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# The Toxicity of Lead in Vulnerable Populations: A Critical Review

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### ABSTRACT

This review critically synthesizes current scientific findings regarding the sources, mechanisms and health impacts of exposure to lead in high risk population. The toxicity of lead has always been a major concern in public health, notably among the at-risk group of the population including children and pregnant women. Its danger is heightened by the fact that it is toxic heavy metal that remains un-decayed in the earth surface for centuries. Exposure to lead occurs mainly through contaminated water, soil, food, and air, with children and women of reproductive age facing the highest risk due to developmental and physiological factors respectively. Of particular concern is endogenous exposure during pregnancy and lactation, as skeletal lead accumulated over decades is remobilized back into the systemic circulation, crosses the placenta and affects fetal development. A systematic approach was used to gather relevant literature from four major sources including Pubmed, Google Scholar, Scopus and Web of Science. Findings show that lead exposure contributes to various health issues affecting women and children. The review provides evidence that will inform public health strategies and intervention to better protect these vulnerable groups from the risk of lead exposure.

**Keywords:** Lead, Women, Children, Pregnancy, Lactation, Calcium Intake

### Introduction

Lead is a toxic metal that is ubiquitously distributed in the environment because of its usefulness in many areas but can be hazardous to health when ingested or inhaled. The characteristics of lead such as its malleability, ductility, corrosion resistance, low melting point and its abundant availability are reasons lead is still being used today despite its hazardous nature (Ara & Usmani, 2015). Since lead is a non-biodegradable environmental pollutant (Kumari & Kumar, 2023), it continues to pose serious threat to human health especially in countries where its use is not well controlled. Though, most countries have phased out the use of leaded gasoline many years

ago, including Nigeria phasing it out in 2003 (Ojo *et al.*, 2010), lead is still being used in the manufacture of household items such as paints, pipes, batteries, children toys, jewelry and cosmetics. Hence, lead persists as a major public health threat due to its widespread use. Lead affects almost all the organs in the body. The nervous system is mostly affected both in children and adults (Ara & Usmani, 2015). Vulnerable groups of the population including reproductive women and children are at higher risk of being affected by lead toxicity. For instance, children absorb more lead than adults (Collin *et al.*, 2022) which makes them to be more susceptible to its neurotoxic effects. Pregnant women exposed to lead can get it transferred to the fetus, posing the risk of miscarriage, preterm birth and impaired

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fetal development. While many research has been conducted on lead toxicity, significant knowledge gap remains especially concerning its effect on children and women reproductive age (which are the vulnerable groups focused in this review). This review is therefore needed to understand existing scientific findings on the exposure pathways, mechanisms of toxicity and health impacts on vulnerable groups. This will guide clinicians and policy makers in making informed decisions that will help to mitigate risks and improve health outcomes in these vulnerable groups of the populace.

## Materials and Methods

This review used a systematic approach to gather and synthesize relevant literature on the toxicity of lead in vulnerable populations. Databases searched are PubMed, Scopus, Web of Science and Google scholar. To tailor the search down to the scope and objective of the review, keywords and Boolean operators used are; 'lead toxicity', 'lead poisoning', 'children', 'pregnant women', 'children', 'lead toxicity AND children', 'lead toxicity AND pregnant women', 'lactating women', 'health effects'. The search was restricted to peer-reviewed articles, government reports, WHO reports and public health databases published in English language. Articles that were not related to human subjects were excluded while the selected articles were further considered based on scientific rigour, sample size, clear objectives and how much relevant they are to the topic. More than 70 studies were reviewed but 40 were eventually considered after filtering to remove those that did not meet the selection criteria

## Results and Discussion

### Lead Exposure Sources

Paint with lead content, polluted soil, contaminated water and air constitute major pathways of environmental lead exposure. A review study to evaluate sources of lead exposure worldwide found that environmental sources of exposure in Nigeria include electronic waste, paint and batteries (Obeng-

Gyasi, 2019). Different studies have identified lead-based paint as a major source of lead exposure, especially in children living in old houses built before 1978 when leaded paint was banned (Body *et al.*, 1991; Obeng-Gyasi, 2019; Gulson *et al.*, 2004; Swearingen *et al.*, 2022; Castro *et al.*, 2019; Jacobs *et al.*, 2002). Leaded paint in old homes can chip to create lead dust when damaged or removed thereby increasing the risk of exposure, mostly in children when they ingest or inhale the lead dust. A handful of study have also found lead in contaminated water, soil and polluted air to be culpable of lead poisoning (Gleason *et al.*, 2019; Redmon *et al.*, 2020; Forsyth *et al.*, 2019). Air lead arises as a result of industrial processes such as smelting, illegal mining and poor waste management. The group of the populace most vulnerable to lead exposure are children and women of childbearing age (Ortega *et al.*, 2021; WHO, 2003). Children are majorly exposed through the eating of contaminated soil (as a result of the frequent mouthing of hands and objects they engage in) and inhalation of air lead. In a nutshell, children are exposed exogenously. Women of child-bearing age, on the other hand, are more prone to endogenous exposure. This is because lead stored in the bones (from past exposures that could date back to decades) are released into the blood during physiological changes such as in pregnancy and lactation (Gundacker *et al.*, 2021). Hence, in addition to ongoing present exogenous exposure, pregnant and lactating women face the risk of lead toxicity from past exposures as the half-life of bone lead spans up to three decades.

### Mechanism of Lead Toxicity

The presence of lead triggers the production of reactive oxygen species (ROS) (Patrick, 2006). ROS are molecules that are capable of damaging cells and tissues. Lead causes increase in the yield of free radicals and decreases the availability of endogenous antioxidant reserves (such as glutathione) responsible for neutralizing the ROS generated in individuals exposed to lead (Sani & Amanabo, 2021). This happens as a result of the binding of lead to sulfhydryl groups on protein leading to the inability of glutathione to neutralize ROS since it has been deactivated (Sani

& Amanabo, 2021). Lead has also been found to be the cause anaemia in exposed individuals due to the inhibition of enzymes associated with the building of the heme group in haemoglobin (i.e. heme synthesis) (Singh *et al.*, 2018). Another mechanism of lead toxicity is its ability to disrupt brain function by inhibiting the action of calcium as a regulator of cell function (Ortega *et al.*, 2021). As seen in Figure 1, Lead is capable of mimicking calcium in some

cellular processes which disrupts its regulation thus leading to the malfunctioning of the blood vessels. Some studies have inferred that there are no safe levels of blood lead as adverse effects have been noticed even at low doses (Glorennec *et al.*, 2010; Oulhote *et al.*, 2011; Lanphear *et al.*, 2005). Hence, blood lead levels previously thought to be safe might still pose some risks, especially to the brain. Succinctly put, lead can be toxic even at assumed safe low levels .

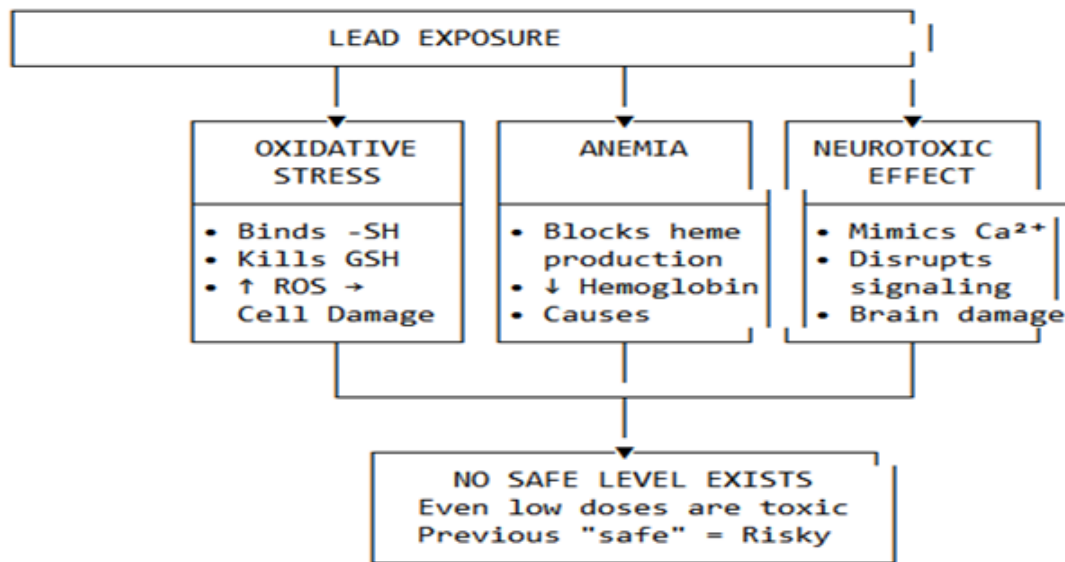


Figure 1: Lead Toxicity Mechanism

### Vulnerable Population Group

Children are the most vulnerable group because of factors such as hand-to-mouth activities (resulting in more lead ingestion) and permeable blood brain barrier (BBB) as a result of yet to be developed tissues (Markowitz, 2021). Since the tissues of children are not fully developed, their absorption rate is significantly more than that of adults; about 40-50% compared to 10% in adults (Ara and Usmani, 2015). Lead, when ingested or inhaled, goes directly into the blood and gets distributed to tissues such as the bones and other soft tissues. The half-life of lead in bone is about 2 decades (Ciosek *et al.*, 2021). Hence, it can be in the bone for an increased period of time without posing any risk as it is considered nontoxic in this tissue except when released back into the systemic circulation. However, there is higher toxicity risk at

the onset of pregnancy and during lactation. This is because at these periods, more calcium obviously needed by women (to meet the demands of fetus and breastfed infants) gets sourced from the bone, particularly in women with inadequate calcium intake. Since lead mimics calcium (due to similarities in their atomic and ionic structures), as calcium is released, lead is also simultaneously released. This explains why men are at lesser risk of exposure, particularly endogenously when compared to women.

### Health Effects

Studies have shown that lead majorly attacks the brain or the central nervous system, leading to neurotoxicity (Cleveland *et al.*, 2008; Naranjo *et al.*, 2020). Long term (chronic) exposure to any amount of lead has been linked to intellectual disability, shortened

attention span, affective disorders (such as anxiety and depression) and visual impairment (Naranjo *et al.*, 2020). Exposure to lead starts from the womb (prenatal exposure) as lead can cross the placenta and impair the development of the fetus due to interference with calcium metabolism (Mandal *et al.*, 2022). Prenatal exposure has been found to be responsible for impairment in neurodevelopment as low levels of prenatal lead exposure correlates with reduction in cognitive function. Some studies have found blood lead levels in neonates to be higher than that of the mother (Bellinger, 2005). In younger children, lead exposure causes both pervasive and permanent effects. According to a study done in 2015, 13% of reading failure and 14.8% of Mathematics failure has been traced to lead exposure (Evens *et al.*, 2015). In a human study by Wang *et al.*, (2009) to assess the adverse health effects of lead in children, it was found that 5.7% and 11.7% of the subjects had ADHD (Attention Deficit Hyperactive Disorders) and mental retardation respectively. Also, it has been established that maternal bone lead levels directly correlate with infant blood lead levels and umbilical cord lead levels (Popovic *et al.*, 2005).

spontaneous abortion, low birth weight, reduced fertility, impaired menstruation, delayed conception and hormonal imbalance (Vigeh *et al.*, 2011; Zhang *et al.*, 2015; Kumar *et al.*, 2020; Gopal *et al.*, 2022). Vigeh *et al.*, (2011) reported that BLL was significantly higher in mothers who delivered preterm babies than in mothers who delivered full term babies ( $4.46 \pm 1.86$  and  $3.43 \pm 1.22$  respectively). It was generally inferred in a review study that long term exposure to lead has adverse effects on fetal viability as well as fetal and childhood development as lead can readily cross the placenta (Kumar *et al.*, 2020). Increased blood lead levels have also been found to be associated with delay in the onset of puberty (Naicker *et al.*, 2010). Blood lead levels of as low as  $3\mu\text{g/dL}$  could cause significant delays in breast and pubic hair development in most girls used as subjects in a study (Selevan *et al.*, 2003). In Nigeria, women with history of miscarriage were found to have blood lead levels of  $7.25\mu\text{g/dL}$  in pregnancy which turned out to be responsible for about 47.6% increase in the incidence of miscarriage (Amadi *et al.*, 2017). A summary of the exposure sources and the resulting health effects are given in Table 1.

Women exposed to lead are at risk of various complications during pregnancy, including

**Table 1:** Lead Exposure Sources and Health Effects in Vulnerable Populations

| Vulnerable Group | Exposure Source and Routes  | Health Effects   | References  |
|------------------|---|--|---|
| Women            | Exogenously (by ingesting contaminated food or water and inhaling polluted air) and endogenously (by increased bone Pb turnover during pregnancy and lactation) | Preterm birth, miscarriage, low birth weight, menstruation imbalance, delay in the onset of puberty,     | Nacker <i>et al.</i> , 2010; Selevan <i>et al.</i> , 2003; Amadi <i>et al.</i> , 2017; Gleason <i>et al.</i> , 2019; Zhang <i>et al.</i> , 2020 |
| Children         | Exogenously mainly through the ingestion of contaminated soil and dust and inhaling polluted air  | Reduced IQ, ADHD, anxiety, depression, shortened attention span, visual impairment, developmental delays | Naranjo <i>et al.</i> , 2020; Ulang <i>et al.</i> , 2009; Castro <i>et al.</i> , 2019   |

**Blood Lead Levels before and after Leaded Gasoline Phase-Out**

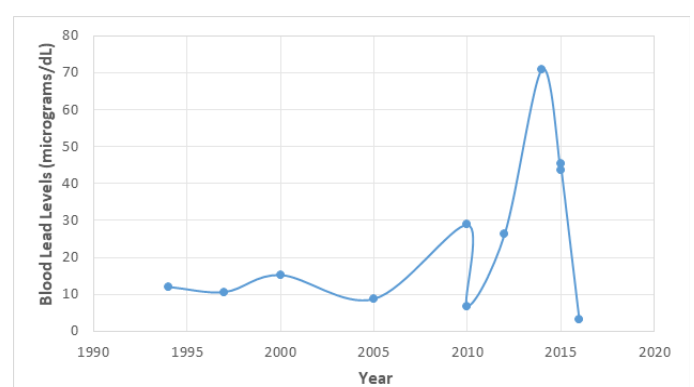
Increase in blood lead levels could be caused by the inhalation of air-borne lead released from leaded gasoline through vehicle exhaust according to human study done by Ojo *et al.*,(2010) after the phase-out of

leaded fuel in Nigeria. The study showed a significant decrease in the BLL of subjects ( $6.81 \pm 2.61 \mu\text{g/dL}$ ) when compared to a prior study done by the same author in the 1990s before the 2003 phase out ( $12.0 \pm 6.0 \mu\text{g/dL}$ ). Though, leaded fuel use has been banned globally, its effect is still being noticed.

**Table 2:** Blood Lead Levels pre Phase-out and post Phase-out of Leaded Fuel

| Year of Study | BLL ( $\mu\text{g/dL}$ ) | References                     |
|---------------|--------------------------|--------------------------------|
| 1994          | 12.0                     | Ojo <i>et al.</i> , 2010       |
| 1997          | 10.6                     | Nriagu <i>et al.</i> , 1997    |
| 2000          | 15.2                     | Pfitzner <i>et al.</i> , 2000  |
| 2005          | 8.7                      | Wright <i>et al.</i> , 2005    |
| 2010          | 6.81                     | Ojo <i>et al.</i> , 2010       |
| 2016          | 3.1                      | Bello <i>et al.</i> , 2016     |
| 2014          | 71 (Exposed)             | Ajumobi <i>et al.</i> , 2014   |
| 2010          | 28.8 (Pregnant)          | Nkolika & Benedict (2010)      |
| 2015          | 43.5 (Exposed)           | Saliu <i>et al.</i> , 2015     |
| 2012          | 26.3 (Pregnant)          | Ikechukwu <i>et al.</i> , 2012 |
| 2015          | 45.43 (Exposed)          | Alli, 2015                     |

From the studies reviewed (Table 2), it was observed that there was significant decrease in blood lead concentrations after phasing out leaded fuel in Nigeria which simultaneously has reduced toxicity across various population groups (Ojo *et al.*, 2010; Ademuyiwa *et al.*, 2007). However, some studies still reported elevated blood lead levels years after phase-out, especially in subjects with ongoing exposure either exogenously or endogeneously (peculiar to pregnant and lactating women) as seen in Figure 2. This agrees with the findings of the author in a recently published study (Karakatose *et al.*, 2025) where the output of the simulation done showed elevated blood lead levels in women of child bearing age several years after the phase-out of leaded gasoline.



**Figure 2:** Graph Showing Blood Lead Levels Pre and Post Phase-out of Leaded Fuel

**Regulatory Measures and Guidelines**

In order to control and minimize the risks of lead toxicity, it will be helpful to put standards in place.

The root cause of toxicity which is primarily exposure must be dealt with. This can be achieved primarily through public awareness campaigns about this pollutant. Public health professional should educate people about the dangers of being exposed to lead after sensitizing them of likely exposure sources. Children should be encouraged to wash their hands frequently to minimize exposure through ingestion. Households should use more of cold water from taps in place of hot/warm water as the latter has been found to contain higher amounts of lead. Hot water taps contain 260 times more lead than cold water tap (Triantafyllidou & Edwards, 2012).

Also, intake of calcium supplements especially by pregnant and lactating women is important to reduce endogenous exposure through bone resorption. Maintaining adequate dietary calcium reduces the mobilization of calcium and consequently, lead from bone stores. This happens because the body, when satisfied with calcium intake, has less need to resorb bone to maintain blood calcium levels. Thus, reducing the risk of bone breakdown. Chelation therapy is another great way of reducing the body burden of lead, particularly when blood lead concentration is very high. Lead-chelating agent including EDTA has more affinity for lead than calcium. Hence, lead binds to the chelating agent and get excreted in the urine, leaving behind only the useful calcium (Parui *et al.*, 2024). This mechanism is described in Figure 3.

For these safety measures to be effective, it is important that environmental remediation is done to reduce ongoing exposure significantly, particularly where people are occupationally exposed through mining or smelting.

### Conclusion

Lead toxicity keeps being a major and persistent challenge to the health of the public, especially in populations at higher risk including children and reproductive women. Despite global efforts to reduce environmental lead exposure, residual and

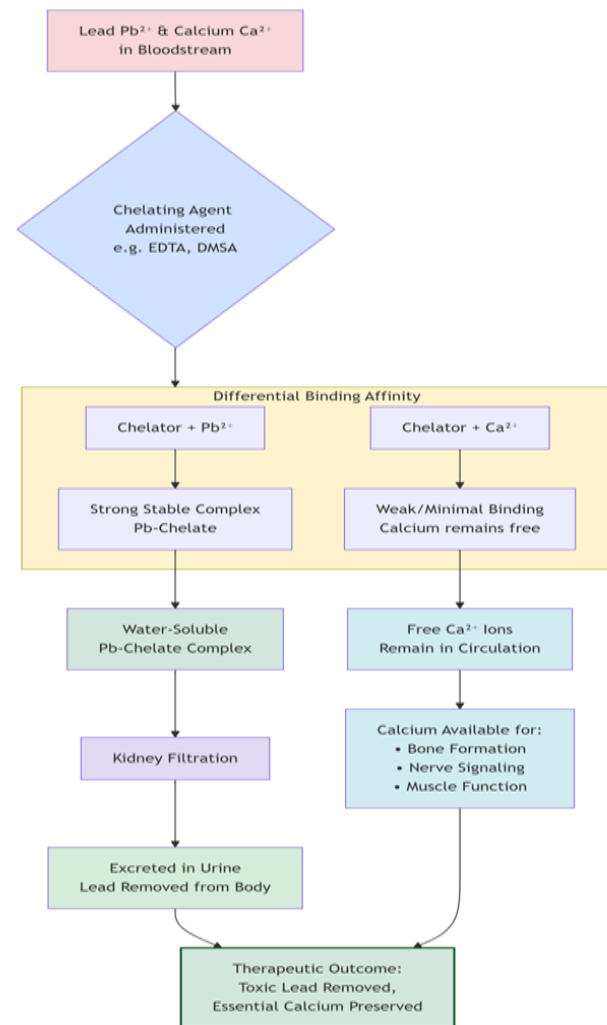


Figure 3: Chelation Therapy

endogenous sources especially those emanating from historical exposure and physiological changes during pregnancy and lactation continue to pose serious risks. This review has highlighted the major pathways of exposure, the biological mechanisms of toxicity, and the wide-ranging health impacts on both mothers and children, including neurodevelopmental disorders, reproductive complications, and hormonal disruptions.

The evidence clearly demonstrates that there is no identifiable safe threshold for lead exposure, particularly for developing brains and physiologically dynamic populations. Therefore, proactive strategies such as public education, policy enforcement,

calcium supplementation, chelation therapy and environmental remediation must be prioritized to reduce both current and future risks. Further research is also needed to close existing knowledge gaps, especially to determine the efficacy of intervention strategies.

## References

- Ademuyiwa, O., Arowolo, T., Ojo, D. A., Odukoya, O. O., Yusuf, A. A., & Akinhanmi, T. F. (2002). Lead levels in blood and urine of some residents of Abeokuta, Nigeria. *Trace elements and Electrolytes*, 19(2), 63-69.
- Amadi, C. N., Igweze, Z. N., & Orisakwe, O. E. (2017). Heavy metals in miscarriages and stillbirths in developing nations. *Middle East Fertility Society Journal*, 22(2), 91-100.
- Ara, A., & Usmani, J. A. (2015). Lead toxicity: a review. *Interdisciplinary toxicology*, 8(2), 55.
- Bellinger, D. C. (2005). Teratogen update: lead and pregnancy. *Birth Defects Research Part A: Clinical and Molecular Teratology*, 73(6), 409-420.
- Body, P. E., Inglis, G., Dolan, P. R., & Mulcahy, D. E. (1991). Environmental lead: a review. *Critical Reviews in Environmental Science and Technology*, 20(5-6), 299-310.
- Castro, I. E., Larsen, D. A., Hruska, B., Parsons, P. J., Palmer, C. D., & Gump, B. B. (2019). Variability in the spatial density of vacant properties contributes to background lead (Pb) exposure in children. *Environmental research*, 170, 463-471.
- Ciosek, Ż., Kot, K., Kosik-Bogacka, D., Łanocha-Arendarczyk, N., & Rotter, I. (2021). The effects of calcium, magnesium, phosphorus, fluoride, and lead on bone tissue. *Biomolecules*, 11(4), 506.
- Cleveland, L. M., Minter, M. L., Cobb, K. A., Scott, A. A., & German, V. F. (2008). Lead hazards for pregnant women and children: part 1: immigrants and the poor shoulder most of the burden of lead exposure in this country. Part 1 of a two-part article details how exposure happens, whom it affects, and the harm it can do. *AJN The American Journal of Nursing*, 108(10), 40-49.
- Collin, M. S., Venkatraman, S. K., Vijayakumar, N., Kanimozhi, V., Arbaaz, S. M., Stacey, R. S., ... & Swamiappan, S. (2022). Bioaccumulation of lead (Pb) and its effects on human: A review. *Journal of Hazardous Materials Advances*, 7, 100094.
- Evens, A., Hryhorczuk, D., Lanphear, B. P., Rankin, K. M., Lewis, D. A., Forst, L., & Rosenberg, D. (2015). The impact of low-level lead toxicity on school performance among children in the Chicago Public Schools: a population-based retrospective cohort study. *Environmental Health*, 14, 1-9.
- Forsyth, J. E., Weaver, K. L., Maher, K., Islam, M. S., Raqib, R., Rahman, M., ... & Luby, S. P. (2019). Sources of blood lead exposure in rural Bangladesh. *Environmental Science & Technology*, 53(19), 11429-11436.
- Gleason, J. A., Nanavaty, J. V., & Fagliano, J. A. (2019). Drinking water lead and socioeconomic factors as predictors of blood lead levels in New Jersey's children between two time periods. *Environmental research*, 169, 409-416.
- Glorennec, P., Peyr, C., Poupon, J., Oulhote, Y., & Le Bot, B. (2010). Identifying sources of lead exposure for children, with lead concentrations and isotope ratios. *Journal of occupational and environmental hygiene*, 7(5), 253-260.
- Gulson, B. L., Mizon, K. J., Davis, J. D., Palmer, J. M., & Vimpani, G. (2004). Identification of sources of lead in children in a primary zinc-lead smelter environment. *Environmental health perspectives*, 112(1), 52-60.
- Gundacker, C., Forsthuber, M., Szigeti, T., Kakucs, R., Mustieles, V., Fernandez, M. F., ... & Saber, A. T. (2021). Lead (Pb) and neurodevelopment: A review on exposure and biomarkers of effect (BDNF, HDL) and susceptibility. *International Journal of Hygiene and Environmental Health*, 238, 113855.
- Jacobs, D. E., Clickner, R. P., Zhou, J. Y., Viet, S. M., Marker, D. A., Rogers, J. W., ... & Friedman, W. (2002). The prevalence of lead-based paint hazards in US housing. *Environmental health perspectives*, 110(10), A599-A606.
- Kumar, A., Kumar, A., MMS, C. P., Chaturvedi, A. K., Shabnam, A. A., Subrahmanyam, G., ... & Yadav, K. K. (2020). Lead toxicity: health hazards, influence on food chain, and sustainable remediation approaches. *International journal of environmental research and public health*, 17(7), 2179.

- Kumari, P., & Kumar, A. (2023). Advanced oxidation process: A remediation technique for organic and non-biodegradable pollutant. *Results in surfaces and interfaces*, 11, 100122.
- Lanphear, B. P., Wright, R. O., & Dietrich, K. N. (2005). Environmental neurotoxins. *Pediatrics in Review*, 26(6), 191-198.
- Mandal, G. C., Mandal, A., & Chakraborty, A. (2022). The toxic effect of lead on human health: A review. *Human biology and public health*, 3.
- Markowitz, M. (2021). Lead poisoning: An update. *Pediatrics in review*, 42(6), 302-315.
- Mayans, L. (2019). Lead poisoning in children. *American Family Physician*, 100(1), 24-30.
- Naicker, N., Norris, S. A., Mathee, A., Becker, P., & Richter, L. (2010). Lead exposure is associated with a delay in the onset of puberty in South African adolescent females: findings from the Birth to Twenty cohort. *Science of the total environment*, 408(21), 4949-4954.
- Naranjo, V. I., Hendricks, M., & Jones, K. S. (2020). Lead toxicity in children: an unremitting public health problem. *Pediatric Neurology*, 113, 51-55.
- Nriagu, J., Oleru, N. T., Cudjoe, C., & Chine, A. (1997). Lead poisoning of children in Africa, III. Kaduna, Nigeria. *Science of the Total Environment*, 197(1-3), 13-19.
- Obeng-Gyasi, E. (2019). Sources of lead exposure in various countries. *Reviews on environmental health*, 34(1), 25-34.
- Ojo, J. O., Oketayo, O. O., & Horvat, M. (2010). Marked lowering in Pb blood levels follows lead phase-out from gasoline in Nigeria. In *Proceedings of 15th International Conference on Heavy Metals in the Environment* (No. INIS-PL--2010-0005, pp. 904- 907).
- Ortega, D. R., Esquivel, D. F. G., Ayala, T. B., Pineda, B., Manzo, S. G., Quino, J. M., ... & de la Cruz, V. P. (2021). Cognitive impairment induced by lead exposure during lifespan: Mechanisms of lead neurotoxicity. *Toxics*, 9(2), 23.
- Oulhote, Y., Le Bot, B., Poupon, J., Lucas, J. P., Mandin, C., Etchevers, A., ... & Glorennec, P. (2011). Identification of sources of lead exposure in French children by lead isotope analysis: a cross-sectional study. *Environmental Health*, 10, 1-12.
- Parui, R., Nongthombam, G. S., Hossain, M., Adil, L. R., Gogoi, R., Bhowmik, S., ... & Iyer, P. K. (2024). Impact of heavy metals on human health. *Remediation of Heavy Metals: Sustainable Technologies and Recent Advances*, 47-81.
- Patrick, L. (2006). Lead toxicity part II: the role of free radical damage and the use of antioxidants in the pathology and treatment of lead toxicity. *Alternative medicine review*, 11(2).
- Pfizer, M. A., Thacher, T. D., Pettifor, J. M., Zoakah, A. I., Lawson, J. O., & Fischer, P. R. (2000). Prevalence of elevated blood lead levels in Nigerian children. *Ambulatory child health*, 6(2), 115-123.
- Popovic, M., McNeill, F. E., Chettle, D. R., Webber, C. E., Lee, C. V., & Kaye, W. E. (2005). Impact of occupational exposure on lead levels in women. *Environmental health perspectives*, 113(4), 478-484.
- Redmon, J. H., Levine, K. E., Aceituno, A. M., Litzenberger, K., & Gibson, J. M. (2020). Lead in drinking water at North Carolina childcare centers: Piloting a citizen science-based testing strategy. *Environmental research*, 183, 109126.
- Sani, A. H., & Amanabo, M. (2021). Lead: A concise review of its toxicity, mechanism and health effect. *GSC Biological and Pharmaceutical Sciences*, 15(01), 055-062.
- Selevan, S. G., Rice, D. C., Hogan, K. A., Euling, S. Y., Pfahles-Hutchens, A., & Bethel, J. (2003). Blood lead concentration and delayed puberty in girls. *Obstetrical & gynecological survey*, 58(9), 592.
- Triantafyllidou, S., & Edwards, M. (2012). Lead (Pb) in tap water and in blood: implications for lead exposure in the United States. *Critical Reviews in Environmental Science and Technology*, 42(13), 1297-1352.
- Vigeh, M., Saito, H., & Sawada, S. I. (2011). Lead exposure in female workers who are pregnant or of childbearing age. *Industrial health*, 49(2), 255-261.
- Singh, N., Kumar, A., Gupta, V. K., & Sharma, B. (2018). Biochemical and molecular bases of lead-induced toxicity in mammalian systems and possible mitigations. *Chemical research in toxicology*, 31(10), 1009-1021.

Swaringen, B. F., Gawlik, E., Kamenov, G. D., McTigue, N. E., Cornwell, D. A., & Bonzongo, J. C. J. (2022). Children's exposure to environmental lead: A review of potential sources, blood levels, and methods used to reduce exposure. *Environmental research*, 204, 112025.

Wang, Q., Zhao, H. H., Chen, J. W., Gu, K. D., Zhang, Y. Z., Zhu, Y. X., ... & Ye, L. X. (2009). Adverse health effects of lead exposure on children and exploration to internal lead indicator. *Science of the total environment*, 407(23), 5986-5992.

Wright, N. J., Thacher, T. D., Pfitzner, M. A., Fischer, P. R., & Pettifor, J. M. (2005). Causes of lead toxicity in a Nigerian city. *Archives of disease in childhood*, 90(3), 262-266.

World Health Organization. (2023). Exposure to lead: a major public health concern. Preventing disease through healthy environments. World Health Organization.

Zhang, R., Wilson, V. L., Hou, A., & Meng, G. (2015). Source of lead pollution, its influence on public health and the countermeasures. *International Journal of Health, Animal Science and Food Safety*, 2(1).