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THE DETRIMENTAL EFFECTS OF MERCURY EXPOSURE ON HUMAN HEALTH

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Abstract: **Mercury is one of the widespread toxic metal detrimental effects on human health. Humans can be exposed to mercury in the workplace, consume large amounts of aquatic product, products containing mercury such as traditional remedies, cosmetics, including skin lightening creams. The aim of this review is to explore the natures and type of mercury, routes of exposure and sources of mercury, toxicity mechanisms of mercury exposure, and the clinical signs of acute and chronic exposure of mercury. The writing methodology used is a literature review. The literature source consists of relevant journals and books from the search engines PubMed and Google Scholar. The major sources of mercury in the environment are divided into two types; natural and anthropogenic sources. Mercury typically exists in in three forms, including elemental mercury, inorganic mercury and organic mercury. Toxic effect of the mercury was documented in several historical accidents that revealed by neurological symptoms, the cardio metabolic syndrome, liver and renal diseases via the oxidative stress mechanisms, due to the strong ability of the mercuric compounds to bind to the sulfhydryl terminal of vital proteins and enzymes.**

Keywords: **Mercury, Exposure, Detrimental, Human Health.**

I. INTRODUCTION

Mercury is a persistent, bioaccumulative, toxic pollutant. Due to its prevalence, toxicity, and high potential for human exposure, Mercury (Hg), is placed third on the 2019 Agency for Toxic Substances and Disease Registry (ATSDR).[1] Mercury occurs naturally as a mineral and is distributed throughout the environment by both natural and anthropogenic processes.[2] When released into the environment, it accumulates in water laid sediments where it converts into toxic methylmercury (MeHg) and enters the food chain.[3] Once released into the atmosphere, mercury can be transported on a global scale, converted to other forms (Hg^{+}, Hg^{2+}), and/or returned to the soil and water by various depositional processes. In the aquatic system, part of the oxidized inorganic mercury (Hg^{2+}) is methylated.[4] The methylation process is believed to occur through a non-enzymatic reaction between Hg^{2+} and methyl-cobalamine produced by bacteria.[5] Formed methyl mercury (MeHg) ca rapidly diffuse and bind to proteins in aquatic biota, leading to its bioaccumulation in fish and marine mammals.[6] In general, mercury can be can be classified into three types: elemental Hg, inorganic mercury (mercury salt), and organic mercury.

Historical accidents resulted from inappropriate use of mercury, caused several disasters. Due to inappropriate use of mercury in construction of mercury rivers in mausoleum of Chinese Emperor Qin Shi Huang at (260-210 BC), this result with sudden premature death after mercuric dose consumption.[7] Minamata disease was discovered for the first time in the world at Minamata City, Japan in 1956. This case was attributed to the methyl mercury that was generated in the process for producing acetaldehyde using mercury as catalyst. Methyl mercury had accumulated in fishes and shellfishes and those who consumed them had been poisoned with it. Such type of exposure to methyl mercury was highly uncommon and unusual, although the number of victims eventually certified with Minimata disease was over 2,200. In Iraq, three epidemic poisonings have been reported in 1955 - 1960 and the largest outbreak in 1971 – 1972. These outbreaks were caused by the

distribution of seed grains treated with methyl mercury. Rural people consumed the grains by using it to make homemade bread, instead of planting the seeds. The total number of victims was 6530, including 459 deaths.[3]

Exposure to mercury can have a very varied effect, depending on the level and duration of exposure, age and health status of individuals. Furthermore, it may cause biocomial damage to tissues and genes through various mechanisms such as interfering with intracellular calcium homeostasis, disruption on membrane potential, change on protein synthesis, inhibiting enzymes and disruption of amino acid pathways in the central nervous system. [8, 9, 10]

The aim of this review is to explore the natures and type of mercury, routes of exposure and sources of mercury, toxicity mechanisms of mercury exposure, and the clinical signs of acute and chronic exposure of mercury.

II. MATERIALS AND METHODS

For this review, literature sources such as scientific journals on search engines (PubMed and Google Scholar) and relevant national books were searched. The keywords used were "Mercury Exposure", "Sources ", "Toxicity", and "Human Health". Inclusion criteria were all reviews on Mercury Exposure. Exclusion criteria were literature published more than 10 years ago. Information was collected, recorded and analysed to assess the validity and reliability of the literature.

III. RESULTS AND DISCUSSION

Natures and Type of Mercury

Mercury comes from a range of natural sources such as volcanoes, soils, undersea vents, mercury-rich geological zones and forest fires, as well as from fresh water lakes, rivers and the oceans. Mercury is also leached from flooded soil at new hydroelectric dam sites, or from any flooded area. This process can add to mercury levels in freshwater aquatic food chains in those areas.[2]

Human activity has increased the amount of mercury in the environment in several ways, including through a variety of combustion and industrial processes like coal-fired power generation, metal mining and smelting and waste incineration (anthropogenic sources). Products such as button batteries, fluorescent tube lights, thermometers, thermostats, switches and relays, barometers and dental amalgam may contain mercury. Highly diluted quantities of mercury are used in some homeopathic medicines. Mercury is also used in various traditional medicines from around the world. Disposing of these products can cause mercury to leach from landfills or be emitted from burning waste, adding to the amount of mercury in the environment.

Mercury is commonly known as quicksilver, because of its silvery white appearance, and an unique element with distinctive properties.[2, 10] The properties of mercury are shown in table 1.

Table 1. Properties of Mercury

Chemical symbol	Hg (hydrorgyrum)
Atomic number	80
Atomic mass	200.59
Freezing point	- 38.83^{0}C
Boiling point	356.73^{0}C
Density	13.5956 g/cm^3
Relative abundance	5 X 10^{-5} %
Specific heat capacity	0.1397 J/g/K
Heat of fusion	11.807 j/g
Heat of evapouration	59.543 kJ/mol
Thermal conductivity	1.063 X 10^{-4} /mm^2
Thermal expansion coefficient	1.826 X 10^{-4} / K
Electrical cinductivity	1.063 X 10^{-4} /mm^2
Viscosity	1.685 mPa
Surface tension	480.3 X 10^{-5} N/cm
A crystal structure	Rhombohedral

Mercury present mainly in three forms, including elemental mercury, inorganic mercury and organic mercury.

1. Elemental mercury (Hg^0, metallic)

Elemental Hg (Hg^0) comes from thermostats, thermometers, dental amalgams, added to latex paint, and to a certain extent entering the atmosphere in a yawning state. The oxidation state of Hg^0 represents the only metal that occurs in a liquid form at room temperature. It's volatile and mainly absorbed through the inhalation in respiratory tract (80%). Mercury can cross the blood brain barrier and is rapidly oxidized to Hg^2 ions which are maintained in brain cells for years.[7, 11]

2. Inorganic mercury (mercury salt)

In the form of Hg^{2+} (mercuric) and Hg^+ (Mercurous) is found in laxatives, cosmetic products, plant powders, diuretics, and antiseptics. This can also be induced from Hg vapour elements or methyl mercury (MEHg) metabolism.[11] Among the two stages, the oxidation of Hg^{2+} is more reactive. It can form complexes with organic ligands, especially sulfurhidryl groups. For example, $HgCl_2$ is very soluble in water and very toxic, whereas HgCl is insoluble and less toxic.

3. Organic mercury (Hg organic)

Organic Hg is considered the most dangerous form of Hg exposure and most often, it is detected as methylmercury (MeHg/ (CH_3Hg^+) and ethyl mercury (EtHg/ $C_2H_5Hg^+$).[12] It has been found in various sources such as fish, poultry, insecticides, fungicides, pesticides, and vaccines containing thimerosal. The most frequent exposures are from consumption of fish holding MeHg and prophylaxis used from vaccines containing preservative thimerosal which is rapidly metabolized to EtHg. MeHg and other organic forms of mercury are particularly hazardous to humans because of their long-term toxicity and ability to cross any cellular barrier.[13]

Routes of Exposure and Sources of Mercury

Relevant routes of exposure for humans vary based on the category of mercury compound:

1. Elemental mercury

The most relevant route of exposure to elemental mercury is through inhalation of mercury vapor. Exposure of workers to elemental mercury vapor has occurred in several occupational settings (i.e., production of chlorine and sodium hydroxide), fluorescent lamp production, gold mining and processing, lithium-6 purification, mercury amalgam dentistry, mercury battery production, natural gas production, recycling, and thermometer production.

2. Inorganic mercury salts

Oral exposure is the primary route of exposure to inorganic mercury salts. Exposure may occur through diet or contaminated environmental media (e.g., soil). The toxic potential of mercury salts is influenced by their solubility. In general, mercurous compounds exhibit lower toxicity compared to mercuric compounds due to their reduced solubility in water.[14] Mercury salts exhibit greater corrosiveness compared to elemental mercury, leading to increased gastrointestinal permeability and absorption.

3. Organic mercury compounds

Methylmercury is by far the predominant form for organic mercury exposure in populations. Exposure to methylmercury occurs worldwide through the diet, with fish as the main dietary source of methylmercury.

The effects of mercury toxicity on humans depend on the chemical form of mercury, dosage, age of people exposed, length of exposure, entry into the body, fish diet and seafood consumption. For example, the form of mercury (HgCl2) is more toxic than mercuro (HgCl), this is because the divalent form dissolves more easily than the monovalent form. In addition, the form of HgCl2 is fast and easily absorbed so that its toxicity is higher. Organic forms such as methyl mercury, about 90% are absorbed by the intestinal wall, which is much greater than inorganic form (HgCl2) that is only about 10%. The organic form can also penetrate the barrier of blood and placenta so that it can cause teratogenic effects and nervous disorders.[10]

Pharmacokinetic Profile and Toxicokinetics of Mercury

Humans are exposed to many forms of mercury, and these exhibit route-dependent and chemical-species-dependent toxicokinetics.[3]

1. Elemental mercury

Absorption of elemental mercury from respiratory tract (inhaled mercury vapor) was estimated to range from 69 to 85% in human adults. Absorption of elemental mercury from gastrointestinal tract (ingested as mercury amalgam) was estimated

to be 0.04% in human adults. Systemic absorption of mercury has been shown to occur in human adults following skin exposure to mercury vapor (approximately 2% of absorption from inhalation during a full-body immersion in mercury vapor). Following inhalation exposure to mercury vapor, mercury distributes throughout the body, with the highest concentrations occurring in the kidneys. Vascular proximity of the heart and brain coupled with a limiting oxidation rate of Hg^0 in blood contributes to a first-pass effect on uptake of Hg^0 in these tissues following inhalation of mercury vapor. Inhaled mercury vapor can be transferred from the mother to the fetus and also from the mother to infants via maternal milk. Absorbed Hg^0 is eliminated in exhaled air and by oxidation to mercuric mercury (Hg^{2+}). The major oxidative pathway for Hg^0 is catalyzed by the enzyme catalase. Following oxidation of Hg0 in blood and tissues, Hg^{2+} is excreted in urine and feces. Following inhalation of mercury vapor, mercury elimination kinetics exhibit multiple phases. The terminal half-time, thought to largely reflect urinary and fecal excretion of Hg^{2+}, has been estimated in humans to range from 30 to 90 days. Several pharmacokinetics models of inorganic mercury have been published. Of these, two models were developed to predict the absorption and distribution of inhaled mercury vapor

2. Inorganic mercuric mercury

After inhalation exposures to mercuric oxide (HgO), mercury was detected in various body regions, including head, kidneys, pelvis, and in the legs, indicating systemic absorption. Absorption of mercuric mercury following ingestion in gastrointestinal tract was estimated to range from 1 to 16% in human adults. Inorganic mercuric mercury was shown to be absorbed across isolated human and pig skin. Following ingestion of mercuric chloride, mercury distributes throughout the body, with the highest concentrations occurring in the kidneys and liver. Inorganic mercury is found in human cord blood, placenta, and breast milk, indicating transfer to the fetus and infant, respectively. The major routes of excretion of absorbed mercuric mercury are feces and urine. Kinetics of elimination of absorbed inorganic mercuric mercury exhibits multiple phases. The terminal half-time has been estimated in humans to range from 49 to 120 days.

3. Inorganic mercurous mercury

No studies were located that provide quantitative information on the absorption, distribution, metabolism, or excretion of mercury following exposure to inorganic mercurous compounds. Pharmacological and cosmetic uses of calomel (mercurous chloride) ointments (skin lightening creams, acne) have resulted in elevated urinary mercury levels and mercury poisoning, indicating that absorption of mercury can occur following oral and/or dermal exposure to inorganic mercurous compounds. Toxicity may have been from absorbed inorganic mercuric mercury, as the low pH and high chloride concentration of the gastric environment favor oxidation of ingested Hg^1 to Hg^{2+}.

4. Organic mercury compounds

No studies have estimated the absorption of organic mercuric mercury in the respiratory tract following inhalation. The results of studies conducted on humans, monkeys, and rodents have demonstrated that the gastrointestinal absorption of mercury is nearly 100% following the ingestion of methylmercury chloride or the incorporation of mercury into fish or other ingested protein. Additionally, the absorption of dimethylmercury through human skin has been shown to occur rapidly. Following ingestion of methylmercury, mercury distributes throughout the body, with the highest concentrations occurring in the liver, kidneys, and brain. Methylmercury is also found in human cord blood, placenta, and breast milk, indicating transfer to the fetus and infant, respectively. A number of studies conducted in humans and in a variety of other mammalian species have observed the presence of both methylmercury and inorganic mercury in tissues and excreta following exposure to methylmercury. Demethylation occurs in the liver, phagocytes, brain, and other tissues. The primary routes of excretion of absorbed methylmercury are faeces, urine, and hair. Following exposure to phenylmercury, absorbed mercury is eliminated in bile, faeces, urine, and hair. The kinetics of elimination of absorbed methylmercury exhibit multiple phases, with terminal half-lives estimated to range from 50 to 130 days in humans. Pharmacokinetic models of methylmercury have been developed for humans and a variety of other animal species.

Toxicity Mechanisms of Mercury Exposure

Mercury compounds have the potential to cause poisoning in the body through a number of different mechanisms. Various forms of Hg can attack thiol groups in proteins or membranes. Once Hg has been connected to one or more sulfur amino acid residues in proteins or membranes, a number of physiological functions, including metabolism, can be weakened or blocked.[10]

Mercury has been demonstrated a significant role in the induction of oxidative stress through the promotion of various protein disturbances at different levels starting with mitochondrial dysfunction, increasing the levels of hydrogen peroxides and lipid peroxidation. The high affinity of methylmercury for binding to the sulfhydryl terminal of proteins has been shown

to result in the blockage of active sites of functional proteins, transporters, enzymes, receptors, and other biomolecules.[15, 16, 17] Concurrently, mercury causes reduction in the defense mechanisms, by induction of apoptosis in human T cells and monocytes. Additionally, the binding of mercury to GSH molecules and lipid peroxidation results in an increased risk of developing cardiovascular risk factors, including an elevation in LDL-C molecules, dysfunctional HDL-C, beta cell dysfunction and insulin resistance. Furthermore, mercury plays a role in the development of hypertension by inhibiting the activity of the enzyme nitric oxide synthase (NOS) and the enzyme angiotensin-converting enzyme (ACE), as well as contributing to obesity-related problems by disrupting the differentiation of pre-adipocytes.[7]

Clinical Signs of Acute and Chronic Exposure of Mercury

Different mercury compounds can cause various clinical symptoms. Mercury poisoning frequently results in a false diagnosis because of its slow onset and ambiguous clinical symptoms. The quantity, length, and mode of exposure all affect how clinically a person who has been exposed to mercury will present.[13] The most frequent cause of acute poisoning is inhalation of elemental mercury or ingestion of inorganic mercury. Chronic poisoning is more likely to result from exposure to organic mercury. Regardless of the type of mercury present, the two main organs affected by poisoning are the kidneys and the central nervous system.[18]

1. Clinical signs of acute exposure

Acute toxicity to elemental mercury by inhalation can cause respiratory symptoms. Acute exposure can cause cough with fever, shortness of breath, headache and muscular pains. Early clinical signs, including shortness of breath, fever, chills, taste of metal, and pleuritic chest pain, may be mistaken for metal fume fever. Other potential clinical manifestations include stomatitis, lethargy, confusion, and vomiting. Although the healing process is possible, inhaled exposure can also cause pulmonary problems such as pneumothorax, interstitial emphysema, pneumatocele, interstitial fibrosis, and pneumomediastinum. In addition, exposure to extremely high levels of elemental mercury might result in lethal acute respiratory distress syndrome.[13]

The most common route of acute exposure to inorganic mercury is through the mouth. Acute symptoms may include ashen-gray mucous membranes due to mercuric salt precipitation, vomiting, melena, hypovolemic shock, and severe abdominal pain. Systemic effects typically appear several hours after administration and might longer for many days. These negative consequences include dental sensitivity, mouth soreness, foul smell, mucosal inflammation, gingival irritation, and renal tubular necrosis, which can cause oliguria or anuria.[19, 20]

2. Clinical signs of chronic exposure

Chronic toxicity is typically resulted by prolonged exposure of workers to elemental mercury that is transformed into the inorganic form. The CNS is quickly penetrated by elemental mercury vapor and short-chain alkylmercury compounds, which bind to and inhibit synaptic and neuromuscular transmission-related proteins and enzymes. The blocking of these signals has the usual degenerative repercussions. As a result, the individual may have mild tremors in their hands and fingers, which may eventually spread to their entire leg. The classic triad of symptoms associated with chronic mercury toxicity is gingivitis, tremors, and erethism (a constellation of neuropsychiatric abnormalities that also includes memory loss, insomnia, sadness, shyness, emotional instability, anorexia, flushing, vasomotor disruption, and uncontrolled sweating). Peripheral neuropathy, headache, salivation, visual disruption, sleeplessness, and ataxia are possible clinical manifestations caused by mercury exposure.

Human exposed to inorganic mercury have a widespread condition called acrodynia. Its symptoms include erythema of the soles and palms, irritability, edema of the hands and feet, hair loss, a desquamating rash, tachycardia, pruritus, diaphoresis, anorexia, hypertension, photophobia, sleeplessness, constipation or diarrhea, and decreased muscular tone. It is also known as Pink Disease.

The most frequent source of organic mercury poisoning is eating contaminated food, especially fish. Long-chain and aryl forms of organic mercury are equally as hazardous to humans as inorganic mercury. The motor and sensory centers, cerebral cortex, cerebellum, and auditory center are all targets for organic mercury. After exposure, symptoms frequently take days or weeks to manifest. Before symptoms manifest, the enzymes to which organic mercury binds must be degraded. Dysarthria, visual disturbances, ataxia, mental deterioration, paresthesias, hearing loss, muscular tremors, movement disorders, and paralysis and death are common toxicity symptoms in extreme cases. Mercury is hazardous to the fetus in any form, but methylmercury most easily have the same clinical manifestations including unsteady walking, ataxia, illegible handwriting, and tremors. A loss of facial muscle tone can also cause slurred speech.[21]

IV. CONCLUSION

Mercury is one of the widespread toxic metals detrimental effects on human health. The major sources of mercury in the environment are divided into two types; natural and anthropogenic sources. Mercury typically exists in in three forms, including elemental mercury, inorganic mercury and organic mercury, and MeHg has been considered the most toxic. Humans can be exposed to mercury in the workplace (who live in proximity to former mercury mining or production sites, secondary production (recycling) facilities, hospital incinerators, or coal-fired power plants), other population groups of people who consume large amounts of fish or marine mammals, people who have mercury amalgam dental restorations, people who use consumer products containing mercury such as traditional or herbal remedies, or cosmetics, including skin lightening creams. Toxic effect of the mercury was documented in several historical accidents that revealed by neurological symptoms, the cardio metabolic syndrome, liver and renal diseases via the oxidative stress mechanisms, due to the strong ability of the mercuric compounds to bind to the sulfhydryl terminal of vital proteins and enzymes.

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