Research Article

BASJVETRES

BASRAH JOURNAL OF VETERINARY RESEARCH, 2024, 23(2): 25-36.
https://bjvr.uobasrah.edu.iq/

Effects of sub-lethal copper sulfate exposure on blood parameters and metabolic enzymes activity in Prussian carp, *Carassius auratus* from river of Shatt Al-Arab, Iraq

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DOI: https://doi.org/10.23975/bjvr.2024.147747.1068

Received: 18 March 2024 Accepted: 23 April 2024.

Abstract

Short- and long-term exposure to sub-lethal copper-sulfate concentrations studied in freshwater fish (*Carassius auratus*). The blood parameters, such as the content of hemoglobin-Hb and the red blood cells-RBC, were examined. Alanine transaminase-ALT and aspartate aminotransferase-AST levels in the serum were also measured. The 96-hour copper sulfate median lethal concentration-LC_{50} for the fish (n= 100) was calculated using different concentrations (1–10 mg/l). Two hundred and forty fish were put in 90 l-glass aquariums (10 in each aquarium) (180 fish in the experimental treatments) and (60 fish in the control treatments). Two groups of fish were formed. The first group was subjected to different concentrations of copper-sulfate for one week: 1 mg/l (0.58 copper sulfate LC_{50}), 2 mg/l (1.15 copper sulfate LC_{50}), and 3 mg/l (1.72 copper sulfate LC_{50}). The second fish group received the same copper-sulfate concentrations but for four weeks. The treatment with zero copper-sulfate LC_{50} served as the control. Three duplicates of each treatment were achieved (A total of 340 fish were used). The results demonstrated that the content of RBC and Hb, as well as the level of the enzymes (AST and ALT), increased significantly with increasing copper-sulfate concentration in both the short- and long-term exposed fish. The current study's findings suggest that sub-lethal copper-sulfate concentrations are responsible for bloody changes and liver dysfunction in fish. The environment's copper contamination can be monitored using these parameters.

Keywords: Fish, AST, ALT, RBC, Metals.

Introduction

Chemicals emitted by urban and agricultural activities continue to pollute the aquatic environment (1). Metal concentrations in many aquatic systems exceed natural levels due to metal releases from sources of agriculture and industry processes. Metals are frequently found in a mix in the environment as a result of their concurrent emissions from industrial processes or mining-operations (2, 1). Copper and zinc are essential for cellular metabolism because they are cofactors in several important enzymes. However, they become toxic in high concentrations when released into the environment (3, 4).

The toxic effects of short-term exposure to only one metal at relatively high concentrations have been the focus of metal toxicity studies in fish, compared to the toxic effects of long-term exposure to metal mixes at concentrations that are realistic for the environment (5- 7). Fish must maintain osmosis and gas exchange in their gills to survive when exposed to high metal concentrations. On the other hand, the liver's capacity to accumulate metals may be more significant in cases of chronic or sub-lethal metal exposure (3,7, 8).

Aquatic organisms may undergo several biochemical and physiological reactions upon uptake of a toxic substance. These reactions may be either compensatory or toxic mechanisms (7, 8). The majority of behavioral, biochemical, and physiological changes observed in fish have been linked to prolonged exposure to metal ions such as copper, cadmium, and zinc. These consequences include decreased aerobic range, death, growth retardation, appetite loss, etc. (9,10).

Hematological indicators are used in many fish species under stress to assess changes in their physiology, anatomy, and function (11, 12). Fish blood is sensitive to pollution-induced stress, so blood markers like hemoglobin-Hb, hematocrit, and red blood cell count-RBC can be used to assess environmental stress brought on by heavy metal pollution (13,14).

Enzyme activity is one of the biochemical markers in fish that is highly sensitive to a variety of toxic substances (15, 16). Estimation of the activity of enzymes such as lactate dehydrogenase-LDH, aspartate aminotransferase-AST, and aspartate aminotransferase-ALT is a good bio-parameter for assessing the level of pollutants in long-term exposure (17, 16). The current study aimed to investigate the hematological and biochemical effects of short- and long-term exposure to sub-lethal concentrations of copper-sulfate on freshwater fish (Prussian carp, *Carassius auratus*).

Materials and Methods

The freshwater fish, Prussian carp (*C. auratus*) (Linnaeus, 1758) (Cypriniformes, Cyprinidae) (length 11.4-13.3 cm and weight 95.6-113.2 g) were used in the current study, kept under constant aeration and fed commercial feeds daily. They were caught in the water of the river of Shatt Al-Arab, located in Basrah city, southern Iraq (Figure 1). Before beginning the experiment, the fish (n= 340) were acclimated for one week and fed daily. They were kept under constant aeration and a 12-hour photoperiod.

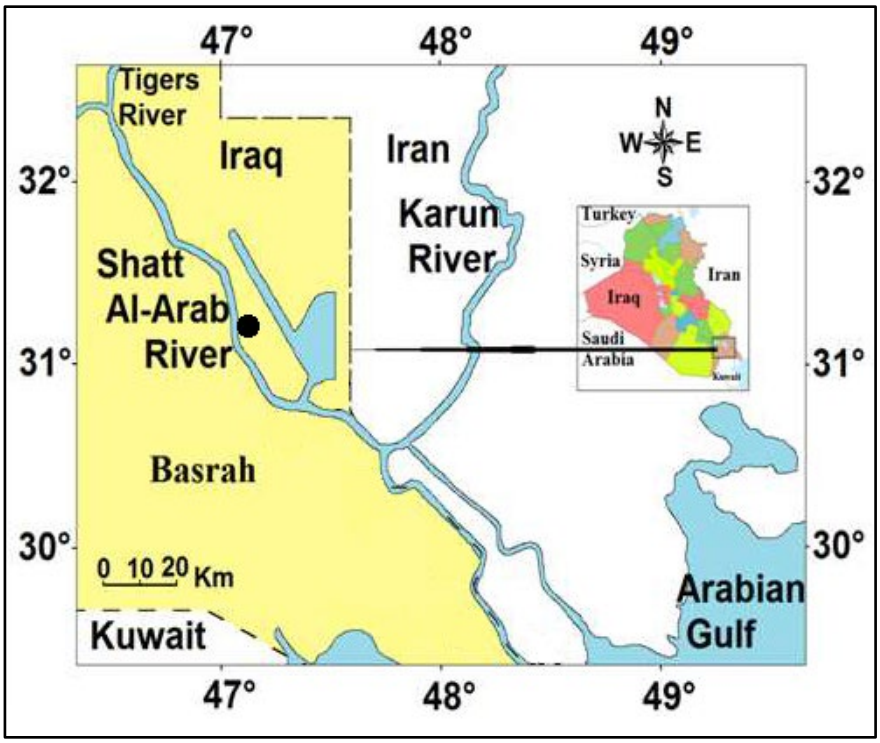

Figure 1. Map of sampling location

One hundred fish were randomly placed into ten aquariums (each holding 90 l) to calculate the 96-hours median lethal concentration-LC_{50}. The copper-sulfate ($CuSO_4$), was dissolved in distilled-water subsequently included in the aquariums, and subjected to the fish at varying concentrations (1-10 mg/l). For 96 hours, fish were exposed to the aforementioned concentrations. Every aquarium's mortality was removed and recorded every day. After recording observations to ascertain the survival time at each copper-sulfate concentration, the LC_{50} value was computed using the regression line designed by (18). The LC_{50} over 96 hours was 5.71 mg/l.

A total of 240 acclimatized fish were separated into two-groups. The first-group was subjected to a short exposure period of one-week to 1, 2, and 3 mg/l of copper-sulfate (0.58, 1.15, and 1.72 of the 96-hour copper sulfate LC_{50}) in addition to the aquaria under similar circumstances but without copper-sulfate (0.00 LC_{50}) as a control treatment. Glass aquaria measuring 70 × 50 × 45 cm and holding 90 l were utilized, with three duplicates for every concentration. For four weeks (a long exposure period), the second-fish group was subjected to the same copper-sulfate concentrations using the same replicates. A diet comprising 32% crude protein was fed to the fish twice a day in a rate of 2% of

their weight of body. The temperature was kept at 25 ±2 °C, and water quality parameters were checked every week (19).

Using a sterile syringe containing the ethylenediaminetetraacetic acid (EDTA) solution as an anticoagulant, the blood samples of fish were drawn from the fish caudal vein at the end of the experiment of both fish groups. Samples of blood were divided into two parts. In the first part, methods of (20, 21) were applied to estimate the Hb content and RBC count, respectively. Using the Reitmen and Frankel (22) method, the second part of the blood samples was used to determine the activity of the AST and ALT enzymes in plasma.

The obtained results were statistically analyzed. The variance in values was evaluated using one-way analysis of variance-ANOVA. With Duncan's multiple range test (Duncan's post hoc test), the means were compared. The level of significance was $p < 0.05$. The statistical software used for all the calculations was IBM-USA's Statistics Package for Social Sciences-SPSS, version 28 for Windows. A calculation was made of the standard error. Microsoft Excel 2007 was used to create the graphs.

Results

Fish exposed to copper sulfate for one-week (short-term exposure) revealed that blood-related parameters significantly different ($p < 0.05$) than the control fish. When fish were exposed to 0.58, 1.15, and 1.72 of copper-sulfate LC_{50}, their RBC count and Hb content increased significantly ($p < 0.05$). The results obtained were 2.43 ±0.05, 2.66 ±0.09, and 2.81 ±0.07 cell/µl (RBC) and 100.33 ±0.86, 102.41 ±0.32, and 105.27 ±0.39 g/l (Hb) for fish subjected to 0.58, 1.15, and 1.72 of copper-sulfate LC_{50}, respectively. They were 1.65 ±0.09 cells/µl (RBC) and 83.54 ±0.43 g/l (Hb) for the control fish.

The results of enzyme activity estimation revealed that the level of AST and ALT in the control fish reached 19.76 ±0.54 and 15.53 ±0.26 IU/l, respectively. Fish treated with copper-sulfate for a week had increased levels of AST and ALT activity. The average activity of copper-sulfate-treated fish (0.58, 1.15, and 1.72 LC_{50}) was 29.48 ±0.87, 30.39 ±0.34, and 36.41 ±0.28 IU/l, respectively, for AST, and 20.89 ±0.51, 23.22 ±0.19, and 28.73 ±0.84 IU/L, respectively, for ALT, which is significantly higher ($p < 0.05$) than the control fish average level. The AST and ALT activities of fish exposed to 0.58 and 1.15 of copper-sulfate LC_{50} were significantly different ($p < 0.05$) from those of fish exposed to 1.72 of copper-sulfate LC_{50} (Table 1 and Figure 2).

The hematological and liver enzymatic parameters of fish exposed to a long-term period (four weeks) of copper-sulfate revealed that RBC counts in copper-sulfate-treated fish (0.58, 1.15, and 1.72 LC_{50}) were significantly higher ($p < 0.05$) (2.64 ±0.05, 2.77 ±0.09, and 2.94 ±0.04 cells/µl, respectively) than in the control fish (1.79 ±0.08 cells/µl). Hb content also increased significantly ($p < 0.05$) in the same direction. The results were 83.75 ±0.83 g/l for the control fish and 101.52 ±0.44, 103.37 ±0.57, and 106.92 ±0.25 g/l for fish exposed to 0.58, 1.15, and 1.72 of copper-sulfate LC_{50}, respectively (Table 2).

The control fish's AST and ALT activity were found to be 18.04 ±0.53and 16.62

±0.82 IU/l, respectively. In contrast, the treated copper-sulfate fish to 0.58, 1.15, and 1.72 LC_{50} revealed significantly higher ($p < 0.05$) average levels of AST and ALT, 30.43 ±0.16, 34.96 ±0.34, and 38.48 ±0.55 IU/l, and 23.78 ±0.29, 29.88 ±0.46, and 34.42 ±0.58 IU/l, respectively (Figure 3).

Table 1. Alterations in RBC count (cell/µl), Hb content (g/l), AST, and ALT activity (IU/l) of *C. auratus* subjected to copper-sulfate concentrations (sub-lethal) for a one-week period.

Treatment	RBC	Hb	AST	ALT
Control	1.65 ±0.09[b]	83.54 ±0.43[b]	19.76 ±0.54[c]	15.53 ±0.26[c]
0.58 LC_{50}	2.43 ±0.05[a]	100.33 ±0.86[a]	29.48 ±0.87[b]	20.89 ±0.51[b]
1.15 LC_{50}	2.66 ±0.09[a]	102.41 ±0.32[a]	30.39 ±0.34[b]	23.22 ±0.19[b]
1.72 LC_{50}	2.81 ±0.07[a]	105.27 ±0.39[a]	36.41 ±0.28[a]	28.73 ±0.84[a]

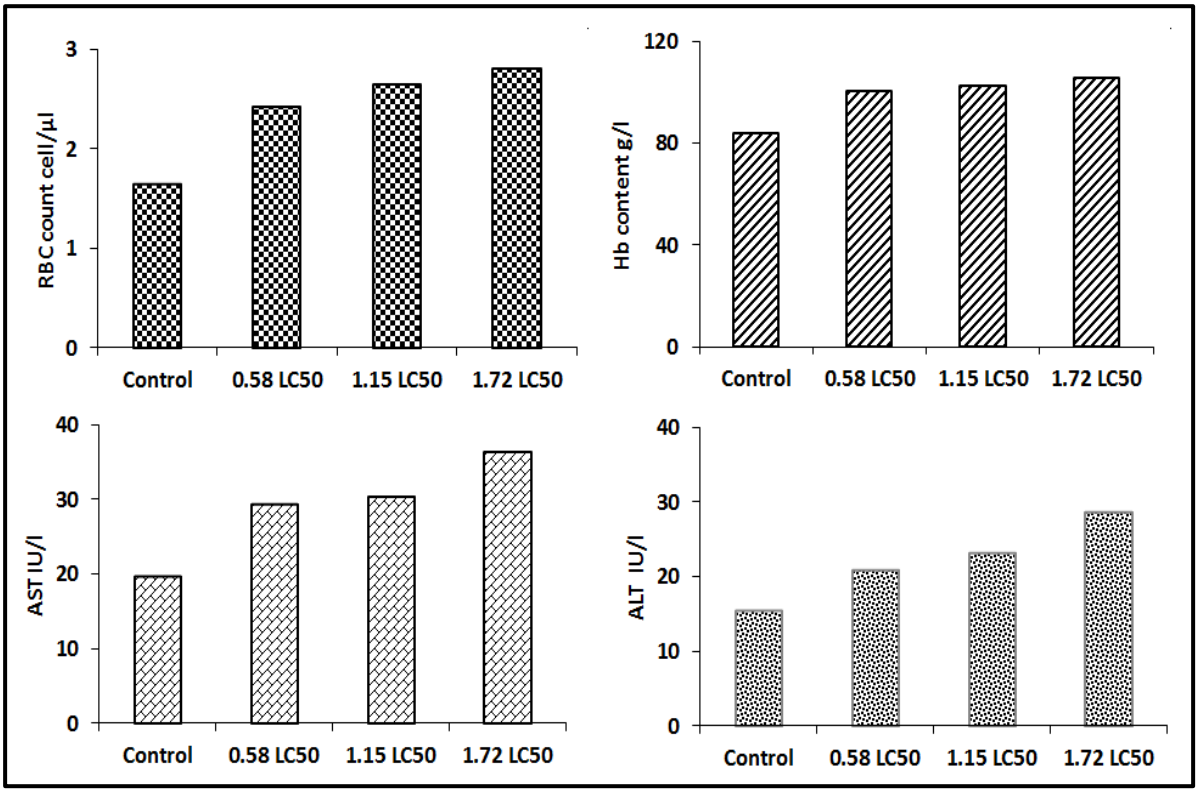

Figure 2. Sub-lethal effects of short-term exposure of copper sulfate in *C. auratus*, in terms of hematological and liver enzymatic stress

Table 2. Alterations in RBC count (cell/µl), Hb content (g/l), AST, and ALT activity (IU/l) of *C. auratus* subjected to copper-sulfate concentrations (sub-lethal) for a four-week period.

Treatment	RBC	Hb	AST	ALT
Control	1.79 ±0.08[b]	83.75 ±0.83[a]	18.04 ±0.53[d]	16.62 ±0.82[d]
0.58 LC_{50}	2.64 ±0.05[a]	101.52 ±0.44[b]	30.43 ±0.16[c]	23.78 ±0.29[c]
1.15 LC_{50}	2.77 ±0.09[a]	103.37 ±0.57[b]	34.96 ±0.34[b]	29.88 ±0.46[b]
1.72 LC_{50}	2.94 ±0.04[a]	106.92 ±0.25[b]	38.48 ±0.55[a]	34.42 ±0.58[a]

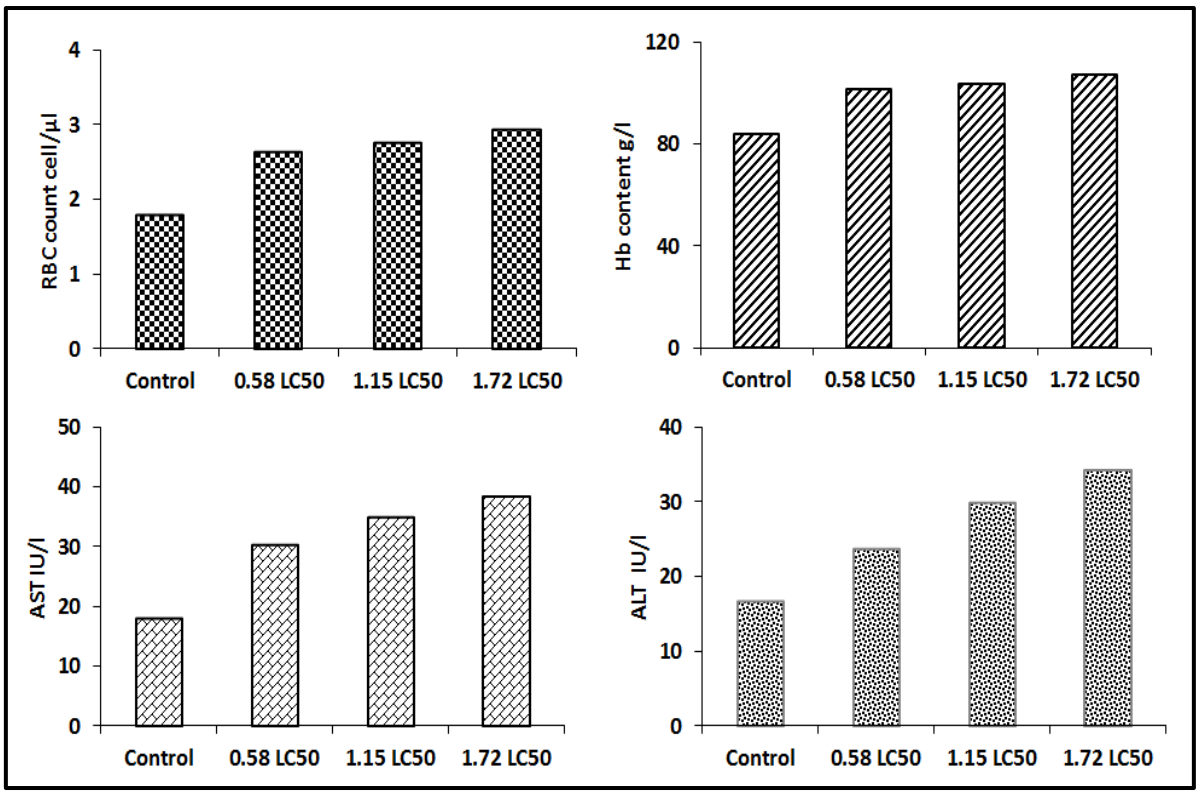

Figure 3. Sub-lethal effects of long-term exposure of copper sulfate in *C. auratus*, in terms of hematological and liver enzymatic stress

Discussion

According to the current study, short- and long-term exposure to copper-sulfate was associated with a statistically significant rise in the RBC count and an increase in Hb content with concentration. These findings are consistent with (23, 24) who found that the content of Hb and hematocrit of *Catla catla* (Indian major carp) treated with arsenic trioxide increased significantly in comparison to the control fish. The noteworthy rise in these hematological indices suggested that the fish's ability to respire was compromised because of the toxic substance's damage to their gills.

Increases in RBC counts due to short-term and long-term exposure to copper-sulfate might be linked to several variables. While *C. auratus* was exposed to copper-sulfate, the fish appeared to become hypoxic. This decrease in dissolved oxygen availability results in the buildup of oxygen debt, and hypoxia, in organisms. Fish also recompense for hypoxia in dominant hypoxic states, which could be attributed to gill lamellae epithelial lifting, by increasing the count of RBC (24). It has been established that metal ions promote erythropoiesis (25). After both short- and long-term sub-lethal exposure to copper-sulfate, there is an increase in RBC, Hb, and hematocrit values. This indicates that *C. auratus* is attempting to survive in surroundings medium with elevated oxygen demand, which is caused by reduced exchange of gases due to the deterioration of gill membranes (26).

The RBC count increased in fish treated with copper-sulfate, along with a significant rise in Hb content. Because of internal hemorrhage, the fish were subjected to oxygen stress. The increased Hb content of their blood likely increased the blood's oxygen capacity, allowing the tissues to receive more oxygen. To meet the increased oxygen demand, the body employs this mechanism to try to absorb more oxygen from the surrounding environment (27).

Toxic substances can accumulate in different tissues after prolonged exposure, changing the activity of enzymes. When toxic substances are administered sub-lethally to fish, the liver plays a crucial role in their absorption, accumulation, biotransformation, and excretion (28, 29). In order to show liver function or hepatotoxicity caused by a toxicant, liver function tests such as serum analysis, AST, and ALT are frequently used (30, 31). The activity of the ALT and AST enzymes increased in *C. auratus* fish exposed to sub-lethal copper-sulfate concentrations over short- and long- period. This suggests that copper accumulation harmed the fish's liver, resulting in the release of the liver enzymes ALT and AST into the bloodstream. The amount of damage caused by a toxic substance depends on the type and duration of exposure (32, 33). Following exposure to As_2O_3, *Channa punctatus* blood showed significant changes in the activity of AST and ALT enzymes. It was proposed that these changes could be the result of histopathological lesions in the liver (34). (35) revealed that fish exposed to zinc experienced liver tissue damage. (30) has been found that the AST-catalyzed molecular rearrangement reaction involving amino acids linked to the citric acid cycle at two points, oxaloacetic and ketoglutaric acids, is the most important mechanism for

the introduction of reduction equivalents into mitochondria. ALT predominates in organs with high glycogenesis, such as the liver (36). The AST and ALT enzymes in blood serum are considered to be useful diagnostic tools from a medical and clinical standpoint for evaluating the effects of toxic environmental pollutants (37).

Heavy metals have been studied for their effects on the activities of ALT and AST enzymes in various fish species' serum or liver (23, 36, 37). (38) has been found that after sub-chronic dieting, a 40-day exposure to copper increased AST and ALT levels in the serum of the rockfish *Sebastes schlegeli* with increasing exposure duration and concentration. The high concentration of AST and ALT in the liver causes large amounts of these chemicals to be released into the bloodstream when the liver is damaged. Thus, elevated AST and ALT activity in *S. schlegeli* serum is thought to be caused by copper-induced liver damage. This study supports the current study, which discovered that fish exposed to copper sulfate for short and long periods of time exhibit increased AST and ALT activity as the concentration of copper-sulfate increases. Furthermore, cadmium exposure has been shown to reduce hepatocyte AST and ALT activities in *Sparus aurata* (39). Copper and cadmium have a significant effect on ALT and AST activities. Although the fact that copper is required for many organisms to function normally, cadmium is a xenobiotic and unnecessary component (35, 40).

Conclusion

Short- and long-term exposure to copper-sulfate causes changes in blood parameters and impaired liver function in *C. auratus*. The biomarkers RBC, Hb, AST, and ALT increased with increasing copper-sulfate concentration and exposure duration. *C. auratus* blood and liver can be used as bio-indicators of prior copper exposure. Long-term exposure resulted in regenerative responses, whereas shorter-term exposure primarily resulted in changes in blood and liver function.

Acknowledgment

The researcher acknowledges the provision of study samples, laboratories, and study facilities by the University of Basrah's Marine Science Center and the Agriculture College's Fisheries and Marine Resources Department.

Conflicts of interest

The authors declare that there is no conflict
. of interest

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تأثيرات التعرض لكبريتات النحاس التحت المميت على مؤشرات الدم ونشاط الانزيمات الأيضية في أسماك الكارب البروسي *Carassius auratus* من نهر شط العرب، العراق

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الخلاصة

تمت دراسة التعرض القصير والطويل الامد لتراكيز كبريتات النحاس تحت المميتة على أسماك المياه العذبة (*Carassius auratus*). تم فحص مؤشرات الدم مثل محتوى الهيموكلوبين وخلايا الدم الحمراء. كما تم قياس مستويات انزيمات الألانين ترانس-امينيز والأسبارتات امينوترانسفيريز في المصل. تم حساب متوسط التركيز المميت لكبريتات النحاس لمدة 96 ساعة اتجاه الأسماك (عدد الاسماك 100) باستخدام تركيزات مختلفة من كبريتات النحاس (1-10 مجم/لتر). ثم تم وضع مائتين وأربعين سمكة في أحواض زجاجية سعة 90 لترًا (10 في كل حوض) (180 سمكة في المعاملات التجريبية) و(60 سمكة في معاملات السيطرة). تم تقسيم الاسماك الى مجموعتين. تم تعريض المجموعة الأولى لتركيزات مختلفة من كبريتات النحاس لمدة أسبوع واحد: 1 ملغم/لتر (متوسط التركيز المميت يساوي 0,58)، و2 ملغم/لتر (متوسط التركيز المميت يساوي 1,15)، و3 ملغم/لتر (متوسط التركيز المميت يساوي 1,72). عرضت المجموعة الثانية من الأسماك الى نفس تراكيز كبريتات النحاس السابقة ولكن لمدة أربعة أسابيع. لم يتم اضافت كبريتات النحاس الى معاملة السيطرة. تم استخدام ثلاث مكررات من كل معاملة (تم استخدام إجمالي 340 سمكة). أظهرت النتائج أن محتوى خلايا الدم الحمراء والهيموكلوبين وكذلك مستوى الإنزيمات، الألانين ترانس-امينيز والأسبارتات امينوترانسفيريز يزداد بشكل معنوي مع زيادة تركيز كبريتات النحاس في كل من التعرض القصير وطويل الامد للأسماك. تشير نتائج الدراسة الحالية إلى أن تراكيز كبريتات النحاس تحت المميتة هي المسؤولة عن التغيرات في محتوى الدم واختلال وظائف الكبد في الأسماك. يمكن مراقبة تلوث النحاس في البيئة باستخدام هذه المؤشرات.

الكلمات المفتاحية: الأسماك، الألانين ترانس-امينيز، الأسبارتيت امينوترانسفيريز، خلايا الدم الحمراء، المعادن.