Lead Toxicity in Human Body: A review

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Abstract

Lead poisoning is a serious environmental illness that has disastrous effects on human health. The human body hardly has any function that is unaffected by lead poisoning. Lead is still widely utilized in poorer nations, despite some degree of regulation over its usage in places like the US and Canada. This is mainly because lead has special physical and chemical characteristics that allow it to be used for a wide range of purposes. Because of this, humans have been using lead for many years, and as a result, it is frequently found in the environment as a contaminant. Due to its widespread usage and extreme persistence in the environment, lead poses a severe concern and is becoming more and more prevalent in practically every nation. This article examines the publications mentioned in the literature that have been updated with new information on lead toxicity. The harmful consequences of lead on the brain system, kidneys, and reproduction are also emphasized. Lastly, some recent changes to the methods for treating lead toxicity are discussed.

Keywords: lead toxicity, lead poisoning, heavy metals, environmental health

Introduction

The most significant hazardous heavy metal in the environment is lead. Its use dates back to historical periods because of its significant physico-chemical characteristics. It is a widely dispersed, significant, and hazardous environmental chemical on a global scale (Mahaffay, 1990a). It's crucial qualities, such as its resistance to corrosion, softness, malleability, ductility, and weak conductivity, make it difficult to stop using it. Because of its continued use and inability to biodegrade, its concentration builds up in the environment, posing a growing risk. Work in industries related to lead, such as leaded gasoline, lead smelting and combustion, pottery, boat building, lead-based painting, lead-containing pipes, battery recycling, grids, arm industry, pigments, book printing, etc., are the main sources of human exposure to lead and its compounds. Its broad use has ceased in many nations, but it is still utilized in a variety of industries, including auto repair, battery production and recycling, refining, smelting, and other processes. Almost all of the body's organs are impacted by the extremely toxic element lead. When lead poisoning occurs, the nervous system is the organ most commonly impacted, in both children and adults. However, children are more affected by poisoning than adults

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are. This is because, in comparison to adult tissues, their internal and exterior tissues are softer. Adults who have been exposed for an extended period of time may perform worse on certain cognitive performance tests that gauge nervous system activities. Young children and infants are particularly vulnerable to even low amounts of lead which may contribute to behavioural problems, learning deficits and lowered IQ (Rubin & Strayer, 2008). Anaemia and elevated blood pressure have been linked to long-term lead exposure, with the majority of affected individuals being middle-aged and older. Fatal exposure to high levels of lead has been connected to severe brain and renal damage in both adults and children. High levels of lead exposure during pregnancy may result in miscarriage. Male fertility has been reported to decrease with chronic lead exposure (Sokol & Berman, 1991). Lead poisoning is associated with an increased risk of blood problems and neurological impairment.

Detection of Lead Toxicity

There are several ways to find high blood lead levels. Lead poisoning can be identified by looking for abnormalities in blood cells that can be seen under a microscope or by removing thick lines from children's X-ray bones. However, measuring the lead content of blood samples is the primary method for identifying high levels of lead in the body. Nevertheless, the amount of lead that is stored in the body cannot be determined by this test; it only reports on the amount of lead that is in the bloodstream. The standard increased blood lead level for adults is 10 µg/dL and for children is 5 µg/dL of whole blood, according to the Centers for Disease Control and Prevention (CDC, 2012). Lead levels for children were previously set at10 µg/dL. Each person's clinical manifestations emerge differently based on additional contextual circumstances. While some people exhibit obvious clinical symptoms even at lower lead levels in their bodily fluids, others remain asymptomatic even at higher levels. Children are particularly vulnerable to the effects of lead since their organs are typically still developing. As a result, blood lead levels need to be lowered and monitored often, especially in areas where exposure is anticipated.

Effect of Lead Toxicity

The symptoms of lead poisoning have been documented as being severe throughout human history. Lead salts used in pottery glazes that are leached by acidic fruit juices have been identified as the source of occasional lead poisoning. Numerous reports have addressed Beethoven's passing. Many of them have come to the conclusion that his doctor's deadly doses of lead-based treatment are what killed him. It was discovered through analysis that he had high lead levels in his hair (Mai, 2006). Additionally, it is believed that lead was illegally added to wine in the eighteenth and early nineteenth centuries in order to sweeten it and give it a fresh appearance (Mai, 2006). Additionally, it is believed that lead was illegally added to wine in the eighteenth and early nineteenth centuries in order to sweeten it and give it a fresh appearance (Mai, 2006). Lead poisoning is thought to be the main factor behind the collapse of the anemia cause in several cases because lead inhibits ferrochelatase and porphobilinogen synthase, which prevents the formation of porphobilinogen and the iron's incorporation into protoporphyrin IX. This, in turn, prevents heme synthesis or results in ineffective

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heme synthesis, which in turn causes microcytic anemia (Cohen et al., 1981). Lead functions as a calcium analogue, interfering with ion channels as one of the ways by which it impairs cognition. Pb2+ has been found to be a strong, selective, reversible blocker of voltage-dependent calcium channels at low concentrations (Busselberg et al., 1993). In a recent study, the authors demonstrated that sodium selenite mitigated the harmful effects of lead nitrate on the blood cells of rats. Additionally, they demonstrated that the effects of lead nitrate were more detrimental to rats with diabetes than to rats without the disease (Bas et al., 2015). Low levels of lead exposure were used to study oxidative stress in first-grade Uruguayan children, and the results suggested that lead may have negative impacts on oxidative stress (Roy et al., 2015). Workers exposed to lead were shown to have impaired respiratory function along with higher blood lead and zinc protoporphyrin concentrations (Jurdziak et al., 2015).

Possible Pathway of Lead Toxicity

Lead poisoning mostly happens when food or water tainted with lead is consumed. Poisoning, however, can also occur by unintentionally consuming tainted dust, soil, or paint that contains lead. Lead is thought to enter the bloodstream swiftly and is known to negatively impact several organ systems, including the immune system, kidneys, central nervous system, and cardiovascular system (Bergeson, 2008). The majority of pharmaceutical firms have established a daily maximum lead consumption of 1.0 μg/g; nonetheless, extended exposure to even this low level of lead can be harmful to humans. Blood lead levels are also raised as a result of occupational exposure. Girls who have higher blood levels tend to go through puberty later (Schoeters et al., 2008). The amount of lead in blood that can be regarded as safe below no threshold has not been determined. Children's cognitive capacity was discovered to be reduced by extremely low but permanent levels of lead exposure (Needlemann et al., 1990). The use of lead in paints has drastically decreased due to the risks of lead poisoning, mostly in children, from paint pigments. Nonetheless, significant levels of lead paint may still be present in older homes (Levin et al. 2008). Lead poisoning in children might occasionally result from this. While yellow lead chromate is still in use, white lead paint has been fully removed from the market in industrialized nations. Sanding should not be used to remove old paint since it releases dust that is inhaled (Marino et al., 1990). Lead and other heavy metals have also been discovered to be present in traditional remedies. Traditional medicine use has been linked to a range of ailments (Karri et al., 2008). It is widely believed that Ayurvedic medications have high levels of heavy metal contamination. Consumers of ayurvedic medicines had their blood lead levels assessed in a recent study. According to Breeher et al. (2015), out of the 115 individuals, 40% had blood lead levels that were elevated and were 10 μg/dL or above, and 9.6% had blood lead levels that were more than 50 μg/dL. It was recently reported that a patient receiving Chinese traditional medicine had dysplastic alterations in erythroid precursors as a result of lead poisoning (Lv et al., 2015). Guidelines on the consumption of certain traditional medicines that have a high concentration of lead and could

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expose users to lead or lead compounds were also released by the Centers for Disease Control and Prevention. For instance, Myanmar (Burma) and Thailand employ Daw Tway as a digestive aid. Lead levels in Daw Tway samples were as high as 970 parts per million (ppm) according to analysis. Additionally, the Daw Tway samples had elevated amounts of arsenic—up to 7,100 ppm. Fruits and vegetables polluted with high amounts of lead from their growing soils may be the source of lead toxicity. Lead levels in the soil are typically caused by pipes, paint, and leftover emissions from leaded gasoline used before to the Environment Protection Agency's rule being implemented in the early 1980s.

Occupational exposure to Lead

Adult lead poisoning is primarily caused by occupational exposure. Over 3 million American workers may be exposed to lead at work, according to estimates from the National Institute of Occupational Safety and Health (NIOSH) (Staudinger & Roth, 1998). Needleman (2004) noted that occupational exposure is the primary cause of lead poisoning and a key cause for concern. Radiation shields, ammunition, specific surgical instruments, producing dental X-ray images before digital X-rays, fetal monitoring, circuit boards, plumbing, jet engines, and ceramic glazes are among the frequent workplace items that contain lead (Patrick, 2006). As exposure increases, all of these raise the potential for toxicity. Moreover, a large number of other professions including lead miners and smelters, plumbers and fitters, vehicle mechanicians, glass manufacturers, construction workers, battery manufacturers and recyclers, firing range instructors, and plastic manufacturers are at risk for lead exposure. There is also a chance of lead exposure in jobs like welding and battery recycling (Sanborn et al., 2002). Children are more likely to be exposed to lead dust if their parents, who are exposed to it at work, carry it home on their clothing or skin (Watts, 2009). Lead and lead products are used in the modern world's industrialization boom. As a result of the widespread industrial use of lead in modern times, it is challenging to pinpoint the precise pathways through which human exposure to lead occurs. One typical environmental contaminant is lead. Lead exposure mostly happens in foundries, metal recycling, lead-acid battery and pipe manufacturing, and occupational settings (Woolf et al., 2007). Children who live close to these locations run the risk of having high blood lead levels. There were riots in August 2009 after it was discovered that 2000 children who lived close to zinc and manganese smelters had been exposed to lead poisoning (Watts, 2009). Lead in the air, home dust, soil, water, and commercial items are other significant sources of lead exposure (Rossi, 2008). Lead frequently accumulates in the highest concentrations in the bones and kidneys after a prolonged period of exposure. A blood lead level of 10 μg/dL or more is considered concerning by the World Health Organization and the US Centers for Disease Control and Prevention. There isn't, however, a cutoff point at which lead exposure is deemed safe. It has been found to impair development and have harmful effects even at lower levels (Rossi, 2008; Barbosa et al., 2005).

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A variety of compounds formed by lead exists in the environment in different forms (Grant, 2009). Poisoning and its features also differ between organic and inorganic lead (Kosnett, 2007). Organic lead poisoning is now very rare around the world because of withdrawal of organic lead compounds as gasoline additives. Nevertheless, such compounds are still used in industrial settings. Organic lead compounds cross the skin and respiratory tract easily and quickly, affecting predominantly the central nervous system.

Symptoms of Lead Toxicity

Different people have different signs of lead poisoning, such as odd behavior, and the duration of exposure is a significant factor (Kosnett, 2005). Additionally, some research indicates that even when the body contains high amounts of lead, there are no signs of lead poisoning (Mycyk et al., 2005). A significant worry is the topic of what causes the human body to differ in such ways. Why do lead and other hazardous chemicals vary so much in their level of toxicity? Why does a concentration rise cause toxicity for one group of persons while having no effect at all for another? There isn't any evidence of these distinctions in the literature at the moment, but hopefully more research in this area will be done in the future. Chronic lead exposure often takes weeks to months to cause the body to develop symptoms; but, brief, high exposures can also cause acute symptoms and indications (Dart et al., 2004). Because organic lead is lipid soluble and has immediate effects, it may be more harmful than inorganic lead (Timbrell, 2008). However, depending on unidentified personal features, the lead concentrations at which symptoms and indications manifest vary greatly (Bellinger, 2004). Neuropsychiatric consequences include headaches, decreased motor nerve conduction, irritability, delayed reaction times, and difficulties concentrating when blood lead levels are between 25 and 60 μg/dL (Merill et al., 2007). Higher than 50 μg/dL blood lead levels might cause anemia. In adults, abdominal colic, involving paroxysms of pain, may appear at blood lead levels higher than 80 μg/dL (Kosnett, 2005). Excessive blood lead levels above 100 μg/dL result in extremely severe symptoms, such as headache, delirium, coma, seizures, and indicators of encephalopathy, a disorder marked by enlargement of the brain and increased pressure inside the skull (Henritig, 2006). On the other hand, children who have lead levels of 70 μg/dL or above exhibit similar signs. While gastrointestinal symptoms typically arise from exposure over extended periods of time, central nervous system and neuromuscular manifestations typically result after intensive exposure (Brunton et al., 2007). Chronic exposure can cause sadness, numbness and tingling in the extremities, nausea, abdominal pain, loss of coordination, and short-term memory or attention problems (Patrick, 2006). Chronic lead poisoning also causes anemia, drowsiness, headaches, fatigue, slurred speech, and stupor (Pearce, 2007). Children who have been poisoned repeatedly typically act aggressively and won't play. Elevated blood lead levels during pregnancy increase the risk of low birth weight or early birth. Even at blood lead concentrations far lower than 25 μg per deciliter, the fetus may suffer harm (Bellinget et al., 1987). It was discovered that the newborn's blood lead levels were greater than the mother's concurrent lead levels (Shannon, 2003; Bellinger, 2005). Women who are underweight and have been

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exposed to high levels of lead before becoming pregnant are thought to be more vulnerable. Due to their bodies' ongoing growth and development, children have been found to be more susceptible to lead poisoning (Chisolm & Harrison, 1956). Additionally, lead is absorbed more quickly in children than in adults. Children, due to their childish behaviour, are more prone to ingest and inhale dust contaminated with lead (Landrigan, 2002). The number of ways how children become easy targets for lead poisoning are illustrated in Fig. 1.

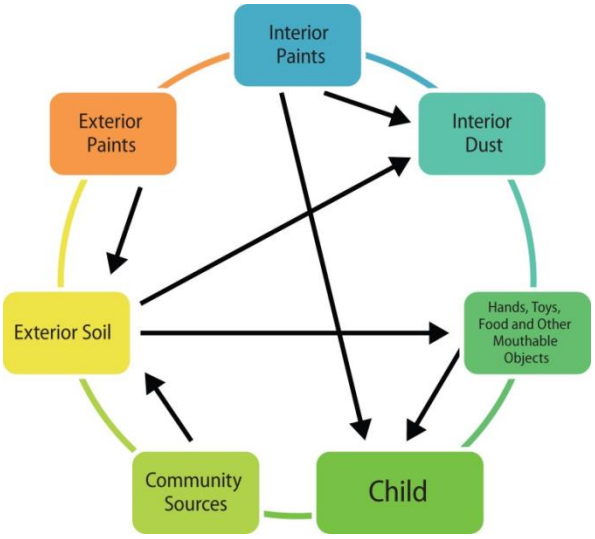

Fig 1 - Scheme how children become easy targets for lead poisoning in the environment (Wani AL, Ara A, Usmani JA. Lead toxicity: a review. Interdiscip Toxicol. 2015)

Path physiology of Lead in human body

There are several ways to become exposed, including by eating, skin contact, or inhalation. Lead levels can also rise as a result of direct contact with lead or compounds containing lead through the mouth, nose, eyes, and skin fissures. Around 35–40% of lead dust inhaled by humans is deposited in the lungs, and approximately 95% enters the bloodstream (Merill et al., 2007). About 15% of inorganic lead that is ingested is absorbed; however, this percentage is higher in children, pregnant women, and individuals with iron, zinc, or calcium deficits (Karri et al., 2008). Because it is not available to other tissues, a certain quantity of lead, which is typically bonded in tissues like bones, teeth, hair, or nails, is regarded as harmless. Lead is induced into the bloodstream long after the first exposure due to its half-life in these organs (Patrick, 2006). In humans, blood lead has a shorter half-life of roughly 40 days. Pregnant women and children whose bones are still growing are more likely to experience this. Lead can be continually reintroduced into the bloodstream in infants due to their developing bones that undergo remodelling (Barbosa Jr et al., 2005).Due to the high rate of lead absorption in bones and teeth—roughly 94% in adults and 70% in children—more lead can be absorbed by soft tissues, which might have detrimental effects on health (Barbosa Jr et al., 2005).

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A far delayed clearance occurs because of years of sustained exposure to lead. This results from a long-term buildup of lead in bones that has been released throughout time (Grant, 2009). The brain, spleen, kidneys, liver, and lungs are only a few of the bodily tissues that retain lead in addition to bones, teeth, and blood (Dart et al., 2004). It was discovered that perspiration, hair, and nails may eliminate trace levels of lead, as well as feces (Rubin & Strayer, 2008). Nevertheless, it's interesting to note that despite having numerous negative consequences, lead has been shown to have no physiological function in the body (Wolf et al., 2007; Rubin & Strayer, 2008). Lead's effects have also been thoroughly researched. at cellular level. Heavy metals, including lead, create reactive radicals which damage cell structures, including DNA and cell membrane (Kosnett, 2006). Lead also affects the enzymes responsible for maintaining the integrity of the cell membrane and for aiding in the creation of vitamin D. It was also discovered that lead obstructed DNA transcription. Anaemia is caused by red blood cells with a broken membrane becoming more brittle due to lead's disruption of the cell membrane's maintenance (White et al., 2007). Lead may also change blood vessel permeability and collagen formation, according to theories (Needlemann, 2004). Reduced immunological activity is the outcome of damaged immune system cells, such as polymorphonuclear leukocytes (Kosnett, 2006). The primary cause of anemia resulting from lead poisoning is the disruption of an important enzyme known as delta-aminolevulinic acid dehydratase, or ALAD. which is important in the biosynthesis of heme, the cofactor found in haemoglobin (Patrick et al., 2006).

Due to their interference with lead, heme precursors like aminolevulinic acid have been reported to accumulate and may cause direct or indirect neuronal damage (Fujita et al., 2002). A recent study by Fang et al. (2014) revealed that the buildup of organic lead in the liver causes oxidative imbalance and protein impairment, which may lead to ER stress and subsequent liver damage. The impact of lead exposure on C57BL/6J mice was noted in a subsequent investigation. Pre-adolescent mice's exploratory behavior was reported to be disrupted by early chronic low-level lead exposure that resulted in BLLs ranging from 1.98 to 14.84 μg/dL (Flores & Sobin, 2014). The authors of a different recent study looked at the interactions between lead exposure and a high-fat diet (HFD. They found that lead and HFD contributed to the suppression of osteoblastogenesis, modification of progenitor cell differentiation, enhancement of adipogenesis, and promotion of osteoclastogenesis. Furthermore, it was revealed that non esterified fatty acids and lead caused MC3T3 cells to have decreased β-catenin activity and enhanced in vitro PPAR-γ activity. They also came to the conclusion that lead and HFD contributed to the selective deficit bone accretion, and that decreased Wnt signaling may have something to do with changes in progenitor cell activity (Beier et al., 2015).

Reproductive System

Lead has an impact on both male and female reproductive systems. If blood lead levels are higher than 40 μg/dL, male sperm count decreases and sperm volume varies. At this level, sperm motility and general morphology are also impacted (Navas-Acien et al., 2007). The reproductive issues that arise from lead exposure in females are more serious. Lead poisoning can cause low birth weight,

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preterm, miscarriages, and developmental issues in children (Park et al., 2008). Because lead in mother blood enters the fetus through the placenta and breast milk, blood lead levels in mothers and infants are often identical (Dart et al., 2004). As a depository for bones the highest content of lead, if metabolic changes mobilise the lead from bones into the blood due to pregnancy, the lead toxic risks increase. However increased calcium intake during pregnancy can help mitigate this phenomenon (Grant, 2009. While high doses of lead increased the proportion of aberrant epididymal sperms and decreased both the sperm count and the percentage of motile sperms, low dosages of lead were shown to dramatically lower the quantity of sperms within the mice's epididymis. Lead specifically affects the sperm in the epididymis that cause reproductive damage as well as testicular spermatogenesis (Wadi & Ahmad, 1999). Serum testosterone, intra testicular sperm counts, and sperm production rates were reported to be decreased in groups treated with lead (Apostoli et al., 1998; Sokol, 1989). Serum testosterone and sperm parameters normalized at the conclusion of the recovery period in prepubertal animals but not in pubertal animals, according to research on the reversibility of the toxic effects of lead on the male reproductive axis (Sokol, 1989). It has been demonstrated that prepubertal rats are less susceptible to the harmful effects of lead than rats whose exposure to lead began after the onset of puberty (Sokol & Berman, 1991). It was shown that rats exposed to lead showed a 32% decrease in plasma luteinizing hormone (LH) and an 80% reduction in testicular and plasma testosterone. Rats exposed to lead showed a significant drop in their testosterone:LH ratio (Thoreux-Manlay et al., 1995). A comprehensive assessment of the mechanism by which lead causes male infertility was conducted (Mohsen et al., 2011). Rather than a chronic developmental problem, other scientists have proposed that growth impacts of lead could result from a delay in the establishment of the sex-specific pituitary growth hormone secretion pattern (Ronis et al., 1996). Long-term low-dose lead exposure was shown to alter the signalling system between the hypothalamus and pituitary gland of male rats. Long-term exposure disrupts this signaling, changing the male rat's gonadotropin-releasing hormone system as a result (Sokol et al., 2002). Zhang et al. (2014) looked at lead's interactions with human chorionic gonadotropin (HCG) in a recent study. Through the destruction and loosening of the HCG skeleton, as well as an increase in the hydrophobicity surrounding Tyr residues, lead acetate altered the secondary structure of HCG, leading to a decrease in the bioactivities of HCG, according to investigations of the hormone using UV-vis absorption spectroscopy, circular dichroism spectroscopy, and ELISA (Figure 2). This study shows how lead directly interacts with sex hormones and offers a potential molecular mechanism for lead-induced reproductive damage.

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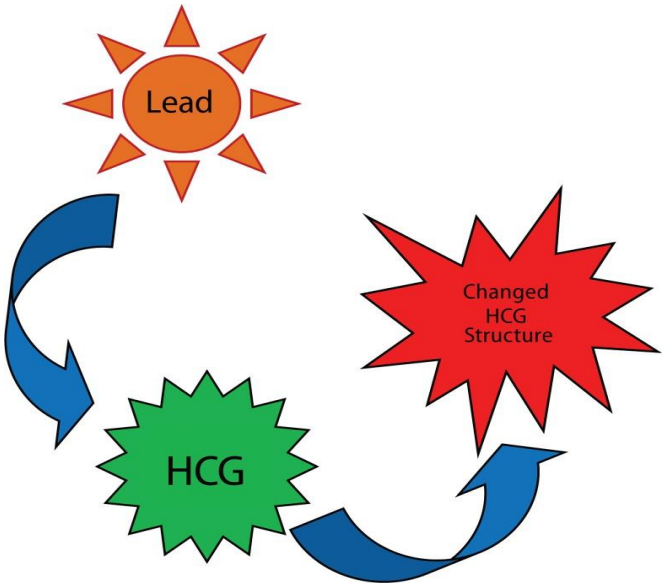

Fig 2 - Depiction of lead changing the structure of HCG
(Wani AL, Ara A, Usmani JA. Lead toxicity: a review. Interdiscip Toxicol. 2015)

Nervous System

The organ most susceptible to lead exposure is the brain (Cleveland et al., 2008). Lead has a major impact on the creation of synapses in the cerebral cortex of a developing child's brain. Lead also disrupts the organization of ion channels and the synthesis of neurochemicals, such as neurotransmitters (Casarett et al., 2007). In addition, loss of the myelin sheath, a decrease in the number of neurons, disruption of neurotransmission, and a reduction in neuronal growth are all consequences of lead poisoning (Pearson & Schonfeld, 2003). Adults who were exposed to elevated lead levels as children also have reduced brain volume, particularly in the prefrontal cortex on MRI (Cleveland et al., 2008). Due to its ability to replace calcium ions, lead can cross the blood-brain barrier's endothelial cells. and be taken up by calcium-ATPase pumps, thereby interfering with synapse formation. According to Brunton et al. (2007), children who have blood lead concentrations higher than 10µg/dL are more likely to experience developmental problems. Lead has very little effect on children's cognitive ability (Xu et al., 2009; Park et al., 2008; Sanders et al., 2009). Lead exposure is considered safe below what appears to be no lower threshold in the dose-response relationship (Meyer et al., 2003). Lower blood lead levels than 5 µg/dL have been linked to worse academic outcomes (Bellinger, 2008; Needlemann et al., 1990). Blood lead concentrations less than 10 µg/dL have been linked, proportionately, to decreased IQ and behavioral issues including aggression (Guidotti & Ragain, 2007). Children's IQ dropped 2-4 points for every µg/dL increase in blood lead levels between 5 and 35 µg/dL (Brunton, 2007). According to Jacobs et al. (2002), elevated blood lead levels are also linked to various psychiatric disorders like anxiety and depression as well as a decline in cognitive function. Adult blood lead levels that rise from 50 to approximately 100 µg/dL have been linked to more serious disorders, such as long-term impairment of central nervous system function

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(Bellings et al., 2004). According to Sanders et al. (2009), there is a correlation between elevated lead exposure in children and a rise in neuropsychiatric illnesses such as attention deficit hyperactivity disorder and antisocial behavior. There has been evidence linking prenatal and early childhood lead exposure to violent crimes in adulthood (Park et al., 2008). According to Neelemann et al. (2004), countries with the greatest air lead concentrations also exhibit abnormal behavior, such as becoming aggressive and violent. As a result, these countries have the highest murder rates. According to one study (Shih et al., 2007; Kosnett et al., 2007), exposure to lead may account for anywhere between 65% and 90% of the difference in violent crime rates in the US. Nevin (2007) found that there was a significant correlation between blood lead levels in preschools and the developments in crime rates over several decades in nine different countries. A region of the brain important in memory and learning is called the hippocampus. The primary causes of lead's interference with learning, especially in young children, are the it damages the cells within the hippocampus.

Additionally, lead interferes with neurotransmitter release (Dart et al., 2004; Needlemann, 2004). Chemicals called neurotransmitters are utilized by neurons to communicate with other cells. The communication between cells is disrupted as a result of this interference. The neurotransmitter glutamate, which is necessary for numerous processes, including learning, is typically interfered with by lead. By inhibiting NMDA receptors, it works. Lead poisoning is assumed to primarily target blocking of these receptors. According to one study, exposure to lead not only inhibited the NMDA receptor but also reduced the gene for this receptor in a specific area of the brain (Kosnett, 2005). According to studies conducted on animals, lead has also been linked to brain cell death (Nedlemann, 2004).

Diagnosis of Lead Toxicity

A fundamental and crucial concern is ensuring accurate diagnosis in order to prevent lead poisoning and toxicity. Investigating potential exposure routes is essential to obtaining an accurate diagnosis (Nevin, 2007). Determining clinical indicators and reviewing medical history should be part of the investigation. Appropriate personnel engagement, such as clinical toxicologists and medical specialists, can aid in the establishment of appropriate diagnosis and treatment. One significant indicator of lead toxicity is basophilic stripping. Under a microscope, this stripping reveals spots in red blood cells (Patrick, 2006). Therefore, a blood film screening for these indicators may be useful in the diagnosis of lead poisoning. A correlation exists between iron deficiency anemia and lead poisoning. Erythrocyte protoporphyrin (EP) levels in blood samples can also be used to assess lead toxicity (Patrick, 2006). When blood lead levels are high, EP is known to rise, however it takes a few weeks to show symptoms (Kosnett, 2007). Nevertheless, below about 35 μg/dL, the EP level is not sensitive enough to detect increased blood lead levels (Patrick, 2006). The use of this approach to identify lead exposure has declined because of the greater threshold for detection and the correlation between elevated EP levels and iron deficiency. Blood lead levels do not represent the entire body load; rather, they are primarily a measure of recent or ongoing lead exposure. The blood lead level is

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only a measure of recent lead exposure; it does not provide an accurate assessment of lead stored in the body. X-ray fluorescence is a noninvasive method of measuring lead in the whole body and may be the best indicator of cumulative exposure and overall body load (Kosnett, 2006). X-rays can also show foreign objects in the gastrointestinal tract that contain lead, like paint chips (Kosnett, 2007; Grant, 2009).

Prevention and Treatment of Lead Toxicity

Lead poisoning is a major worry that can have severe consequences; nevertheless, it is preventable. Preventing lead exposure is the best course of action (Rossi, 2008). It is advised to give the kids more calcium and iron in addition to often washing their hands. Discouragement of youngsters from routinely placing their hands which may be contaminated in their mouths is also advised, as this can increase the risk of lead poisoning. Exposures can also be avoided by regularly vacuuming the home and removing any lead-containing items, such as jewellery and blinds, from usage or presence. To prevent lead poisoning of drinking water, lead-containing household pipes or plumbing solder installed in older homes should be replaced. Dimercaprol and succimer are the two main treatments for lead poisoning (Park et al., 2008). Given the ongoing research on the cognitive impairments associated with lead poisoning, especially in youngsters, widespread exposure reduction ought to be required.

The chelating salt disodium calcium edentate, which is the calcium chelate of the disodium salt of ethylene-diamine tetracetic acid (EDTA), is typically used to treat lead poisoning. These chelating agents are very affine to the agent that is being removed. The lead chelate is created by exchange because the lead chelating agent has a higher affinity for lead than calcium. After that, it is eliminated in the urine, leaving behind calcium that is safe. It has been demonstrated that administering succimer as chelation therapy to children exposed to lead lowers blood lead levels.

Nevertheless, succimer did not improve cognition test scores, despite being shown to assist in lowering blood lead levels (Rogan et al., 2001). Numerous antioxidants are thought to counteract the toxicity of substances like lead and its derivatives. Through solubilisation, a novel method known as nano-encapsulation of antioxidants may increase the bio distribution and bioavailability of poorly soluble medicines (Flora et al., 2012). Curcumin encapsulated in a pluronic block copolymer exhibited anticancer efficacy equivalent to that of free Curcumin, as well as a delayed and prolonged release of Curcumin (Sahu et al., 2010). Numerous human disorders may be treated with these novel procedures Akt and GSK-3β phosphorylation in PC12 cells exposed to lead acetate in a fairly recent study. The study's authors came to the conclusion that puerarin, a phytoestrogen, might be a desirable therapeutic and prevention strategy for long-term illnesses linked to lead neurotoxicity. Another recent discovery revealed that beta-carotene, without the need for chelation, has an antioxidant function and can help treat lead poisoning. (Dobrkowski et al., 2014). The injection of beta-carotene to lead-exposed workers resulted in significantly lower homocysteine levels, according to the authors' findings. In a recent study, blood lead levels significantly decreased in individuals treated with N-

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acetylcysteine (NAC) among a group of workers exposed to lead at work. In a recent study, blood lead levels significantly decreased in individuals treated with N-acetylcysteine (NAC) among a group of workers exposed to lead at work. Furthermore, it was demonstrated that the glutamate dehydrogenase activity was significantly higher in all groups who received NAC. It was also stated that taking NAC reduced oxidative stress and returned homocysteine levels to normal. Thus, it was determined that NAC might be suggested as a substitute treatment for long-term lead poisoning in people (Kasperczyk et al., 2015).

Conclusions

It seems that lead poisoning is the most common heavy metal toxicity. Lead has been used since antiquity, and there is ample proof of its toxicity. Its significant physico-chemical qualities have led to its global application. When industrialization began in the seventeenth century, its use multiplied, increasing its toxicity for human consumption. Youngsters are more vulnerable, especially in areas where their play areas are close to lead-related jobs. Lead poisoning is also more common among workers who are exposed to lead at work. To reduce the risk of lead poisoning, children whose parents work in environments where lead is present should have regular blood tests. Literature clearly shows that lead is harmful, and practically no bodily function is unaffected by lead.The reproductive, neurological, pulmonary, and digestive systems, among others, are all negatively impacted by lead poisoning. Lead also inhibits enzymes from carrying out their regular functions. Lead even damages bones by interfering with the regular transcription of DNA. Lead has no physiological function in the body as a whole, and even low concentrations of the metal can be harmful. The good news is that there are currently several ways that can reverse it and lower the body's lead levels. Among these, N-acetylcysteine (NAC), nano-encapsulation, and chelation therapy are the most well-known. Antioxidants in general aid in the body's elimination of lead. Even if there are many therapeutic options accessible today, it is unquestionably preferable to avoid direct exposure to toxins and avoid any negative effects down the road. Additionally, it is advised that parents teach their kids how to avoid unintentional lead poisoning. The effectiveness of treatment options varies amongst patients due to a variety of factors, including genetics, environment, and food.

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