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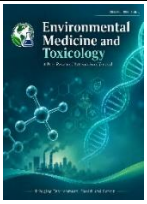

Toxic effects of tin(II) chloride on hematological parameters and histopathology of gills and intestines in common carp (*Cyprinus carpio*)

Javad Kharkan[1], Mohammad Hossein Sayadi[2*]

1. Department of Environmental Engineering, Faculty of Natural Resources and Environment, University of Birjand, Birjand, Iran
2. Iranian National Institute for Oceanography and Atmospheric Science (INIOAS), Tehran 1411813389, Iran

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Abstract

Background and Objectives: Tin(II) chloride ($SnCl_2$) was selected because of its industrial applications and the potential release of inorganic tin into aquatic environments, motivating assessment of its toxic effects in freshwater fish. Therefore, the aim of this study was to investigate the toxic effects of sublethal concentrations of tin chloride ($SnCl_2$) on blood parameters and gill and intestinal tissues of common carp.

Methods: For this purpose, the 96-h median lethal concentration (LC_{50}) of $SnCl_2$ in common carp weighing 40 ± 3 g was determined through an acute toxicity test conducted according to OECD Test Guideline 203. Then, 10, 25, and 50% of the LC_{50}-96h were selected as sublethal concentrations of SnCl2. After the end of the experimental period (28 days), hematological and histological examinations were performed.

Results: The 96-h LC_{50} of $SnCl_2$ was 131.82 mg/L. Compared with the control, red blood cell (RBC) counts decreased significantly and white blood cell (WBC) counts increased significantly at 25% and 50% of the LC_{50}, whereas hematocrit (HCT) and hemoglobin (HGB) decreased significantly at 50% of the LC_{50} ($p < 0.05$). Gill lesions included shortening and curvature of secondary lamellae, oedema, and necrosis. Intestinal changes included increased goblet cell numbers, goblet cell swelling, increased blood cell presence, and expansion of villous structures.

Conclusion: Under the experimental conditions, 28-day exposure to $SnCl_2$ was associated with altered hematological parameters and histopathological damage in the gills and intestines of common carp.

Highlights

- The 96-h LC_{50} of $SnCl_2$ for common carp was 131.82 mg/L.
- $SnCl_2$ exposure reduced RBC, HGB, and HCT but increased WBC counts.
- Gill lesions included secondary lamellar shortening, oedema, necrosis, and curvature.
- Intestinal lesions included goblet cell swelling and expansion of villous structures.
- $SnCl_2$ exposure produced tissue damage in carp gills and intestines across the tested concentrations.

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* **Corresponding author:** Mohammad Hossein Sayadi (**E-mail:** mh_sayadi@inio.ac.ir)

Introduction

Assessing metal toxicity in freshwater fish can help identify physiological and tissue-level responses to contaminant exposure. At the same time, large amounts of heavy metals enter aquatic ecosystems as a result of human activities [1]. Heavy metals including copper (Cu), manganese (Mn), zinc (Zn), iron (Fe), lead (Pb), cadmium (Cd), tin (Sn), and mercury (Hg) are natural components of the Earth's crust and enter the environment through natural activities such as rock weathering, erosion, and hydrodynamic processes. However, their increase due to human activities has made them a major growing concern worldwide [2]. Heavy metals are persistent, non-biodegradable compounds that can accumulate in sediments, have a long half-life in the environment, and can accumulate in the tissues of living organisms, leading to toxic effects in any food source [3]. Furthermore, they cannot be removed through oxidation, sedimentation or bioremediation like organic compounds [4]. Pollution of aquatic ecosystems with heavy metals is not only dangerous for aquatic animals, but also for humans as consumers of fish [5]. Tin (Sn) is an important metal with a wide range of applications in various industries including soldering, metallurgy, packaging, alloys and electronics. It is used as a catalyst in organic reactions, stabilizer in plastics and reagent in organic synthesis. As a result, tin is a vital element with a wide range of applications due to its desirable properties such as corrosion resistance, malleability and low toxicity [6]. Therefore, during various stages of extraction and use as a raw material in the industrial sector, there is a possibility of entering aquatic environments and exposure to fish. Environmental monitoring data demonstrate the occurrence of tin in aquatic environments. The World Health Organization reported that, in a historical survey of Canadian waters, nearly 80% of samples contained inorganic tin at concentrations below 1 µg/L, whereas concentrations reached 37 µg/L near pollution sources [7]. These measurements refer to inorganic tin rather than specifically to $SnCl_2$ and provide environmental context without establishing that the experimental concentrations represent typical environmental exposure. Tin(II) chloride ($SnCl_2$) was selected as an industrially used inorganic tin salt to investigate the effects of waterborne tin exposure under controlled laboratory conditions. Its applications include use as a reducing agent, a soldering flux, and in the preparation of surfaces for metallization [7]. Previous fish studies summarized by WHO have examined both lethal and developmental effects of $SnCl_2$: Taylor et al. investigated acute mortality in dab (*Limanda limanda*) [8], whereas Kapur and Yadav assessed hatching success in common carp (*Cyprinus carpio*) [9]. These studies provide a basis for further investigation of hematological and tissue-level responses to repeated $SnCl_2$ exposure. Fish are among the biomarkers and by examining the conditions of these biomarkers, it is possible to monitor the entry of pollution into aquatic ecosystems, even at low concentrations [10]. The toxic effects of heavy metals in fish cause changes at the structural and functional levels of various organs [11]. Metal accumulation in fish causes the generation of reactive oxygen species (ROS) and can cause oxidative stress and morphological and biochemical changes in various fish organs [12].

Histology as a biomarker is a common and useful approach for assessing pollution and its effects on aquatic ecosystems [13]. This method is a microscopic examination of the cells and tissues of a living organism and can be used to determine both quantitative and qualitative tissue abnormalities [14]. Histological damage can result from various biochemical and physiological interactions within the cell following exposure to foreign substances such as heavy metals. The main organs susceptible to heavy metal toxicity are the gills, liver, and intestine. Gills are the primary target of waterborne pollutants due to their close contact with the external environment [15]. Whereas, the intestines are of particular importance in histological studies due to their digestion and absorption of nutrients [16]. Various studies prove that different heavy metals cause histological damage to different organs of fish. For example, Naz et al. stated that Cu and Cd

metals in the gill tissue of common carp (*Catla catla*) caused histological damage of telangiectasia and necrosis of lamellar epithelial cells, while in the intestine, villous atrophy, epithelial villi loss, and congestion were observed after 30 days of exposure [2]. Aliza et al. stated that mercuric chloride ($HgCl_2$) causes several changes such as Oedema, hyperemia, lamellar hyperplasia, and fusion of secondary lamellae in the gill tissue of tilapia (*Oreochromis mossambicus*), and reported that the severity of histological damage increases with increasing concentration and duration of exposure to $HgCl_2$ [17].

Another biological monitoring method to assess the effects of pollutants on the health of fish populations is hematological studies. Blood is important as a key indicator for assessing the physiological status of body organs, in diagnosing health or disease, and in monitoring the biological processes of living organisms, including fish [2]. Blood markers such as red blood cells (RBC) and white blood cells (WBC) form part of the non-specific cellular immune system and changes in their numbers can be considered as a suitable indicator of fish response to stressors. Hematology is used to assess the physiological conditions of blood cells. By collecting blood and determining the biochemical parameters of blood serum and comparing it with natural conditions, it can be used as a paraclinical tool in diagnosing the effects of pollutants on fish [18]. Common carp (*C. carpio*) is one of the most important species of freshwater fish that is distributed in Asia and Europe. When fish is exposed to various contaminants such as heavy metals, it can lead to changes in biochemical and hematological parameters. For example, Habib et al. stated that hematological parameters such as the number of RBCs and hematocrit (HCT) were reduced in common carp exposed to lead nitrate ($Pb(NO_3)_2$) compared to the control group [19]. Vo et al. stated that Pb reduces HCT and RBCs in tilapia fish [20]. Shah et al. reported that chromium metal causes a decrease in hemoglobin (HGB) and RBCs levels in the fish *Ctenopharyngodon idella* [1]. Previous investigations demonstrate that $SnCl_2$ toxicity in fish is not restricted to acute mortality. Şişman reported developmental abnormalities and reduced survival in zebrafish (*Danio rerio*) embryos and larvae, together with increased micronucleus frequency in peripheral erythrocytes of exposed adults [21]. More recently, Barkhordari et al. included $SnCl_2$ in a comparative study of tin compounds using zebrafish embryos and assessed survival and developmental endpoints over 96 h [22]. Therefore, the aim of this study was to investigate the toxic effects of tin(II) chloride on hematological indices and the histopathology of the gills and intestine in common carp (*Cyprinus carpio*).

Materials and Methods

Experimental design

The study comprised two experimental stages: a 96-h acute toxicity test to determine the median lethal concentration (LC_{50}) of $SnCl_2$ and a 28-day exposure experiment to assess hematological parameters and gill and intestinal histopathology. Mortality was recorded at 24, 48, 72, and 96 h during the acute test, whereas hematological and histopathological assessments were conducted at the end of the 28-day experiment.

Preparing common carp

In this study, 300 common carp (*Cyprinus carpio*) weighing approximately 40 ± 3 g were obtained for the acute toxicity assessment [23], while 40 fish weighing approximately 100 ± 8 g were used for the chronic toxicity assessment of $SnCl_2$ [24]. The fish were purchased from a fish farm in Birjand, Iran, and subsequently transported to the Limnology Laboratory of the Faculty of Natural Resources and Environment at the University of Birjand. In order to acclimate the fish to laboratory conditions, they were kept in 200-liter tanks for two weeks and during acclimation, fish were fed a commercial diet (Faradaneh Co., Iran) twice daily at a total daily ration of 4.9–5.2% of fish body weight. The physicochemical parameters of water such as temperature (23.2 ± 1.1 °C), pH (7.1 ± 0.2) and

dissolved oxygen (7.3 ± 0.1 mg/L) were measured.

Preparation of $SnCl_2$

$SnCl_2$ powder was purchased from Merck Company (Germany) and a 1000 mg/L solution (Stock solution) was prepared from it.

Acute Toxicity Test

The acute toxicity of $SnCl_2$ to common carp was evaluated using OECD Test Guideline 203 [25]. Based on this method, 10 pieces of carp were exposed to different concentrations (1, 5, 10, 30, 50, 100, 200, 300 and 400 mg/L) of $SnCl_2$ for 24, 48, 72, and 96 hours. Each $SnCl_2$ concentration was tested in three independent replicate tanks. During this period, the fish were not fed. The number of fish that died in each time interval was recorded and they were immediately removed from the environment. Mortality data were used to estimate LC_{50} values at each observation time, as described in the Statistical Analysis section.

Sublethal Toxicity Assessment

Haematological and Histopathological Effects

After calculating the acute toxicity of $SnCl_2$, 10, 25, and 50% of the 96-hour median lethal concentration ($LC_{50\text{-}96h}$= 131.82 mg/L) were selected as sublethal concentrations. Then, fish were exposed to sublethal concentrations of $SnCl_2$ in 30 L of tap water used directly with dechlorination for 28 days in 4 groups (one control group) of 10 fish.

- Group A: Control group (normal water and commercial feed).
- Group B (10 %): 13.18 mg/L $SnCl_2$.
- Group C (25 %): 32.95 mg/L $SnCl_2$.
- Group D (50 %): 65.91 mg/L $SnCl_2$.

Measurement of hematological parameters

At the end of the experimental period (28 days), blood samples were collected for hematological analyses. In each treatment, five fish were randomly selected and anesthetized in a solution of clove powder at a concentration of 500 mg/L. Then, in the anesthetized state, blood samples were collected from the caudal peduncle of the fish. RBCs counting was performed manually by diluting the sample with isotonic saline and a Neubauer slide. For WBCs counting, a white melanjour pipette and Marcano diluent solution were used. Then, the number of WBCs on the Neubauer slide was calculated. HCT was also measured using the microhematocrit method or capillary method [26]. The HGB level in the blood was measured after cell lysis and conversion of hemoglobin to cyanmethemoglobin using Drabkin's solution. For this purpose, the blood sample was mixed with Drabkin's solution at a dilution of 1:250. Then, the absorbance of the samples was measured using a spectrophotometer at a wavelength of 540 nm. The hemoglobin concentration was calculated based on the absorbance of the sample using equation 1 [27]:

Eq (1)

$$HGB = (A_{540} - A_{BLANK})\, W_{Hb}\, F_D\, (C_E\, d\, 1000)^{-1}$$

In this equation, A_{540} is the absorbance of the sample at 540 nm, A_{BLANK} is the absorbance of pure Drabkin solution at 540 nm, W_{Hb} is the molecular mass of hemoglobin, F_D is the dilution factor, C_E is the millimolar extinction coefficient of cyanomet hemoglobin at 540 nm, d is the optical path length in centimeters, and 1000 is the conversion factor from mg to g.

Histological studies

After 28 days, three carp from each treatment were caught completely randomly and anesthetized using clove powder. Then, their gill and intestinal tissues were removed completely without the slightest physical damage. First, the tissues were fixed in Bouin's solution for 24 hours and then preserved in 70% alcohol. After dehydration, clarification, and paraffin embedding of the tissues were performed in an automated device (Duplex processor, Shandon Elliott). The samples were molded and 5 µm-thick sections were prepared using a microtome. Hematoxylin-eosin was used to stain tissues. Three coverslips were prepared from each sample and examined using a Nikon light microscope (eclipse-E200). Tissue changes were evaluated based on a semi-quantitative method, in which (-)

is no change, (+) is mild, (++) is moderate, and (+++) is severe change [26].

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 24. Acute toxicity mortality data were analysed using probit analysis to estimate the median lethal concentrations (LC_{50}) at 24, 48, 72, and 96 h. For the 28-day sublethal exposure experiment, normality was assessed using the Kolmogorov–Smirnov test, and homogeneity of variances was evaluated. Haematological parameters were analysed using one-way ANOVA, followed by Duncan's multiple range test to compare treatment means. Results are presented as mean ± standard deviation, with statistical significance set at $p < 0.05$.

Results

Acute toxicity of $SnCl_2$

The LC_{50} of $SnCl_2$ for common carp is shown in Fig 1. Based on the obtained results, the median lethal concentration decreases from 24 hours to 96 hours, but its acute toxicity increases.

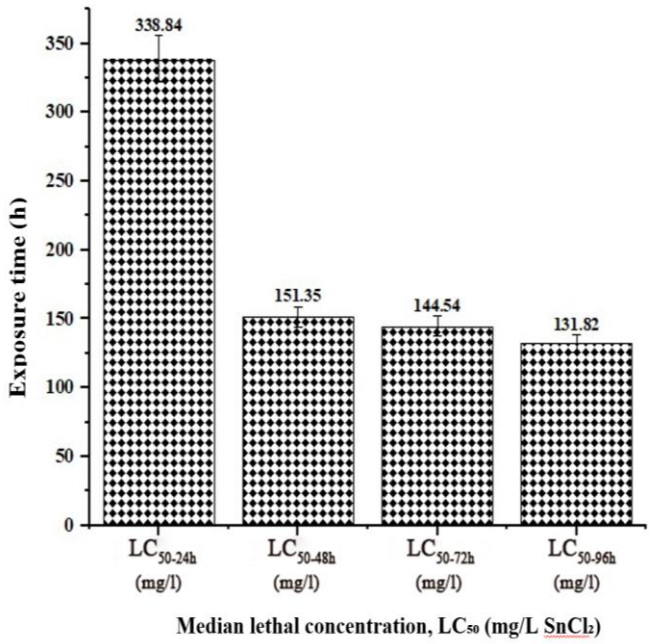

Fig. 1 The lethal concentration (LC_{50}) of stannous chloride ($SnCl_2$) for common carp (*C. carpio*)

Effects of $SnCl_2$ on hematological parameters

Changes in hematological parameters of carp exposed to sublethal concentrations of $SnCl_2$ are shown in Fig. 2. According to the results, the number of RBCs ($10^6/mm^3$) in the B (1.18 ± 0.10), C (1.03 ± 0.12) and D (0.88 ± 0.07) groups has decreased compared to the control group (1.27 ± 017). The findings show that increasing the concentration of $SnCl_2$ decreases the number of RBCs. This reduction was statistically significant in groups C and D compared to the control group ($p < 0.05$).

Mean WBC counts increased from 112.08 ± 17.93 × $10^3/mm^3$ in the control to 118.21 ± 16.59, 124.83 ± 11.23, and 127.19 ± 15.26 × $10^3/mm^3$ in groups B, C, and D, respectively (Fig. 2). The decrease in the number of WBCs in the C and D groups is significant ($p < 0.05$) compared to the control group. The results show that there is a direct relationship between the concentration of $SnCl_2$ and the number of WBCs in such a way that with the increase in the concentration of $SnCl_2$, the number of WBCs also increases.

The level of HCT (%) and HGB (mg/dL) decreased in all studied groups (B, C and D) compared to the control group. The results show that with increasing $SnCl_2$ concentration, HCT and HGB levels decrease which. is significant ($p < 0.05$) in group D compared to the control group (Fig. 2).

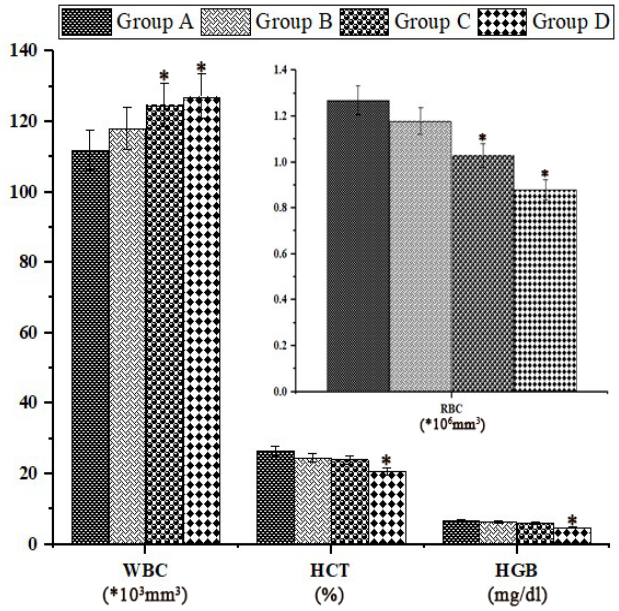

Fig. 2 Changes in RBC count, WBC count, HGB, and HCT levels in the blood of common carp (C. carpio) exposed to the control group, 10% (B), 25% (C), and 50% (D) of the LC_{50}-96h of SnCl2 after 28 days. (*Indicates a significant difference ($p < 0.05$) compared to the control group).

Effects of $SnCl_2$ on gill tissue

Gill lesions included shortening of secondary lamellae, curvature, oedema, and necrosis (Fig. 3; Table 1). Lamellar shortening was scored as severe in groups B and D and moderate in group C. Curvature was moderate in groups C and D, whereas mild oedema and moderate necrosis were recorded only in group D. No listed lesions were recorded in the control. The lesion-specific scores therefore did not show a consistently increasing pattern across exposure concentrations.

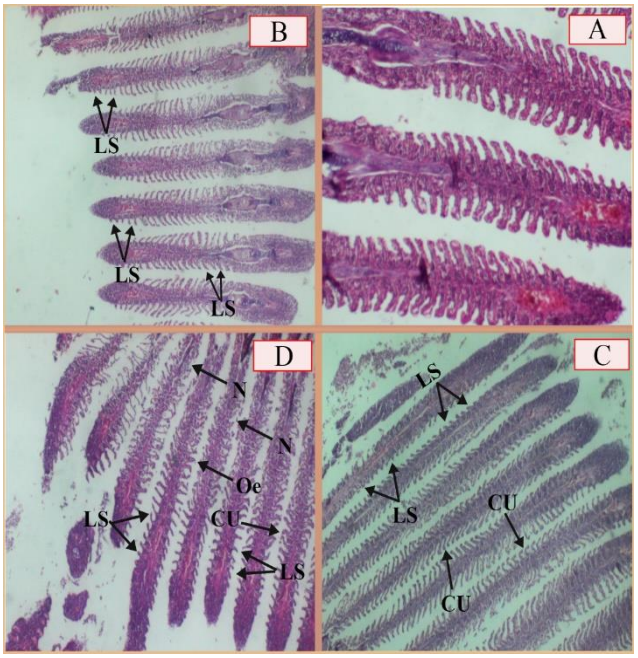

Fig. 3 Histopathological damages of gill tissue of common carp (*C. carpio*) exposed to the control group (A), 10% (B), 25% (C), and 50% (D) of the LC_{50}-96h of $SnCl_2$ after 28 days. CU: Curvature, N: Necrosis, Oe: Oedema and LS: Shortening of secondary lamellae.

Table 1 Histopathological damages of gill tissue of common carp (*C. carpio*) in all groups*

Gill Tissue	Histopathological Damages			
	Oe	CU	LS	N
Control (A)	-	-	-	-
10% (B)	-	-	+++	-
25% (C)	-	++	++	-
50% (D)	+	++	+++	++

*Exposed to the control group (A), 10% (B), 25% (C), and 50% (D) of the LC50-96h of $SnCl_2$ after 28 days. CU: Curvature, N: Necrosis, Oe: Oedema and LS: Shortening of secondary lamellae (-: No change, +: Mild, ++: Moderate, +++: Severe)

Effects of $SnCl_2$ on intestine tissue

Intestinal changes included increased blood cell presence (INBC), increased goblet cell numbers (INGC), goblet cell swelling (SG), and expansion of villous structures (EVS) (Fig. 4; Table 2). Goblet cell swelling was severe in all exposed groups. Increased goblet cell numbers were absent in group B, mild in group C, and severe in group D. Expansion of villous structures was mild in groups C and D, while increased blood cell presence was mild in groups B and D but absent in group C. No listed lesions were recorded in the control. These findings indicate variation among lesion types rather than a uniform concentration-dependent increase in severity.

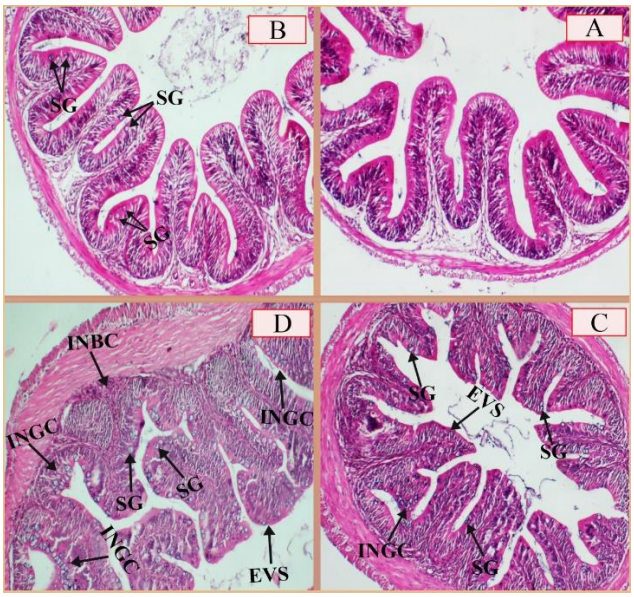

Fig. 4 Histopathological damages of intestine tissue of common carp (*C. carpio*) exposed to the control group (A), 10% (B), 25% (C), and 50% (D) of the LC50-96h of SnCl2 after 28 days. INGC: Increase in number of goblet cells, EVS: Expansion of villi structure, SG: Swelling of goblet cells, INBC: Increase in the number of blood cells.

Table 2 Histopathological damages of intestine tissue of common carp (*C. carpio*) in all groups*

Intestine Tissue	Histopathological Damages			
	INBC	INGC	SG	EVS
Control	-	-	-	-
10% (B)	+	-	+++	-
25% (C)	-	+	+++	+
50% (D)	+	+++	+++	+

*Exposed to the control group (A), 10% (B), 25% (C), and 50% (D) of the LC_{50}-96h of $SnCl_2$ after 28 days. INGC: Increase in number of goblet cells, EVS: Expansion of villi structure, SG: Swelling of goblet cells, INBC: Increase in the number of blood cells. (-: No change, +: Mild, ++: Moderate, +++: Severe)

Discussion

Acute toxicity of $SnCl_2$

The estimated 96-h LC_{50} of $SnCl_2$ in common carp was 131.82 mg $SnCl_2$/L. The decrease in LC_{50} with increasing exposure duration indicates that lower concentrations were sufficient to produce 50% mortality over longer observation periods. This finding emphasizes the importance of exposure duration when interpreting acute toxicity. However, the 96-h LC_{50} does not establish a safe exposure concentration for the separate 28-day experiment. In this study, the LC_{50}-96h of Sn for common carp weighing 100 ± 8 g was calculated to be 131.82 mg/L. Previous studies have examined inorganic tin toxicity in fish. As summarized by WHO, Taylor et al. (1985) reported a 96-h LC_{50} of 35 mg Sn/L in dab (*Limanda limanda*) exposed to tin(II) chloride [8], whereas Kapur and Yadav (1982) reported a 96-h EC_{50} of 295 mg Sn/L for hatching success in common carp [9]. These values should not be compared directly with our LC_{50} of 131.82 mg $SnCl_2$/L because the concentration basis differs, and hatching inhibition is distinct from mortality. Species, developmental stage, and exposure conditions also require consideration when interpreting these comparisons.

Based on the obtained results, the median lethal concentration decreases from 24 hours to 96 hours, but its acute toxicity increases. Various studies on determining the lethal range of heavy metals on fish prove that the LC_{50}-96h of heavy metals is different for different fish species. Because different fish species may differ in their absorption, accumulation, diffusion, metabolism, and excretion of chemicals [29]. For example, Sayadi and Kharkan, (2023) stated that the LC_{50}-96h for Cd, Cr, Co, and Cu metals in blackfish (*C. fusca*) is 37.14, 292.49, 106.95, and 10.73 mg/L, respectively [29]. They reported that as time increased from 24 hours to 96 hours, the median lethal concentration decreased, but acute toxicity increased because lower concentrations of heavy metals caused fish mortality. Mansouri et al. reported that the LC_{50}-96h of Hg and Ag metals for blackfish (*C. fusca*) weighing 2.95 ±0.55 was 0.24 and 0.013 mg/L, respectively [30]. Sayadi et al. stated that the median lethal concentration of 96 hours of ferric nitrate, ferric chloride, and ferric sulfate in blackfish is 1.6, 3.3, and 5.2 mg/L, respectively, and these results indicate that ferric nitrate is more toxic to blackfish than its chloride and sulfate [31]. Roy et al. reported that the 96-hour lethal concentration of cadmium chloride for the fish *Trichogaster (Colisa) fasciata* was 49.5 mg/L [32]. Al-Kshab and Yehya, reported that the 24-, 48-, 72-, and 96-hour lethal concentrations of lead chloride in *Gambusia affinis* were 59.4, 55.9, 51.1, and 49.0 mg/L, respectively. They stated that LC50 decreases as time increases from 24 to 96 hours [33]. Ghosh and Saha, reported that the 24, 48, 72, and 96-hour lethal concentrations of Cr for *Nile tilapia* were 121.06, 108.30, 99.31, and 93.49 mg/L, respectively. They also reported that fish mortality was directly proportional to the concentration of metals exposed [34]. Islam et al. stated that the 96-hour lethal concentration of chromium in striped catfish *Pangasianodon hypophthalmus* is 32.47 mg/L [35]. Ullah et al. stated that the LC_{50}-96h of copper sulfate for *Oreochromis niloticus* is 30 mg/L. They stated that as the metal concentration increased, the fish mortality response gradually increased [36].

The present findings complement previous $SnCl_2$ investigations in zebrafish. Şişman demonstrated developmental toxicity and genotoxic effects [21], whereas Barkhordari et al. examined $SnCl_2$ alongside organotin compounds in a zebrafish embryo model [22]. Our study extends this context by documenting hematological alterations and gill and intestinal lesions following 28-day exposure in common carp. Nevertheless, differences in species, developmental stage, exposure duration, and measured endpoints prevent direct comparisons of sensitivity. In particular, changes in blood cell counts do not establish genotoxicity, and the observed tissue lesions do not identify a molecular mechanism.

Effects of $SnCl_2$ on hematological parameters

Hematological studies are useful in diagnosing diseases and assessing damage to living organisms. Blood parameters serve as efficient indicators for monitoring health status, physiological changes, and metabolic disorders in fish. They also serve as biomarkers in the field of environmental toxicology, water quality, and stress-induced diseases in fish populations [37]. Changes in blood parameters in fish exposed to pollutants may vary and depend on the degree of toxicity, concentration and time of exposure, environmental conditions and intrinsic factors

such as fish species, age and size [38]. The concurrent reductions in RBC count, HCT, and HGB indicate an anemia-like response in $SnCl_2$-exposed common carp. These changes suggest a possible reduction in blood oxygen-carrying capacity, although oxygen transport was not measured directly. The available data cannot distinguish erythrocyte destruction from reduced erythrocyte production or changes in plasma volume. Because erythrocyte indices such as MCV and MCHC were not reported, the response cannot be classified as normocytic or assigned a specific morphological subtype. The onset of anemia symptoms may be due to destruction of RBCs or damage to hematopoietic tissues following exposure to the toxin [39].

The anemia caused is normocytic. Anemia may be caused by two reasons: hemolysis and decreased production of RBCs by the bone marrow [40]. Suganthi et al. stated that a decrease in HCT indicates a condition of "hydremia," which means there is too much fluid in the blood, leading to reduced oxygen delivery [41]. In similar studies, Sayadi and Kharkan, stated that Cd, Cr, Co, and Cu metals cause a decrease in the level of RBCs, HCT, and HGB in the blood of black fish [29]. This similarity provides comparative context but does not establish that the same mechanism operated during $SnCl_2$ exposure. The present measurements demonstrate hematological changes without identifying their cellular cause. The increased WBC counts indicate that the hematological response to $SnCl_2$ differed between erythrocyte-related parameters and leukocytes. Higher WBC counts may reflect stress-associated changes or inflammation accompanying tissue injury, but total counts alone cannot establish enhanced immune function, immunosuppression, or increased phagocytic activity. Oxidative injury could also contribute to the observed blood changes; however, this remains an untested explanation because ROS production, antioxidant activity, and oxidative damage biomarkers were not measured. The findings should therefore be interpreted as evidence of altered blood parameters rather than proof of a particular immune or oxidative mechanism.

Effects of $SnCl_2$ on gill histological changes

Fish are a very useful biomarker for detecting abnormal entry of pollutants into aquatic ecosystems. Histology is a very sensitive and important tool for determining changes in tissues or organs under stress. Examination of gill tissue changes is very important for assessing the effects of various toxins such as heavy metals. The direct contact of gills with exposure water makes branchial histopathology relevant to the assessment of waterborne pollutants [42]. Gills are organs with a large contact surface and related to the external environment. Gills are directly affected by changes such as pH and increasing or decreasing the amount of ions and pollutants in the water [43]. Gill responses to toxicant exposure may include deformation of the secondary lamellae and epithelial proliferation within the interlamellar spaces. Progressive narrowing of these spaces can result in lamellar fusion. Together with increased mucus secretion, such changes may restrict contact between pollutants and the epithelial surface [44].

In the present study, gill lesions comprised shortening and curvature of secondary lamellae, oedema, and necrosis. Oedema and necrosis were recorded only in group D, whereas lamellar shortening was severe in groups B and D and moderate in group C. Thus, group D displayed a broader range of lesions, but individual lesion scores did not increase consistently with concentration. These structural changes suggest possible functional consequences for the gills, although respiratory performance and ion regulation were not measured. In similar studies, Kharkan et al. stated that nickel oxide nanoparticles and its various salts cause damage such as shortening of secondary lamellae, curvature, and edema in the gill tissue of black fish [44]. Sayadi et al. stated that iron oxide nanoparticles and its salts cause curvature, fusion of secondary lamellae, and shortening of lamellae in the gill tissue of black fish [31]. Mansouri et al. reported that Hg and silver nanoparticles cause vacuolation of secondary blades, fusion, hypertrophy, mucus secretion, and necrosis in zebrafish gill tissue [45]. Mansouri et al. stated that sublethal effects of diazinon in exposure to TiO_2 nanoparticles cause histological changes such as hyperplasia, gill blade fusion, aneurysm, and necrosis in the gill tissue of common carp [46]. Javed et al. stated that iron and nickel metals in the gills of *Channa punctatus fish* cause damage such as necrosis, epithelial lifting, lamellar fusion, hyperplasia and syneching [47].

Kakakhel et al. stated that histological examination showed that exposure of common carp to silver nanoparticles resulted in changes in gill tissue. The observed histological changes included atrophy, shortening of secondary blades, degeneration, and necrosis at different concentrations of silver nanoparticles [48]. Noureen et al. stated that histological changes of copper nanoparticles in common carp gill tissue include necrosis of primary and secondary blades, edema, degeneration, and epithelial lifting [49]. All of the above studies show that toxic substances, especially heavy metals, can cause histological damage in the gills of various fish by causing oxidative stress. Studies involving other metal salts provide additional context for the gill alterations observed here. Naz et al. reported lamellar fusion, atrophy, disorganization, secondary lamellar curling, and necrosis of lamellar pillar cells in *Labeo rohita* exposed to copper sulfate [50]. These findings show that structural gill damage also occurs following exposure to metal salts. However, similarities in histological lesions do not establish equivalent toxicity or a shared mechanism between copper sulfate and $SnCl_2$.

Effects of $SnCl_2$ on intestine histological changes

Based on the obtained results, the histological damages observed in the intestinal tissue of common carp exposed to sublethal concentrations of SnCl2 include swelling of goblet cells, increase in the number of goblet cells, increase in the number of blood cells and expansion of the villi structure. Lesion severity varied among treatments, without a consistently increasing concentration–response pattern across all assessed changes. The intestine is one of the main metabolic organs of fish and makes it the most susceptible organ to the toxic effects of pollutants. Pollutants such as heavy metals can cause serious damage to intestinal tissue. Therefore, examination of intestinal tissue is very useful in toxicological studies [51]. The first defense mechanism of the intestinal tissue exposed to harmful substances is the increase in the number of goblet cells, which temporarily reduces the vulnerability of the intestine. Goblet cells act as a protective barrier and prevent the penetration of pathogens and the occurrence of inflammation. Recent studies have shown that these cells are not inactive structures, but play an active role in maintaining the local immunity of the intestine [52]. Expansion of the villi structure is one of the most common histological damages in intestinal tissue exposed to pollutants [53]. Past studies have shown that chemical compounds can cause various histological damages in fish intestinal tissue. Kharkan et al. reported an increase in the number of goblet cells, swelling of goblet cells, and expansion of the villi structure in an investigation of the effect of nickel oxide nanoparticles and nickel salts on blackfish (*C. fusca*) [44]. Abbaszadeh et al. also showed that exposure of blackfish (*C. fusca*) to a combination of PVC and paraquat resulted in the expansion of the villi structure, an increase in the number of goblet cells, and swelling of goblet cells, and the severity of these lesions was concentration-dependent [54].

In similar study, Mansouri et al. stated that the combination of copper oxide and titanium dioxide nanoparticles causes histopathological lesions such as degeneration, swelling of goblet cells, and necrosis-erosion in the intestinal tissue of common carp [55]. Naz et al. reported that Cd and Cu metals in the intestine of *Catla catla* fish caused atrophy of villi, sloughing of epithelial villi, and congestion were seen after 30 days of exposure [2]. Younis et al. stated that Cd increased the number of goblet cells in the intestinal tissue of Nile tilapia (*Oreochromis niloticus*) and reported that the increase in the number of goblet cells represents a defense mechanism against severe pathological changes [56]. Shi et al. stated that arsenic exposure causes histopathological changes including shortening of intestinal villi, thinning of the intestinal muscle layer, and infiltration of inflammatory cells in the intestinal tissue of common carp [57]. Necrosis, increased number of goblet cells, attachment and shortening of villi were reported in the intestinal tissue of fish (*Labeo rohita Hamilton*) exposed to different concentrations of cypermethrin [58].

Several limitations should be considered. Hematological and histopathological assessments involved five and three fish per treatment, respectively, limiting the characterization of biological variability. These fish-level sample sizes should be distinguished from the number of independent exposure tanks when interpreting treatment comparisons. The histological assessment used ordinal severity categories,

which do not quantify proportional changes in injury or establish statistically significant differences between treatments. Tissue tin concentrations and oxidative stress biomarkers were not measured, preventing conclusions about bioaccumulation or oxidative mechanisms. Furthermore, exposure concentrations were selected as fractions of an acute LC_{50} rather than from field measurements, limiting extrapolation to environmental exposure conditions.

Conclusion

Exposure to $SnCl_2$ for 28 days was associated with decreased RBC counts, HCT, and HGB and increased WBC counts in common carp. Gill lesions included shortening and curvature of secondary lamellae, oedema, and necrosis, while intestinal changes involved goblet cells, blood cell presence, and villous architecture. Histological severity varied among lesion types and did not show a uniformly increasing concentration–response pattern. These findings characterize hematological and tissue-level responses under the tested laboratory conditions without establishing their underlying molecular mechanisms. Longer-term studies, mechanistic investigations, and field validation are needed to assess the relevance of these responses under environmental exposure conditions.

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Authors' Contributions

Javad Kharkan: Investigation, Methodology, Formal analysis, Writing – original draft. Mohammad Hossein Sayadi: Methodology, Data curation, Conceptualization, Supervision, Writing – review & editing. Both authors reviewed and approved the final manuscript.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of interest

The authors declare that they have no financial or personal competing interests related to this study.

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Ethics approval and consent to participate

The experimental procedures involving fish were approved by University of Birjand. Consent to participate was not applicable because this study did not involve human participants.

Abbreviations

ANOVA, analysis of variance; EC_{50}, median effective concentration; HCT, hematocrit; HGB, hemoglobin; LC_{50}, median lethal concentration; OECD, Organisation for Economic Co-operation and Development; RBC, red blood cell; ROS, reactive oxygen species; SD, standard deviation; $SnCl_2$, tin(II) chloride; WBC, white blood cell.

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