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Research Article

HISTOPATHOLOGICAL IMAGE OF THE NUMBER OF NEURONS IN THE BRAIN OF MICE (*MUS MUSCULUS*) INDUCED BY LEAD HEAVY METAL

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Abstract
Lead is a known heavy metal from human history to date for various uses, abuses and toxicities. Lead poisoning has been recognized as one of the most serious environmental health issues worldwide, especially children living in developing countries. Lead can damage or affect the organs of the body, among others, the nervous system, kidneys, reproductive system, and heart, as well as disturbances in the brain so that intellectual and mental disorders in the brain. This study aims to see the effect of lead on the number of neurons in the hippocampus in mice experimental animals. The mice used amounted to 10 tails and divided into two for the control group and the treatment group. The control group, each of which was 5 tails, was then given unbleached drinking water while the treatment group was given drinking water mixed with acetate with concentration of 1000ppm for 17 days in adlibitum. The observations were taken from each of the 5 fields of view on each preparation with a magnification of 40x. The calculation of the number of neuron cells is then analyzed descriptive statistics and different test. The result showed that control group with mean ± SD were 127,6 ± 7,9, and for treatment group that was 80,0 ± 20,9. T independent test result showed p = 0,001 (p <0,05) which means that there was a significant difference between control and treatment group. The conclusion of this research is the existence of neuron cell damage in mice that get treatment. By showing the number of neurons less than the control mice. Suggestion to develop this research hence need to continue research to find neuro protector compound specially from lead work in brain.

Keywords: Brain, Hippocampus, Lead

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INTRODUCTION

Lead (Pb) has a low melting point, is chemically active, and can be used to coat metals to prevent corrosion (Alyami et al., 2026). Lead is a heavy metal known throughout human history for its various uses, misuse, and toxicity (Kiris et al., 2023). With the enactment of laws and regulations regarding its usage, the excessive use of lead is now reported as a consumable material and environmental pollutant (Generalova et al., 2025). Lead poisoning has been known since ancient Egypt and Greece around 5000 years ago (Maddusa et al. 2023). It is one of the oldest diseases in the history of human civilization (Xu et al., 2025). In recent years, lead poisoning has become a significant environmental health issue worldwide, particularly among children living in developing countries (Hardiansyah 2025). Lead (Pb) naturally exists in the Earth's crust but can also be produced by human activities, reaching levels 300 times higher than natural Pb.

Sources of lead poisoning can stem from vehicles using leaded fuel, as well as from metal mining, smelting, lead disposal factories, industrial recycling products, dust, soil, paint, toys, jewelry, drinking water, candies, ceramics, traditional medicine, and cosmetics (World Health organization 2024). Several heavy metals, including lead, are known to induce the production of reactive oxygen species (ROS) and alter unsaturated fatty acids in membranes (An et al., 2026). In addition, ROS are highly reactive with lipid membranes, proteins, and DNA (Generalova et al., 2025). ROS are also considered major contributors to oxidative stress and the rapid cellular damage in the body.

Lead in the environment has negative impacts. Villalva (2025) stated that lead disrupts organs with its primary targets being the bones, brain, blood, kidneys, and thyroid glands (Gonzalez-Villalva et al. 2025), while Maria (2025) found that lead can damage or affect several body organs, including the nervous system, kidneys, reproductive system, endocrine system, and heart, as well as causing brain dysfunction, resulting in impaired cognitive abilities and mental health issues (Maria et al. 2025). According to Rainone G et al (2025) 25% of children affected by lead encephalopathy may suffer permanent damage to the central nervous system (Rainone et al. 2025). Lead neurotoxicity has been identified in both humans and animals, with acute encephalopathy suspected to cause permanent damage to the central nervous system, which can specifically affect children (Bao et al., 2026). This damage is often reflected in abnormal behavior and learning difficulties, with or without accompanying retardation.

The brain is a particularly sensitive target for lead poisoning because it contains relatively low concentrations of enzymes, which are less capable of defending against stress (Cacabelos et al., 2025). A recent study observed behavioral changes during a period of lead exposure with a dose of 4% lead acetate in water provided ad libitum (Son et al. 2025). These behaviors included aggressive actions, while histopathological measurements revealed neuron damage, brain fiber damage, and an increase in glial cell numbers (Bellver-Sanchis et al., 2026). This study aims to observe changes in the neuron cells of rat brains in the hippocampus region induced by lead.

RESEARCH METHOD

Research Design

This study uses a qualitative approach to investigate the impact of lead exposure on neuron cells in the hippocampus of mice (Cavestro et al., 2026). A posttest-only control group design was employed to compare the number of neurons between the control and treatment groups. The research was conducted over a period of 17 days, with the treatment group receiving drinking water mixed with 1000ppm lead acetate while the control group received unbleached drinking water (Yadav et al., 2023). The research took place at the Faculty of Medicine, Universitas Pembangunan Nasional Veteran Jakarta. The mice were housed in appropriate animal care facilities, and the study adhered to ethical standards for animal research.

Research Target/ Subject

The subjects of this study were 10 male mice (Mus musculus), selected to ensure genetic consistency (Naskar et al., 2024). These mice were divided into two groups: a control group consisting of 5 mice and a treatment group consisting of 5 mice. The mice were chosen based on their health and age, ensuring they were mature, with similar physical characteristics (Feng et al., 2026). The sampling technique employed was random selection, ensuring unbiased group assignments. The treatment group was exposed to lead acetate while the control group was not exposed to any treatment.

Research Procedure

The procedure followed an experimental research design, with a posttest-only control group approach. After 17 days of exposure, the mice were euthanized using a humane method, and their brains were carefully removed for analysis (Gu et al., 2026). The brains were preserved in 10% Neutral Buffered Formalin and processed for histopathological examination (Wāng, 2025). The prepared tissue samples were stained using Hematoxylin-Eosin and then analyzed for neuron count in the hippocampus at a magnification of 40x.

Instruments and Data Collection Techniques

The primary data collection involved histopathological analysis of brain tissue samples. The neurons in the hippocampus were counted under a microscope at 40x magnification (Ma et al., 2024). For each brain sample, five fields of view were examined (Huang et al., 2026). The number of neurons in each field was recorded, and descriptive statistics were used to analyze the data. The data were collected using a microscope, Hematoxylin-Eosin stain, and standard histological techniques.

Data Analysis Technique

The data were analyzed using descriptive statistics (mean, standard deviation, minimum, and maximum) and compared between the control and treatment groups (Jiao et al., 2026). An independent t-test was used to assess the statistical significance of differences between the two groups (Levi et al., 2024). The normality of the data was verified, with a p-value greater than 0.05, indicating that the data followed a normal distribution, thus allowing the use of the t-test for analysis.

RESULTS AND DISCUSSION

This study aimed to determine the difference in the number of neurons in the brains of mice exposed to 1,000 ppm of lead administered ad libitum for 17 days. The results are as follows:

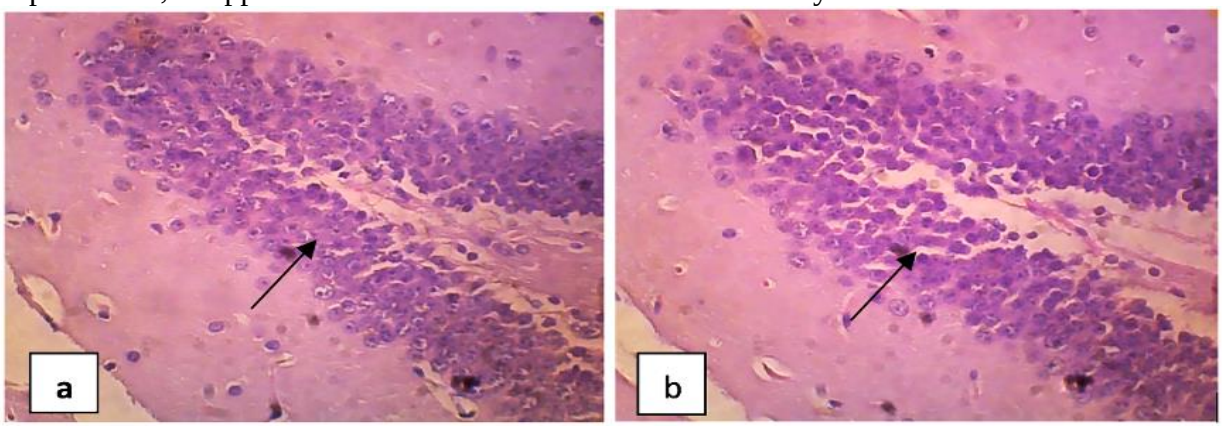

Figure 1. Microscopic Image of the Dentate Gyrus of a Mouse at 40x magnification

(a) shows a picture of a normal mouse neuron, (b) shows a picture of a mouse neuron exposed to the heavy metal lead acetate for 17 days. The number of neuron cells in (a) appears denser

Figure 1 clearly illustrates the contrast between the neuronal structure in the control and treatment groups. In panel (a), the control group displays a dense arrangement of neurons, indicative of healthy, functional neuronal activity. Conversely, panel (b) depicts the treatment group, where lead exposure resulted in fewer neurons and noticeable structural alterations (Lin et al., 2026). The neurons in the treated group appear shrunken and spaced further apart, which suggests that lead exposure led to neuronal loss and disruption of the brain's cellular architecture (Liang et al., 2026). This difference is quantified in Table 1, where the neuron count in the treatment group was significantly lower than in the control group, reinforcing the neurotoxic effect of lead acetate.

Table 1. Statistical Analysis of Neuron Cell Number Parameters

Group	Normality (p-value)	X±SD	Score
Control	0.196	127.6 ± 7.9	0.001
Treatment	0.206	80.0 ± 20.9	

The results of the preparation were then calculated the number of neurons in each visual field of 5 visual fields for each mouse. The results of the calculation were then calculated for normality to determine the distribution of the data used. The results of the normality test (table 1) showed a p value > 0.05 for both groups, so the distribution of the data used was X and SD. The results of the normality test as a requirement for conducting an independent T test were accepted because they showed a value greater than 0.05 so that the data could be continued with an independent T test.

Lead is a toxic heavy metal that can cause neurological disorders in the form of encephalopathy, such as frequent headaches, fainting, impaired thinking, and even coma (Naskar et al., 2024). However, the effects on children are hyperactive and have difficulty concentrating (Parithathvi et al. 2025). Research has found differences in neuron density in the dentate gyrus (Figure 1). The number of neurons decreases due to apoptosis in rat hippocampal cells (Rojo et al., 2025). Degeneration of some neurons in several brain regions and behavior are linked to degeneration. New evidence suggests apoptosis in PC 12 cells by increasing BAX and caspase-3 activation (Molecular mechanisms of lead neurotoxicity 2021; Neuwirth and Emenike 2024). However, several neurotoxicity mechanisms can cause apoptosis, including breakdown of the blood-brain barrier, modification of synaptogenesis, disruption of energy metabolism, disruption of calcium homeostasis, disruption of cell signaling, changes in neurotransmitter systems, particularly the glutamate system, and effects on N-Methyl-D-Aspartate (NMDA) glutamate receptors (Rautela et al., 2024). NMDA receptors play a crucial role in brain development, regulating cell morphology, differentiation, migration, and synaptogenic circuitry. Inhibition of NMDA receptors by NMDA antagonists can induce apoptosis and neurodegeneration in the rat brain. Lead can cause disruption by disrupting neurotransmitters via NMDA receptors (Molecular mechanisms of lead neurotoxicity 2021).

Excess calcium can also contribute to cell damage by increasing neurotransmitter levels, potentially damaging cells (Nam et al. 2024). One pathway affected is the activation of protein C-kinase. Protein C-kinase is a serine/threonine protein kinase involved in numerous processes essential for synaptic transmission, such as neurotransmitter synthesis, ligand-receptor interactions, ionic channel conductance, and dendritic branching (Molecular mechanisms of lead neurotoxicity 2021). The protein C-kinase family consists of 12 isozymes, each with distinct enzymatic cofactor requirements, tissue expression, and cellular distribution. One form of protein C-kinase is the γ-isoform. The γ-isoform is a calcium-dependent form of protein C-kinase and is

a target for lead-induced neurotoxicity (Li et al. 2025). The specific neurons involved in protein C-kinase have the potential for long-term pathways and memory processes. This is supported by Tamagno W et al (2025), who conducted research on animals exposed to lead. The results showed nerve cell damage due to excessive calcium or lead entering the brain barrier, resulting in increased neurotransmitter activation and glutamate receptor activation. Lead crosses the brain barrier because lead can mimic and replace calcium (Tamagno and Freeman 2025). This study demonstrated the effect of lead acetate on neuron number. This was demonstrated by the fact that the neuron count in the control group of normal mice was higher than in the mice exposed to lead acetate.

CONCLUSION

From the research results, it can be concluded that there is a difference in the number of neuron cells in mice exposed to lead acetate at a dose of 1000ppm for 17 days, which is less than in control mice in the hippocampus orally. The observed decrease in neuronal density indicates the neurotoxic effects of lead exposure, particularly in the hippocampal region, which is essential for cognitive functions such as memory and learning. The significant reduction in the number of neurons, as shown by the statistical analysis, underscores the harmful impact of lead acetate on neuronal survival and function.

DECLARATION OF AI AND AI ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the authors used an AI-assisted tool to improve language clarity and readability. After using this tool, the authors reviewed and edited the content as necessary and took full responsibility for the content of the publication.

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AUTHOR CONTRIBUTIONS

Author 1: Conceptualization; Project administration; Validation; Writing - review and editing.

Author 2: Conceptualization; Data curation; In-vestigation.

Author 3: Data curation; Investigation.

Author 4: Formal analysis; Methodology; Writing - original draft.

Author 5: Supervision; Validation.

Author 6: Other contribution; Resources; Visuali-zation.

Author 7: Other contribution; Resources; Writing - original draft.

DECLARATION OF COMPETING INTEREST

The authors declare that they have no competing financial interests or personal relationships that could have influenced the work reported in this study. The research was conducted independently without any commercial or financial relationships that could be construed as a potential conflict of interest.

REFERENCES

Al-khafaf, A., Ismail, H. Kh., & Alsaidya, A. M. A. (2021). Histopathological effects of experimental exposure to lead on nervous system in albino female rats. Iraqi Journal of Veterinary Sciences, 35(1), 45–48. https://doi.org/10.33899/ijvs.2019.126248.1273

Alyami, N. M., Alonaizan, R., Alobaid, H., Maodaa, S., Alothman, N. S., Alshiban, N. M., Alnakhli, Z. A., Alyami, M. M., & Almeer, R. (2026). Dose-inverted neurotoxicity: La_2O_3 nanoparticles cause redox dysregulation at low concentrations but excitotoxic catastrophe at high doses. *Toxicology*, *520*, 154358. https://doi.org/10.1016/j.tox.2025.154358

An, S., Ge, Y., Liu, Y., Sun, Z., Chen, M., Chi, C., Yin, Z., Liu, J., Li, J., & Chen, L. (2026). Regulatory role of GLUT1 in lead-induced neurotoxicity and the neuroprotective effect of Morinda officinalis oligosaccharides. *iScience*, *29*(4), 115342. https://doi.org/10.1016/j.isci.2026.115342

Bao, M.-Y., Li, X.-Q., Sun, Q.-Q., He, Y., Yin, Y.-J., Li, S.-H., Du, R.-Y., Ma, G.-X., Feng, C.-Y., Han, B., Jia, R., Wang, X., Wang, L.-B., Yan, Y.-P., Li, X., & Zhang, Y. (2026). Oligodendrocyte-encoded lactate dehydrogenase A couples glycolysis to remyelination via protein lactylation. *Neuron*. https://doi.org/10.1016/j.neuron.2026.02.032

Bellver-Sanchis, A., Valle-Garcia, D., Barbaraci, C., Romero-Becerra, F., Singh, R. K., Jarne-Ferrer, J., Vasilopoulou, F., Irisarri, A., Martínez-Fernández, C., Fafián-Labora, J. A., Arufe, M. C., Wüst, C., Castellanos, A., Soto, D., Casals, N., Fadó, R., Pocock, J. M., Navarro, G., Val, C., … Griñán-Ferré, C. (2026). First-in-class SAM-competitive G9a inhibitor FLAV-27 as a disease-modifying therapy for Alzheimer disease. *Molecular Therapy*, *34*(4), 2372–2407. https://doi.org/10.1016/j.ymthe.2025.12.038

Cacabelos, R., Tellado, I., Cacabelos, N., Martínez-Iglesias, O., & Naidoo, V. (2025). Chapter 1—Epigenetic machinery. In R. Cacabelos (Ed.), *Pharmacoepigenetics (Second Edition)* (Vol. 10, pp. 1–117). Academic Press. https://doi.org/10.1016/B978-0-443-18722-3.00021-9

Cavestro, C., Cascone, F., Legati, A., Izzo, R., Catania, M., Vergara, C., Rodríguez-Pascau, L., Pizcueta, P., Tiranti, V., & Di Meo, I. (2026). PPARγ activation by leriglitazone counteracts neurodegeneration and neuroinflammation in a disease-relevant mouse model of COASY dysfunction. *Pharmacological Research*, *227*, 108193. https://doi.org/10.1016/j.phrs.2026.108193

Feng, S., Sun, B., Tian, L., Li, M., & Sun, G. (2026). Nrf2 modulates the IRF7–SLC31A1 axis to suppress neuronal cuproptosis after traumatic brain injury. *Experimental Neurology*, *401*, 115747. https://doi.org/10.1016/j.expneurol.2026.115747

Generalova, A., Davidova, S., & Satchanska, G. (2025). The mechanisms of lead toxicity in living organisms. Journal of Xenobiotics, 15(5), 146. https://doi.org/10.3390/jox15050146

Gonzalez-Villalva, A., et al. (2025). Lead systemic toxicity: A persistent problem for health. Toxicology, 515, 154163. https://doi.org/10.1016/j.tox.2025.154163

Gu, Z., Qian, L., Jiang, W., Lu, S., Lin, Y., Ge, X., Liu, Z., Qu, F., & Miao, L. (2026). Iron overload induces hepatic iron deposition and oxidative damage in freshwater fish Megalobrama amblycephala by inhibiting ferroportin 1 gene expression. Comparative Biochemistry and Physiology Part B: Biochemistry and Molecular Biology, 282, 111188. https://doi.org/10.1016/j.cbpb.2025.111188

Hardiansyah, A. (2025). Tinjauan sistematis faktor lingkungan dan perilaku terhadap keracunan timbal pada anak. Journal of Medical and Health Sciences, 1(2), 55–61. https://doi.org/10.71094/jmhs.v1i2.171

Huang, J., Qin, Y., Chen, H., & Liu, F. (2026). Preclinical evidence of adenosine for early intervention in Bungarus multicinctus envenomation. Journal of Advanced Research, 81, 769–780. https://doi.org/10.1016/j.jare.2025.06.012

Jiao, D., Zhang, Y., Guo, W., Liu, S., Su, P., & Huang, L. (2026). Cardiac glycosides: Structural diversity, chemical ecology, bioactivity, and artificial synthesis. Natural Product Reports, 43(1), 167–199. https://doi.org/10.1039/d5np00050e

Kiris, I., Kukula-Koch, W., Karayel-Basar, M., Gurel, B., Coskun, J., & Baykal, A. T. (2023). Proteomic alterations in the cerebellum and hippocampus in an Alzheimer's disease mouse model: Alleviating effect of palmatine. Biomedicine & Pharmacotherapy, 158, 114111. https://doi.org/10.1016/j.biopha.2022.114111

Levi, U. I., Bintu, M. M., Daniella, O. C., Oyenike, O.-A. F., Agbonu, A. O., Adedamola, A. M., Ndidi, E., Saka, S. F., Gela, B. V., Mbagwu, S. I., Edem, E. E., Olukayode, O. J., & James, C. (2024). Neurobehavioral deficits, histoarchitectural alterations, parvalbumin neuronal damage and glial activation in the brain of male Wistar rat exposed to Landfill leachate. Journal of Chemical Neuroanatomy, 136, 102377. https://doi.org/10.1016/j.jchemneu.2023.102377

Li, Y., et al. (2025). Protein kinase C family: Structures, biological functions, diseases, and pharmaceutical interventions. MedComm (Beijing), 6(11). https://doi.org/10.1002/mco2.70474

Liang, Y., Liu, M., Jiang, H., Wang, X., Shao, H., & Wang, L. (2026). Pathophysiological and behavioral consequences of dichlorvos-induced oxidative stress in rodent models: A systematic review of mechanisms and protective strategies. Ecotoxicology and Environmental Safety, 310, 119600. https://doi.org/10.1016/j.ecoenv.2025.119600

Lin, J., Zeng, X., Su, Z., Li, X., Yu, Y., Qian, S., Hou, Q., Duan, W., Wang, Z., Liu, C., Zhang, J., Huang, C., & Liao, L. (2026). Hyodeoxycholic acid relieves neuropathic pain by activating farnesoid X receptor signaling. Journal of Advanced Research, 82, 881–900. https://doi.org/10.1016/j.jare.2025.07.017

Maddusa, S. S., Joseph, W. B. S., Pomantow, S. M. T., Asrifuddin, A., Akili, R. H., & Bahrain, N. E. A. (2023). Environmental health risks assessment of human exposure to Pb pollution around Soyoan River, Indonesia. Diversity: Disease Preventive of Research Integrity, 87–94. https://doi.org/10.24252/diversity.v3i2.34891

Maria, H., Jessica, A., Gabriella, E., Aprillia, C., & Yudiarso, A. (2025). Impact of lead (Pb) on health. Journal of Global Research in Public Health, 10(1), 1–14. https://doi.org/10.30994/jgrph.v10i1.549

Ma, W., Ren, H., Meng, X., Liu, S., Du, K., Fang, S., & Chang, Y. (2024). A review of the ethnopharmacology, phytochemistry, pharmacology, pharmacokinetics and quality control of Paeonia lactiflora Pall. Journal of Ethnopharmacology, 335, 118616. https://doi.org/10.1016/j.jep.2024.118616

Nam, D., Kim, H., Han, S. J., Son, I., & Ho, D. H. (2024). Effects of calcium ion dyshomeostasis and calcium ion-induced excitotoxicity in Parkinson's disease. Journal of Molecular Pathology, 5(4), 544–557. https://doi.org/10.3390/jmp5040037

Naskar, R., Ghosh, A., Bhattacharya, R., & Chakraborty, S. (2024). A critical appraisal of geroprotective activities of flavonoids in terms of their bio-accessibility and polypharmacology. Neurochemistry International, 180, 105859. https://doi.org/10.1016/j.neuint.2024.105859

Neuwirth, L. S., & Emenike, B. U. (2024). Neurotoxicity and outcomes from developmental lead exposure: Persistent or permanent? Environmental Health Perspectives, 132(4). https://doi.org/10.1289/EHP14809

Parithathvi, A., Harshitha, P., Mumbrekar, K. D., & Dsouza, H. S. (2025). Systematic review on neurotoxic implications of lead-induced gene expression alterations in the etiology of Alzheimer's disease. Cellular and Molecular Neurobiology, 45(1), 98. https://doi.org/10.1007/s10571-025-01613-6

Rainone, G. J., Johansen, P. M., Pressman, P., & Hayes, A. W. (2025). Putative effects of lead on the endocannabinoid system: A literature review and summary. International Journal of Molecular Sciences, 26(18), 8994. https://doi.org/10.3390/ijms26188994

Rautela, A., Chatterjee, R., Yadav, I., & Kumar, S. (2024). A comprehensive review on engineered microbial production of farnesene for versatile applications. Journal of Environmental Chemical Engineering, 12(2), 112398. https://doi.org/10.1016/j.jece.2024.112398

Rojo, A. I., Buttari, B., Cadenas, S., Carlos, A. R., Cuadrado, A., Falcão, A. S., López, M. G., Georgiev, M. I., Grochot-Przeczek, A., Gumeni, S., Jimenez-Villegas, J., Horbanczuk, J. O., Konu, O., Lastres-Becker, I., Levonen, A.-L., Maksimova, V., Michaeloudes, C., Mihaylova, L. V., Mickael, M. E., … Dinkova-Kostova, A. T. (2025). Model organisms for investigating the functional involvement of NRF2 in non-communicable diseases. Redox Biology, 79, 103464. https://doi.org/10.1016/j.redox.2024.103464

Son, Y., et al. (2025). Behavioral and neurochemical changes in mice induced by low-level lead exposure: Implications for ADHD and conduct disorders. Ecotoxicology and Environmental Safety, 302, 118735. https://doi.org/10.1016/j.ecoenv.2025.118735

Tamagno, W. A., & Freeman, J. L. (2025). Glutamate-mediated neural alterations in lead exposure: Mechanisms, pathways, and phenotypes. Toxics, 13(7), 519. https://doi.org/10.3390/toxics13070519

Wāng, Y. (2025). Physicochemical properties and combined toxic effects of micro- and nanoplastics on gut and gut-organ axes. Trends in Food Science & Technology, 165, 105265. https://doi.org/10.1016/j.tifs.2025.105265

World Health Organization. (2024). Lead poisoning and health. https://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health

Xu, Y., Liu, L., Ma, Y., Wang, C., Duan, F., Feng, J., Yin, H., Sun, L., Cao, Z., Jung, J., Li, P., & Li, Z.-H. (2025). Biotransport and toxic effects of micro- and nanoplastics in fish model and their potential risk to humans: A review. Aquatic Toxicology, 279, 107215. https://doi.org/10.1016/j.aquatox.2024.107215

Yadav, B., Chavan, S., Atmakuri, A., Tyagi, R. D., & Drogui, P. (2023). Chapter 3—Environmental effects of microplastics and nanoplastics exposure. In R. D. Tyagi, A. Pandey, P. Drogui, B. Yadav, & S. Pilli (Eds.), Current Developments in Biotechnology and Bioengineering (pp. 59–78). Elsevier. https://doi.org/10.1016/B978-0-323-99908-3.00008-7

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