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Research Article

Neural Toxicity And Deviating Behaviors: A Review Study

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ABSTRACT

Background: neural toxicity can be mediated by several agents including the accumulation of heavy metal. Neural toxicity could cause deviating behaviors and violent actions.

Study objectives: the main objectives of the present study were to review the literature regarding the impacts of neural toxicity on deviating behaviors and violent actions and to explore the impacts of heavy metals including lead (pb) on violent actions.

Methodology: literature was reviewed for articles involved in the neural toxicity and their impacts on deviating behaviors such as violence. Articles were studied and summarized to extract the appropriate information, this helped in making up the current study.

Results: reviewed literature showed that exposure to heavy metals is associated with violent behaviors and deviating actions. Previous studies indicated that prisoners had higher levels of lead (pb) compared with their counter controls. It was also shown that schoolchildren who had poor academic achievement and committed to violent actions has higher levels of lead (pb).

Conclusions: violent actions and deviating behaviors have their environmental origin such as exposure to heavy metals. assessment of heavy metals may be a future indicator to evaluate persons with violent attitudes.

Keywords: violence, deviating behaviors, environment, heavy metals, lead (pb)

1. Introduction

Heavy metals (HMs) are prevalent in the environment and may accumulate in the body unnoticed until they cause a chronic illness. Evidence suggests that metal toxicity can impact all age groups and organs, but in developing and adult brains, the central nervous system is most severely affected and the effects persist for a prolonged period of time. There is a lack of clinically validated treatments for HM toxicity (1).
The increasing prevalence of HMs on a global scale has become a substantial concern (2). The contamination of HMs can arise from a multitude of sources, including waste disposal, contaminated chemical fertilizers and pesticides, and contaminated water sources like rivers (3).
Due to their elevated atomic mass and density, HMs such as cadmium, zinc, mercury, arsenic, silver, chromium, copper, iron, and platinum can cause damage to both human health and the environment (4). A significant environmental concern that affects plants, animals, and humans is the contamination of water with HMs (5). HMs present risks even at low concentrations due to their non-biodegradable nature (6).

2. Environmental toxins and their health impacts

Certain chemical constituents found in human sustenance are hazardous in number in the hundreds. Both heavy metals and organophosphates (OPs) are toxic. Fuel and water are contaminated by compounds that

readily accumulate in the ecosystem. After entering the body, OPs and heavy metals are capable of influencing the CNS and immune system. By augmenting oxidative stress and diminishing antioxidant defenses, these chemicals induce neurotoxicity. Neurodegenerative and developmental disorders may result from exposure to heavy metals and OP. Persistent alterations in behavior may result from exposure to these hazards. On account of CNS redundancy, which may compensate for initial injuries, early exposures may not become apparent until much later in life. Antioxidant-rich diets have demonstrated unexpected therapeutic benefits. According to numerous studies, neurodegenerative diseases caused by environmental pollution can be mitigated by antioxidant foods. **(7).**

3. Industrial revolution and chronic diseases resulting from exposure to HMs

Heavy metal exposure has been linked to a constant increase in acute and chronic health problems since the industrial and agricultural revolutions. Socioeconomic and environmental factors contribute to the prevalence of this issue in developing nations (e.g., China, India, Africa, South America), but it also impacts developed nations (e.g., the Flint catastrophe in the United States). Mental disabilities, cognitive and neurological abnormalities, and mental disorders that are caused by HMs are frequently chronic, progressive, and permanent. The accumulation and impact of heavy metals on organisms, species, and biosystems, which may result in irreversible harm to physical and mental health, have been the subject of numerous studies (8-10).
The impact of heavy metal bioaccumulation on the central nervous system (CNS) and peripheral nervous system (PNS) in human beings has been demonstrated to be more pronounced (1).

4. Developmental impacts of exposure to heavy metals

The immature blood-brain barrier and blood-cerebrospinal fluid interfaces permit heavy metal compounds to penetrate the central nervous system (CNS) via bypass or sequestration mechanisms during prenatal and neonatal development. This results in chemical-induced neurotoxicity. Certain toxic metals are transported by ligands across the developing body, while others pass through calcium and zinc membrane channels in the form of free ions. There were detected levels of mercury, lead, arsenic, nickel, copper, and iron in the bodily fluids (such as urine, saliva, and perspiration) and tissues of infants and developing embryos (11).
Recent studies have provided confirmation that mental neurotoxicity can occur as a consequence of mercury exposure during pregnancy. This neurotoxicity manifests as malformations of the central nervous system (12-14). These studies indicate that early-life neurotoxicity induced by HMs may have long-lasting consequences. Extensive research has determined that lead, a heavy metal, contributes to neurotoxicity during development (15). During development, low lead concentrations (5 mg/dL) were associated with changes in brain volume, decreased IQ, shorter stature, behavioral issues, and hearing loss; at 75 mg/dL, coma and mortality were observed (16). Although there has been a gradual reduction in acceptable lead levels in children, concentrations below 5 mg/dL continue to be associated with adverse effects such as impulsive behaviour, speech impairment, delayed reaction times, compromised non-verbal reasoning, attention deficit disorder, and low academic performance (17). This is supported by recent assessments conducted on thousands of children across different continents (18-20). There may come a time to declare that there is no clinically tolerable level of lead in infants and pregnant women. Ingestion of endocrine disruptors such as lead, cadmium, mercury, and arsenic during pregnancy may result in adverse long-term consequences for both the mother and the fetus, including spontaneous miscarriage and CNS injury, respectively (21). According to research (22), children whose drinking water contained manganese levels exceeding 0.24 mg/mL exhibited diminished levels of school readiness, anxiety, melancholy, and ADHD. Prenatal mercury and lead exposure was found to have a significant positive correlation with early neurodevelopmental performance in children aged 1 to 2 years (mental, psychomotor, social, and behavior assessment scores), according to a comprehensive South Korean study (23).

5. Exposure to heavy metals and deviating behaviors

A study was conducted by Alkhatib et al (24) to investigate lead levels in two prisons located in Northern Jordan and to ascertain whether there was a correlation between lead levels and crime-related characteristics. The study employed a methodology that involved conducting in-person interviews with 46 detainees, visiting two institutions in Jordan, and collecting blood samples. A 27-person reference group was also investigated. The assessment of blood lead levels was conducted utilizing atomic absorption spectroscopy. Inmates had elevated blood lead levels (0.924±1.79 μg/dL) in comparison to the control group (0.570 ±0.560 μg/dL), according to the study. There was no statistically significant variation observed among the research groups (P=0.480). Convict lead exposure was positively correlated with congested traffic, according to the study (P = 0.038). Additionally, monthly income, blood lead levels, and family size were all positively correlated (P=0.000). Although no significant correlation was found between the blood lead concentrations of prisoners and reference participants, the concentration of lead in the blood of prisoners is approximately double that of reference participants; therefore, these results may lend credence to the environmental hypothesis that lead removal from gasoline reduces crime rates in the United States.

The United States experienced a progressive increase in violent crime rates until the mid-1990s, at which point they reportedly began to decline by 3-4% annually. A 13% decline was documented in 2010 (24).
The sudden decline in violent crime defied the explanations put forth by crime scientists, who relied on demographic, cultural, economic, and law enforcement theories (25). The environmental hypothesis was formulated in lieu of the unexpected decline in violent crime rates. It is consistent with the neurotoxicity hypothesis, which posits that aggressive and violent behavior is caused by lead (Pb) exposure and impacts neurotransmitter and hormonal systems (24). A correlation between adolescent antisocial behavior and early lead exposure was identified by Dietrich et al. (26).

Alalawneh and Alkhatib (27) recently undertook a study in response to the growing body of evidence linking lead (pb) exposure to mental disorders and violence. In Qatari institutions, the researchers sought to determine the prevalence of lead (pb) and the impact of that element on academic achievement and attitudes toward violence. It was a case-control investigation. On the basis of their academic performance, 170 candidates were selected at random. A tailored questionnaire was developed specifically for the purpose of this inquiry. Urine lead (pb) was quantified using atomic absorption across a spectrum. Pb content, slumber duration, and preservative utilization exhibited an inverse correlation with grades. Additionally, student performance was correlated with foul and violent conduct. Strong performers have lower grades (50-60) which are significantly correlated with lower concentrations of Pb in their urine. Further, this research demonstrates that academic achievement can be predicted by quantitative physiological indicators and observable behaviors. A correlation is established between the number of hours students slumber and their consumption of preservatives in an effort to educate students, school administrators, and the general public. Academic performance and conduct may be adversely affected by toxins such as Pb, necessitating increased validation and public health standards. Lead exposure is, in general, a social concern.
The neurological system is adversely affected by lead (Pb), a substance that is commonly found in the environment (27). Elevated concentrations of lead (Pb) in the subclinical range have been found to induce encephalopathy, which has profound consequences for social behavior and cognitive, affective, and motivational functions (27). After adverse exposure, lead (Pb) is absorbed and retained in the blood and bones. Exposure to lead (Pb) has the potential to induce modifications in bodily systems, such as the central nervous system, which may result in academic underachievement and heightened attitudes towards school violence. Numerous studies conducted in the United States have discovered that children who have dangerously high levels of Pb in their bloodstream experience stunted development, learning challenges such as difficulties with reading and writing, and hearing impairments. Children 1-2 years of age and black (non-Hispanic) children 1-5 years of age who have toxic levels of Pb are more likely to experience delays in the development of their nervous systems and consume non-nutritional foods, according to CDC data (27). Aggression and criminal activity are associated with dangerously high Pb blood levels (28). Furthermore, it gives rise to cardiovascular issues and mental cognition impairments in 16.8 million individuals (29).
A substantial correlation was discovered between cumulative air Pb levels and cognitive impairment and delayed brain development in children, according to one study. The study investigated the correlation between urinary Pb concentrations and violent tendencies. The correlation was confirmed and a correlation was established by the study. Poisonous urine in infants is caused by an excess of Pb. 250 individuals were examined by Wrieht et al (30). A correlation was discovered between Pb exposure and violent behavior and criminal activity. In addition, hostile behavior increases by 50% for every six years that the decimeter rate increases by 50 micrograms. Pregnant rodents that were provided water containing 10µg/ml of Pb throughout gestation, nursing, and weaning exhibited neurobehavioral impairments in their severely obese young (31).
Between 1921 and 1936, James and Christopher (32) described how Pb pipelines affected homicides. They discovered that Pb exposure from these pipelines is the leading cause of homicides.

6. Impacts of Prenatal Exposure to Heavy Metals on Infant Development

Prenatal development is susceptible to neurotoxicity due to environmental flexibility and intensive fetal brain development (33). The DOHaD hypothesis posits that prenatal and perinatal environmental adversity may have long-lasting detrimental effects on health and development via a variety of mechanisms (34). First, heavy metals can cause damage to both the mother and fetus through epigenetic modifications to the genome (35). Secondly, certain heavy metals can cause fetal neurotoxicity by crossing the placental and blood-brain barriers (36). Furthermore, the transfer of nutrients may be impeded or perinatal endocrine function compromised by heavy metals (37). The emotional development of children could potentially be adversely affected in the long run by these practices (7).
The emotional development of children in relation to perinatal heavy metal exposure has been the subject of a single study. Stroustrup et al. (38) investigated the impact of lead and mercury in the prenatal blood and bone of the mother on the emotion expression and management of 500 infants. Prenatal lead exposure was associated with more intense emotional reactions and difficulty regulating them in children, but not mercury exposure.
Prenatal heavy metal exposure may have detrimental effects on the neurocognitive development and mental health of children, according to research. Prenatal lead and cadmium exposure in cord blood was associated

with behavioral and emotional problems, such as aggression and depression, in children aged 7 to 8 years, according to a study (N = 270) (39). An additional investigation (N = 233) discovered that one-year-olds' psychomotor development was impeded by elevated levels of mercury in the umbilical cord and maternal blood (40). Prenatal methylmercury exposure in cord blood and mother hair predicted difficulties with attention, expressive language, and long-term memory in 7-year-olds (41). ADHD and autism spectrum disorders have also been associated with prenatal heavy metal exposure (42).

Metal accumulation can occur within the body, despite the fact that the majority of research focuses on the developmental effects of particular metals (43). Seven heavy metals (cadmium, chromium, cobalt, lead, mercury, nickel, and silver) negatively impacted the health and cognitive development of infants whose mothers were pregnant, according to an American study (N = 92). The children of the exposed mother exhibited elevated rates of infections, ailments, as well as deficits in language, perception, and motor skills. In particular, heavy metals emitted by contemporary weaponry may co-occur. Tissues obtained from war wounds are composed of carcinogenic, teratogenic, and noxious elements such as uranium, mercury, vanadium, chromium, and strontium (44). The researcher investigated the effect of perinatal heavy metal exposure on the emotional development of infants, with a particular emphasis on heavy metals that may be associated with weapons.

In recent decades, contamination of animal-based commodities with heavy metals and trace elements has posed a worldwide threat to food safety (45). Heavy metals pose significant environmental and health hazards as a result of their persistent nature, toxicity, and capacity to accumulate in ecosystems; furthermore, they can infiltrate human bodies via the food chain (46). Hazardous chemical contaminants include arsenic, cadmium, lead, mercury, chromium, and nickel; possibly essential contaminants include vanadium; and essential contaminants include copper, zinc, iron, manganese, selenium, and cobalt (47). Depending on their nature, quantity, and level of exposure, heavy metals can cause both beneficial and detrimental effects on the body; therefore, their presence must be investigated (48). Extended exposure to these metals has the potential to induce a range of adverse health effects, such as mutagenic and teratogenic consequences, bone and cardiovascular disorders, infertility, neurotoxicity, renal complications, depression, hypertension, psychological disorders, gastrointestinal cancer, gastric ulcers, sideroblastic anemia, liver dysfunction, and sensory impairments (e.g., loss of taste, smell, and appetite). The contamination of food, particularly feed and feed additives in poultry products, with metals has become a matter of concern due to the escalation of environmental pollution caused by industrialization, deforestation, and waste disposal (49, 50).

7. Impacts of heavy metal toxicity

The globalization of heavy metal contamination is occurring. Heavy metals are capable of being absorbed by fish via the gills, body surface, and digestive tract (51).

HMs have the potential to deplete energy and cause damage to various organs, including the liver, brain, lungs, kidneys, and blood (43). Extended periods of exposure may induce degenerative processes in bodily tissues, nerves, and tissues, emulating the symptoms of various diseases such as Alzheimer's, Parkinson's, muscle dystrophy, and multiple sclerosis (52). Acute lead (Pb) exposure has the potential to induce a range of adverse effects, including but not limited to nausea, vomiting, renal failure, fatigue, insomnia, arthritis, hallucinations, and vertigo (53). Acrodynia, also known as pink disease, is brought on by mercury poisoning. Mercury exposure has the potential to induce structural changes in the brain, which may manifest as cognitive impairment, irritability, timidity, tremors, vision or hearing problems (54). Excessive levels of metallic mercury vapors can induce lung damage, vomiting, diarrhea, vertigo, skin rashes, and hypertension in individuals exposed to them for a brief period of time. Headache, depression, memory loss, palpitations, fatigue, and hair loss are symptoms of organic mercury toxicity. Identification of these symptoms may present a difficulty owing to their concomitant presentation with other disorders (55).

Manganese, an essential element, is involved in numerous bodily physiological processes. Acute exposure has the potential to mitigate apoptotic cell death and exert a neuroprotective effect. However, overexposure can lead to neurological complications such as Alzheimer's and Parkinson's disease, which are characterized by cell death and disruptions in homeostasis (56). To maintain cellular Mn homeostasis, sufficient cellular uptake, storage, and excretion via ion channels and receptors are necessary. Homeostatic processes downregulate metal uptake receptors and upregulate cell discharge receptors in response to excessive Mn exposure. Nonetheless, prolonged manganese accumulation contributes to mitochondrial dysfunction by increasing ROS production. Cytochrome c, which is liberated by dysfunctional mitochondria, induces caspase-9 activation and caspase-3 cleavage. The fragment of cleaved caspase-3 interacts with pro-apoptotic PKCd. Caspase-3 cleaves PKCd, resulting in DNA fragmentation and death (55).

Central nervous system cognitive impairment is the result of arsenic ingestion. It is correlated with a range of neurologic disorders, such as neurodegenerative diseases and neurodevelopmental abnormalities. Synaptic transmission and neurotransmitter equilibrium are both altered by arsenic poisoning (58). Neurotoxicity caused by arsenic is associated with multiple apoptotic mechanisms. Following mitochondrial apoptosis, arsenic and its methylation metabolites induce caspase-mediated death in brain cells via MAPK signaling pathways including ERK2, JNK, and p38. Arsenic also induces an increase in intracellular calcium, a factor that controls apoptosis. Conversely, inhibition of mTOR and stimulation of AMPK can induce autophagy and

result in cellular demise. Autophagy is a homeostatic process in which cellular components are released by double-membraned autophagosomes for lysis (58).
Amyotrophic lateral sclerosis, Parkinson's disease, Alzheimer's disease, and multiple sclerosis are neurodegenerative disorders induced by cadmium neurotoxicity (59). Cadium significantly impairs the functions of the peripheral nervous system (PNS) and central nervous system (CNS), as evidenced by preclinical research. Symptoms observed in adults and children include peripheral neuropathy, olfactory dysfunctions, neurological disturbances, learning disabilities, mental retardation, motor function impairment, and behavioral changes (60). Furthermore, numerous biological processes are affected, such as cell proliferation, differentiation, and mortality. Cadmium induces apoptosis-induced death of brain cells, which has been found to have neurotoxic effects on endocrine function, gene expression, neurogenesis, and epigenetics (61). Pathological investigations have revealed that thallium poisoning causes damage to the brain and peripheral nerves in both animals and humans. Certain regions of the brain develop necrosis, vascular engorgement, cerebellar edema accompanied by Purkinje cells, and edema (62).
In addition to cadmium, arsenic, and manganese, numerous heavy metals pose risks. Similar to iron, excessive amounts of copper and zinc in the brain can impede neurodegeneration (63). Wilson's disease, an inherited malady that induces neurobehavioral difficulties similar to those of schizophrenia, is attributed to an overabundance of copper retention. Neurodevelopment is adversely affected by zinc deficiency, whereas the consequences of zinc excess remain uncertain (64). Copper exacerbates the neurotoxicity induced by zinc, as demonstrated in the research of Tanaka and Kawahara (65).

8. Conclusions

Deviating behaviors may be caused by the accumulating effects of heavy metals such as lead and mercury. Increased levels of heavy metals impacts various systems in the body including nervous system.The crime rates have been lowered when heavy metals such as lead (pb) had been removed or their existence reduced in environment. Classical theories of social impacts in criminology could not explain the lowering rates of crimes and accordingly the environmental theory of crime committing found acceptance. The studies in schoolchildren and prisons showed that the existence of lead (pb) to be associated with violent actions. According to this context, we recommend testing of heavy metals to be considered in analysis and understanding of crimes and violent actions.

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References:

1. Kothapalli, Chandrasekhar R. Differential impact of heavy metals on neurotoxicity during development and in aging central nervous system. Current Opinion in Toxicology, 2021; 26, 33-38.
2. Ali, M.M., Islam, M.S., Islam, A.R.M.T., Bhuyan, M.S., Ahmed, A.S., Rahman, M.Z., Rahman, M.M., 2022. Toxic metal pollution and ecological risk assessment in water and sediment at ship breaking sites in the Bay of Bengal Coast, Bangladesh. Marine Pollution Bulletin 175, 113274.
3. Ali MM, Ali ML, Jahan Rakib MR, Islam MS, Bhuyan MS, Senapathi V, Chung SY, Roy PD, Sekar S, Md Towfiqul Islam AR, Rahman MZ. Seasonal behavior and accumulation of some toxic metals in commercial fishes from Kirtankhola tidal river of Bangladesh - A health risk taxation. Chemosphere. 2022 Aug;301:134660. doi: 10.1016/j.chemosphere.2022.134660. Epub 2022 Apr 22. PMID: 35469901.
4. Gu, Y.-G., Ning, J.-J., Ke, C.-L., Huang, H.-H., 2018. Bioaccessibility and human health implications of heavy metals in different trophic level marine organisms: a case study of the South China Sea. Ecotoxicol. Environ. Saf. 163, 551–557.
5. Wang, N., Qiu, Y., Hu, K., Huang, C., Xiang, J., Li, H., Tang, J., Wang, J., Xiao, T., 2020. One-step synthesis of cake-like biosorbents from plant biomass for the effective removal and recovery heavy metals: effect of plant species and roles of xanthation. Chemosphere 129129.
6. Ferrey, M.L., Coreen Hamilton, M., Backe, W.J., Anderson, K.E., 2018. Pharmaceuticals and other anthropogenic chemicals in atmospheric particulates and precipitation. Sci. Total Environ. 612, 1488–1497.
7. Caridad López-Granero, Michael Aschner, Fernando Sánchez-Santed (2023). Chapter 22 - Environmental toxicants (OPs and heavy metals) in the diet: What are their repercussions on behavioral/neurological systems? Diet and Nutrition in Neurological Disorders. Academic Press, https://doi.org/10.1016/B978-0-323-89834-8.00038-6.
8. Iqubal A, Ahmed M, Ahmad S, Sahoo CR, Iqubal MK, Haque SE: Environmental neurotoxic pollutants: Review. Environ Sci Pollut Res Int 2020, 27:41175–41198.
9. Rahman Z, Singh VP: The relative impact of toxic heavy metals (thms) (arsenic (as), cadmium (cd), chromium (cr)(vi), mercury (hg), and lead (pb)) on the total environment: an overview. Environ Monit Assess 2019, 191:419.

10. Sall ML, Diaw AKD, Gningue-Sall D, Aaron SE, Aaron JJ: Toxic heavy metals: impact on the environment and human health, and treatment with conducting organic polymers, a review.Environ Sci Pollut Res Int 2020, 27:29927–29942.
11. Zheng W, Aschner M, Ghersi-Egea JF: Brain barrier systems: a new frontier in metal neurotoxicological research. Toxicol Appl Pharmacol 2003, 192:1–11.
12. Wang J, Wu W, Li H, Cao L, Wu M, Liu J, Gao Z, Zhou C, Liu J, Yan C: Relation of prenatal low-level mercury exposure with early child neurobehavioral development and exploration of the effects of sex and dha on it. Environ Int 2019, 126:14–23.
13. Wang X, Qi L, Peng Y, Xia W, Xu S, Li Y, Zhang H: Urinary concentrations of environmental metals and associating factors in pregnant women. Environ Sci Pollut Res Int 2019, 26:13464–13475.
14. Engwa GA, Ferdinand PU, Nwalo FN, Unachukwu MN: Mechanism and health effects of heavy metal toxicity in humans. In Poisoning in the modern world. New tricks for an old dog?. Edited by Karcioglu O, Arslan B, IntechOpen; 2019.
15. Ahmad F, Liu P: (ascorb)ing pb neurotoxicity in the developing brain. Antioxidants 2020, 9:1311.
16. Anyanwu BO, Ezejiofor AN, Igweze ZN, Orisakwe OE: Heavy metal mixture exposure and effects in developing nations: an update. Toxics 2018, 6:65.
17. Rocha A, Trujillo KA: Neurotoxicity of low-level lead exposure: history, mechanisms of action, and behavioral effects in humans and preclinical models. Neurotoxicology 2019, 73: 58–80.
18. Desrochers-Couture M, Oulhote Y, Arbuckle TE, Fraser WD, Seguin JR, Ouellet E, Forget-Dubois N, Ayotte P, Boivin M, Lanphear BP, Muckle G: Prenatal, concurrent, and sex-specific associations between blood lead concentrations and iq in preschool canadian children. Environ Int 2018, 121: 1235–1242.
19. Polanska K, Hanke W, Pawlas N, Wesolowska E, Jankowska A, Jagodic M, Mazej D, Dominowska J, Grzesiak M, Mirabella F, Chiarotti F, et al.: Sex-dependent impact of low-level lead exposure during prenatal period on child psychomotor functions. Int J Environ Res Publ Health 2018, 15:2263.
20. Gagnon-Chauvin A, Bastien K, Saint-Amour D: Environmental toxic agents: the impact of heavy metals and organochlorides on brain development. In Handbook of clinical neurology, vol. 173; 2020:423–442.
21. Malin AJ, Wright RO: The developmental neurotoxicity of cadmium. In Handbook of developmental neurotoxicology; 2018, 407–412.
22. Bjørklund G, Dadar M, Peana M, Rahaman MS, Aaseth J: Interactions between iron and manganese in neurotoxicity. Arch Toxicol 2020, 94:725–734.
23. Kim S, Eom S, Kim HJ, Lee JJ, Choi G, Choi S, Kim S, Kim SY, Cho G, Kim YD, Suh E, et al.: Association between maternal exposure to major phthalates, heavy metals, and persistent organic pollutants, and the neurodevelopmental performances of their children at 1 to 2 years of age- check cohort study. Sci Total Environ 2018, 624:377–384.
24. Ahed J Alkhatib, Haitham Mohammad Alta'any, Qasem Mohammad Abdelal (2014). Lead exposure and possible association with violent crimes: a field study in two Jordanian prisons. European Scientific Journal October, 10 (30), 1-8.
25. Rick Nevin (2007). Understanding international crime trends: The legacy of preschool lead exposure. Environmental Research 104, 315–336.
26. Dietrich KN, Ris MD, Succop PA, Berger OG, Bornschein RL (2001). Early exposure to lead and juvenile delinquency. Neurotoxicol Teratol, 23:511–8ر
27. Alalawneh MA and Alkhatib AJ. Studying the Prevalence of Lead among Schoolchildren in Qatar and its Impacts .on School Performance and Attitudes towards Violence in School. Adv Clin Toxicol 2022, 7(4): 000250.
28. Mbonane TP, Mathee A, Swart A, Naicker N (2019) study protocol to determine the association between lifetime lead exposure and violent criminal behaviour in young males in conflict with the law. BMC Public Health 19: 932. .2
29. Naicker N, Jager P, Naidoo S, Mathee A (2018) Is There a Relationship between Lead Exposure and Aggressive .Behavior in Shooters? Int J Environ Res Public Health 15(7): 1427. 3
30. Wright JP, Dietrich KN, Ris MD, Hornug RW, Wessel SD, et al. (2008) Association of prenatal and childhood .blood lead concentrations with criminal arrests in early adulthood. PLoS Med 5(5): e101. 4
31. Swati B, Maher TJ)2012(.Neurobehavioral effects of lead and manganese individually and in combination in .developmentally exposed rats. Neurotoxicology 33(5): 1117-1127. 5.
32. James JF, Christopher M (2016) Lead Exposure and Violent Crime in the Early Twentieth Century. Explorations in Economic History 62: 51-86. 6.
33. Andersen, S. L. (2003). Trajectories of brain development: point of vulnerability or window of opportunity? Neuroscience & Biobehavioral Reviews, 27, 3–10. https://doi.org/10.1016/S0149
34. Darney, S., Fowler, B., Grandjean, P., Heindel, J., Mattison, D., & Slikker, W. (2011). Prenatal Programming and Toxicity II (PPTOX II): Role of environmental stressors in the developmental origins of disease. Reproductive Toxicology, 271–276. https://doi.org/10.1016/j.reprotox.2010.10.010.
35. Cheng, T. F., Choudhuri, S., & Muldoon-Jacobs, K. (2012). Epigenetic targets of some toxicologically relevant metals: A review of the literature. Journal of Applied Toxicology, 32, 643–653. https://doi.org/10.1002/jat.2717.

36. Vahter, M. (2009). Effects of arsenic on maternal and fetal health. Annual Review of Nutrition, 29, 381–399. https://doi.org/10.1146/annurev-nutr-080508-141102.
37. Kippler, M., Hoque, A. M., Raqib, R., Ohrvik, H., Ekström, E. C., & Vahter, M. (2010). Accumulation of cadmium in human placenta interacts with the transport of micronutrients to the fetus. Toxicology Letters, 192, 162–168. https://doi.org/10.1016/j.toxlet.2009.10.018.
38. Stroustrup, A., Hsiao-Hsien, H., Svensson, K., Schnaas, L., Cantoral, A., Solano González, M., & Hsu, H. (2016). Toddler temperament and prenatal exposure to lead and maternal depression. Environmental Health: A Global Access Science Source, 15, 1–12. https://doi.org/10.1186/s12940-016-0147-7.
39. Sioen, I., Den Hond, E., Nelen, V., Van de Mieroop, E., Croes, K., Van Larebeke, N., … Schoeters, G. (2013). Prenatal exposure to environmental contaminants and behavioural problems at age 7–8years. Environment International, 59, 225–231. https://doi.org/10.1016/j.envint.2013.06.014.
40. Jedrychowski, W., Jankowski, J., Flak, E., Skarupa, A., Mroz, E., Sochacka?Tatara, E., & Perera, F. (2006). Effects of prenatal exposure to mercury on cognitive and psychomotor function in one-year-old infants: epidemiologic cohort study in Poland. Annals Of Epidemiology, 16(6), 439–447. https://doi.org/10.1016/j.annepidem.2005.06.059.
41. Grandjean, P., Weihe, P., White, R. F., Debes, F., Araki, S., Yokoyama, K., Murata, K., Sørensen, N., Dahl, R., & Jørgensen, P. J. (1997). Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. Neurotoxicology and Teratology, 19(6), 417–428.
42. Roberts, A. L., Lyall, K., Hart, J. E., Laden, F., Just, A. C., Bobb, J. F., … Weisskopf, M. G. (2013). Perinatal air pollutant exposures and autism spectrum disorder in the children of nurses' health study II participants. Environmental Health Perspectives, 121(8), 978–984. https://doi.org/10.1289/ehp.1206187.
43. Jaishankar, M., Tseten, T., Anbalagan, N., Mathew, B. B., & Beeregowda, K. N. (2014). Toxicity, mechanism and health effects of some heavy metals. Interdisciplinary toxicology, 7(2), 60–72. https://doi.org/10.2478/intox-2014-0009.
44. Skaik, S., Abu-Shaban, N., Abu-Shaban, N., Barbieri, M., Barbieri, M., Giani, U., … Manduca, P. (2010). Metals detected by ICP/MS in wound tissue of war injuries without fragments in Gaza. BMC International Health and Human Rights, 10, 17–31. https://doi.org/10.1186/1472-698X-10-17.
45. Xin, Z., Wenchao, Z., Zhenguang, Y., Yiguo, H., Zhengtao, L., Xianliang, Y., Xiaonan, W., Tingting, L., Liming, Z., 2015. Species sensitivity analysis of heavy metals to freshwater organisms. Ecotoxicol. (London, England) 24 (7–8), 1621–1631. https://doi. org/10.1007/s10646-015-1500-2.
46. Roy, D., Rahman, F., Islam, S.S., et al, 2022. Bioaccumulation of heavy metal in urban pond reared Oreochromis niloticus, water, sediment, and handmade feed in Bangladesh and human health risk implications. Arab. J. Geosci. 15, 959. https://doi.org/10.1007/ s12517-022-10260-w.
47. Makedonski, L., Peycheva, K., Stancheva, M., 2017. Determination of heavy metals in selected black sea fish species. Food Control 72 (B), 313–318. https://doi.org/10.1016/j.foodcont.2015.08.024.
48. Shaheen, N., Ahmed, M.K., Islam, M.S., et al, 2016. Health risk assessment of trace elements via dietary intake of 'non-piscine protein source' foodstuffs (meat, milk and egg) in Bangladesh. Environ. Sci. Pollut. Res. 23, 7794–7806. https://doi.org/10.1007/s11356-015-6013-2.
49. Ullah, A.K.M.A., Afrin, S., Hosen, M.M., et al, 2021. Concentration, source identification, and potential human health risk assessment of heavy metals in chicken meat and egg in Bangladesh. Environ. Sci. Pollut. Res. 29, 22031–22042. https://doi.org/10.1007/s11356- 021-17342-4.
50. Briffa, J., Sinagra, E., Blundell, R., 2020. Heavy metal pollution in the environment and their toxicological effects on humans. Heliyon 6 (9), e04691.
51. Dane, H., Sisman, T., 2020. A morpho-histopathological study in the digestive tract of three fish species influenced with heavy metal pollution. Chemosphere 242, 125212. https://doi.org/10.1016/j.chemosphere.2019.125212.
52. Järup L. Hazards of heavy metal contamination. Br Med Bull. 2003;68(1):167–182.
53. Jan, A. T., Azam, M., Siddiqui, K., Ali, A., Choi, I., & Haq, Q. M. (2015). Heavy Metals and Human Health: Mechanistic Insight into Toxicity and Counter Defense System of Antioxidants. International journal of molecular sciences, 16(12), 29592–29630. https://doi.org/10.3390/ijms161226183.
54. Guzzi, G., Ronchi, A., Pigatto, P., 2020. Toxic effects of mercury in humans and mammals. Chemosphere 263, 127990.
55. Atti, S.K., Silver, E.M., Chokshi, Y., Casteel, S., Kiernan, E., Dela Cruz, R., Kazzi, Z., Geller, R.J., 2020. All that glitters is not gold: Mercury poisoning in a family mimicking an infectious illness. Curr. Probl. Pediatr. Adolesc. Health Care, 100758.
56. Goldhaber, S.B., 2003. Trace element risk assessment: Essentiality vs. toxicity. Regul. Toxicol. Pharmacol. 38 (2), 232–242. https://doi.org/10.1016/S0273- 2300(02)00020-X.
57. Harischandra, D.S., Ghaisas, S., Zenitsky, G., Jin, H., Kanthasamy, A., Anantharam, V., Kanthasamy, A.G., 2019. Manganese-induced neurotoxicity: New insights into the triad of protein misfolding, mitochondrial impairment, and neuroinflammation. Front. Neurosci. 13. https://doi.org/10.3389/ fnins.2019.00654.

58. Garza-Lombó, C., Pappa, A., Panayiotidis, M.I., Gonsebatt, M.E., Franco, R., 2019. Arsenic-induced neurotoxicity: a mechanistic appraisal. J. Biol. Inorg. Chem. 24 (8), 1305–1316. https://doi.org/10.1007/s00775-019-01740-8.
59. Branca, J.J.V., Morucci, G., Pacini, A., 2018. Cadmium-induced neurotoxicity: Still much ado. Neural Regen. Res. 13, 1879–1882. https://doi.org/10.4103/1673- 5374.239434.
60. Marchetti, C., 2014. Interaction of metal ions with neurotransmitter receptors and potential role in neurodiseases. Biometals 27 (6), 1097–1113. https://doi.org/ 10.1007/s10534-014-9791.
61. Wang, B., Du, Y. (2013). Cadmium and its neurotoxic effects. Oxidative medicine and cellular longevity, 2013, 898034. https://doi.org/10.1155/2013/898034.
62. Saikat Mitra, Arka Jyoti Chakraborty, Abu Montakim Tareq, Talha Bin Emran, Firzan Nainu, Ameer Khusro, Abubakr M. Idris, Mayeen Uddin Khandaker, Hamid Osman, Fahad A. Alhumaydhi j, Jesus Simal-Gandara.Impact of heavy metals on the environment and human health: Novel therapeutic insights to counter the toxicity. Journal of King Saud University – Sciencem, 34 (2022) 101865.
63. Prohaska, J.R., 2000. Long-term functional consequences of malnutrition during brain development: copper. Nutrition 16 (7-8), 502–504. https://doi.org/ 10.1016/S0899-9007(00)00308-7.
64. Cai, L.u., Li, X.-K., Song, Y.e., Cherian, M.G., 2005. Essentiality, toxicology and chelation therapy of zinc and copper. Curr. Med. Chem. 12 (23), 2753–2763. https://doi.org/10.2174/092986705774462950.
65. Tanaka, K.I., Kawahara, M., 2017. Copper enhances zinc-induced neurotoxicity and the endoplasmic reticulum stress response in a neuronal model of vascular dementia. Front. Neurosci. 11. https://doi.org/10.3389/fnins.2017.00058.