contrast, Tian et al. found that serum transaminase and alkaline phosphatase activities in Nile tilapia decreased significantly during 28 days of starvation and remained below initial levels even after 21 days of refeeding. Shui et al. observed no significant changes in serum transaminase or alkaline phosphatase in tiger puffer (*Takifugu flavidus*) during 15 days of starvation. These discrepancies may relate to species, age, size, detection methods, and environmental factors.

3.3 Effects of Starvation and Refeeding on Antioxidant Capacity

Total anti-oxidation capacity reflects the metabolic capacity of both enzymatic and non-enzymatic antioxidant systems to cope with external stress and is closely related to health status. Yengkokpam et al. demonstrated that increased free radicals reduce total anti-oxidation capacity. In this study, hepatic total anti-oxidation capacity did not differ significantly after starvation but was lower in the refed group than the control, possibly due to persistent malondialdehyde accumulation. Malondialdehyde accumulated excessively after 14 days of starvation, and its toxic effects persisted even after refeeding. Environmental stress generates excessive reactive oxygen species (ROS) that damage protein structures, exacerbate membrane lipid peroxidation, and accelerate aldehyde/ketone accumulation. Malondialdehyde can cross-link with proteins and nucleic acids, gradually inactivating antioxidant enzymes, and its content serves as an important indicator of free radical scavenging capacity and cellular damage severity. Additionally, hepatic superoxide dismutase activity was significantly higher after starvation, likely due to activation by low-level ammonia stress. Superoxide dismutase is a primary indicator of oxidative stress, converting superoxide anion radicals to hydrogen peroxide (H_2O_2), which is further decomposed to water and oxygen by glutathione peroxidase and catalase. The absence of significant differences in superoxide dismutase activity between groups after refeeding suggests that refeeding can alleviate antioxidant enzyme system damage caused by starvation.

3.4 Effects of Starvation and Refeeding on Immune Response

As evolutionarily lower vertebrates, fish rely heavily on non-specific immunity as the first and second lines of defense against pathogens. Lysozyme is an important non-specific defense factor that destroys and eliminates foreign invaders and activates complement and phagocytes. In this study, serum lysozyme activity decreased significantly after starvation, indicating immune suppression. Lou et al. reported that serum and spleen lysozyme activities in Japanese sea bass (*Lateolabrax japonicus*) did not change significantly after 5, 10, or 15 days of starvation, though head-kidney lysozyme increased after refeeding in the 10-day starvation group and decreased in the 15-day group but recovered after 5 days of refeeding. Caruso et al. found no significant changes in serum or head-kidney lysozyme activities in European sea bass after 31 days of starvation, while blackspot sea bream showed significantly decreased serum lysozyme. These variations may relate to species, age, size, detection methods, and environmental factors. In this study, after 28 days of refeeding, no significant differences were observed in head-kidney macrophage respiratory burst, serum total complement, or lysozyme activity between groups, while serum total immunoglobulin content and head-kidney macrophage phagocytic index were significantly higher in the experimental group, suggesting hyper-compensatory or full compensatory immune responses.

Conclusion

Under ammonia stress, starvation causes growth retardation, blood health deterioration, and suppression of antioxidant enzyme activities and immune responses in juvenile yellow catfish. Refeeding after starvation induces partial compensatory growth and alleviates blood degradation, antioxidant enzyme suppression, and immune inhibition to varying degrees.

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