HEAVY METALS AND HEMATOLOGIC DISEASE ON WETLANDS : A LITERATURE REVIEW

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ABSTRACT
Heavy metals are metal elements with relatively high density compared to water. One area prone to heavy metal pollution is the wetland environment. In South Kalimantan, a wetland area, pollution is mainly caused by mining and palm plantation activities. The presence of heavy metals in the sediments of Martapura River, have passed the sediment pollution threshold, include Mn, Fe and Hg. Heavy metals can cause oxidative stress by generating reactive oxygen species (ROS), are associated with increased systemic inflammation, can lead to impaired immune function and accumulation of immune complexes. Mechanisms including direct toxicity to marrow precursors, inhibition of enzymatic processes essential for cell division and maturation, impairment of erythrocyte membrane transport, and immune-mediated cell destruction. However, research results are still contradictory. Presence of heavy metals still poses health risks in wetlands. The effect of heavy metal on health, especially hematological diseases, requires further research.

KEYWORDS
Environmental pollution; Heavy metals; Hematological disease; Oxidative stress; Wetland

INTRODUCTION

The term Wetlands, as a translation of "wetland" was only known in Indonesia around 1990. Previously, Indonesians called wetland areas based on the physical shape/name of each type such as: swamp, lake, rice field, pond, and so on. In addition, various sectoral departments also define wetlands based on their respective sectors of employment area. The physical definition of wetlands used to equalize the perceptions of all parties has become known by default since the ratification of the Ramsar Convention in 1991, namely: "Swampy, brackish, peatland, and aquatic areas; fixed or temporary; with stagnant or running water; unsalted, brackish, or salty; including the area of marine waters that are no more than six meters deep at low tide[1].

Pollution is currently happening everywhere, water crises are also happening everywhere, in general the water crisis occurs due to pollution of industrial, household or agricultural liquid waste. In addition to the decline in water quality due to pollution, the water crisis also occurs due to reduced water availability and erosion due to the clearing of forests upstream and changes in land use upstream and downstream. The shrinking of water supply in several large rivers in Kalimantan has become a terrible phenomenon, these rivers have experienced siltation due to the lack of water during droughts and added erosion and sedimentation[2].

In South Kalimantan, the water crisis is mainly caused by mining activities, especially coal mines and by oil palm plantation activities, both of which are very widely cultivated in South Kalimantan Province. Mining activities cause polluted water quality in terms of chemistry, namely metals, because mining activities dismantle layers of rock in the soil in which many heavy metals are covered. Metals can be classified into two types, namely heavy metals and light metals. Heavy metals are metals weighing more than equal to 5 g/cm3. Heavy metals are named as non-essential metals and can to some degree be toxic to living things. Heavy metals are natural components in the soil. This component cannot be degraded (non degradable) or destroyed. These compounds can enter the human body through food, drinking water, and air. Some heavy metals that are harmful to humans include lead, copper, mercury, cadmium, and chrome[3]. In this review will be discussed further about heavy metals and hematological diseases of wetlands.

Wetlands and Heavy Metals in South Kalimantan

South Kalimantan is one of the provinces rich in natural resources in the form of minerals, both coal, petroleum, gold and various natural products. In terms of topography, South Kalimantan consists of 4 (four) parts of soil types, alluvial yaki, rawamperhilan plains and mountains, with the dominant soil types, namely red yellow and alluvial podolic. Wetlands have an important role in environmental conservation, including as a contributor to biodiversity, climate balance, water circulation sources and food sources for local communities. People use wetlands to meet various needs, especially the needs of life. Banjar people in South Kalimantan, use wetlands for farming, raising fish and farming. However, the use of wetlands is also the cause of damage and loss of wetlands[4].

The results of Dwiyatno's research in 2008 showed that the content of heavy metals (Hg, Pb, Cd, and Cu) in fish from the estuary waters of S. Barito is still below the allowable threshold so that it is safe for consumption. However, the level of consumption for haruan fish should receive attention because the Hg metal content is already relatively high. Other studies in South Kalimantan waters only showed that Pb (Lead) cadavers were still below the maximum of heavy metal contamination, but this study did not examine the content of other metals[5].

In a 2020 study on the Martapura River, South Kalimantan, the Mn heavy metal content obtained ranged from 494-2,142 ppm with an average of 248.77 ppm. When compared to the threshold of Mn metal in sediments, the entire sampling site point has been polluted by Mn metal. High value of Mn can be caused by the rest of coal mining activities, where toxic materials left over from coal mining and processing contain heavy metals, one of which is Mn. Besides being derived from mining, Mn can also be produced from the process of weathering rocks in water basins or the presence of volcanic activities, derived from the active substance material where the battery stones that have been used up are discharged into rivers and coasts. The heavy metal content of Fe in martapura river sediment samples ranged from 48,000-68,800 ppm with an average of 60,133.33 ppm. When compared to the threshold, the Fe metal at all sampling sites has been above the sediment contamination threshold of 68800ppm. The heavy metal content of Fe in sediments elsewhere such as in the Ombilin River shows values ranging from 71,840-111,900 ppm with an average of 96,181 ppm which has exceeded the sediment contamination threshold, while in the Citarum River it ranges from 44,900-56,000 ppm indicating it is still below the sediment contamination threshold. The high content of Fe in river sediments can be caused by the weathering of bedrock in this area, where it is known that the Martapura River has its head in the Bobaris Mountains, one of the bedrock is ultrabasic igneous rock[6].

The existence of a gold mine in Mataraman District, Banjar Regency, South Kalimantan is also suspected to cause a high Hg content in the Martapura River, where the river flow from the area enters the Martapura River[6].

The presence of heavy metals in the Martapura River sediments in South Kalimantan that have crossed the sediment contamination threshold include Mn, Fe and Hg. High content of these three heavy metals, besides being suspected to be derived from the weathering process of bedrocks, is also suspected to be caused by the remaining coal and gold mining processes and also the use of pesticides around the research area[6].

Table 1. Heavy Metal Content in Martapura River Sediment[6]

	Heavy Metal Concentration (ppm)				
Sampel	**Cu**	**Zn**	**Mn**	**Fe**	**Hg**
1	69	113	921	61.100	5.775
2	51	93	1.299	58.600	0.108
3	40	79	1.026	48.000	0.092
4	75	106	2.142	68.800	0.115
5	82	103	1.786	63.100	0.115
6	68	147	815	58.600	0.205
7	72	131	991	64.800	0.154
8	67	161	797	67.300	0.169
9	43	110	494	50.900	0.123
Average	63	115,88	**1.141,22**	**60.133,33**	**0.761**
Minimum Value	40	79	494	48.000	0.092
Maximum	82	161	2.142	68.800	5.775

	Heavy Metal Concentration (ppm)				
Value					
Threshold	108	271	248.77	20	0.02-0.35

Effects of Heavy Metals on Health

Heavy metals are natural components in the soil. This component cannot be degraded (non degradable) or destroyed. These compounds can enter the human body through food, drinking water, and air. At low levels, heavy metals are required by living beings for the regulation of various chemical and physiological functions of the body. It is commonly known in the term trace element, which is a chemical element needed by living organisms in very small quantities (less than 0.1% of the volume). As a trace element, some heavy metals such as copper (Cu), selenium (Se), Iron (Fe) and zinc (Zn) are very important for the body. Heavy metals can become harmful or toxic when they are in excessive levels in the body[7].

Logam yang bersifat racun bagi Living things are only classified as heavy metals. Heavy metals include essential ones such as Zn, Cu, Se and non-essential ones such as Hg, Pb, Cd, and As. Cases of heavy metal poisoning are often caused by environmental pollution by heavy metals themselves, such as the use of metals as pest repellents (pesticides), fertilization or due to the disposal of factory waste that uses metal. Essential metals such as Cu and Zn at certain levels are needed as nutritional elements for animals, but non-essential metals such as Hg, Pb, Cd, and As are currently unknown. When heavy metals enter the human body, they will accumulate in body tissues and cannot be excreted outside the body anymore. At already high levels in the human body, it will cause serious negative impacts[6.7]

Meanwhile, the microelement group is a group of heavy metals with no function at all for the body. Examples are lead (Pb), mercury (Hg), arsenic (As) and cadmium (Cd). These compounds are even very dangerous and toxic in humans . Heavy metals are non-biodegradable and easily absorbed[3].

Heavy metals can also cause damage to aquatic organisms. Sources of metal pollution mostly come from mining, metal smelting, other industries, and can also come from domestic waste that uses metals, as well as agricultural land that uses fertilizers containing metals. The retention of pollutants depends on their biological half-life. Thus, a polluter must show a relatively high resistance to destruction or disposal by living beings to allow for sufficient picking time to achieve high concentration. The heavy metal content will increase over time. For example, the first o rganism affected due to the heavy metal content in the soil is the plants in that habitat. If the plant is consumed by humans, then heavy metals can accumulate in the body and remain for a long time as toxins accumulate, or this is commonly called bioaccumulation. This process can occur by direct absorption from the environment or through foodstuffs. Pollutants in living things through foodstuffs can arise from the same source. So in a natural food chain, pollution can be moved from one level to another[3,4].

Bioaccumulation is an increase in the concentration of chemicals in the body of living things over a long period of time, compared to the levels of chemical substances in nature. Excessive levels cause heavy metals to be non-metabolized and unable to undergo biotransformation to other forms of compounds. Heavy metals can only be excreted by the body through the kidneys in the form of ions and these ions can cause impaired renal function[3]. In addition to naturally, the causes of heavy metal content in the soil include agrochemical materials in the form of fertilizers and pesticides, contamination from motor vehicle smoke, fuel oil, household waste disposal, industrial waste, and mining. The content of heavy metals in the soil is influenced by environmental factors such as soil acidity, organic matter, temperature, texture, clay minerals, and other elemental content. The degree of acidity (pH) is an important factor in the process of metal transformation. At low pH, the availability of some heavy metals may increase[3,8].

The knowledge gained about heavy metal homeostasis has been growing rapidly for more than a decade. Although heavy metals do not have a known metabolic function, when present in the body they disrupt normal cell processes, which causes poisoning in a number of organs. Heavy metals have poor absorption, but after absorption they are slowly removed and accumulate in the body causing organ damage. So, its toxicity is largely due to their accumulation in biological tissues, including the diet of animals such as fish and cows as well as humans. The distribution of heavy metals in the body depends on their binding to carrier molecules in circulation. Its main impact on human health is mainly through exposure to work, environmental pollution, and food buildup, especially on vegetables grown in contaminated soil. Arsenic and cadmium, in addition to mercury and lead, have been identified as the

most likely causes of heavy metal-related diseases observed in primary care medicine. Exposure to one heavy metal contaminant is often accompanied by exposure to another. Therefore, combined interactions may occur in populations exposed to metal mixtures[8].

Heavy metals are toxic because they may have cumulative damaging effects that can cause chronic degenerative alterations, especially on the nervous system, liver and kidneys, and, in some cases, also have teratogenic and carcinogenic effects. The mechanism of toxicity of some heavy metals is still unknown, although enzymatic inhibition, antioxidant metabolic disorders, and oxidative stress may play a role. Heavy metals produce many adverse health effects through the formation of free radicals, resulting in DNA damage, lipid peroxidation, and depletion of sulfhydryl proteins[8,9].

Heavy metals as environmental pollutants and promoters of oxidative stress are associated with many detrimental impacts on human health. There is growing concern about the physiological and behavioral effects of environmental heavy metals on human populations. Heavy metal poisoning in humans has an acute and chronic effect on health and the environment. Although the toxicity of heavy metals at high levels of exposure is well known, the main concern at the moment is the possibility that continued exposure to heavy metals with relatively low levels can cause adverse health effects. Nevertheless, his contribution to hematological diseases is still not fully understood. Recent studies have shown that the vascular effects of heavy metals can contribute to a variety of pathological conditions including diabetes mellitus and hypertension, however the mechanism of action of heavy metal exposure remains unclear[12].

Prevalence of Hematological Disorders in South Kalimantan

Hematology is a branch of science that studies blood and its disorders. Hematological disease can be interpreted as an abnormality in the blood, its functions, and systems. Hematological disorders can range from mild abnormalities such as anemia to severe cases and malignancies, as in the case of leukemia[13].

The prevalence of anemia in Indonesia is still quite high. The Ministry of Health of the Republic of Indonesia (2013) shows that the prevalence rate of anemia nationally in all age groups is 21.70%.4 Basic Health Research Data (Riskesdas) 2018 shows that the percentage of anemia in WUS in Indonesia has increased compared to Riskesdas 2013 data to 48.9%[14].

The incidence of anemia in young women in South Kalimantan Province in 2019 was 27.03% highest in Hulu Sungai Utara Regency (57.51%), Barito Kuala (41.88%), Balangan (40.31%), Kota Baru (37.80%), Tanah Bumbu (32.26%), Tanah Laut (27.56%), Banjarmasin (25.7%), Hulu Sungai Tengah (24.27%), Banjar (22.51%), Banjarbaru (21.13%), Tapin (7.06%), Hulu Sungai Selatan (4.43%) and Tabalong (4.42%)[15].

Another case of hematological disorder that occurs quite a lot in South Kalimantan is Leukemia. In the 2016 non-communicable disease profile issued by the ministry of health, South Kalimantan was the fourth most province for leukemia cases (Figure 1)[16].

Heavy Metals and Hematological Diseases

The potential link between chronic heavy metal exposure and hematological diseases has a number of implications. Although hematological systems are not usually specifically discussed as The main targets of heavy metal toxicity, review articles and the main attention of most reviews have focused on imbalances in antioxidant protection mechanisms causing oxidative stress in cells as the main effects of their environmental exposure. Gene expression is altered by environmental influences, especially food components of gene regulation that can be influenced by metal toxicity[17,18].

Patients differ in the time of onset and dynamics of risk factors, exhibiting complex pathophysiology in hematological disorders. Differently, genetically determined susceptibility to the environment, risk factors, interaction of the hematopoethic, cardiovascular systems with other organs such as the immune system, and perhaps the interaction between these risk factors in a person are possible causes of such differences. Despite an increased understanding of genes, proteins, signaling pathways, cell-cell interactions, and systemic processes involved in hematology (initiation, development, and outcomes), the relevance of environmental factors has hardly been discussed[19].

Heavy metals (As, Cd, Hg, and Pb) can cause oxidative stress by producing reactive oxygen species (ROS), including superoxide radicals, hydrogen peroxide, and nitric oxide[7,20].

Heavy metals can be deposited in the bone marrow, and induce aplasia or immunological reactions. For example, gold is phagocytosed by the reticuloendothelial system, accumulates in the

lysosomes of bone marrow cells, and provokes the formation of antibodies. Various heavy metals inhibit in normal metabolic pathways. Lead inhibits some enzymes essential for the synthesis and degradation of porphyrins, by binding to enzymes containing thiol and sulfhydryl, thereby causing disruption of disulfide bonds. Other heavy metals besides lead can inhibit glutathione synthetase and interfere with cells' ability to tolerate oxidative stress, resulting in hemolysis. Heavy metals can also compete with essential minerals that have similar properties, for example calcium and iron, in metabolic pathways. Cadmium has a high affinity for hemoglobin and can compete with iron in the formation of hemoglobin[21].

Heavy metals are associated with an increase in systemic inflammation, by which deficiency of essential metals and excess toxic metals can lead to impaired immune function and the accumulation of immune complexes, and through a series of interrelated processes, , including the release of inflammatory cytokines, kidney damage, and stimulation of the central nervous system. Metals increase oxidative stress. As it has been associated with an increase in intravascular inflammation by increasing the regulation of interleukin 6 (IL-6), tumor necrosis factor a (TNF-a), monocyte chemotactic proteins, vascular cell adhesion molecule 1 (VCAM-1), and intercellular adhesion molecule (ICAM) . Cd has also been linked to inflammatory and coagulation disorders, including elevated levels of blood reactive C-reactive protein (CRP) and fibrinogen in the U.S. general population, and VCAM-1 in animal studies. Both oxidative stress and increased systemic inflammation caused by exposure to toxic metals contribute to the development of the disease as a result of exposure to heavy metals[1].

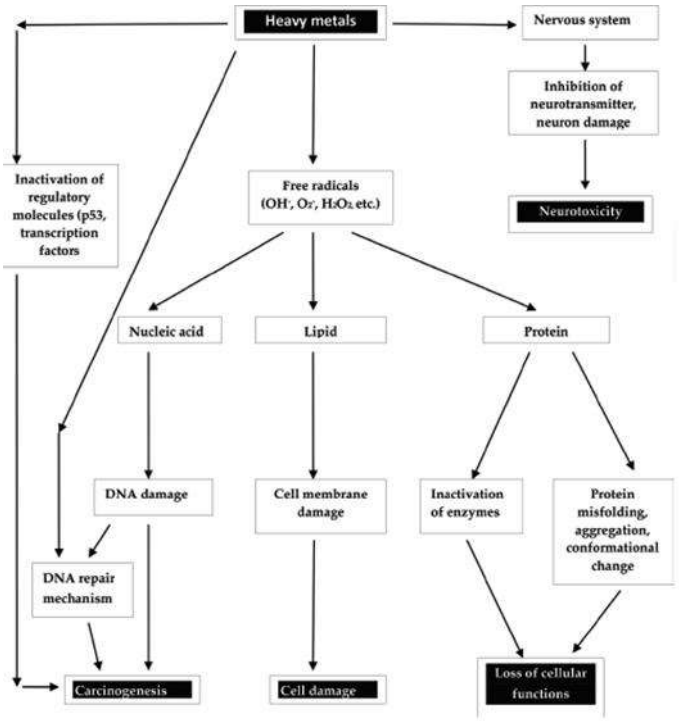

Figure 1. Heavy Metal Toxicity Mechanism[21]

Mercury is one of the metal contents that is crushed past the threshold on the Martapura River. Mercury is one of the most toxic heavy metals and has no known physiological role in humans. The presence of different entry routes of mercury (air, water, food, vaccines, medicines and cosmetics) causes its easy accessibility to humans. In particular, in populations whose diet is mainly based on fish consumption, the risk of mercury exposure increases. In many developing countries, mercury is still a big problem.

Various efforts must be made to reduce the use of mercury globally. Exposure to mercury and its compounds has resulted in harmful effects on human health. Mercury and methylmercury induce mitochondrial dysfunction, decrease ATP synthesis, deplete glutathione, and increase phospholipids, proteins, and DNA peroxidation. Selenium and fish, rich in omega-3 polyunsaturated fatty acids, fight mercury toxicity[3,10].

The Ian Observer in Nigeria stated that there was a decrease in the number of red blood cells in

Artisans in Nigeria, most likely as a result of a sequelae of oxidative stress that inevitably led to peroxidative hemolysis in the membranes of red blood cells[22].

Exposure to high concentrations of mercury vaporsisa syndrome characterized by fatigue, fever, chills, and an increased number of leukocytes. This syndrome is similar to the fever seen after exposure to other metals. After acute inhalation exposure to mercury metal, moderate to high leukocytosis with neutrophilia is observed in a number of studies. Studies on workers exposed to elemental mercury also reported a decrease in ALAD activity in erythrocytes, and a significant increase in α-2-macroglobulins and ceruloplasmin (α-globulin proteins active in copper storage and transport) compared to unexposed workers[23].

Although in some studies, it was found that exposure to mercury and other heavy metals is not always related, there are several explanations for some of the hematological effects of mercury, such as: pancytopenia due to direct toxic effects on the bone marrow, anemia due to apoptosis, blood loss due to direct effects on the gastrointestinal mucosa and hemolysis, polycythemia due to an increase in levels of erythropoietin, leukopenia, lymphopenia neutropenia and bastopia due to passing inflammatory reactions and apoptosis, leukocytosis and neutrophilia due to pulmonary inflammatory reactions (pneumonitis), eosinophilia related to hypersensitivity and idiosyncrasi, lymphocytosis due to increased calcium content in the cytoplasm, and immunologically mediated thrombocytopenia[23].

Heavy metals compete with essential metals for various physiological functions and influence the risk of disease occurrence. Heavy metals compete with essential metals for absorption and excretion; transport of metals in the body, i.e. the anchoring of target proteins; and metabolism and sequestration of toxic metals. Part of the toxicity of Pb, for example, comes from its ability to mimic other essential metals, for example, Ca, Fe, and Zn, since it binds to and interacts with many of the same enzymes as this essential metal and thus interferes with the ability of the enzyme to catalyze its normal reaction. Cd and Pb have chemical and physical properties similar to Zn, and compete for metal absorption and enzymatic protein binding sites. Therefore, in the event of Zn deficiency and increased exposure to this toxic metal, the body will use Cd and Pb instead of Zn. Cd also competes with Fe for access to intestinal metal absorption transporters. Fe deficiency can lead to greater absorption and toxicity of Cd and Pb. At low concentrations it can decrease ace poisoning through the excretion of AseSe compounds, but excessive Se can increase the toxicity of As.Ca and Mg also competes with Pb or Cd for intestinal absorption to reduce the burden of toxic metals and prevent tissue damage caused by toxic metals competitively binding to the active sites of enzymes. Trace metal essential with its antioxidant properties at a normal level has the ability to resist oxidative stress caused by toxic metals, thereby reducing the toxicity of toxic metals[14,15].

The vascular effects of mercury include increased oxidative stress and inflammation, reduction of oxidative defenses, thrombosis, and mitochondrial dysfunction, depolarization, and autoxidation of the inner mitochondrial membranes. Another mechanism by which mercury exerts a toxic effect on the cardiovascular system is the inactivation of paraoxonase, which causes HDL dysfunction to reduce the transport of cholesterol back to the liver. This enzyme plays an important role as an LDL antioxidant, so it is directly involved in atherosclerosis, myocardial infarction, and cardiovascular disease. Mercury toxicity is said to correlate strongly with hypertension, coronary heart disease, myocardial infarction, cardiac arrhythmias, carotid artery obstruction, and general atherosclerosis[3,10]. Mercury can bind to other compounds such as chlorine, sulfur or oxygen. The bond will form inorganic mercury compounds or salts. Most inorganic mercury compounds are in the form of white powders or solutions except mercury sulfide (synabar). Sinabar has a red color and turns black when exposed to light. Mercury is commonly found in nature in the form of metallic mercury, mercury sulfide, mercury chloride and methyl mercury[3,10].

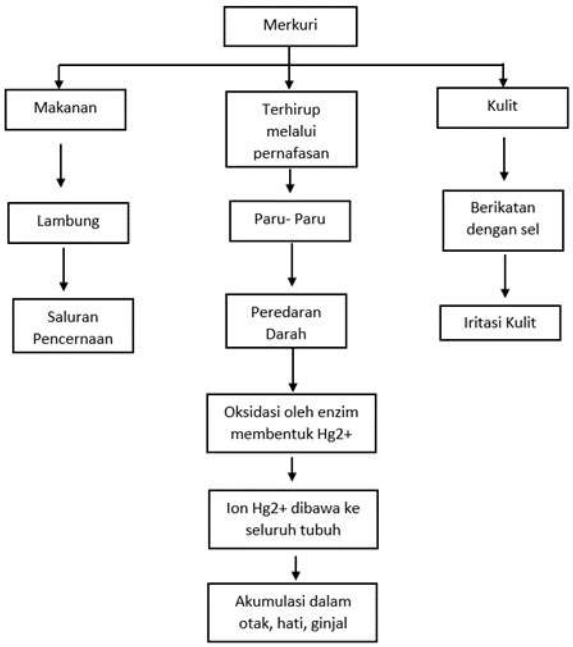

Figure 2. Mercury Toxicity Mechanism[3]

The release of mercury from natural sources remains the same for many years. However, the concentration of mercury in the environment has increased due to human activities. Most of the mercury from human activities is released into the air through the burning of fossil fuels, mining, smelting and burning solid waste. Some human activities cause the release of mercury directly into the soil or water, for example the use of fertilizers and the disposal of industrial waste. All mercury released in the environment will end up on the ground or the surface of the water. Mercury is not found naturally in foodstuffs. However, mercury can appear in food because it is spread in the food chain of small organisms consumed by humans such as fish. The concentration of mercury in fish is biased beyond the concentration in water. Products of farm cattle may also contain mercury. Mercury is not commonly found in plants, but can enter the human body through vegetables and other plants. This can happen when pesticides contain mercury[10,13].

Over the past few years or so, the toxic effects of mercury have been associated mainly with the central nervous system, kidneys and brain. It is reported that exposure to mercury compounds, caused by frequent fish consumption by the Amazon basin population in Brazil, has a strong correlation with an increase in arterial blood pressure. Studies on BP and heart rate variability , among aboriginal populations from Quebec, who are indirectly exposed to mercury and methyl mercury, have been reported by Valera and co-authors. The study showed the damaging impact of mercury and MeHg on BP and HRV in Inuit adults, while exposure to MeHg during childhood affected HRV among Nunavik Inuit children without regard to BP. Thurston et al. report that prenatal exposure to MeHg from seafood consumption increases children's blood pressure. Grandjean et al. study whether heart function in childhood is affected by MeHg exposure from seafood[3,10,13].

Methylmercury exposure is associated with a decrease in sympathetic and parasympathetic low-frequency modulation of HRV. Other studies link exposure to mercury toxins with an increased risk of myocardial infarction, atherosclerosis, hypertension, and coronary dysfunction. Mercury levels in the hair are predictors of low-density lipoprotein levels that are often found in atherosclerosis lesions and are associated with atherosclerosis and are associated with atherosclerosis and acute coronary insufficiency. The toxic effect of mercury in all its forms has been proven both in vitro, in animals and humans. Exposure to mercury increases the production of free radicals, reactive oxygen species (ROS), and superoxide anions due to the Fenton reaction. Mercury binds to thiol-containing molecules (-SH) and binds to selenium, forming selenium-mercury complexes, reducing the activity of glutathione peroxidase, catalase, and superoxide dismutase due to the absence of selenium in the active site of this enzyme. Increased ROS and decreased activity of antioxidant enzymes increase the risk of developing diseases. In addition, mercury promotes LDL oxidation and destroys the integrity of plasma membrane phospholipids by externalization of phosphatidylserin. In addition, the translocation of

phosphatidylserine from the leaflets of the inner mitochondrial membrane to the outside leads to modification of the mitochondrial membrane with the loss of mitochondrial potential and the occurrence of apoptosis. As a result, mitochondrial function changes, mitochondrial permeability (MPT) transitions are affected by membrane potential reduction, oxidative phosphorylation, and ATP production[12,14].

Human exposure to mercury is associated with anthropogenic activity. Mercury is one of the earliest elements found to be non-essential for biological processes, and is considered toxic due to its accumulation in the organism. Mercury exerts an effect on neurological, retrimental, respiratory, immunological, dermatological, reproductive and developmental. Mercury induces oxidative stress that causes membrane damage, enzymatic damage and oxidation of biomolecules. Mercury exposure decreases the catalytic activity of GPx, and promotes H2O2 product synthesis and lipid peroxidation (LPO)[12].

Mercury induces oxidative stress that causes membrane damage, enzymatic damage and oxidation of biomolecules. Exposure to mercury decreases the catalytic activity of GPx, and promotes the synthesis of H2O2 products and lipid peroxidation (LPO) in the renal and mitochondrial membranes. Thus, mercury stimulates the production of malondialdehyde (MDA), 4-hydroxyalkena (4-HOA) and advanced protein oxidation products such as dityrosine, which enhance the inflammatory response[18,25].

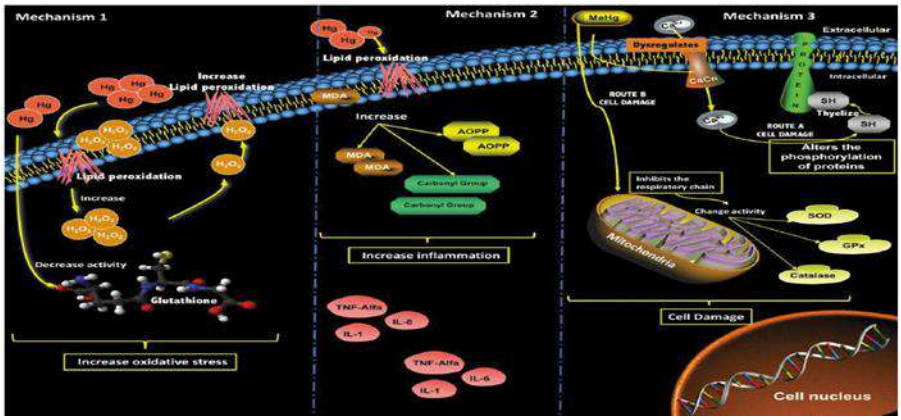

Figure 3. Mekanisme Mercury Induced Oxidative Stress[12]

A prospective study found a positive association with the incidence of coronary disease, but no dose-response relationship, and no association with overall cardiovascular mortality. Two cross-sectional studies found a positive association with cardiovascular disease, and another with arterial stiffness. But the cross sectional design could not establish causality, and both of these studies measured blood rather than mercury concentrations in toenails. Blood samples do not last as well as nail samples, red blood cells usually have an alternation of 17 weeks, compared to 26-52 weeks for toenails[2,6,27].

Chowdury et al, 2018, and Downer et al 2017 research showing no link between mercury exposure and increased risk of disease in adults should not alter ongoing public health and policy efforts to reduce mercury contamination in fish and the environment, which still have the potential to compensate, at least in part, nets. Manfaat from fish consumption. These findings should still not change the recommendations addressed to women who are pregnant or breastfeeding, as exposure to methylmercury from the consumption of certain fish species can cause neurodevelopmental damage[18,28]. Although in populations that consume fish, the risk of mercury exposure increases, and experimental studies in humans show that the consumption of even the slightest amount of fish and seafood significantly reduces heart disease and mortality. Selenium and fish rich in omega-3 polyunsaturated fatty acids can fight mercury toxicity. Fish consumption can balance the toxic effects of mercury with the benefits derived from the consumption of omega-3 polyunsaturated fatty acids. Mercury toxicity should be evaluated in any patient with anemia, hypertension, coronary heart disease, cerebral vascular disease, or other vascular disease and in patients who have a history of exposure or clinical evidence on an over-mercury test[16].

CONCLUSION

The presence of heavy metals is still one of the health risks that exist in wetlands. The results of

a 2020 study for the heavy metal content of the Martapura River sediment in South Kalimantan that has passed the sediment contamination threshold include Mn, Fe and Hg. Metal content that crosses this threshold, especially mercury (Hg) can be a cause or risk factor for hematological diseases.

The effects of heavy metal content, such as mercury on health, especially hematological diseases are still not fully understood, so they require further tracing and research.

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