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Assessment of Gill Histology in the Nile Tilapia (*Oreochromis niloticus*) and Greenback Mullet (*Planiliza suviridis*) Exposed to Environmental Stressors in East Hammar Marsh and Shatt al-Basra Canal, Southern Iraq

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ABSTRACT

Gills are composed of specialized components such as primary and secondary lamellae, chloride cells, epithelial cells, mucous cells and venous sinuses, which play essential role for respiration and regulation of osmotic pressure. The results highlight significant histopathological changes induced by exposure to pollution, including capillary dilation, edema, necrosis and hyperplasia, indicating that fish are exposed to significant physiological stresses. These alterations in addition to affecting the efficiency of gas exchange also disrupt normal cellular functions, potentially leading to long-term health consequences for fish populations. Furthermore, the presence of inflammatory responses such as leukocyte infiltration and subepithelial edema underscores the impact of environmental toxins on aquatic organisms.

INTRODUCTION

Fish are widely used as indicators of aquatic ecosystem degradation based on the principle that fish abundance and diversity are strongly influenced by changes in biotic and abiotic factors within their habitats **(Fausch *et al.*, 1990)**. Pollutants entering fish organs cause damage to metabolic pathways **(Kondera *et al.*, 2013)**. Fish are commonly used to study the pathogenic potential of pollutants and cellular changes **(Strzyzewska *et al.*, 2016)**. According to their biological responses, fish are used to determine the health of aquatic ecosystems by serving as biomarkers of environmental pollution. Histological analysis provides a great biomarker for assessing toxicity levels due to its comprehensive assessment **(Moeller, 1985)** and the relationship between molecular and organismal levels **(Srivastava *et al.*, 1990)**.

Histopathological changes have been widely used as biomarkers in assessing the health of fish exposed to pollutants, both in laboratory and field studies. One of the significant advantages of using histopathological biomarkers in environmental monitoring is that they allow for the examination of specific target organs, including gills,

kidneys and liver, responsible for vital functions, such as respiration, excretion and accumulation of organisms **(Gernhofer *et al.*, 2001)**. Monitoring programs for measuring bioaccumulation serve as a biomarker for fish in polluted areas, providing information about environmental conditions. They are designed for different aspects of environmental risk assessment **(Van der Oost *et al.*, 2003)**.

Histopathological changes serve as warning signs of damage to fish health **(Hinton & Laurén, 1990)**. Therefore, they have been widely used as biomarkers in assessing the health of fish exposed to pollutants, both in laboratory **(Wester & Canton, 1991)** and field studies **(Hinton *et al.*, 1992; Schwaiger *et al.*, 1997; Teh *et al.*, 1997)**. Histopathological Changes in fish organs are usually easier to identify than functional changes **(Fanta *et al.*, 2003)**.

The gills are a critical organ for fish respiration, especially the secondary gill lamellae, which represent active structures for gas exchange due to contain respiratory cells and chloride cells **(Salman *et al.*, 1991; Carmona *et al.*, 2004)**. Several studies have investigated histopathological changes in different fish species such as North African garfish (C. *garieinus*), Arctic salmon (*Salvelinus alpinus*) and common carp (*Cyprinus carpio*), focusing on microscopic analysis of tissues from different affected organs such as gills, kidneys, liver and olfactory epithelium **(de Oliveira Ribeiro *et al.*, 2002; Al-Tamimi *et al.*, 2015; Alimba *et al.*, 2019)**.

The family Cichlidae (order Cichliformes) consists of 1786 species belonging to 254 genera **(Froese & Pauly, 2024)** distributed from South Africa to northern Syria. The Nile Tilapia has been introduced to many countries for aquaculture. These fish are now widely distributed in the water bodies of many tropical and subtropical countries, where they have been cultured **(Altun *et al.*, 2006)**. The Nile tilapia (Linnaeus, 1758) *Oreochromis niloticus* is endemic to Africa, but has been introduced to many parts of the world for aquaculture **(Emmanuel *et al.*, 1998)**. Moreover, the Nile tilapia was first introduced into fish ponds on the Tigris River near Baghdad, but did not survive the winter **(Herzog, 1969)**. However, **Al-Sa'adi *et al.* (2012)** reported the presence of the Nile tilapia in the Euphrates River in Al-Musayyib city, Babylon Governorate, since 2006.

The Mugilidae family (order Mugiliformes) consists of 78 species belonging to 26 genera **(Froese & Pauly, 2024)** distributed in tropical and temperate seas, mainly in coastal marine waters and some in fresh water. The greenback mullet *planiliza subviridis* is distributed in the Indian and Pacific Oceans, the Arabian Gulf, South Africa and the Red Sea, and this species enters the inland waters of Iraq.

MATERIALS AND METHODS

The East Hammar Marsh is distinct from other Iraqi marshes due to its tidal influence, which facilitates presence of marine fish species in addition to river species in it, resulting in increasing the biodiversity. The longitudinal column of the Shatt Al-Basrah Canal is 29km **(Hassan *et al.*, 2018)**. The main objective of this canal is to control

flood waves that cause river levels to rise and mitigate the impact of tides on areas west of Basrah and reduce its impact on adjacent lands.

Fish sampling

Samples of the Nile tilapia and greenback mullet fish were collected from the East Hammar Marsh and the Shatt Al-Basrah Canal during the winter (December 2023) and summer (May 2024) seasons. Fishing was done by using fishing boats and nets that are 9m in diameter with a mish size of 15 × 15mm. The collected fish were classified using the method of **Froese and Pauly (2024)**.

Laboratory procedures

Fish were dissected in the laboratory after anesthetizing directly killing. The parts designated were removed and marked. The samples were fixed by using Zenger's solution to fix the gills due to their hardness. Zenger's solution was prepared instantly by mixing 2.5g potassium dichromate with 5g mercuric chloride, 1g sodium sulfate, 5ml glacial acetic acid, and 100ml distilled water. After fixation, the samples were washed with distilled water for an hour to remove fixative residues. The microscopic slides were prepared according to the methods used in optical preparation techniques as outlined by **Humason (1967)** as follows: Dehydration, Clarification, Infiltration, Embedding, Sectioning, Staining, and Mounting.

RESULTS

Histological section of gill tissues of tilapia fish from South East Hammar Marsh (BH)

The histological section shows the gill structure of tilapia fish in a healthy state indicating good environmental conditions. The gills are the primary respiratory organ which exhibit an organized structural arrangement that facilitates efficient gas exchange and osmoregulation. Histological examination reveals that the primary lamellae (PL) are elongated and flattened which resulted in increasing the surface area for gas exchange. These primary lamellae act as the main site for oxygen intake and carbon dioxide releases. The secondary lamellae (SL) are small look like finger-like projections that assist in increasing the surface area for allowing effective gas diffusion.

The venous sinus (V) participates in blood circulation by pushing deoxygenated blood into the gill capillary network for gas exchange. Mucous cells (M) secrete mucus, which lubricates the gill surfaces to protect against pathogens and facilitates gas exchange. Chloride cells (C) regulate osmotic pressure through the active transport of ions for maintaining ionic balance in different fish environments. Epithelial cells (EC) form a membrane that regulates gas and ion exchange and provide protection to internal organs from environmental stressors. Histological analysis indicates a healthy gill structure for effective respiration and osmoregulation, which reflects optimal performance under ambient environmental conditions.

Hematoxylin and eosin (H&E) staining and 0.2µm scale provide a clear visualization of these structures.

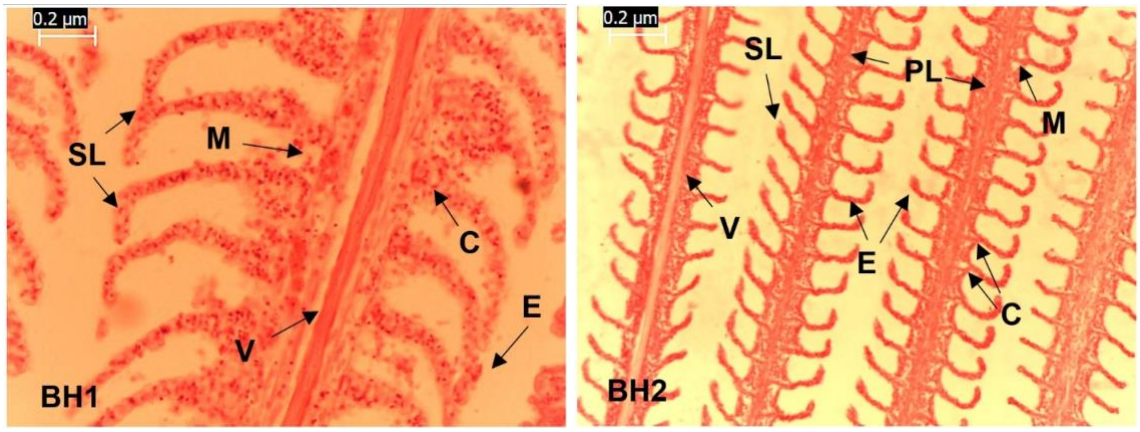

Fig. 1. A histological section of the gill tissues of tilapia fish inhabiting East Hammar marsh (BH). This section showcases normal gill architecture, comprising primary lamellae (PL), secondary lamellae (SL), mucous cells (M), chloride cells (C), epithelial cells (Ec), and venous sinus (V). The section was stained with hematoxylin and eosin (H&E) and viewed at a drawing scale of 0.2µm

Histological changes in gill tissues of tilapia from Shatt Al-Basrah canal. The histological section (BS1) of the gill tissues of tilapia fish from the Shatt Al-Basrah Canal (BS) reveals several histological changes. Large vacuoles (LV) within the epithelial cells indicate cellular edema which is a response to environmental stress caused by toxic substances in polluted water. The formation of vacuoles disrupts ionic regulation and osmotic regulation which affectes the cells' ability to maintain fluid balance. Basophilic inclusions (BI) appear as dense dark-colored basal structures within the cytoplasm that representi accumulations of waste materials and pollutants or dead cellular components. These inclusions are an early sign of necrosis or degenerative processes. The secondary lamellae show apical fusion (AF) which reduce the surface area available for gas exchange and compromise the fish's ability to breathe. Usully, these histological changes result from chronic exposure to pollutants whch lead to hypoxia (respiratory distress) or suffocation. The histopathological changes observed in the gill tissues such as vacuole formation, basophilic inclusions and apical fusion of lamellae reflect the negative impact of poor environmental conditions on the respiratory system in tilapia. These changes are stress responses to pollutants such as heavy metals and industrial waste or other pollutants.

Congestion is observed in the histological section (BS2) at the apices of the gill filaments (C) which indicate excessive blood accumulation within the capillary network. This congestion results from poor blood flow or inflammation due to pollutants or external environmental factors. Edema in the gills (E) is visible with significant swelling due to fluid accumulation within the gill tissue. This condition indicates damage to the epithelial cells and capillary membranes due to exposure to abnormal environmental factors. An increase in white blood cells (leucocytes) within the gill tissue {leucocyte infiltration (LI)} is observed especially in areas surrounding edema and swollen areas.

This infiltration represents an immune response to combat potential pathogens or repair damage.

The gill tissue section (BS3) shows marked capillary dilation (CD) within the gill filaments that indicates abnormal dilation of blood vessels in response to long time exposure to environmental toxins. Hyperplasia of epithelial cells (HE) is observed which results in a thickening of the epithelial layer covering the gills. These two major histological findings (capillary dilation and epithelial cell hyperplasia) highlight the physiological stress suffered by tilapia in the Shatt Al-Basrah canal.

The histological section (BS4) shows several severe histopathological changes that indicate extensive damage to the gill tissues of tilapia fish. Subepithelial edema (SR) is characterized by fluid accumulation between the epithelial layer and the underlying lamellar structures of the gills. Necrotic areas (N) are observed and characterized by the death of cells and tissues in many areas of the gills. These pathological changes reflect the body attempt to respond to unfavorable environmental conditions which ultimately reduce the fish's ability to absorb oxygen and release carbon dioxide efficiently.

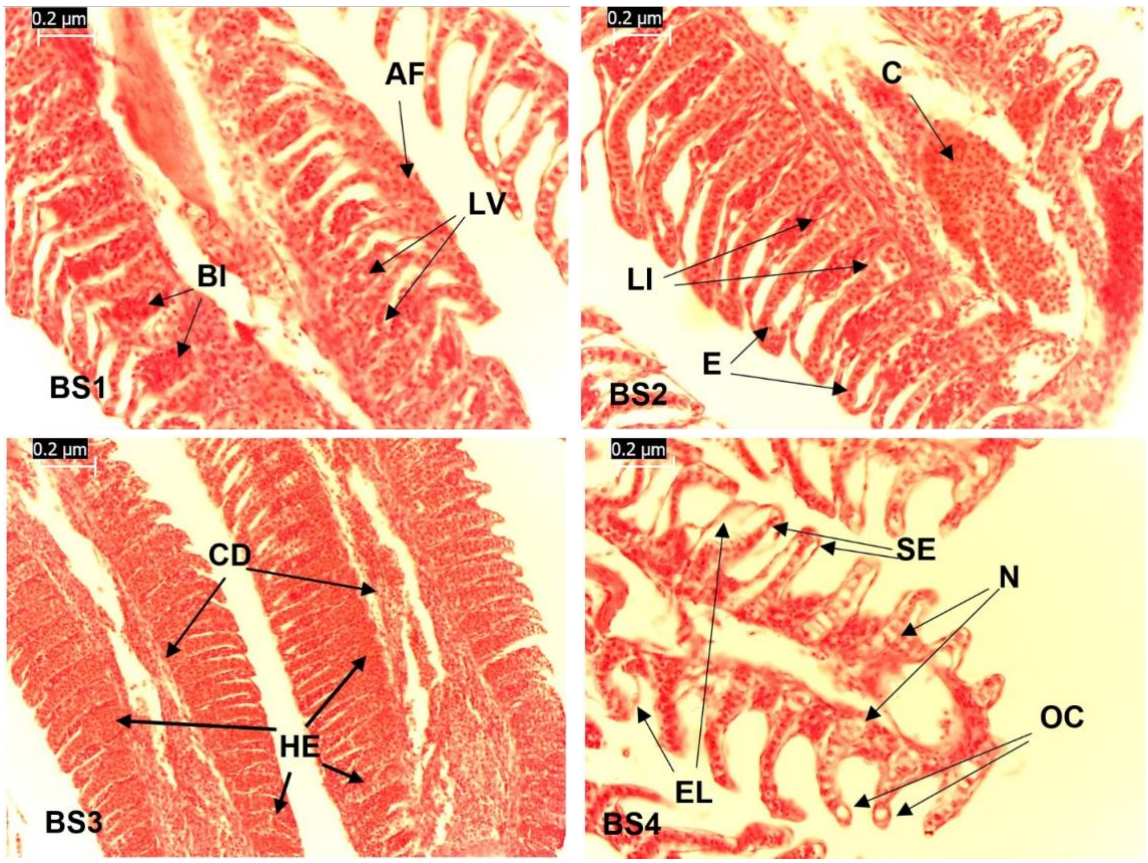

Fig. 2. Histological sections of gill tissues of tilapia fish living in the Shatt Al-Basrah Canal (BS) (H&E, scale: 0.2µm). Large vacuoles (LV), basophilic inclusions (BI), and apical fusion of lamellae (AF) are shown in BS1. Congestion in the tips of the gill filaments (C), edema in the gills (E), and leukocyte infiltration (LI) are observed in BS3. Capillary dilation (CD) and hyperplasia of epithelial cells (HE) are also observed in BS3.

In BS4, subepithelial edema (SR), necrotic areas (N), epithelial lifting of lamellae (EL), and edematous changes (OC) are observed

The histological examination of greenback mullet fish gills shows several components that are important for respiration and osmotic pressure regulation (Fig. 3). The primary lamellae (PL) are the main structural units which consist of long and thin filaments that provide a great surface area for gas exchange. The secondary lamellae (SL) are thin plate-like attached to gill filaments which increase the surface area for effective gas exchange. Chloride cells (C) are specialized epithelial cells serving as osmotic pressure regulation. They maintain ion balance by transporting ions specifically sodium chloride across the epithelial tissue.

The epithelial tissue consist of squamous epithelial cells (Ec) that provide a barrier that assist in gas exchange and protect internal tissues from pathogens and environmental stresses. Mucous cells (M) secrete mucus to blocks harmful particles and reduces the friction for protecting the gills. The venous sinus (V) collects blood from the gills capillaries to ensure a functional gas exchange and oxygen delivery throughout the fish body. Blood cells (B) within the capillaries transport oxygen and carbon dioxide due to the dense network of blood vessels ensures oxygen saturation.

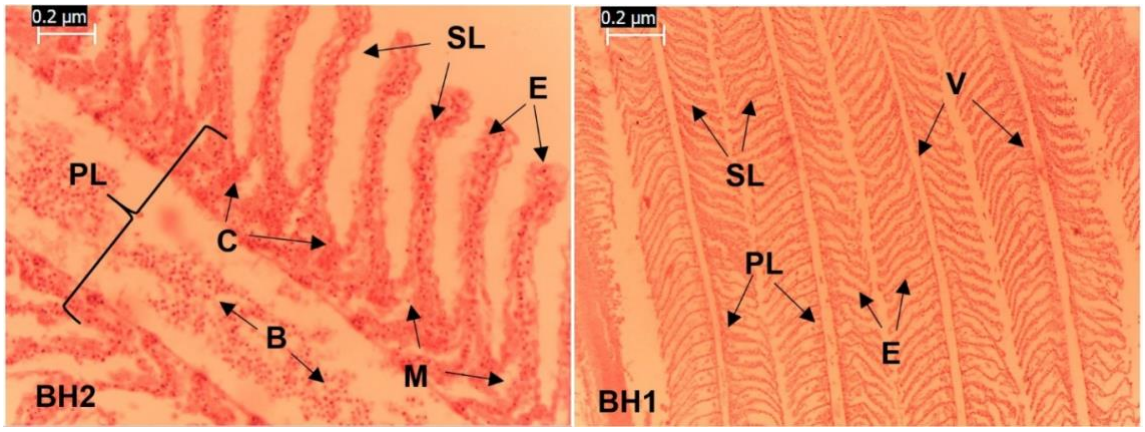

Fig. 3. Histological section of gill tissues of greenback mullet fish from East Hammar marsh (BH). This histological section shows normal gills with primary lamellae (PL), secondary lamellae (SL), chloride cells (C), epithelial cells (EC), mucous cells (M), venous sinus (V), and blood cells (B) (H&E, scale: 0.2µm).

Histological examination of (BS1) revealed capillary dilation (CD) which increases the blood vessel diameter within the gill tissue. This is often caused by inflammatory responses to environmental toxins which result in increasing blood flow, however it may cause a congestion and reduce oxygen transport. Gills have also been observed with edema (E) which is characterized by excessive accumulation of fluid in the interstitial spacing of gill tissues. This refers to increased capillary permeability caused by the

exposure to toxins which could lead to swelling and structural alterations that have an effect on gas exchange efficiency.

Fig. (2) shows oedematous changes (OC) which is a severe form of edema with greater fluid accumulation that affect gill lamellae structure. This results from continuous exposure to pollutants or unfavorable environmental factors leading to cellular dysfunction. Necrotic areas (N) in gill tissue indicate irreversible cell damage that is often caused by long time exposure to harmful substances. Dead cells appear shrunken or fragmented which indicate a significant physiological stress that may weaken the respiratory function.

Fig. (3) shows large vacuoles (LV) indicating cellular injury or metabolic disturbance. These vacuoles damage normal cellular processes and are associated with external environmental stresses. Basophilic inclusions (BI) are emblematic of aberrant protogenic activity or accumulation resulting from cellular stress or damage. They disturb normal cell functions and are treated as an indicator of disease state. Apical fusion (AF) of lamellae was observed. It occurs when adjacent gill lamellae adhere which causes a reducing in surface area available for gas exchange. Apical fusion is formed in response to environmental stresses significantly decrease respiratory efficiency.

Fig. (4) shows a congestion (C) at the tips of the gill filaments which indicate blood cell accumulation due to poor venous return or increased vascular permeability. Congestion can also be caused by low environmental oxygen levels that increases stress on the gill tissues. Leucocyte infiltration (LI) into gill tissue indicates activity for immune response to infection or injury which is essential for combating pathogens. Chronic inflammation can cause additional tissue damage. Hyperplasia of epithelial cells (HE) refers to the increased cellular proliferation seen as a protective adaptation to various environmental stresses. As a consequence, the thickness of the epithelial layers is compromised, causing impairment of the normal gill function. The subepithelial edema (SE) is the fluid accumulating beneath the epithelial layer indicating poor vascular integrity and increased permeability due to the presence of toxins. Additionally, it appears that the SE might also act in aggravation of other pathological conditions due to the altered communication of nutrient exchanges with other infections and diseases. The histopathological changes of gill cells from Greenback mullet fish that had suffered such environmental stresses shows the major impacts on their health. Such findings point to the need for monitoring pollution in aquatic ecosystems and its possible effects on populations of fish and biodiversity.

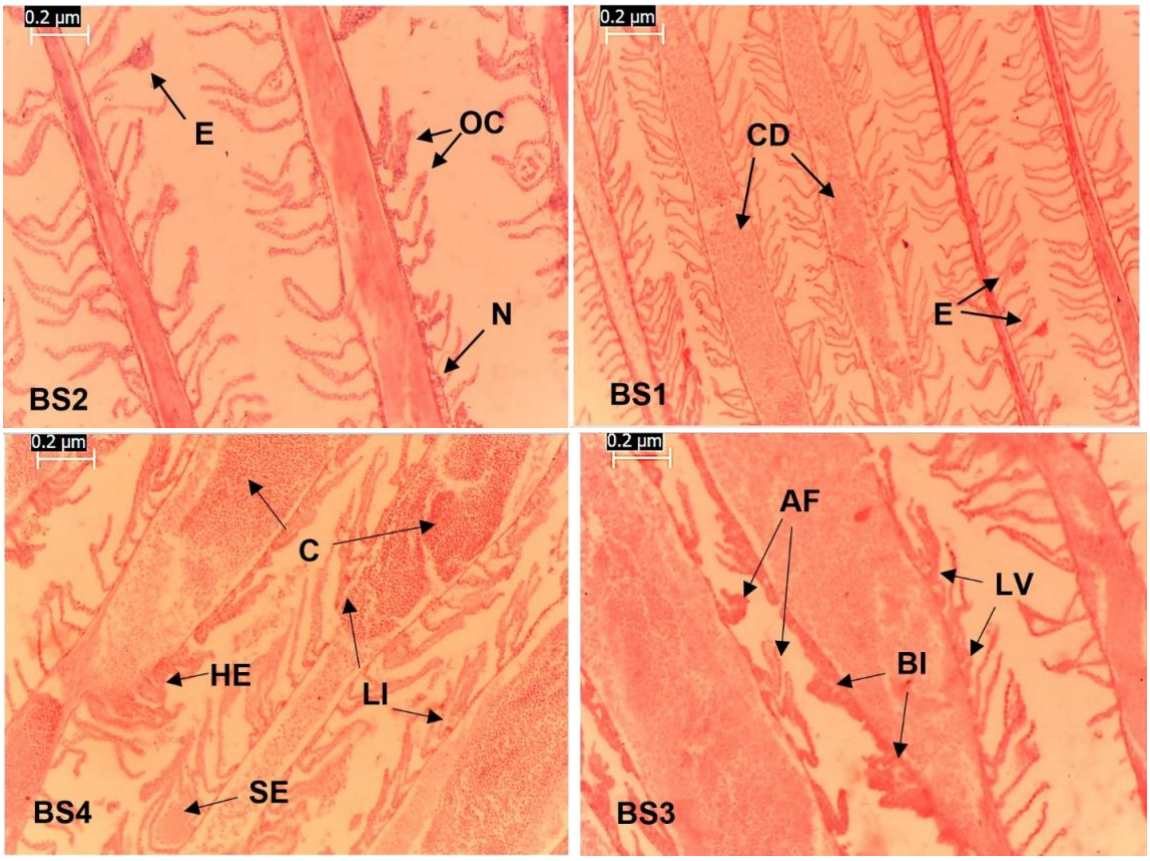

Fig. 4. Histological sections of gill tissues of greenback mullet fish from the Shatt Al-Basrah Canal (BS) (H&E, scale: 0.2 µm). In section BS1, capillary dilation (CD) and edema in the gills (E) are evident. Section BS2 shows oedematous changes (OC) and necrotic areas (N). Section BS3 reveals large vacuoles (LV), basophilic inclusions (BI), and apical fusion of lamellae (AF). Finally, section BS4 exhibits congestion at the tips of the gill filaments (C), leucocyte infiltration (LI), hyperplasia of epithelial cells (HE), and subepithelial edema (SE)

DISCUSSION

Gills are vital organs in fish. They are responsible for respiration, osmoregulation, and excretion of nitrogenous wastes **(Heath, 1987)**. Gills are susceptible to damage from unsuitable environmental conditions such as salts, heavy metals, pesticides, sewage, and fertilizers **(Temmink *et al.*, 1983)**. The gills' direct exposure to external environmental conditions makes them prone to disease **(Mallatt, 1985)**. They are considered the primary interface between fish and their environment, which makes them sensitive to changes in water quality and makes them a major target for pollutants **(Camargo & Martinez, 2007)**.

The gills of tilapia in the present study from the Shatt Al-Basrah canal exhibited large vacuoles within epithelial cells indicating cellular edema and environmental stress. Vacuole formation disrupts ionic regulation and osmoregulation that affect cellular fluid balance. This is consistent with previous findings **(Karlsson *et al.*, 1985)**. Increasing the

thickness of epithelium may lead to delayed blood flow and lamellar fusion **(Kantham & Richards, 1995; Figueiredo-Fernandes *et al*., 2007)**. Gill lamellae fusion and hypertrophy may occur due to toxin exposure, which causes altering glycoproteins in the mucous membrane of epithelial cells **(Ferguson, 1989)**. Epithelial hypertrophy and necrosis are often caused as a result of heavy metal exposure **(Mallatt, 1985)**. As well as various factors, that can cause similar structural changes, where a harem of environmental conditions or pollutants can induce distinct histopathological changes **(Haaparanta *et al*., 1997)**. Epithelial hypertrophy and elevation are considered defense mechanisms against harem environmental conditions **(Mallatt, 1985; Roberts & Rodger, 2012)**. Although these mechanisms can reduce respiratory surface area and increase dispersal distance. Gill tissue recovery is expected after stopping exposure to these conditions **(Bernet *et al*., 1999)**. Gills edema lamellae reduces the surface area available for gas exchange, which impairs fish respiration. Chronic exposure to pollutants can lead to hypoxia and respiratory distress, or even suffocation in fish **(Darmono, 1995)**. Damage of gill tissue structure impairs fish respiration and reduces oxygen content in the blood. This leads to difficulties in binding oxygen to blood due to damage to the secondary lamellae. There are three primary mechanisms by which toxic substances enter an organism, absorption, distribution, and accumulation in the respiratory system. Heavy metals can cause significant damage to organs associated with fish gills. It led to heavy metal poisoning, which can cause damage to gill tissues or even tissue death. The damage caused by pollutants to fish gills begins with edema, followed by hypertrophy, lamellar fusion, epithelial lifting, and then necrosis **(Rennika *et al*., 2013)**. These finds are consistent with the current study, which observed histopathological changes in gill tissues of tilapia fish from the Shatt Al-Basrah canal, where it indicates cavitation, cellular inclusions, and lamellar fusions. These changes reflect the negative impact of poor environmental conditions on health and competence of the respiratory system.

Congestion (characterized by a change in red color) indicates a change in blood oxygenation levels. The present study also observed a marked increase in epithelial hyperplasia of the epithelial cells lining the gill lamellae. Hyperplasia is a defensive response to chronic irritation or injury caused by pollutants or pathogens, aimed at protecting underlying tissues from further damage. This excessive growth compromises the primary functions of the gills, such as facilitating oxygen uptake and ion regulation, which led to stress in respiration. Hyperplasia results in thickening of the epithelial tissue at the tip of the filament, which appears as baseball bat-like shapes, or thickening of the epithelial tissue near the base of the lamella. Gill enlargement is a common sign of chronic toxicity caused by various chemical pollutants. It has been suggested that such hyperplastic reactions may increase the thickness of the epithelium to obstruct or prevent the toxic ions from entering into the bloodstream or to compensate for osmotic imbalance **(Laurent, 1984)**.

Necrotic areas, which are characterized by disorganized and fragmented tissue, indicate cell death due to severe stresses such as oxygen deficiency and toxic accumulation or direct cellular damage caused by environmental pollutants. The presence of necrotic areas indicates that parts of the gills have been severely damaged, which causes respiratory failure. The gill degeneration and necrosis reflect the direct effect of pollutants on fish health **(Garcia-Santos *et al*., 2007)**. These lesions may reduce the gills functional surface for gas exchange **(Mallatt, 1985)** to impairing respiratory function. The elevation of the respiratory epithelium is one of the common lesions found in fish, which is characterized by displacement of the lining epithelium of the secondary lamellae, where a space called edema occurs. It is linked to the presence of chemical pollutants that reduce the surface of the gills and compromise the gas exchange process. The hematoma causes proliferation of cells of the adjacent lamellae, which reduces the space between the lamellae **(Fracário *et al*., 2003)**.

This study observed chloride cells (specialized epithelial cells) located primarily on the secondary lamellae and played a crucial role in regulating osmotic pressure through active ion transport across the gill epithelium. Epithelial cells are lining the gill filaments, and secondary lamellae are typically cuboidal or columnar in shape. These cells form a membrane that regulates gas and ion exchange as well as providing protection to internal parts **(Peebuaa *et al*., 2006)**. Chloride cells are identified by their light cytoplasm, which are usually found at the base of the lamellae. Mucous cells are present at the base of the lamellae, which lack light cytoplasm and are smaller than chloride cells **(Bonga & Lock, 1991)**. Fish gills are highly susceptible to toxic chemicals from environmental pollutants due to direct contact. Absorption of toxic chemicals through the gills is enhanced by increasing the permeability of the gill epithelium and inhibiting the ion exchange activity of chloride cells **(Liao *et al*., 2006; Camargo & Martinez, 2007; Authman, 2008)**. **Avella *et al*. (1993)** observed a large number of chloride cells present in gill filaments in halophilic bony fishes transferred to salt water **(Assem & Hanke, 1983)**. Also, **Azab *et al*. (1999)** discussed the morphology and ultrastructure of gill epithelial cells of the halophytic cyprinids and their variation during adaptation to both hypersaline and freshwater. Since gills are in direct contact with polluted water, they are an important tissue for histological research. Some chemicals can rapidly damage gill lamellae after only a few hours of exposure **(Pariza *et al*., 2019)**. Gill tissues of fish exposed to mercury and cadmium showed lamellar disintegration, vacuolization, secondary lamellae fusion, and epithelial hypertrophy **(Selvanathan *et al*., 2013)**.

In contrast, the Nile tilapia gills from East Hammar Marsh show an organized structure that is reflected in the gas exchange process and osmotic pressure regulation. Histological examination reveals elongated and flattened primary lamellae that increase the surface area for gas exchange. The secondary lamellae are covered by a layer of epithelial cells that allow gases to diffuse efficiently.

The present study of the greenback mullet gill fish in the Shatt Al-Basrah channel revealed capillary dilation, which is characterized by an increase in the diameter of blood vessels within the gill tissue. This is often caused by environmental toxins that led to increased blood flow, congestion, and impaired oxygen transport. The study also showed the presence of gill edema that represent the accumulation of excess fluid in the interstitial spaces of the gill tissue. This can be attributed to increased capillary permeability due to exposure to toxins which cause swelling and disturbances in the normal structure of the gills which affect the efficiency of gas exchange. Edema, epithelial elevation, and lamellar fusion are defense mechanisms that reduce the branched surface area contacting to the external medium to increasing the diffusion barrier to pollutants **(Van Heerden *et al*., 2004; Pane *et al*., 2004)**.

Necrotic areas in the gill tissues were observed, which indicate irreversible cell damage resulting from long-term exposure to harmful substances. Dead cells appear shrunken or fragmented. Their presence indicates significant physiological stress, which may impair respiratory function. Furthermore, histopathological changes in the gills lead to hypoxia, respiratory failure, and problems with ionic and acid-base balance **(Alazemi *et al*., 1996; Rosidah *et al*., 2020)**. The histological section of greenback mullet gills in the East Hammar Marsh showed healthy gill filaments which are the main structural units of the gills. These filaments provide a large surface area for gas exchange that facilitates oxygen extraction from the water. The secondary lamellae are attached to the gill filaments, which are thin folds that increase the surface area even more and are supplied with dense blood vessels for efficient gas exchange. Chloride cells, which are specialized epithelial cells essential for osmotic pressure regulation, were observed. These cells help maintain ion balance through the active transport of ions, especially sodium and chloride, across the epithelial tissue of the gills. Their presence indicates adaptation of the fish to its aquatic environment **(Foskett *et al*., 1983)**. Chloride cells play an important role in ion transport in both freshwater and saltwater adapted bony fishes by absorbing ions and Ca2 +Na + CI **(Laurent & Dunel, 1980; Flike *et al*., 1984; Perry & Wood, 1985; Avella *et al*., 1987)**.

CONCLUSION

The current research compared the histological differences concerning gill tissues in tilapia and greenback mullet from two distinct aquatic ecosystems of southern Iraq, namely the East Hammar marsh and the Shatt Al-Basrah canal. The findings revealed that fish of East Hammar marsh had normal gill histology; this meant that they had really good environmental conditions. In contrast, fish from the Shatt Al-Basrah canal had several histopathological changes such as capillary dilations, edema, necrotic areas in the tissues, and hyperplasia of epithelial cells. These changes imply that the fish in the Shatt Al-Basrah canal are suffering from severe environmental stress due to pollution. This research emphasizes the importance of monitoring the water quality and urgently demands conservation activities that protect aquatic ecosystems and biodiversity.

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