Caspian Journal of Environmental Sciences
Online ISSN: 1735-3866
Print ISSN: 1735-3033

Determination of acute toxicity of sodium chloride and its effect on pathological lesions of gills, liver and kidney of fingerling kutum, *Rutilus kutum* (Kamensky, 1901)

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ABSTRACT

In this study, the tolerable concentration of sodium chloride in juvenile kutum, *Rutilus kutum* and its effect on the fish gill, liver and kidney were examined. So, LC_{50} of sodium chloride in 96 h in a standing environment on 200 juveniles of 1-3 g was determined to be 8.12 g L^{-1} using Probit analysis statistical method. The experiment was performed according to the OECD method with five treatments and one control (each with three replications). Toxicity test was performed in 30-L glass aquariums. Ten juvenile kutum were stocked in each aquarium. LC_{10}, LC_{50} and LC_{90} of sodium chloride for 96 h with logarithmically-determined concentrations (5, 6.46, 8.36, 10.81 and 14 g L^{-1}, respectively) were 3.37, 8.12, 19.52 g L^{-1}. During the experiment, water temperature (22 ± 0.7 °C), dissolved oxygen (7.5 ± 0.3 mg L^{-1}) and pH (7.52 ± 0.2) were measured daily. Histopathological examination on kutum juveniles in the gill by the increased sodium chloride concentration, included hyperemia, hyperplasia, the secondary lamellae adhesion, cell necrosis and lifting secondary lamellar epithelial layers. In addition, hyperemia and fat degeneration, cell necrosis, bleeding and hypertrophy were observed in the fish liver. Histopathologically, hemorrhage, cell necrosis, hemorrhage, melanoma macrophages and increased urinary space were observed in the fish kidneys. Behaviorally, group swimming and aggregating at the corners of aquariums and floors, and by elevating the sodium chloride concentration, impatience and suffocation of fish increased. In addition, by uprising the sodium chloride concentration, lethargy and mucus secretion on the surface of juvenile fish body increased.

Keywords: Kutum, Sodium chloride, Acute toxicity, Gills, Liver, Kidney.
Article type: Research Article.

INTRODUCTION

Kutum, *Rutilus kutum* (Kamensky, 1901) is of special importance among the bony fishes of the Caspian Sea, as it accounts for the highest annual fishing in the sea (Razavi Sayyad 1997). The very first attempts to artificially reproduce the Kutum were made in 1939, but a large annual project started from 1981 in more than 12 rivers in the east and west of Bandar Anzali, Iran to introduce millions of the kutum fry, weighing 1-2 g, to the sea every year (Shariati 2003). Therefore, researchers aim to increase the survival rate of fries after they are introduced to the sea. One of the ways to increase the survival rate of fry and juveniles is to study physiological indicators and environmental factors. Physicochemical factors of water can greatly affect the growth, survival, and metabolism of fish, as any deviation from the permissible limits of these factors can be a challenge for aquaculture (Chakraborty & Mirza 2007). Salinity is an important factor that can affect the growth and survival of fries and juveniles through osmoregulation. Since the osmotic pressure of body fluids at low salinities is almost equal to

Caspian Journal of Environmental Sciences, Vol. 20 No. 2 pp. 253-264 Received: July 08, 2021 Revised: Sep. 25, 2021 Accepted: Dec. 13, 2021
DOI: 10.22124/CJES.2022.5554

Publisher: University of Guilan, Iran

that of the environment, fishes need to consume less energy for osmoregulation in such environments and, consequently, save more energy for their growth (Sadok *et al.* 2004). Therefore, the measurement and correct understanding of the water physicochemical parameters can help us to increase the efficiency of preservation, stock enhancement, and breeding and rearing of economically valuable fish species, including kutum. The mortality rate is not a good measure to determine the resistance and adaptation of a fish to physiological and nutritional bottlenecks, because death is the endpoint of an animal's endurance. Small physiological and nutritional bottlenecks can considerably affect the growth and reproduction of fishes (Amini & Oryan 2003). Therefore, physicochemical parameters of water should be maintained in the optimum range for fishes as much as possible to increase their growth and survival. Salinity is also one of the environmental factors that affect the physiology, growth efficiency, and food absorption in fish species (Boeuf & Payan 2001). Sodium chloride is the most common disinfectant used in aquaculture, as it is sometimes known as aquaculture aspirin (Swann & Fitzgerald 1993). Sodium chloride (NaCl) is used in aquaculture to enhance osmoregulation and to control protozoan parasites, fungal infections such as Saprolegnia, Columnaris, Ichthyophthirius, viral infections, and bacterial infections (Schnick *et al.* 1998). Salt is an inexpensive and almost harmless substance for fish (Noga 2000; Robert 2001) that can improve diseases of aquatic organisms by increasing the production of mucus and dehydrating the pathogens. It is also effective in maintaining osmotic balance by reducing the release of ions into water and thus reducing the stress level (Carneiro & Urbinati 2001). Salt also intervenes in the competition between Cl^- and NO_{2-} to prevent and reduce the toxic effects of nitrite (Atwood *et al*. 2001). Enayat Gholampour *et al.* (2011) studied the effects of different salinity levels on growth indices, survival rate, foot intake, and blood parameters of the Kutum. Jeged (2007) reported that the increased salinity level damaged the gills and kidneys of the Nile tilapia. Fathollahi *et al.* (2021) showed that the LC_{50} of sodium chloride to sturgeons was equal to 7.67 g L^{-1}. Histopathological changes caused by the increased sodium chloride concentration include hyperemia, secondary lamella elongation, and secondary lamellae adhesion (fusion) in gill tissue and mucus secretion. The behavioral changes caused by low or high sodium chloride concentrations are abnormal swimming, imbalance, lordosis or scoliosis, lethargy, and high density of individuals on the sides and bottom of the aquarium. The study conducted by Boeck *et al.* (1996) on carp fries showed that they tolerated a salinity range of 0 to 1% for several weeks but all of them died under the salinity level of 1.1%. By contrast, the results of studies conducted on *Oreochromis mossambicus* by Jamil *et al.* (2004) showed that the fish easily tolerated and recorded no mortality under the salinity levels of 5, 10, 15, and 20 g L^{-1}. Vosyliene (2006) reported that the LC_{50} of sodium chloride to rainbow trout, with a mean weight of 12-14.4 g, was 20.38 g L^{-1}. Yohana & Pabloe (2007) conducted a study on juvenile and mature *Metynnis orinocensis* and found that 96 h of exposure of gills to salinity levels higher than 10 g L^{-1} caused lesions such as hyperplasia, lamella fusion, bleeding, and hyperemia. Also, Valente *et al.* (2021) studied the effects of salinity on juvenile platy, *Xiphophorus maculatus*; The other studies included those of Takata *et al.* (2021) on *Lophiosilurus alexandri*; Shirangi *et al.* (2016) on *Acipenser persicus* and Handayani *et al*. (2020) on *Oreochromis niloticus.* Considering the importance of the preservation and stock enhancement of the Kurume, this study aims to determine the acute toxicity of sodium chloride and its subsequent pathological lesions in gills, liver, and kidney of kutum fry.

MATERIALS AND METHODS

Experimental design:

This study was conducted in Ansari Bony Fishes Breeding and Stock Enhancement Center, Rasht, Guilan Province, Iran, in 2020. In this study, 200 kutum fry, with a mean weight of 1-3 g, were provided and kept in fiberglass tanks for further experiments. Feeding was stopped before the trial. According to OECD, the trial was conducted in 30-L glass aquariums with well water (TRC 1984). At first, the aquariums were thoroughly washed with potassium permanganate to keep the environment free of any contamination or disease. After several pilot studies, five treatments (with three replicates) were established including control (T_1), 4.46 (T_2), 8.36 (T_3), 10.81 (T_4), and 14 g L^{-1} (T_5) solar salt. The mortalities were recorded every 24, 48, 72, and 96 h and the dead fish were removed from the aquariums at the end of each day. Probit analysis was employed to determine LC_{10}, LC_{50}, and LC_{90} based on daily data. In addition, the 96-h probit diagram with concentration logarithm was drawn by calculating the regression line and correlation coefficient. Temperature, pH, and dissolved oxygen of aquariums were measured and recorded every day. The mean value of these three parameters throughout the trial was 22 ± 0.7 °C, 7.5, and 7.5 ± 0.3 mg L^{-1}, respectively. After the end of the first stage (96 h), 9 fish from each treatment

(3 from each replicate) were randomly selected and their gills, liver, and kidney were removed for histopathological studies, immersed in bouin fluid, and finally kept in McCarthy containers labeled with the tissue name, sodium chloride concentration, time, and date (Happaranta *et al*. 1997). The tissue samples were then transferred to the Histology Laboratory of the International Sturgeon Research Institute, Rasht, Iran. Then, 1 cm of the tissue sample was removed (Rojhan 1997) to prepare tissue slides through a process of fixation, dehydration, clarification, paraffinization, molding, microtoming, staining by hematoxylin and eosin (H & E), and mounting (Akahundov & Fedorovo 1995), followed by examining under a Nikon light microscope (E600) equipped with a monitor and a camera. Several tissue fields in each slide were studied and different parts were photographed at different magnifications.

Data analysis (LC_{50})

After recording the mortalities every 24, 48, 72, and 96 h, alterations were compared to the control group and the probit value was extracted from the ANOVA table. The logarithms of sodium chloride concentrations were used for probit analysis (Finney 1971). The regression equation (y = a + bx) was established by acquiring *a* (constant value) and *b* (line slope) coefficients. To solve the equation and determine LC_{10}, LC_{50}, and LC_{90}, probit value was extracted from the ANOVA table to replace *y* in the equation [(Probit Value = a + b (LogC)]. By solving the equation and calculating the anti-Log of Log C, LC_{10}, LC_{50}, and LC_{90} at 24, 48, 72, and 96 h were determined. All data were recorded in an Excel 2015 data sheet and statistically analyzed in SPSS 27, 2019.

RESULTS AND DISCUSSION

LC_{50} experiments

After several pilot studies on the kutum fry, with a mean weight of 1-3 g, the logarithmic method was employed to establish five treatments in the range between 5 and 14 g L^{-1} of sodium chloride. The treatments included the control (T_1), 4.46 g L^{-1} (T_2), 8.36 g L^{-1} (T_3), 10.81 g L^{-1} (T_4) and 14 g L^{-1} (T_5; Table 1). The LC_{10}, LC_{50}, and LC_{90} of sodium chloride were calculated every 24, 48, 72, and 96 h based on the regression line. The results showed that the 96-h LC_{50} of sodium chloride to the kutum fry was equal to 8.12 g L^{-1} and the maximum allowable concentration of this substance was 0.812 g L^{-1} (Table 2, Figs. 1 - 4).

Table 1. Comparison of the effect of different sodium chloride treatments on kutum juvenile mortality of 1-3 g during 96 h (Average of three repetitions).

Treatment	Concentration of Sodium Chloride (g L^{-1})	24h		48h		72h		96h		Changes compared to control				Concentration logarithm	Probit value			
		Death	Live	Death	Live	Death	Live	Death	Live	24h	48h	72h	96h		24h	48h	72h	96h
Control	0	0	10	0	10	0	10	0	10	0	0	0	0	0	0	0	0	0
I	5	0	10	1	9	1.6	8.7	3	7	0	-10	-16	-30	0.69	3.49	3.71	4.01	4.47
II	6.46	1.3	8.7	1.6	804	2.3	7.7	3.6	7.4	-13	-16	-23	-36	0.81	3.87	4.01	4.26	4.64
III	8.36	1.6	8.4	2.3	7.7	3.6	7.4	5.3	4.7	-16	-23	-36	-53	0.92	4.01	4.26	4.64	5.07
IV	10.81	2.3	7.7	3.6	7.4	4.3	5.7	7.6	2.4	-23	-36	-43	-76	1.03	4.26	4.64	4.82	5.70
V	14	2.6	7.4	4.3	5.7	6.6	7	9	1	-26	-43	-66	-90	1.14	4.36	4.82	5.41	6.28

Table 2. Lethal concentrations of sodium chloride over 4 days on kutum (Average of three repetitions) (g L^{-1})

Time	LC	24 h	48 h	72 h	96 h
	10	5.94	5.02	4.02	3.37
	50	28.33	21.19	13.57	8.12
	90	135.04	89.34	45.78	19.52

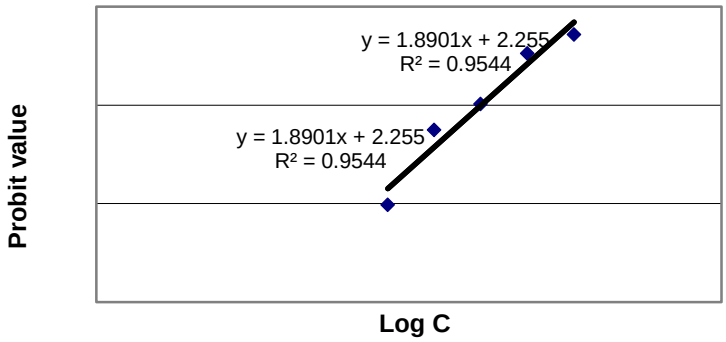

Fig. 1. Regression line equation and logarithm correlation coefficient of sodium chloride concentration and probit value (Average of three repetitions in 24 h).

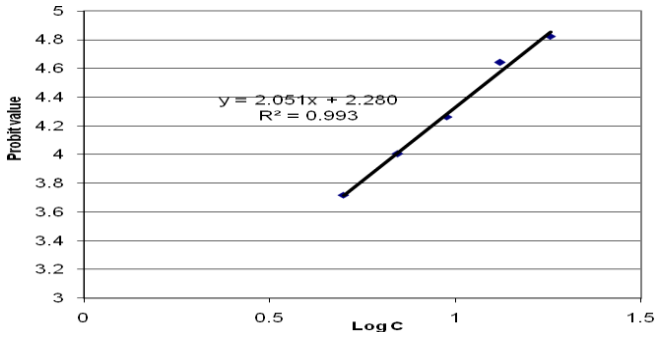

Fig. 2. Regression line equation and logarithm correlation coefficient of sodium chloride concentration and probit value (Average of three repetitions in 48 h).

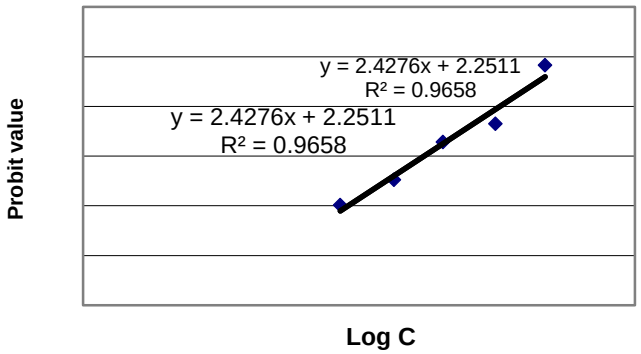

Fig. 3. Regression line equation and logarithm correlation coefficient of sodium chloride concentration and probit value (Average of three repetitions in 72 h).

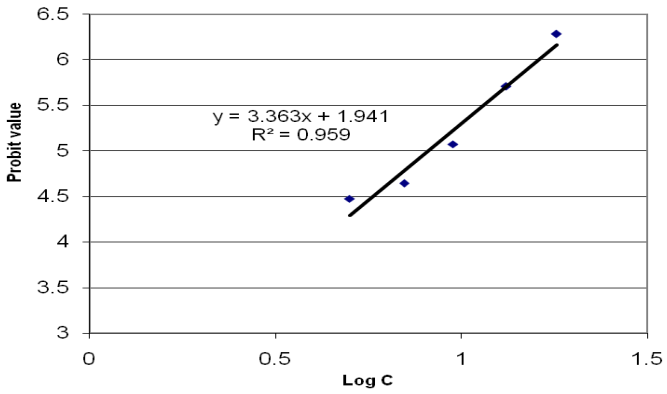

Fig. 4. Regression line equation and logarithm correlation coefficient of sodium chloride concentration.

Physical and clinical signs

The most prevalent clinical or behavioral signs of the kutum fry in exposure to sodium chloride were group swimming and high density of individuals on the sides and bottom of the aquarium. As the sodium chloride

concentration increased in treatments T_4 and T_5, the prevalence of restlessness, suffocation, lethargy, and body mucus increased among them. These symptoms caused the severe mortality of the fish in T_5 (14 g L^{-1}) after 72 h, such that no live fish remained in this treatment. Moreover, the mortality rate increased by the elevated sodium chloride concentration and reduced by the upraised weight of the fish, such that the highest mortality was observed among smaller (1 g) fish.

Pathological examination of gills

The results of histological evaluation of gills showed that exposure to sodium chloride caused histopathological lesions such as hyperemia, hyperplasia, secondary lamellae adhesion, cellular necrosis, and lifting epithelial layers of secondary lamellae. The most common lesions in T_1 (control) were hyperemia, hyperplasia, and secondary lamellar adhesion. The same lesions, but more severe, along with cellular necrosis were observed in T_2 (6.46 g L^{-1}). The severity of hyperemia, hyperplasia, secondary lamellar adhesion, and cellular necrosis increased in T_3 (8.36 g L^{-1}). The prevalence of hyperemia, hyperplasia, secondary lamellar adhesion, cellular necrosis, and lifting epithelial layers of secondary lamellae considerably increased in T_4 (10.81 g L^{-1}) and caused high mortalities. Finally, there was no live fish in T_5 (14 g L^{-1}) after 96 h of exposure to sodium chloride due to the high severity of tissue lesions. Therefore, it was not possible to take samples for histopathological studies (Figs. 5-9).

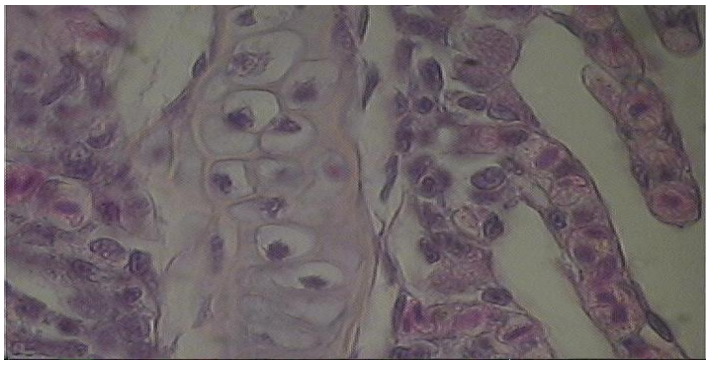

Fig. 5. Gill control group (H & E; 100 X).

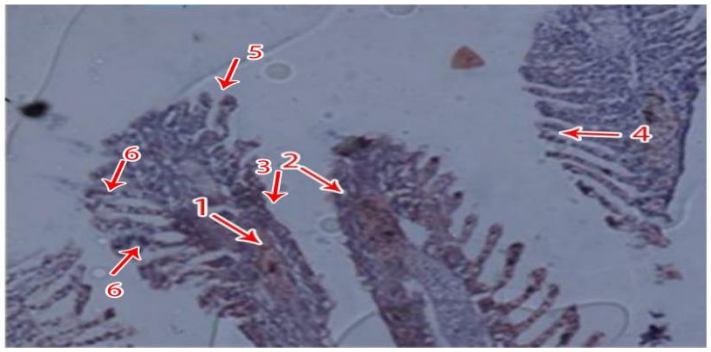

Fig. 6. Treatment 1 Gill: 1. Hyperemia, 2. Cellular necrosis, 3. Hyperplasia, 4. Adhesion of gill secondary filaments, 5. Curling secondary lamellae, 6. Detachment of epithelium layer in the secondary lamellae (H & E; 20 X).

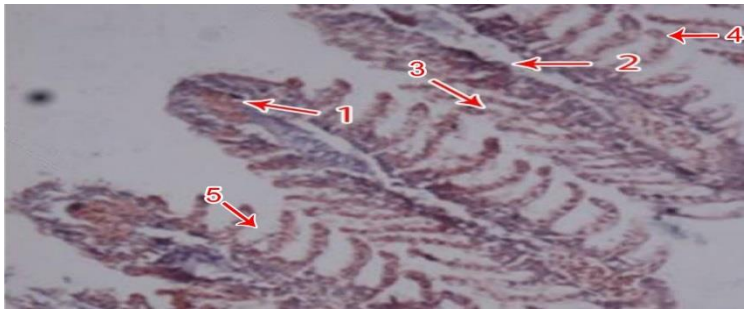

Fig. 7. Treatment 2 Gill: 1. Hyperemia; 2. Opening of the gill epithelial layer; 3. Adhesion of gill secondary lamellae; 4. Curling secondary lamella; 5. Detachment of epithelium layer in the secondary lamella (H & E; 20 X).

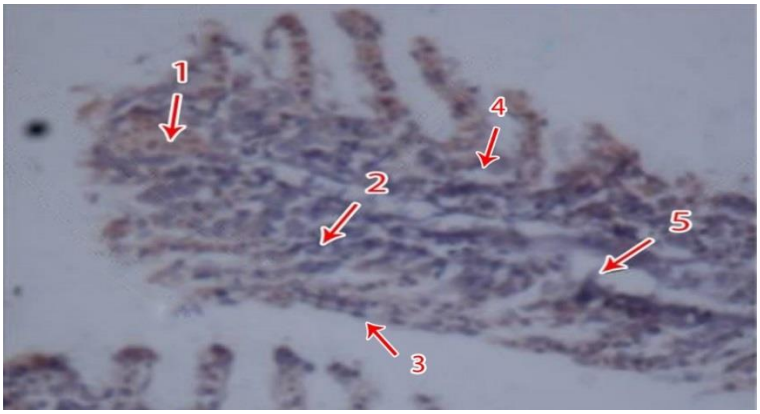

Fig. 8. Treatment 3 Gill: 1. Hyperemia; 2. Hyperplasia; 3. Adhesion of gill secondary lamellae; 4. Cellular necrosis; 5. Opening of the gill epithelial layer (H&E; 40X).

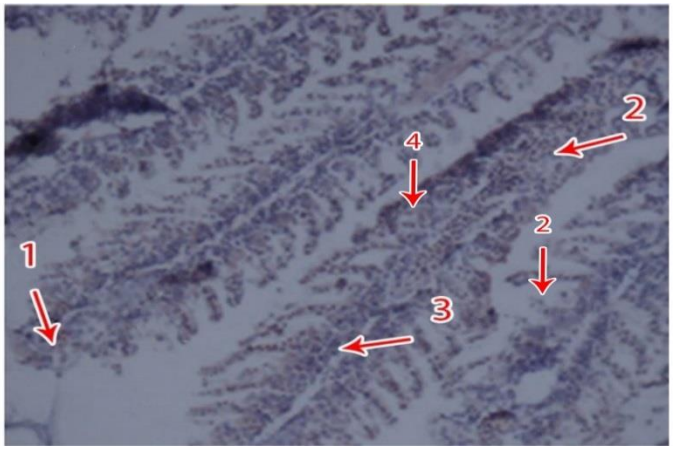

Fig. 9. Treatment 4 Gill: 1. Hyperemia; 2. Cellular necrosis; 3. Opening of the gill epithelial layer; 4. Adhesion of gill secondary lamellae (H & E; 20X).

Pathological evaluation of liver

The results of histological evaluation of the liver showed that exposure to sodium chloride caused histopathological lesions such as hyperemia, fatty degeneration, cellular necrosis, hemorrhage, and hypertrophy. The results indicated that the liver tissue of most fish in T_1 (control) was healthy, however, hyperemia was observed in a few of them. The most common lesions observed in T_2 (6.46 g L^{-1}) were hyperemia, fatty degeneration, and cellular necrosis. The histopathological lesions observed in T_3 (8.36 g L^{-1}) were the same, however more severe than those in T_2 and T_1. The most prevalent histopathological lesions of the liver in T_4 (10.81 g L^{-1}), i.e. cellular necrosis, hemorrhage, hypertrophy, and fatty degeneration, were more severe compared to the control (T_1) and T_3, and the liver was seriously damaged. There was no live fish in T_5 (14 g L^{-1}) after 96 h exposure to sodium chloride due to the high severity of tissue lesions. Therefore, it was not possible to take samples for histopathological studies (Figs. 10 -14).

Fig. 10. Liver control group (H & E; 40 X).

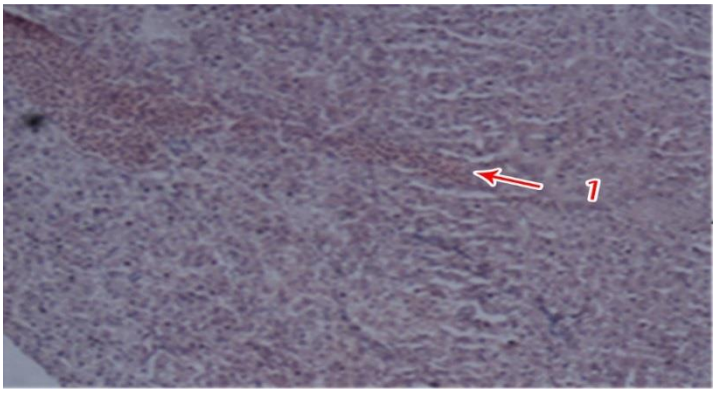

Fig. 11. Treatment 1 Liver: 1. Hyperemia (H&E; 20 X).

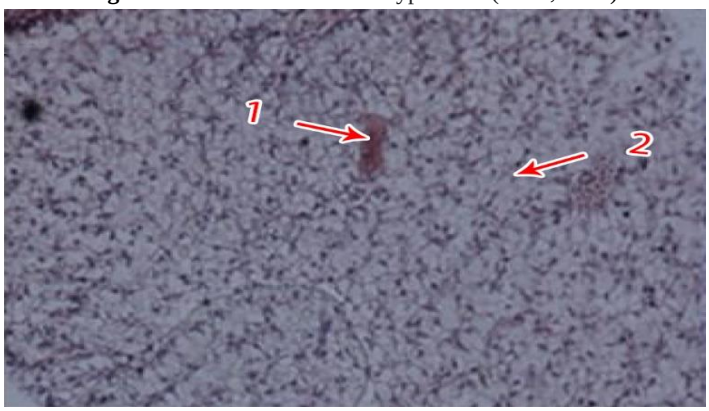

Fig. 12. Treatment 2 Liver: 1. Hyperemia, 2. hydropic degeneration (H & E; 20X).

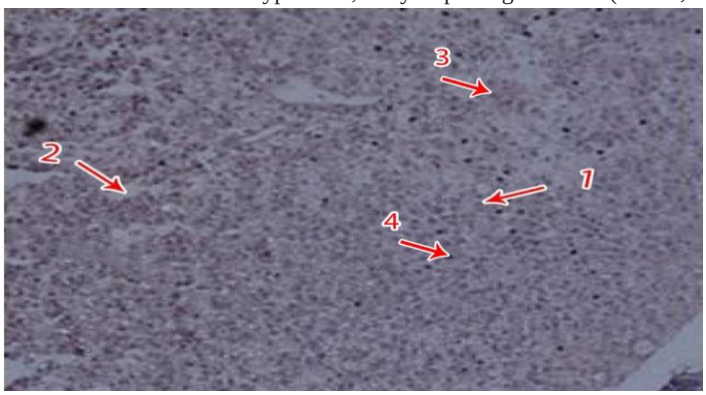

Fig. 13. Treatment 3 Liver: 1. Cellular necrosis, 2. Hydropic degeneration, 3. Bleeding, 4. Pyknosis (H & E; 20 X).

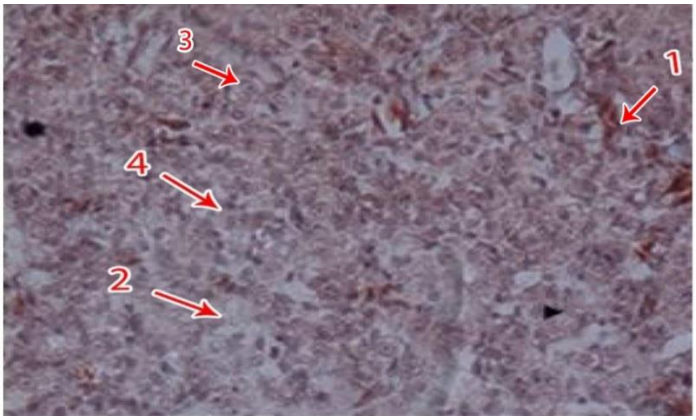

Fig. 14. Treatment 4 Liver: 1. Bleeding, 2. Cellular necrosis, 3. Cell swelling; 4. Hydropic degeneration (H & E; 40X)

Pathological evaluation of kidney

The results of histological evaluation of kidneys showed that exposure to sodium chloride caused histopathological lesions such as hyperemia, cellular necrosis, hemorrhage, melanoma-associated macrophages,

and increased urinary space. The results showed that the kidney tissue of most fish in T_1 (control) was healthy. The most prevalent lesions in T_2 (6.46 g L^{-1}) were cellular necrosis, increased urinary space, and a little hyperemia. The histopathological lesions observed in T_3 (8.36 g L^{-1}) were more severe compared to T_2 and T_1 and included cellular necrosis, hyperemia, melanoma-associated macrophages, hemorrhage, and increased urinary space (less prevalent). The more severe histopathological lesions, including cellular necrosis, hyperemia, melanoma-associated macrophages, hemorrhage, and increased urinary space, caused high mortalities in T_4 (10.81 g L^{-1}). There was no live fish in T_5 (14 g L^{-1}) after 96 h exposure to sodium chloride due to the high severity of tissue lesions. Therefore, it was not possible to take samples for histopathological studies (Figs. 15-19).

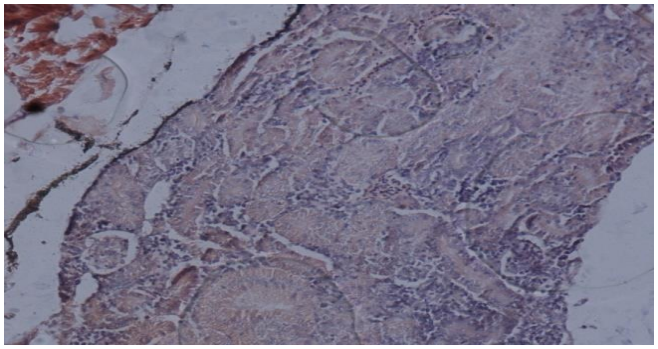

Fig. 15. Kidney control group (H & E; 20 X).

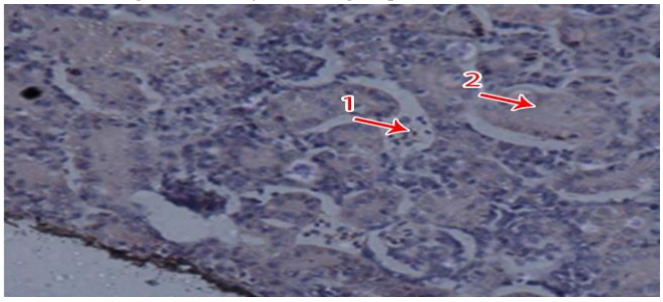

Fig. 16. Treatment 1 Kidney: 1. Hyperemia; 2. Occlusion of the tubular lumen (H & E; 20 X).

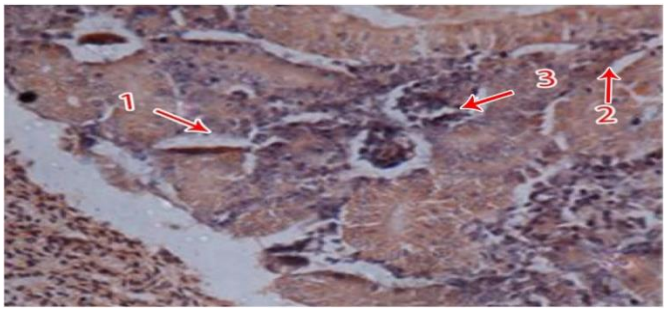

Fig. 17. Treatment 2 Kidney: 1: Increased urinary space; 2. Hyperemia; 3. Cellular necrosis in glomeruls (H & E; 40 X).

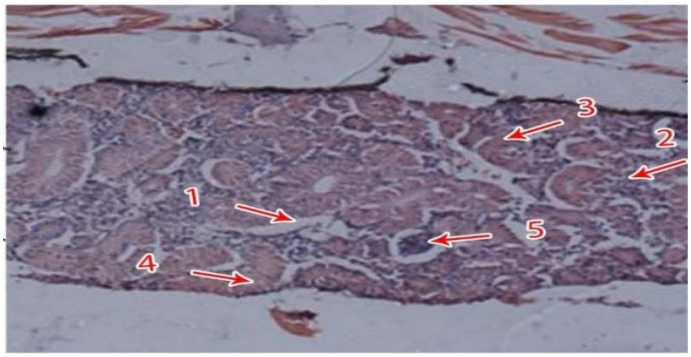

Fig. 18. Treatment 3 Kidney: 1: Increased urinary space; 2. Melanomacrophages; 3. Bleeding; 4. Hyperemia; 5. Dilation of bowmans space (H & E; 40 X).

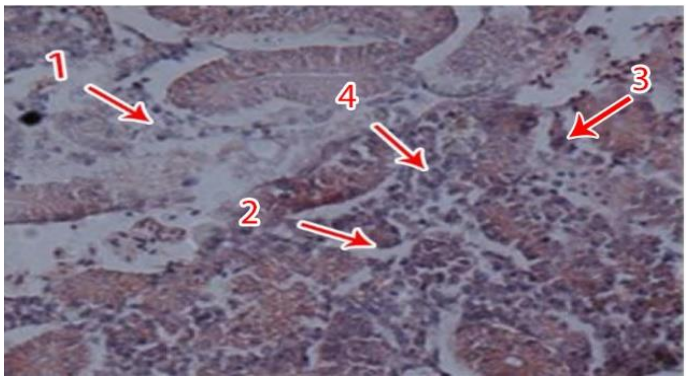

Fig. 19. Treatment 4 Kidney: 1. Cellular necrosis; 2: Increased urinary space; 3. Bleeding; 4. Melanomacrophages (H & E; 40 X).

DISCUSSION

The study results showed that the 96-h LC_{50} of sodium chloride in the kutum fry was equal to 8.12 g L^{-1}. The mortality rate also significantly increased by the elevated sodium chloride concentration. Previous studies have shown that the fish species whose physiological alterations affecting osmoregulation are consistent with the above-mentioned pattern of changes, can withstand the increasing salinity and survive in the new environment. However, if the physiological capacity is limited or once a sharp increase in salinity, fish species cannot adapt to the new conditions using the above-mentioned pattern of physiological changes and may die (Altinok *et al.* 1998). This can justify the very negligible mortalities in T_1 and T_2 and high mortalities in T_5. Studies have shown that the mortality rate of fish species elevates by the upraised salinity and reduces by the raise in their weight. This has been reported in bony fishes, such as the Chinook salmon (*Oncorhynchus tshawytscha*; Wagner *et al.* 1969) and the flathead grey mullet (*Mugil cephalus*; Nordlie *et al.* 1982), and chondrosteans such as the Persian sturgeon (*Acipenser persicus*; Kazemi *et al.* 2005), two tropical fishes, *Oreochromisaureus* and *O. niloticus* (Avella *et al.* 1993) and fringebarbel sturgeon (*Acipenser nudiventris*; Farabi *et al.* 2007). There are also several reports about the LC_{50} of sodium chloride in different fish species. For example, the LC_{50} of sodium chloride has been reported to be 11.33 g L^{-1} in Nile tilapia (Jeged 2007); 20.38 g L^{-1} in rainbow trout (Vosyliene 2006); 7.67 g L^{-1} in Persian sturgeon (Fatollahi *et al*., 2010); and 7.6 g L^{-1} in bighead carp (Garcia *et al.* 1999). In a study conducted by Nafisi Bahbadi *et al*. (2011) on rainbow trout juveniles (with an initial weight of 50.29 ± 4.61 g) at different salinities (0, 10, 20, 30, and 40 g L^{-1}), it was found that the growth performnace and survival rate reduced by elevating the salinity up to 20 g L^{-1} and mass mortalities were observed in salinities higher than 20 g L^{-1}. Jamil *et al.* (2004) reported that the Mozambique tilapia, *Oreochromis mossambicus*, was easily tolerated to the salinities of 5, 10, 15, and 20 g L^{-1} with no mortality. Histological examination is a comprehensive parameter that fully determines the health status of fish (Van der Oost *et al.* 2003). The study of behavioral, swimming, and respiratory patterns as well as internal and external reactions of the body is a good way to assess responses to environmental stresses (Kane and Salierno 2005), whereas histopathological examination is a suitable tool for determining morphological changes (Gernhofer *et al*. 2000; Takata *et al.* 2021). Since the respiratory system (gills) are the most vulnerable tissues of fish, hence this tissue was first sampled for histopathological studies. Gills are constantly affected or damaged by various dissolved or suspended irritants and stimuli in water and are a good tissues for exhibiting lesions due to their intimate contact with ambient water and their rich vascularization. In this study, the most prevalent tissue lesions in gill were found in T_3 (8.36 g L^{-1}) and T_4 (10.81 g L^{-1}) including hyperemia, hyperplasia, secondary lamellae adhesion, cellular necrosis, and lifting epithelial layers of secondary lamellae. The severity of these lesions was significantly higher in T_4. This is consistent with the findings of Yohana & Pabloe (2007) who reported that the most prevalent tissue lesions on the gills of juveniles and adults *Orinocensis metynnis* exposed to salinities over 10 g L^{-1} for 96 h were hyperplasia, secondary lamellae adhesion, hemorrhage, and hyperemia. Fathollahi *et al.* (2021) also found that the increased sodium chloride concentration caused hyperemia, secondary lamella elongation, and secondary lamellae adhesion in gills and increased body mucus in Persian sturgeon. They also reported that the severity of lesions elevated by the upraised sodium chloride concentration, consistent with the findings of the present study. Damages to the gill tissue actually reduce its ability to absorb oxygen and excrete carbon dioxide, and severe lesions can even lead to the suffocation and death of fish. However, these changes are reversible if the lesions are less severe (Roberts 2001; Valente *et al.* 2021). This study showed that the exposure of the kutum fry to sodium chloride caused hyperemia, fatty degeneration, cellular necrosis, hemorrhage, and

hypertrophy in the liver tissue. These lesions were significantly more severe in T_3 (8.36 g L^{-1}) and T_4 (10.81 g L^{-1}) than in the other treatments. The liver is a very important body organ of fish because it is involved in metabolism and processes such as biological transfers. Since the liver is also highly sensitive to contaminants and vulnerable to lesions caused by chemicals, it is a suitable organ for studying the effect of environmental stimuli on animals. Therefore, alterations in the liver structure are good measures for assessing the health status of fish species (Heidari 2009; Shirangi *et al.* 2016; Handayani *et al.* 2020). Cloudy swelling, atrophy, necrosis, vacuolar degeneration, fatty degeneration, cholestasis, liver swelling, cirrhosis, hyperemia, tumor, adenoma, hepatoma, and cholangiomaniaoplasia are some examples of pathological liver lesions whose continuation can lead to serious liver injuries, disruption of important hepatic physiological mechanisms such as metabolism of protein, carbohydrate, and fat, production of plasma proteins, formation and secretion of bile, and detoxification, and finally affect the overall metabolism of aquatic organisms (Sattari 2002). The most prevalent tissue lesions in the kidney of the fish in this study were hyperemia, cellular necrosis, hemorrhage, melanoma-associated macrophages, and increased urinary space. These lesions were significantly more severe in T_3 (8.36 g L^{-1}) and T_4 (10.81 g L^{-1}) than in the other treatments. Given the importance of the kidney as an effective tissue in osmoregulation and based on the study results, it can be stated that the elevated concentration of sodium chloride can damage and disrupt the function of kidney, consistent with the findings of Jeged (2007), Valente *et al.* (2021) and Takata *et al.* (2021). Given the different aspects of physiological processes, water salinity is of special importance in the introduction of fish fry to the sea in terms of both osmoregulation and tissue lesions. Based on the study results about the LC_{50} of sodium chloride to the kutum fry (8.12 g L^{-1}) and tissues lesions caused by this substance, it is recommended to introduce this fish species fry to the sea at a salinity under 5 g L^{-1}, i.e., LC_{10} of sodium chloride obtained in this study (3.37 g L^{-1}).

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Bibliographic information of this paper for citing:

Nazari, Y, Sadeghpour, A, Khara, H 2022, Determination of acute toxicity of sodium chloride and its effect on pathological lesions of gills, liver and kidney of fingerling kutum, *Rutilus kutum* (Kamensky, 1901). Caspian Journal of Environmental Sciences, 20: 253-264.