Biological Effects of Common Toxins: A Complete Review by Shreya Rangaswamy

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Abstract

The prevalence of poisons today poses a significant threat to human health, as there have been more than 2 million poisoning cases in the US each year and the number increases every year. This paper will detail the different mechanisms of action and the associated health implications of different poisons such as anthrax, arsenic, mercury, and lead. Each poison exhibits a unique mode of action, influencing specific cellular proteins and pathways. Although there has been a lot of research conducted on these matters, there are not many details available on specific cellular molecules poisons interact with and how health symptoms occur. Understanding these interactions is crucial for developing treatment. This scientific review seeks to raise awareness about the pervasiveness of poisons and toxins in modern society and the potential for adverse health outcomes. Poisons are not only capable of causing death but also play a crucial role in the emergence of various diseases. It is also very important for implementing effective public health measures and minimizing their impact on human well-being. This research contributes to the broader scientific community as it answers the fundamental questions of how poisons work and what detrimental effects they inflict on the body.

Introduction to Common Toxins

Understanding the profound impact of poisons on human health is crucial for protecting people from potential harm and promoting public health and safety. A poison can be defined as any substance if taken in enough quantity causes physiological or anatomical harm (Amir 2019). Poisons have been a longstanding concern throughout history due to their capability of creating various distressing illnesses or death when absorbed by the body. Whether it be natural toxins from plants and animals, or synthetic chemicals developed for various reasons, the diverse characteristics of poisons require thorough research of their effects on the human body.

Historical events where poisons have been deployed have been instances of bioterrorism where poisons were intentionally used to inflict death or illness to humans, livestock, or crops (Rathjen, 2021). The research of the poisons and their effects includes analyzing the exposure pathways which are the routes through which the poisons enter the body and target specific organs. Certain toxins produce spores, which are cells that attack particular organ systems and spread the poison throughout the body (Vyas, 2023).

The poisonous spores disperse across the entire organism by interacting with various enzymes, which are proteins that facilitate chemical reactions with specific biological compounds within cells. In doing so, the poisons start to disrupt several cellular pathways, such as cellular respiration and DNA synthesis and repair processes. Such reactions can trigger diseases in the body and ultimately affect the health and well-being of the individual. Knowledge of the development of preventive measures and treatment procedures to reduce the harm and risks associated with poisons is extremely important for both healthcare professionals and the

general public. In this next section, common poisons such as anthrax, arsenic, mercury and lead will be evaluated for their specific mechanisms and effects on the human body.

Anthrax causative agent: *Bacillus anthracis*

Anthrax is spread by spores produced by a bacteria called *Bacillus anthracis*, which can enter the body through touch, consumption, or inhalation. The poison has commonly been used in several situations as a weapon of bioterrorism, such as in the 2001 anthrax attacks. *B. anthracis* is an aerobic, immobile bacterium enclosed in a thick cell wall (Lowe, 2012). The bacterium is also non-hemolytic, which means that it does not cause hemolysis or the destruction of blood cells in the body. Spores released by these bacteria are resistant, or unaffected by heat and several types of disinfectants and radiation. These spores develop and grow in animal tissues or blood, but they do not survive well outside of the host, where they cannot obtain necessary nutrients like amino acids, glucose, and nucleosides (Lowe, 2012).

When anthrax spores are introduced into the body, they are phagocytosed; indicating that they are engulfed by cells of the immune system called macrophages. In situations where surroundings support the growth and reproduction of bacteria, the dormant spores become active and start producing virulence factors, or molecules that are required for a bacterium to cause disease while infecting eukaryotic hosts such as humans (Abedon, 2009). At this point, the spores enter the bloodstream and are called vegetative bacteria, denoting the fact that they can grow and reproduce. For the bacteria to develop full virulence, it requires an antiphagocytic capsule to protect themselves from the macrophages (Lowe, 2012). *B. anthracis* produces three toxin components, and they bond together to form 2 toxins: edema toxin and lethal toxin (Lowe, 2012). These are the poisonous toxins that cause observable symptoms and possibly death to the individual. Edema toxin causes edema in the body tissues, which is swelling due to too much fluid trapped in the cells as water homeostasis is disrupted. It also weakens the neutrophil cells of the immune system, which makes the host more vulnerable to infections (Lowe, 2012). Lethal toxin causes the release of proinflammatory cytokines, which are small proteins that act to make diseases worse (Dinarello, 2000). This also results in a circulatory collapse, where almost all the blood circulation in the body is interrupted. Other results of the toxins are tissue edema and profuse hemorrhage, which is copious loss of blood from blood vessels (Johnson, 2023). There are different ways in which anthrax can be absorbed into the body, and these include inhalational, cutaneous, and gastrointestinal anthrax.

Inhalational Anthrax

One of the types of anthrax is inhalational anthrax, where the spores are deposited in the alveolar spaces in the lungs. The spores must be between one and five micrometers in size. If the particles are smaller than 1 micrometer, they get stuck to the mucus on the walls of the bronchi in the lungs, and if they are larger than 5 micrometers are trapped by mucus in the trachea and never reach the alveoli (Lowe, 2012). The spores do not cause infections in the lungs but are engulfed by macrophages in the alveoli and carried to lymph nodes. In the lymph node, the spores germinate and produce the toxins mentioned above, which are released into the blood and cause edema, hemorrhage, and other conditions like necrosis (death of body tissue) and septic shock (a dangerous drop in blood pressure). Inhalational anthrax is usually a biphasic disease, having two phases of symptoms. The first stage of symptoms includes flu-like

symptoms like fatigue, fever, chills, sweats, and others (Lowe, 2012). Pulmonary complaints, like wheezing or breathing issues, are not very common, but gastrointestinal symptoms such as nausea and vomiting are often experienced. The second stage of inhalational anthrax includes chest pains, dyspnea (difficulty in breathing), and stridor (abnormal respiratory sound because of irregular airflow in the airway) (Sicari, 2023). Eventually, the host would develop hemorrhage and multiorgan failure, which would lead to death. However, the infected individual can have a higher chance at survival if they are treated with antibiotics, glucose infusion, mechanical ventilation, and other methods (Lowe, 2012).

Cutaneous Anthrax

Cutaneous anthrax, or anthrax obtained by contact with the skin, is the most common type of anthrax infection. It is often acquired by people who have had exposed skin contact to animals or animal products, and the spores can also be obtained from cuts or injuries in the skin. Within a few weeks of exposure, lesions, or damaged regions in the skin start forming. The lesions then produce vesicles, which undergo necrosis to form a black eschar (a cluster of dry dead tissue) on the skin surrounded by purple vesicles and edema (Lowe, 2012). Another condition related to cutaneous anthrax is lymphangitis, which is the inflammation of lymphatic vessels. If left untreated with antibiotics, anthrax can cause conditions like hyponatremia (abnormally low sodium levels in the body), thrombocytopenia (lack in number of platelets, resulting in bleeding problems), septic shock, and eventually death (Lowe, 2012).

Gastrointestinal Anthrax

Gastrointestinal anthrax is often caused by the consumption of incompletely cooked or contaminated meat from diseased animals (Lowe, 2012). This type of anthrax can be further divided into two syndromes: abdominal and oropharyngeal anthrax. In abdominal anthrax, spores are deposited in the lower gastro-intestinal tract, causing lesions in the intestine, and the bacteria spreads from there (Lowe, 2012). Symptoms include abdominal pain, nausea, vomiting, bloody diarrhea, and others. A condition called ascites also develops, in which fluid collects in spaces in the abdomen. Also, massive blood loss, fluid imbalance, intestinal perforation (when a small hole in the intestine causes the contents to leak into the abdomen) and sepsis syndrome (in which the body doesn't respond properly to an infection) can cause mortality in the host. Oropharyngeal anthrax is less common and less severe than abdominal. In this case, lesions form in the middle part of the pharynx. Symptoms for this include fever, cervical edema, and respiratory difficulties.

The Impact of Mercury on Cellular Systems and Health

Mercury poisoning can affect several organ systems in the body, like the cardiological, pulmonary, neurological and several others. Mercury is recognized as an extremely toxic substance, and mercury poisoning has increased as the levels of mercury in the environment's soil, water and air have increased from processes like burning fossil fuels or volcanic eruptions (Ankrah). There have been over a thousand cases of mercury poisoning reported in 2022 in the US, and it is much more prevalent in other countries, especially among mining communities or seaside towns where food could become contaminated. Elemental mercury is used in various industries, such as thermometers, sphygmomanometers, electrical materials, and

electrochemical operations (Rice, 2014). Occupational exposure, particularly in industries like mining and dentistry, can lead to mercury poisoning. Mercury can also be obtained by consuming fish from waters that have been methylated. Mercuric mercury compounds, resulting from mercury combining with chlorine, sulfur, or oxygen, include highly toxic substances like mercuric chloride and mercury fulminate (Azevedo, 2012). Some organomercury compounds were used as pesticides and antiseptics.

Different Forms of Mercury

Mercury exists in several forms of elemental, organic and inorganic compounds in the environment. Elemental mercury (HgO) in its liquid form poses low health risks as it is poorly absorbed in the body. However, in vapor form, it can be easily absorbed through the lungs and can pass through cell membranes, the blood-brain barrier, and placental barriers, reaching vital organs (Azevedo, 2012). In the bloodstream, mercury oxidizes and transforms into inorganic mercuric mercury (Hg++) and mercurous mercury (Hg+). Mercuric mercury in the bloodstream binds to various substances and accumulates in placenta and fetal tissues but does not efficiently cross the blood-brain barrier. Evidence suggests its transport via amino acid transporters and accumulation in the brain through binding to cysteine (Azevedo, 2012). Organic mercury compounds, also known as organometallic, form through a covalent bond between mercury and a carbon atom in an organic functional group like methyl, ethyl, or phenyl. Methylmercury (CH_3Hg^+) is the most common form absorbed by the body, primarily produced by microorganisms through the methylation of inorganic mercuric ions in soil and water (Azevedo, 2012).

The Systemic Effects of Mercury

At the cellular scale, mercury can cause changes in the permeability of the cellular membrane, changes in the molecular structures of macromolecules, and damage in the DNA. Mercury can also cause oxidative stress, where antioxidant levels in our body are low, and mitochondrial dysfunction, where the mitochondria malfunction and ATP synthesis decreases Additionally, the homeostasis of calcium in the cell is disrupted, and lipid peroxidation occurs, which in turn leads to membrane rupture and cell death (Cai, 2005). Therefore, understanding the consequences of mercury exposure remain crucial for protecting cellular cohesion and overall health.

Mercury can be inhaled and enter the circulatory system through the lungs. Elemental mercury vapor that is inhaled also deposits in large amounts in the brain. Methylmercury doesn't reach the brain as effectively, but significant amounts of it are still deposited in tissues throughout the body. Mercury salts mainly damage the lining of the intestinal tract and the kidneys. Many forms of mercury can slow down the production of enzymes that help in the digestive process like trypsin, chymotrypsin, and pepsin. Symptoms of mercury in the digestive system include abdominal pain, indigestion, and bloody diarrhea, among others. Mercury can also destroy microbes and bacteria that live in the intestines, which would cause undigested food particles to enter the bloodstream, which in turn would cause disruptions in immune reactions. Mercury poisoning causes a variety of kidney diseases, like renal cancer and chronic renal disease among several others. Mercury poisoning impairs immune system functioning by causing damage to polymorphonuclear white blood cells, hindering their ability to eliminate foreign substances. This makes the host more vulnerable to infections. People who are infected with

mercury are also more likely to develop immune and autoimmune conditions like allergies, arthritis, eczema, epilepsy, and several others (Rice, 2014).

When mercury accumulates in the heart, it can lead to cardiomyopathy, angina (a type of chest pain because of less blood flow to the heart), and other types of heart disease. Mercury might also cause anemia as it can compete with iron molecules to bind with hemoglobin in the blood (Rice, 2014). According to some research, mercury might also be linked to forms of cancer such as leukemia and Hodgkin's disease. Mercury induces oxidative stress, generating free radicals and reducing antioxidant enzyme activity, increasing the risk of cardiovascular disease (Azevedo, 2012). Chronic inhalation of mercury can cause tremors, sleep disturbances etc. Several pulmonary conditions like bronchitis and pneumonia can also be caused by mercury poisoning.

The nervous system is the main target for mercury poisoning. One problem mercury causes in the nervous system is that it impairs the cellular detoxification process, which is vital in removing harmful chemicals from the body, so the cells either exist in chronic malnutrition or die off (Rice, 2014). Inorganic mercury can hinder the synthesis of some important components of a nerve cell structure, like tubulin and actin. Another significant impact of mercury is that it's known to increase reactive oxygen species in the body, which results in lipid peroxidation and apoptosis (Azevedo, 2014). Individuals who were exposed to mercury prenatally, the toxin can alter the cytoarchitecture, or the cellular composition of the central nervous system's tissues which was shown to damage motor and memory functioning following birth (Im, 2021) . Mercury can obstruct the organization of microtubules in the cytoskeleton and interfere with intracellular signaling (Azevedo, 2014). Studies have shown that the poison can cause many disturbances in intestinal peristalsis (the involuntary muscle movement in the digestive tract) by increasing activity of nitrergic nerves in the body (Bódi, 2019). Additionally, mercury can damage the barrier between blood and the brain and assist other toxic substances to enter and infect the brain. Some symptoms caused by mercury poisoning of the nervous system are depression, paranoia, hallucinations, memory loss and others. The toxin can also affect the sensory nervous system, causing blindness, reduced sense of smell and motor and behavioral dysfunctions that are related to autism (Rice, 2014). In summary, mercury poisoning detrimentally affects several organ systems by impairing various processes, disrupting structural components of cells and inducing a wide array of symptoms.

Health Effects of Arsenic Exposure

Arsenic is a deadly toxin that can affect almost all organ systems in the body as a poison and as well as a carcinogen, or a compound that can cause cancer in organisms (Schrenk 2018). Arsenic is used in several industries, such as the manufacturing of paints, cosmetics, and agriculture pesticides. An estimated 140 million people in at least 70 countries have been drinking water containing arsenic at levels above the WHO provisional guideline value of 10 µg/L. Like anthrax, arsenic is also commonly used as a weapon for murder and bioterrorism, as several arsenic compounds look similar to white sugar. Since ancient Greek times, arsenic was also for healing purposes, and the long-term usage of it caused many diseases as people continued to poison themselves thinking of it as medicine. In recent times, arsenic is also used reduce the symptoms of leukemia in patients because of its ability to cause apoptosis, or the

process of organized death of cancer cells in the patients. The toxin does this by releasing an apoptosis-inducing factor (AIF) from the mitochondrial intermembrane space to the nucleus of the cell. There, it executes apoptosis by breaking and separating the DNA and condensing the chromatins, which eventually leads to the cell dying off. Arsenic is also used in many Asian traditional medicines, but overall arsenic is more commonly used as a toxin rather than for helpful medicinal intentions.

Routes of Arsenic Exposure

Just like anthrax, people can get infected by arsenic through inhalation, ingestion, and absorption through skin. The most common cause of arsenic exposure is from ingesting contaminated water, but arsenic is also commonly ingested through solid foods like seafood and algae. Arsenic compounds can also enter the plant food chain from agricultural pesticides and other products. Most of the arsenic that enters the body is absorbed through the small intestine, because the pH in that area is optimal for arsenic absorption. The poison molecules then go through a process called hepatic biomethylation, after which they form acids called monomethylarsonic acid and dimethylarsinic acid. After arsenic enters the body, it can result in either acute or chronic poisoning depending on the degree of its effects on the body.

Arsenic is found mainly in two forms of compounds – arsenite (As III) and arsenate (As V). Arsenite is about 60 times more poisonous than arsenate. When arsenic infects the cell, it deactivates up to 200 enzymes that are involved in DNA replication and repair processes. It also disrupts cellular energy pathways by taking the place of Phosphorus in energy compounds like ATP. Additionally, arsenic also causes lipid peroxidation and DNA damage. These are only some instances of how it affects functioning of the body at the cellular level, and which will be presented in further higher detail in the upcoming sections.

Acute Arsenic poisoning

During acute arsenic poisoning, the arsenic is mostly concentrated in the liver and the kidneys. The poisonous acid can cause acidosis, or the buildup of excess acid in body fluids. When less than 5 mg of arsenic is ingested, it is usually excreted from the body within a day (Ratnaike, 2003). The patient would face symptoms like vomiting and diarrhea, but no treatment is necessary. In the deadliest cases of acute poisoning anywhere from 100-300 mg of arsenic is absorbed. In this case, depending on how much was consumed, the patient can die in one to four days, usually because of extreme dehydration, reduced blood volume and consequently circulatory collapse. More severe symptoms would be experienced such as nausea, abdominal pain, acute psychosis, cardiomyopathy, seizures, and others. Several conditions in the body like hepatic steatosis (fatty liver disease) and inflammation of the gastrointestinal tract have been reported (Ratnaike, 2003). Many abnormal blood conditions also occur from poisoning, like hemoglobinuria (blood in urine), intravascular coagulation (abnormal blood clotting in blood vessels) and bone marrow depression (when the bone marrow doesn't produce enough blood cells) among others. Respiratory failure and renal failures are also frequent symptoms. Relating to the nervous system, the most common symptom is peripheral neuropathy, where the nerves in the peripheral nervous system are damaged. Encephalopathy, or any form of brain damage and dysfunction, is also a frequent result of arsenic poisoning (Ratnaike, 2003).

Chronic arsenic poisoning

When a person is exposed to arsenic for long periods of time, it can cause disease and even cancerous tumors as the toxin accumulates in the heart, lungs, muscles, and several other organs. Some of the initial signs of arsenic poisoning are changes in the skin like hyperpigmentation (darkening of certain spots in the skin, characteristically in a raindrop shape for arsenic poisoning) and keratosis (rough patches on patches on skin). As mentioned, arsenic is associated with several types of cancer and malignancies in several organs. Though the mechanism of action that results in cancer is not complete, arsenic might be involved in disrupting DNA repair, DNA methylation, and/or cause abnormalities in the structure of chromosomes in the cell. Arsenic is known to disrupt various pathways in the cell like ones involved in cell migration, cell adhesion, cell survival and others (Singh, 2011). In serious conditions, chronic arsenic exposure can cause a form of cancer known as Bowen's disease, which is cancer in the squamous cells in the epidermis of the cell (Ratnaike, 2003). It also promotes other types of skin cancer by acting with sunlight, blocking DNA repair, and dysregulating cell cycle control and DNA methylation. Arsenic can act as a tumor-promoter and helps tumors grow larger by changing the expression of genes involved in cell growth and resulting in rapid reproduction of cells (Singh, 2011). Moreover, it acts as a teratogen, which means that it can cause fetal abnormalities when high levels are consumed in pregnancy.

Impacts of Arsenic on the Cell

Within the cell membrane of vascular endothelial cells and smooth muscle cells, arsenic triggers the enzyme NADPH (nicotinamide adenine dinucleotide phosphate) to increase the production of reactive oxygen species (known as ROS) like superoxides and hydrogen peroxide. These ROS can bind with nitric oxide to form peroxynitrite, which is known to cause several cellular symptoms like modulations of cell signaling and necrosis/apoptosis. Arsenic activates the expression of genes related to atherosclerosis (inflammatory diseases characterized by thickening or hardening of arteries), like HO-1, MCP-1 and IL-6. It also does this by increasing the synthesis of inflammatory mediators (molecules part of the immune response that induce inflammation), an example of which is leukotriene E4. Additionally, arsenic stimulates the protein kinase C alpha, which after many chemical reactions leads to increased permeability of the endothelium (Singh, 2011). Arsenic causes vascular endothelial dysfunction and many other cardiac issues by lessening the activity of endothelial nitric oxide (eNOS) which plays a critical role in regulating and maintaining a healthy cardiovascular system (Tran, 2022). Moreover, arsenic brings about vasoconstriction, or the narrowing of blood vessels, which results in elevated blood pressure and ventricular arrhythmias. Arsenic poisoning is also linked to cardiomyopathy and ischemic heart disease (weakened heart due to reduced blood flow). A peripheral vascular disease (where blood flow is reduced to peripheral system) called Blackfoot disease is a unique condition caused exclusively by arsenic poisoning (Singh, 2011).

High concentrations of arsenic can induce type II diabetes in the individual by reducing the expression of a protein called PPAR-γ, which reduces the responsiveness of insulin in the body (Singh, 2011). Arsenic interrupts the production of glucose and restricts the ATP-dependent insulin secretion by replacing a phosphate group from ATP and instead forms ADP-arsenate. Arsenic also binds with disulfide groups of insulin, insulin receptors and enzymes involved in

glucose metabolism. Furthermore, high levels of arsenic can cause hypoglycemia, as it decreases the activity of the protein glucose-6-phosphatase in the liver and kidneys. Arsenic diminishes the expression of insulin mRNA, the gene that encodes insulin, and increases the expression of several stress mediators like NF-κB (Singh, 2011). Arsenic can cause hyperglycemia too, by slowing down insulin-dependent glucose uptake. The main targets of arsenic in the body are the liver and the kidneys. In chronic poisoning, the symptoms of diarrhea and vomiting are worsened, along with a condition called hepatomegaly, where the liver is enlarged beyond its normal size. Arsenic can cause acute tubular necrosis in the kidneys, where the tubule cells are damaged. As mentioned above, when arsenic increases the production of ROS, the toxin increases lipid peroxidation and cellular damage in renal and hepatic (liver) tissues, in addition to prompting apoptosis in them by increasing pro-apoptotic proteins in the organs. Furthermore, arsenic induces hepatotoxicity by upregulating various enzymes in the liver (Singh, 2011).

Neurotoxic effects

For many poisons, the nervous system is the main target, and arsenic causes several effects in it such as Parkinson's Disease, peripheral neuropathy, cognitive impairment, memory loss, and changes in behavior (Singh, 2011). Cerebrovascular disease, where blood flow to the brain is reduced, and cerebral infarction, another name for a type of stroke, are also associated with arsenic toxicity (Ratnaike, 2003). The poison can also bring about apoptosis in the cerebral neuron cells and DNA damage in the brain. Arsenic can cross the blood-brain barrier freely, and it also affects the nervous system by increasing reactive oxygen species, decreasing superoxide dismutase (an enzyme that catalyzes many chemical reactions in the cell), and decreasing glutathione levels (a molecules that's involved in tissue repair, protein synthesis and other functions in the body). Arsenic also causes changes in the functioning of neurotransmitters like monoamines, acetylcholine, and glutamate. Arsenic can cause axonal degeneration, or the loss of communication between neurons, by destabilizing the framework of the cytoskeleton. (Wang, 2009). That causes thiamine deficiency in the body, which leads to encephalopathy and other neuronal problems. Arsenic inhibits the NMDA receptors in the hippocampus of the brain, which detect neurotransmitters and plays a crucial role in learning and memory (Singh, 2011). Ultimately, this results in cognitive dysfunction and neurobehavioral diseases.

Sources of Lead Exposure

Lead is an important and essential metal that has been used since ancient times for various purposes such as in food production, plumbing systems, battery manufacturing, fuel and in many more industries (de Souza, 2018). Despite a decline in its use, it remains a serious public health concern, particularly in developing countries. It is one of the most prevailing poisons in today's world, as an estimated one million people a year die from lead poisoning. Lead compounds primarily enter the body through the respiratory and gastrointestinal tracts, with respiratory absorption being more effective. Additionally, lead can also be absorbed through the skin, particularly in the form of hydrophobic organic compounds like tetraethyl lead, a compound consisting of four ethyl groups bound to a central lead atom. In the bloodstream, about 90% of the lead compounds bind with red blood cells and interact with plasma and many proteins containing thiol and sulfhydryl (de Souza, 2018). Notably, bones and teeth can store lead, and

its release into the bloodstream can occur from processes like bone remodeling. Hormonal changes like lactation and pregnancy can stimulate lead mobilization in the body, which increases lead exposure to the fetus, which in turn can impair its growth and development. (de Souza, 2018) Lead also tends to accumulate in soft tissues, particularly the liver, kidneys, and the brain. Lead can cross the blood-brain barrier through protein channels that transport ions to brain tissues, though the endothelial cells of the choroid plexus of the brain fight the lead molecules to protect the brain from them. Lead poisoning has a wide array of symptoms, as it affects the nervous, cardiovascular, immune, skeletal, reproduction, renal and other systems by interacting with various chemicals and proteins and disrupting metabolic pathways in the cell.

Lead's Impact on Cellular Pathways

There are 23 proteins that bind to lead, and they are categorized into 3 types: stimulation (when lead increases protein activity), inhibition (when lead decreases the protein activity), and binding (where the lead's effect on the protein's function hasn't been observed yet). These proteins are an integral part of several cellular pathways, and the interference of lead affects them in many ways, like it can cause anemia and disrupt cell homeostasis in blood cells. A protein called d-aminolevulinic acid dehydratase (ALAD) is one of the main targets of lead, and it helps in forming d-aminolevulinic acid (ALA), which is a crucial component in the heme synthesis pathway. ALAD also acts in the process of protein degradation and interacts with proteasome complexes, and the presence of lead strongly slows down all these major processes and increases the acid level in blood. Another protein that is inhibited by lead is Pyrimidine 50-nucleotidase, which results in the disruption of RNA catabolism and the accumulation of a large amount of RNA in red blood cells. Lead is also known to cause oxidative stress and lipid peroxidation in cells, which brings about serious mutations in the DNA, and eventually apoptosis (de Souza, 2018).

Lead often replaces metals like zinc or calcium in some cellular processes, and this can either hyperactivate or deactivate the function of proteins. Scientists have hypothesized that the way lead crosses the blood brain barrier is by pretending to be calcium. Additionally, lead replacing calcium can damage many pathways containing calmulodin, a calcium-binding protein that is an integral part of metabolic functions like intracellular movement, muscle contraction, nerve growth, immune response, and several others. Lead also impairs the function of another protein called cyclic adenosine monophosphate, or cAMP, which leads to neurotoxic effects as it impairs neurotransmitter release and axonal growth in nerves (de Souza, 2018). When lead ions in the cytosol of the cell reach the nucleus, they affect many nucleic proteins, like apyrimidinic endonuclease 1 (APE1), a major protein involved in DNA repair, and many others (de Souza, 2018). Overall, lead can diminish the transcription of genes involved in the immune system, neurodevelopment, and xenobiotic metabolism (the elimination of useless compounds from the body) .

Many proteins in the body act as chelators, which are compounds that form complexes with metal ions (Johnson 2010) and prevent the toxic effects of metals like lead. Metallothionein is an example of a chelator, as it binds lead ions in the cell and acts as a biomarker for lead poisoning, which means that it shows what is happening in the cell at a given moment. Additionally, when metallothionein is present in the placenta (the organ in which the fetus

develops), it can prevent lead from entering it and affecting the fetus. Other chelator proteins include thymosin b4 and a2l-microglobulin. In the kidneys and glial cells of the body, lead often forms structures called inclusion bodies, which are large aggregates of lead molecules and cellular proteins (de Souza, 2018). These inclusion bodies are first formed in the cytoplasm and can then move to the nucleus of the cell. It is suggested that these inclusion bodies can protect the cellular structures from the poison, as they sequester or isolate the lead molecules in the cell. An example of this type of protein is GRP78 (78 kDa glucose-regulated protein). The protein is found in the endoplasmic reticulum, and along with being a binding site for lead, GRP78 performs other functions such as ensuring the correct folding of new proteins and maintaining calcium levels in the cell (de Souza, 2018). These types of interactions with lead and certain proteins still need to be researched and understood better, because the studies as of now aren't completely conclusive.

Clinical manifestations of lead poisoning

The most studied effect of lead in the body is its chronic and acute damage on the nervous system. When the nervous system is infected with poisons like lead in its early developing stages, it is the most vulnerable to permanent damage for the rest of the individual's life. It can cause severe cognitive diseases and deficiencies in spatial and learning and memory pathways. When an individual is infected by lead in later years of life, it can cause neurodegenerative diseases like Parkinsons's and Alzheimer's (de Souza, 2018).

Predominantly, the symptoms of lead poisoning are associated with the central nervous system, causing dullness, irritation, and poor attention. The damage in the blood-brain barrier can result in conditions like cerebral edema and hemorrhages in the brain. (de Souza, 2018). When the individual is exposed to lead prenatally as a fetus, their brain-blood barrier is significantly more ineffective in protecting the nervous system during development. Some severe symptoms include seizures, paralysis, and coma. High levels of lead exposure can also cause kidney diseases like chronic nephritis and nephropathy, which is characterized by the failure of the functioning of the kidneys (de Souza, 2018). Another effect of prenatal lead exposure is the impairment of the axial skeleton. Also, when lead accumulates in the bone marrow it can hinder heme synthesis (heme is a compound that is a key part of hemoglobin).

There have been experiments conducted on how a compound of lead, lead (II) acetate, stimulates and increases the production signaling proteins like Interleukin 8, Macrophage Inflammatory Protein-1 Alpha, , Monocyte Chemoattractant Protein-1 and others from PMBC, (peripheral blood mononuclear cells, which are a major part of the immune system). This can considerably alter DNA transcription processes, which in turn can affect the expression of several cellular metabolic enzymes and chelator proteins. The changes in expression of genes can be used as biomarkers to assess lead exposure effects. Research shows that even blood lead levels lower than 10 µg/dL (the CDC recommended concern level) can cause cognitive dysfunction, neurobehavioral disorders and renal impairment (Gillis, 2012). Overall, high levels of lead concentration in the bloodstream can lead to several changes in gene expression and cellular responses.

Conclusion

In conclusion, this paper explores the complex details of the cellular effects and health implications of common toxins such as anthrax, mercury, arsenic, and lead to protect public health and advance the knowledge of problems of poison exposure. The research includes the various modes of entry of each of the toxin into the body, the specific organs they target, and the important cellular pathways that they disrupt. Each poison poses distinct challenges to the overall health and well-being of the human affected. While there was a lot of valuable information available for the cellular mechanisms of actions of some poisons, the research was still limited in the way that there wasn't a large amount of information available for some other poisons. Additionally, many hypotheses are not completely concluded yet. Despite these limitations, the significance of this study is its contribution to the broader understanding of poisons' impact on human health. The detailed analysis of anthrax, mercury, arsenic, and lead enhance awareness among the public. This knowledge can serve as a foundation for development for preventive measures, treatment strategies and environmental regulations to reduce the risks associated with these poisons.

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