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REVIEW

BIOACCUMULATION AND EFFECTS OF LEAD (Pb) ON METABOLIC PROCESSES AT CELLULAR AND TISSUE LEVEL OF TILAPIA (*Oreochromis niloticus*)

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ABSTRACT

Aquaculture environments have faced major problems due to the accumulation of various contaminants and pollutants due to various industrial activities. Lead (Pb) is one of the heavy metals that is very dangerous for the sustainability of the food chain. Early producers and consumers are the chains most vulnerable to being directly affected by heavy metal exposure in aquatic environments. Their large numbers in the ecological pyramid allow heavy metal accumulation to occur in higher consumers due to chain processes and food webs. Tilapia (*Oreochromis niloticus*) is an omnivore species of fish that feeds on various early producers and consumers in the ecological pyramid. When fish consume foods that contain toxic substances, the substances will accumulate and settle for a long time in fish cells and tissues. Pb can also enter the body of fish through gills, then will be carried by blood flow through arteries to all organs involved in fish blood circulation. In the end, Pb will be deposited in the liver of the fish for a long time. *O. niloticus* exposed to heavy metals in the environment is reported to cause damage to several vital organs of fish such as gills, kidneys, and liver. Organ failure will cause chronic pain, make it easier for pathogens to enter the tissues, weaken the body's immune system and death in fish. Exposure to Pb is reported to inhibit active transport processes, suppress oxidation-reduction reactions, and protein synthesis. Other reports suggest that Pb residues can cause hematological, neural, and gastrointestinal dysfunctions in fish. Exposure and accumulation of Pb in fish is in fact very dangerous for the survival of fish. A dangerous threat is food security for humans. We, humans who consume tilapia as a source of protein must consume food that is free from various exposures to harmful heavy metals that generally manifest as hazardous waste in industry. In the future, factories and industries must thoroughly manage industrial waste so that it is completely safe and free of various heavy metals before being discharged into the aquatic environment.

Keywords: Bioaccumulation, Food security, Lead, Metabolism, *Oreochrimus niloticus*, Toxic

INTRODUCTION

Because it is produced during the processing of ore, smelting, and refining processes, as well as during the production of cement, gasoline, batteries, and paint, as well as when coal is burned, lead (Pb) is a toxic metal that still poses a risk to aquatic ecosystems (**Adhim *et al*., 2017**). Because of their toxicity, bioaccumulation, and biomagnification through the food chain, Pb is one of the heavy metals whose presence in aquatic systems has had a significant negative impact on fish (**Ahmad *et al*., 2015**). Recent studies state that the presence of Pb can cause disruption of SOD (Superoxide Dismutase) function in zebrafish liver tissue (**Green & Planchart, 2018**).

Fish tissue histopathological changes can be used as biomarkers to identify heavy metal toxicity, such as Pb (**Udotong, 2015**). Fish liver tissue undergoes changes after exposure to Pb due to the presence of Melan macrophage and vacuole degeneration, which also affects fish cells (**Sirimongkolvorakul *et al*., 2012**). The mechanism of action of some xenobiotics, especially heavy metals, can initiate the formation of specific enzymes that cause changes in metabolism, thus causing cellular poisoning and death at the cellular level, namely necrosis, which can be used as a histopathological biomarker at the tissue level **(Bakhiet, 2015**). Freshwater fish, particularly tilapia (*Oreochromis niloticus*), have been exposed to Pb in the water, according to findings in numerous research reports. The purpose of this paper is to review studies on how the tissues and cells of the tilapia (*Oreochromis niloticus*) react to the buildup of Pb heavy metal.

LEAD (Pb) ENTERS THE BODY OF TILAPIA

Pb deposits can form in aquatic sediments as a result of human activity, particularly industry, which releases the metal into the environment (**Ezeabasili *et al*., 2015; Rumanta, 2014)**. Heavy metals in the waters will enter the body of fish which can affect the metabolism, distribution and biological function of fish (**Ciftci et al., 2015**). The absorption of heavy metals Pb can be through various pathways namely through gills and skin or by ingestion of water and food contaminated by Pb, which can cause many biochemical and histological changes in fish that survive resulting in high mortality rates (**Doaa & Hanan, 2013; Udotong, 2015**).

Heavy metals, particularly Pb, enter the bodies of freshwater fish initially through the gill organs **(Iyabo & Immaculate, 2015; Roomiani *et al*., 2015**). The most important physiological role of gills is to take oxygen from water and expel carbon dioxide so that large amounts of metal are passed on the gills at any time, which increases metal uptake (**Langston & Bebianno, 1998**). Pb enter through gill filaments when fish must bind oxygen from the waters by passing large volumes of water over the gills, so that both essential and non-essential metals will enter through the filaments (**Akan *et al*., 2012**). Active ion uptake through gill filaments and prevents and inhibits ion loss through membranes and water flow to their tissues, causing heavy metals to affect oxygen uptake and osmoregulation in fish (**Aldoghachi *et al*., 2016**).

Pb will enter the body through the mouth, first entering the bloodstream and then specifically entering the fish liver, the main organ for detoxification. The liver is where heavy metal biotransformation, bioaccumulation, and enhanced elimination occur **(Hadi & Alwan, 2012; Nikinmaa, 2014**). Heavy metal was absorbed through the digestive tract, will diffuse passively or catalyst or active and transported to the target person or react **(Hapsari *et al*., 2017**). The distribution of metals in this organ is related to absorption and elimination rates, as well as time, where the influence of time on metal distribution in fish is a complex problem due to the different affinities of various metals to the tissues of various fish species **(Jezierska & Witeska, 2006**).

BIOACCUMULATION OF LEAD (Pb) IN TILAPIA

Fish have the capacity to absorb heavy metals along their gill surfaces and intestinal tract walls to concentrations greater than toxic levels in their environment (**Annabi *et al*., 2013**). The level of bioaccumulation and toxicity of heavy metals in fish depends on absorption through the surface and the concentration and properties of heavy metals (**Ahmad *et al*., 2015**). The accumulation of metals in various organs and tissues depends on the mode of exposure such as through feed or different rates of retrieval, deposition and excretion (**Hossain *et al*., 2016**). Gills, liver and kidneys accumulate heavy metals in higher concentrations compared to muscles, which indicate the lowest rate of metal accumulation **(Al-Kahtani, 2009; Kamaruzzaman *et al*., 2010**).

The gills are organs that are the site of temporary bioaccumulation of Pb, because gills are the main site of osmotic and ionic regulation in fish that have properties that are susceptible to heavy metals **(Adhim *et al*., 2017; Taweel *et al*., 2011**). The liver is the main site of bioaccumulation of Pb, where the liver has the capacity to accumulate metals through transport by blood from other parts of the body including gills and muscles, where metal-binding proteins such as metallothionein, which has a role to bind heavy metals, are produced **(Jabeen & Chaudhry, 2013**). The organ most vulnerable to the harmful effects of Pb is the liver, because it works as a detoxification organ and is essential for metabolism as well as excretion of toxic substances (**Bakhiet, 2015**). Lower concentrations in muscles suggest that fish muscles are important excretory organs also serving as a protective barrier between the external environment and muscles **(Javed & Usmani, 2013**).

Previous research has shown that the highest concentrations of Pb in fish are kidney and liver followed by *O. niloticus* bone and muscle (**Badr *et al*., 2014; Salah *et al*., 2013; Yacoub & Gad, 2012**). The BCF Pb value in tilapia liver is 8.10 ppm, with the concentration of Pb in the liver is 1.523 ± 0.02 **(Kaoud & El-Dahshan, 2010**). Another study showed that the highest Pb value in fish muscle was 0.81 µg/g wet wt during winter in the western sector, while the lowest concentration of Pb in fish muscle was 0.21 µg/g wet wt during winter in the eastern sector, with a maximum BCF Pb of 122.94 observed in the intermediate sector (**El-Batrawy *et al*., 2018**).

RESPONSE OF TILAPIA TISSUES AND CELLS TO LEAD (Pb)

Figure 1 depicts how tilapia tissue reacts to Pb. The tilapia liver exhibits congestion damage, degeneration of pancreatic tissue, turbid swelling, and vacuole degeneration, and the tilapia kidneys exhibit melanomacrophage, which is accompanied by renal cell necrosis and tubular disintegration. Secondary lamellae fusion, epithelial removal, and intense rupture are histopathological changes in the gills **(Abdel-Khalek, 2015**). In the gills of fish exposed to Pb, edema damage by 0-25%, lamellar fusion by 1-75%, hyperplasia by 0-50%, epithelial damage by 0-50%, and necrosis by 0-50% (**Utami *et al*., 2018**). The most numerous changes in the liver after lead exposure are cytoplasmic vacuolation, melanomacrophage,

congestion in sinusoids and cell degeneration (**Doaa & Hanan, 2013**). Tubule damage was 7%, glomerular vacuolation 13% and glumeruli shrinkage 12% in tilapia kidneys exposed to Pb (**Bakhiet, 2015**).

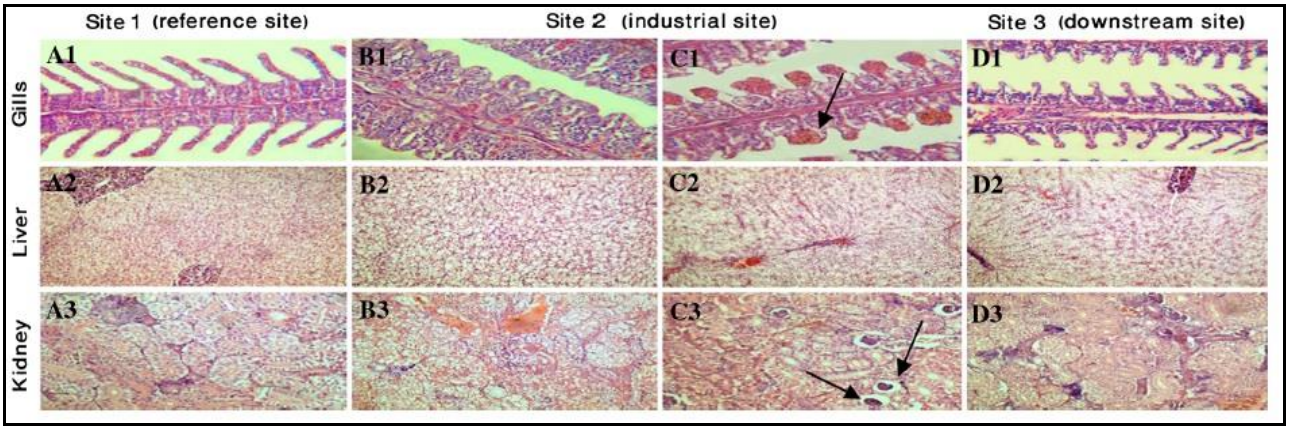

Figure 1 Changes in tilapia tissue to heavy metals. Where A1, A2 and A3 are normal tissues and others are metal-damaged tissues (**Abdel-Khalek, 2015**)

Damage to the lamellar epithelium and lamellar fusion occurs due to being protective because it reduces the gill surface area vulnerable to Pb (**Jabeen & Chaudhry, 2013**). Cell necrosis includes changes in cells that result in cell death, whereby cells will shrink and be damaged, causing cell contents to diffuse out and into the general blood circulation (**Udotong, 2015**). Vacuolation caused by Pb can inhibit impulse conductivity by inhibiting the activity of monoamine oxidase and acetylcholine esterase, thus causing pathological changes in tissues (**Salah *et al*., 2013**).

Changes in fish hematological parameters typically occur at the cellular level, particularly in blood cells, as a result of osmotic changes that cause hemoconcentration (an increase in the concentration of blood cells produced) or hemodilution (a decrease in the concentration of red blood cells due to an increase in plasma volume) (**Cogun & Sahin, 2012**). In Pb blood cells with a range of 35% - 80% can bind to δ-aminolevulinic dehydratase acid (ALAD), which is an enzyme present in red blood cells and responsible for hemoglobin synthesis (**Ribeiro *et al*., 2014**). A significant increase in white blood cell count could come from increased production of antibodies that aid the survival and recovery of tilapia exposed to Pb (**Adhim *et al*., 2017**). Changes in red blood cells, where there is a decrease indicate compensation for oxygen deficit in the body due to gill damage and the nature of changes indicate the release of erythrocytes, which causes disruption of water balance in the body (**Al-Rudainy, 2015**).

TOXIC EFFECTS OF LEAD (Pb) ON TILAPIA

Pb is an element that gives rise to various toxic effects, where the concentration is 10 µg/g d.wt. on the liver cause subclinical effects (**Jakimska *et al*., 2011**). Pb is known to induce oxidative damage through direct effects on cell membranes, interactions between lead and hemoglobin, which promote auto-oxidation of hemoglobin, auto-oxidized δ-aminolevulinic acid, interactions with GR, or through complex formation with selenium, which decreases GPx activity (**Sevcikova *et al*., 2011**). Intoxication can be produced in living cells by Pb, by following ionic mechanisms and oxidative stress (**Jaishankar *et al*., 2014**). Oxidative stress can be caused by the formation of ROS (Reactive Oxygen Species) which can damage polyunsaturated fatty acids in cell membrane phospholipids causing damage to cellular function (**Wang *et al*., 2014**). Pb is also known to inhibit enzyme, physiological and metabolic activities of fish (**Al-Balawi *et al*., 2013; Baharom & Ishak, 2015**).

Pb in tilapia (*Oreochromis niloticus*) is known to inhibit active transport mechanisms, involving ATP, to suppress cellular oxidation-reduction reactions and inhibit protein synthesis (**Yacoub & Gad, 2012**). Pb residues can cause hematological, gastrointestinal and neurological dysfunctions in fish, and severe or prolonged exposure to Pb can also lead to chronic nephropathy, hypertension and reproductive disorders (**Al-Kahtani, 2009**). Tilapia exposed to Pb will experience lipid peroxidation damage, DNA damage and enzyme inactivation with consequent inhibition of metabolic processes, hematological changes, and decreased fertility and survival (**Kaoud & El-Dahshan, 2010**).

CONCLUSION

Lead (Pb) is a poisonous element that harms aquatic life, particularly tilapia (*Oreochromis niloticus*). The article explains that Pb bioaccumulation is dependent on fish intake and dispersal. Additionally, the presence of Pb in the tilapia's body will trigger reactions in a number of crucial organs, including the gills, liver, and kidneys, as well as in cells and tissues like the blood and the liver and kidney tissues. Tilapia will experience toxic consequences as a result of the bioaccumulation and reaction to Pb, making tilapia dangerous for human consumption.

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