Evaluating the Dose-Dependent Impact of Lead Acetate on Hippocampal Structure and Cholinergic Neurotransmission in Adult Male Wistar Rats

A.Y. Solomon, *E.M. Olayiwola and J.S. Kumbet

Department of Anatomy, College of Health Sciences, Nile University, Abuja, Nigeria

***Corresponding Author**

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ABSTRACT

Background: Lead (Pb) is a pervasive environmental neurotoxicant with well-documented deleterious effects on the central nervous system. Despite decades of research, the dose-response relationship governing hippocampal structural integrity and cholinergic neurotransmission following subacute oral lead acetate exposure remains incompletely characterized. **Objective**: This study investigated the dose-dependent effects of oral lead acetate administration on brain and body weight, hippocampal histoarchitecture, and acetylcholine levels in adult male Wistar rats over a 21-day exposure period. **Methods**: Twenty adult male Wistar rats (150–200 g) were randomly assigned to four groups (n = 5 each): a control group receiving distilled water, and three treatment groups receiving lead acetate at doses of 100, 200, and 400 mg/kg body weight per day via oral gavage. Animals were sacrificed at the end of the experiment; brain and body weights were recorded, hippocampal sections were processed for haematoxylin and eosin (H&E) staining, and acetylcholine levels were quantified. **Results**: Lead acetate administration produced dose-dependent reductions in body weight, with the highest dose (400 mg/kg) causing a 6.43% reduction from baseline. Brain weight showed limited variation across groups (1.58–1.79 g). Histological examination revealed progressive hippocampal degeneration characterized by pyramidal cell layer disorganization, reduced neuronal density, pyknotic nuclei, and extensive neuropil vacuolation, most pronounced in the 400 mg/kg group. Acetylcholine levels increased significantly across treatment groups in a dose-dependent manner ($p < 0.05$). **Conclusion**: Subacute oral lead acetate exposure induces dose-dependent neurotoxic effects in the rat hippocampus, with marked structural degeneration and cholinergic perturbation. These findings provide experimental evidence for the neurodegeneration-promoting capacity of lead and highlight the hippocampal cholinergic system as a sensitive target of lead neurotoxicity.

Keywords: lead acetate, neurotoxicity, hippocampus, acetylcholine, Wistar rats, neurodegeneration, cholinergic system

INTRODUCTION

Lead (Pb) is one of the most extensively studied environmental neurotoxicants and continues to pose a significant global public health burden, particularly in low- and middle-income countries where occupational and environmental exposures remain inadequately regulated [28, 30]. It is a naturally occurring heavy metal with no established biological function, yet its widespread industrial use in paint, batteries, plumbing, and gasoline has resulted in pervasive human and animal contamination [4, 29]. Once absorbed, lead distributes broadly across body compartments, with particular affinity for bone, blood, and nervous tissue [22].

The central nervous system (CNS) is the primary target organ of lead toxicity, and the brain exhibits heightened vulnerability due to the unique metabolic demands of neural tissue and the critical roles of metal ion homeostasis in neuronal signaling [10, 20]. Lead readily crosses the blood-brain barrier (BBB) through calcium transport mechanisms, accumulating preferentially in the hippocampus, cortex, and cerebellum [3, 11]. The hippocampus, a brain region integral to learning, memory consolidation, and spatial navigation, is particularly susceptible to

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lead-induced neurodegeneration, as its dense pyramidal neuron population and high metabolic activity render it especially vulnerable to oxidative insult and excitotoxicity [18, 8].

The mechanisms underlying lead neurotoxicity are multifactorial and interconnected. Lead displaces essential divalent cations such as calcium (Ca^{2+}), zinc (Zn^{2+}), and iron (Fe^{2+}) from their binding sites, disrupting enzyme function and receptor-mediated signaling [6]. It generates reactive oxygen species (ROS), overwhelms endogenous antioxidant defenses, and induces lipid peroxidation, protein carbonylation, and DNA strand breaks within neuronal tissue [7, 13]. Additionally, lead impairs mitochondrial function, triggers apoptotic cascades, and promotes neuroinflammation through activation of glial cells [15, 26].

The cholinergic system, which plays a central role in learning, memory, and attention, is a well-recognized target of lead toxicity [25]. Acetylcholine (ACh) is the primary neurotransmitter of the cholinergic system, and its dysregulation has been implicated in cognitive disorders including Alzheimer's disease [9]. Studies have demonstrated that lead exposure alters both the synthesis and degradation of acetylcholine, with downstream effects on synaptic plasticity and cognitive performance [21, 9].

Despite accumulating evidence for lead-induced neurotoxicity, dose-response data characterizing the hippocampal histological and cholinergic consequences of subacute oral lead acetate exposure in adult Wistar rats remain limited. Establishing such relationships is essential for understanding the neurological threshold effects of lead and informing toxicological risk assessment frameworks. This study was therefore designed to evaluate the dose-dependent effects of 21-day oral lead acetate administration on body and brain weight, hippocampal histoarchitecture, and acetylcholine levels in adult male Wistar rats.

MATERIALS AND METHODS

Study Design and Animals

This study utilized an experimental, parallel-group, controlled in vivo animal design to evaluate the dose-dependent toxicity of lead acetate. Twenty adult male Wistar rats with an initial weight range of 150–200 g were used. The animals were housed in standard polypropylene cages under controlled environmental conditions (temperature: 22 ± 2°C; 12-hour light/dark cycle) with ad libitum access to a standard laboratory diet and water. Following a one-week acclimatization period, the rats were randomly divided into four experimental groups (n = 5 per group):

• Group 1 (Control): Received distilled water orally for 21 days.

• Group 2 (Lead-100): Received lead acetate at 100 mg/kg body weight per day via oral gavage.

• Group 3 (Lead-200): Received lead acetate at 200 mg/kg body weight per day via oral gavage.

• Group 4 (Lead-400): Received lead acetate at 400 mg/kg body weight per day via oral gavage.

All experimental procedures were conducted in accordance with internationally accepted principles for laboratory animal care and use, and with institutional ethical approval granted by the Nile University of Nigeria Animal Care and Use Committee, Abuja, Federal Capital Territory, Nigeria (Approval Code: NILE/IAEC/2026/034).

Body and Brain Weight Measurements

Body weights were recorded at baseline (Day 0) and at the end of the 21-day exposure period. Animals were sacrificed by cervical dislocation under anesthesia. Brains were carefully dissected, blotted dry, and weighed on an analytical balance. Relative brain weight (brain weight/body weight) was calculated for each animal to assess organ-to-body weight relationships.

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Hippocampal Histology

Following sacrifice, brains were fixed in 10% neutral buffered formalin for 72 hours, processed through ascending grades of ethanol, cleared in xylene, and embedded in paraffin wax. Coronal sections (5 µm) through the hippocampus were cut using a rotary microtome and mounted on glass slides. Sections were stained with haematoxylin and eosin (H&E) and examined under light microscopy at ×100 magnification. Histopathological features assessed included the organization of the pyramidal cell layer, neuronal density, nuclear morphology (presence of pyknotic nuclei), and degree of neuropil vacuolation.

Acetylcholine Quantification

Brain homogenates were prepared in ice-cold phosphate-buffered saline (PBS, pH 7.4) at a 1:10 (w/v) ratio and centrifuged at 3,000 rpm for 15 minutes at 4°C. The supernatant was collected and used specifically for acetylcholine quantification. Acetylcholine levels were determined using a commercial enzyme-linked immunosorbent assay (ELISA) kit according to the manufacturer's instructions. Absorbance was read at 450 nm using a microplate reader, and results were expressed as nanomoles per milligram of protein (nmol/mg protein).

Statistical Analysis

Experimental data are presented as Mean ± Standard Error of Mean (SEM). Statistical analyses were performed using SPSS version 20.0 (SPSS Inc., Chicago, IL, USA). Differences among groups were assessed by one-way analysis of variance (ANOVA), and post-hoc comparisons were conducted using Duncan's Multiple Range Test. A significance level of $p < 0.05$ was used throughout.

RESULTS

Body and Brain Weight Changes

Lead acetate administration produced a dose-dependent reduction in final body weight. The control and low-dose groups showed only marginal decreases from baseline, while the medium-dose group exhibited a modest reduction. The high-dose group (400 mg/kg) recorded the most pronounced body weight loss, with a reduction of 20.0 g from baseline, corresponding to approximately 6.43% of initial body weight (Chart 2). These findings suggest that lead acetate exerts a greater suppressant effect on overall body mass at higher doses.

Brain weights ranged from 1.580 g in the low-dose group to 1.790 g in the high-dose group, representing relatively small inter-group variation (Chart 1). This limited variation in absolute brain mass indicates that acute neuronal loss did not translate into gross organ-level weight changes within the 21-day exposure window. However, the relative brain weight (brain-to-body weight ratio) showed a mild downward trend, declining from 0.0076 in the control group to 0.0062 in the high-dose group, suggesting that body weight reduction outpaced any changes in brain mass with increasing lead exposure.

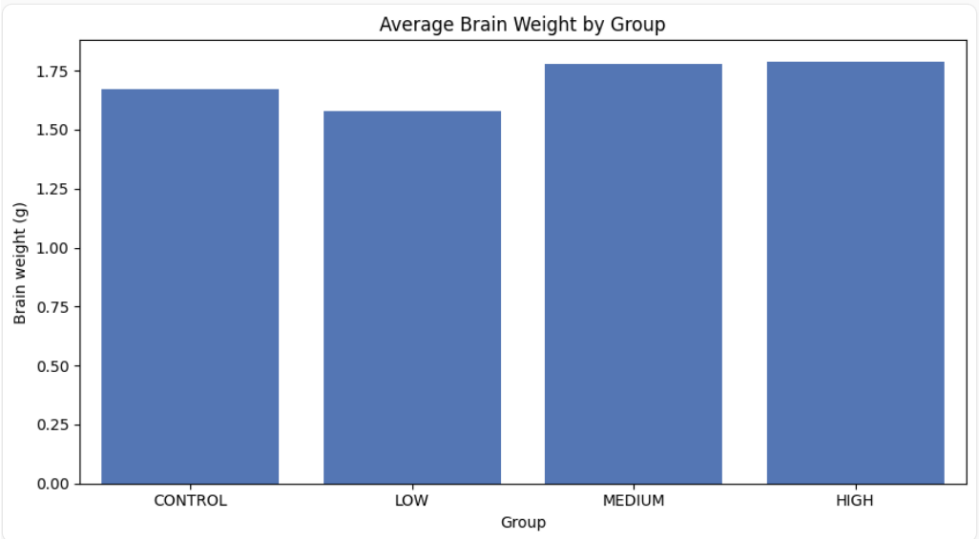

Chart 1: Showing limited influence on absolute brain mass

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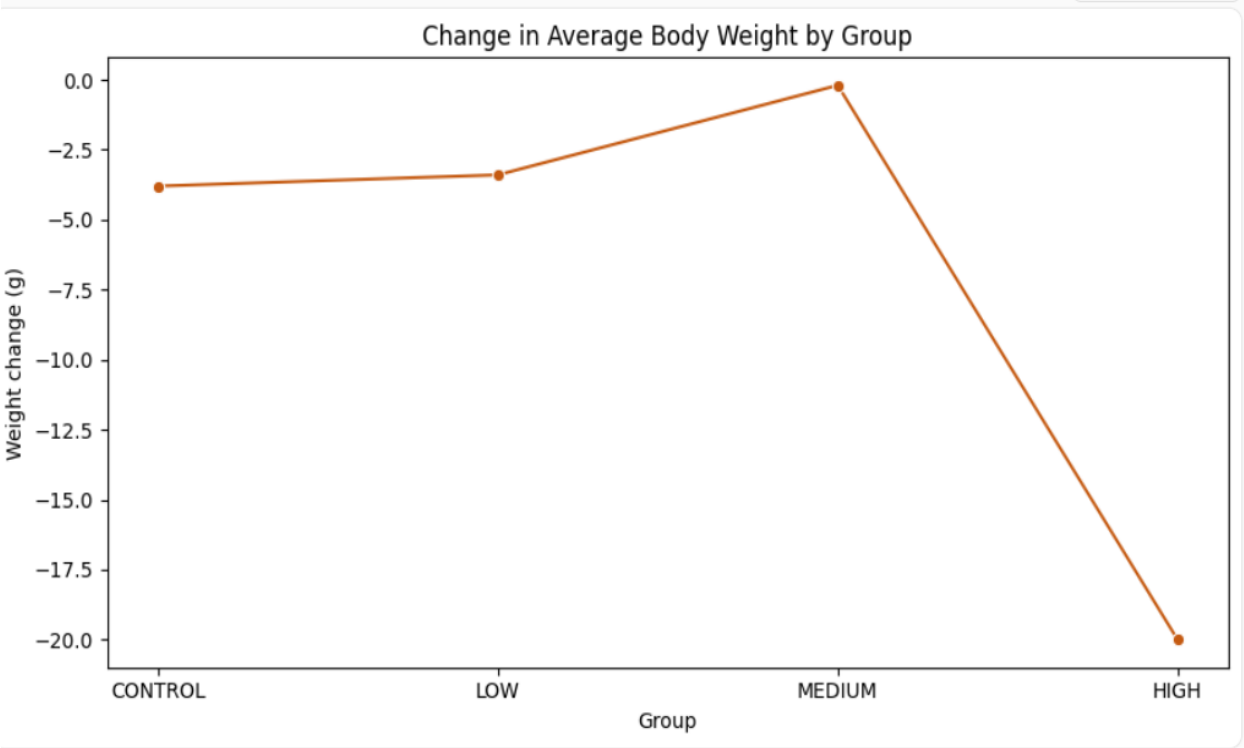

Chart 2: Showing decrease in body weight across groups, most noticeable in the high-dose group.

Hippocampal Histological Findings

Histological examination of H&E-stained hippocampal sections revealed progressive, dose-dependent structural alterations across treatment groups.

Fig. 1 Control Group: Hippocampal sections from control animals exhibited normal cytoarchitecture, with a well-defined pyramidal cell layer composed of densely packed, uniformly arranged neurons. Neuronal morphology was intact, with prominent, round nuclei and well-preserved cytoplasm. The neuropil was homogeneous and free from vacuolation, consistent with healthy hippocampal tissue.

Fig. 2 Low-Dose Group (100 mg/kg): Mild hippocampal alterations were observed, characterized by slight disorganization of the pyramidal cell layer and a modest reduction in neuronal density. Some neurons displayed pyknotic nuclei, indicative of early-stage degeneration. Mild neuropil vacuolation was noted, suggesting the onset of cytotoxic edema at this exposure level.

Fig. 3 Medium-Dose Group (200 mg/kg): More pronounced histopathological changes were evident. The pyramidal cell layer showed marked disorganization, with significant neuronal loss and a notable reduction in cellular density. Degenerating neurons with pyknotic nuclei were numerous, and neuropil vacuolation was increased relative to the low-dose group, reflecting moderate hippocampal damage consistent with escalating oxidative and excitotoxic injury.

Fig. 4 High-Dose Group (400 mg/kg): Severe hippocampal degeneration was observed. The pyramidal cell layer architecture was profoundly disrupted, with extensive neuronal loss and markedly reduced cell density. Widespread presence of shrunken, intensely pyknotic neurons was noted, along with severe neuropil vacuolation with large clear spaces suggesting extensive tissue rarefaction. In several fields, near-total loss of normal hippocampal organization was observed, reflecting pronounced neurotoxic damage consistent with advanced neurodegeneration.

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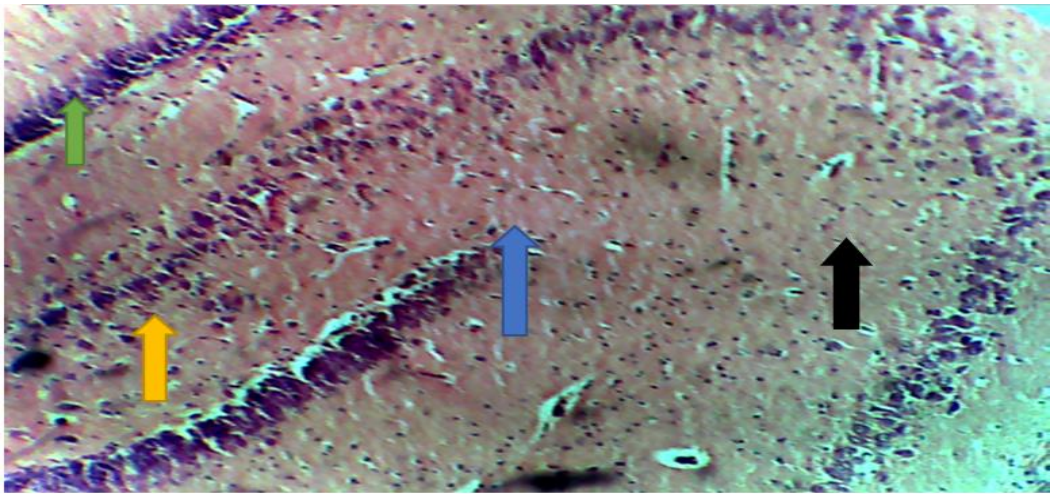

Fig. 1 Control group: Normal hippocampal architecture with a well-defined pyramidal cell layer (Yellow arrow), densely packed and uniformly arranged neurons (Green arrow), intact neuronal morphology with prominent nuclei (Black arrow), and preserved neuropil without vacuolation (Blue arrow).

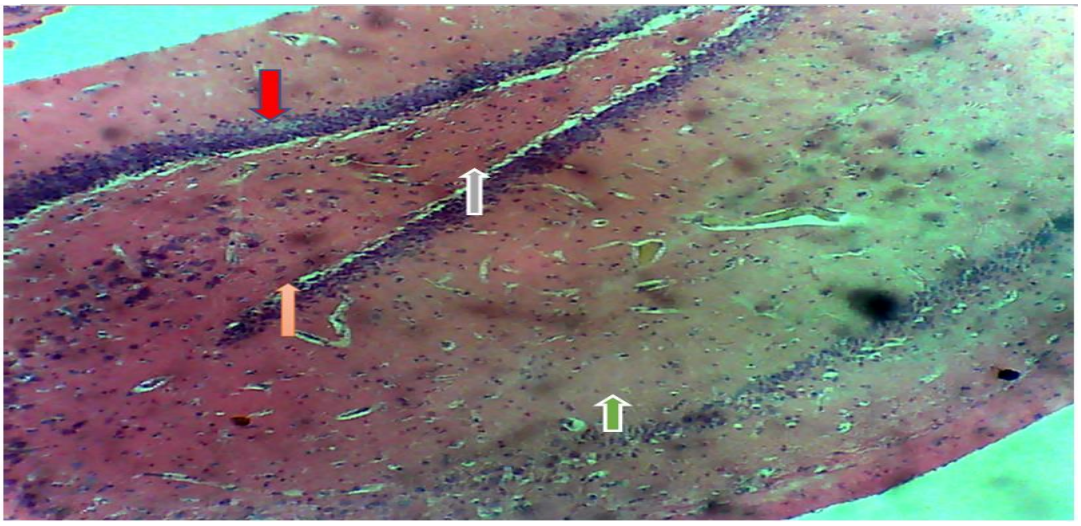

Fig. 2 Low-dose lead acetate group 100 mg/kg, x100: Mild hippocampal alterations characterized by slightly disorganized pyramidal cell layer (Red arrow), reduced neuronal density (Ash arrow), neurons with pyknotic nuclei indicating early degeneration (Pink arrow), and mild neuropil vacuolation (Green arrow).

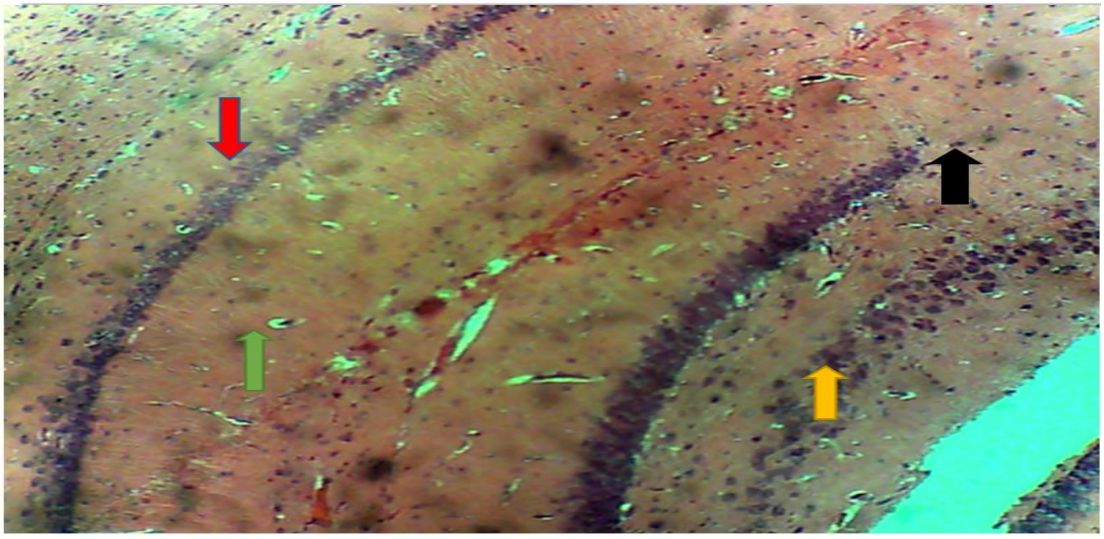

Fig. 3 Medium-dose lead acetate group 200 mg/kg, x100: Moderate hippocampal damage characterized by markedly disorganized pyramidal cell layer (Red arrow), significant neuronal loss and reduced density (Black

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arrow), numerous degenerating neurons with pyknotic nuclei (Yellow arrow), and increased neuropil vacuolation (Green arrow).

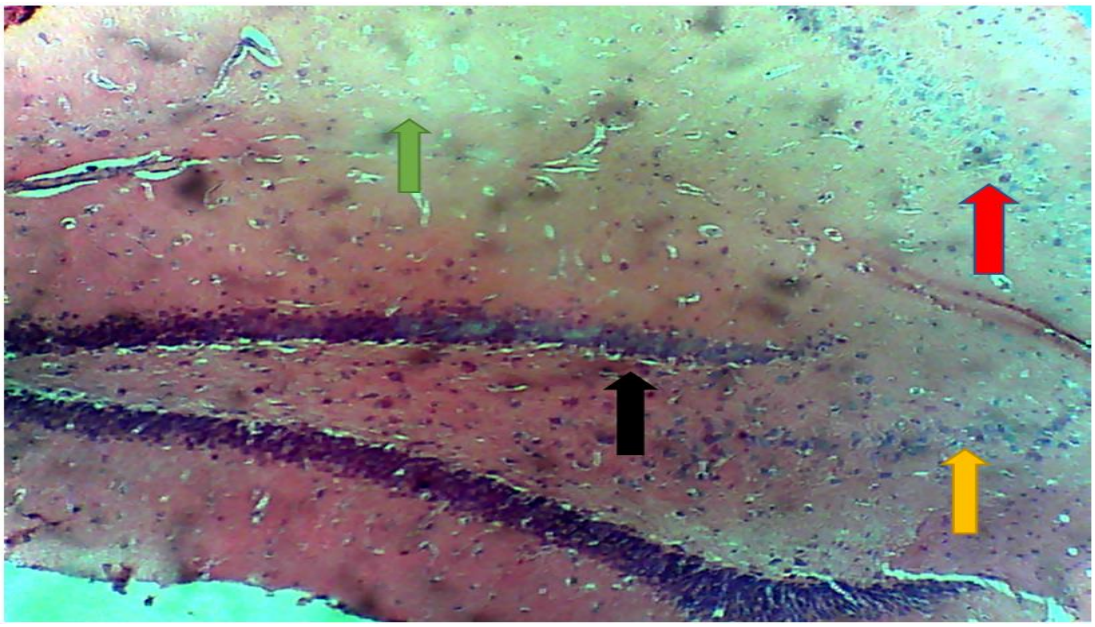

Fig. 4 High-dose lead acetate group 400 mg/kg, H&E x100: Severe hippocampal degeneration characterized by profound disruption of the pyramidal cell layer architecture (Red arrow), extensive neuronal loss with marked reduction in cell density (Black arrow), widespread presence of shrunken and intensely pyknotic neurons indicating advanced degeneration (Yellow arrow), and severe neuropil vacuolation with large clear spaces suggesting extensive tissue rarefaction (Green arrow).

Acetylcholine Levels

Brain acetylcholine levels increased significantly across treatment groups in a dose-dependent manner ($p < 0.05$; Chart 3). The control group displayed the lowest acetylcholine concentrations, while successive elevation was observed in the Lead-100, Lead-200, and Lead-400 groups. The most substantial increase was recorded in the high-dose group, suggesting that lead acetate progressively impairs cholinergic neurotransmission by disrupting acetylcholine degradation, likely through inhibition of acetylcholinesterase (AChE) activity.

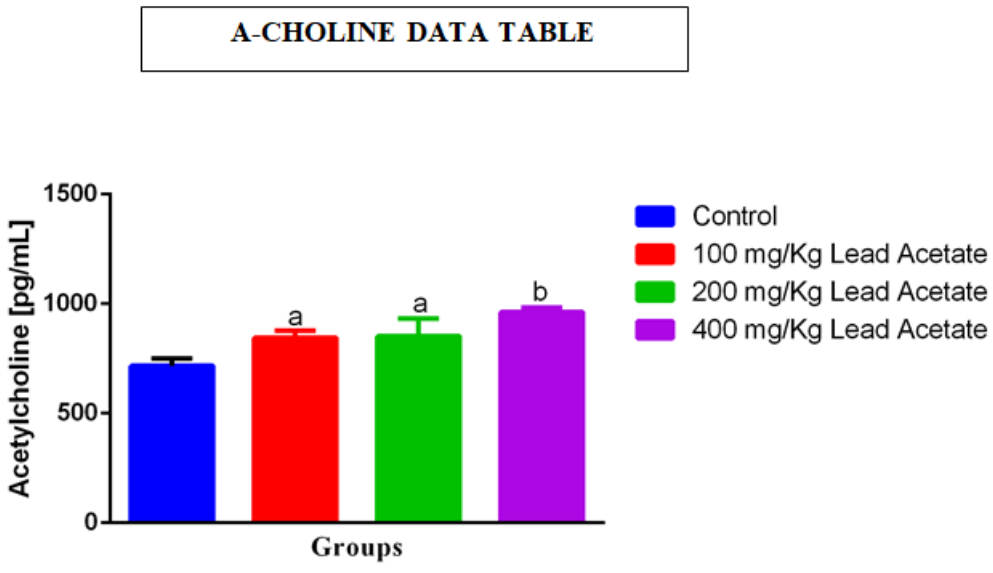

Chart 3: Chart showing significant increase in acetylcholine across groups. Bars are represented as Mean ± Standard Error of Mean (SEM). $P < 0.05$.

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DISCUSSION

The present study demonstrates that subacute oral administration of lead acetate produces dose-dependent neurotoxic effects in adult male Wistar rats, manifesting as progressive hippocampal degeneration, altered body weight, and significant dysregulation of cholinergic neurotransmission. These findings are consistent with and extend the existing literature on lead-induced CNS toxicity [4, 8, 18, 30].

The dose-dependent reduction in body weight observed in this study aligns with prior reports documenting the systemic toxicity of lead, which impairs multiple physiological processes including nutrient absorption, energy metabolism, and hormonal regulation [14, 16]. The more substantial weight loss at the 400 mg/kg dose suggests that high-level lead exposure triggers widespread metabolic disruption that compromises overall growth and body composition [28]. Notably, brain weight remained relatively stable across groups, a finding consistent with the well-recognized capacity of the brain to maintain gross structural integrity even as microstructural and cellular damage accumulates. The declining relative brain weight ratio, however, underscores the systemic nature of lead toxicity and reflects the disproportionate impact of lead on somatic growth [8].

The hippocampal histopathological findings represent perhaps the most striking outcome of this study. The progressive disorganization of the pyramidal cell layer, neuronal loss, widespread pyknosis, and neuropil vacuolation observed with increasing lead doses are consistent with the established mechanisms of lead neurotoxicity [17, 18, 26]. Lead-induced oxidative stress is central to these histological changes. By generating reactive oxygen species and depleting glutathione and superoxide dismutase activity, lead promotes lipid peroxidation of neuronal membranes, protein oxidation, and mitochondrial dysfunction, culminating in apoptosis and necrosis of hippocampal neurons [7, 10]. The pyknotic nuclei observed in treatment groups are a morphological hallmark of neuronal apoptosis and are consistent with activation of caspase-mediated cell death pathways reported in experimental models of lead neurotoxicity [12, 18].

Neuropil vacuolation, which was most severe in the 400 mg/kg group, reflects cytotoxic edema arising from disruption of ionic homeostasis and failure of the Na^+/K^+-ATPase pump, both of which are known targets of lead [6]. In addition, lead-induced disruption of the blood-brain barrier may amplify these effects by permitting entry of inflammatory mediators and exacerbating neuroinflammation [3, 11]. The activation of reactive astrocytes and microglia in response to lead-induced neural damage further compounds hippocampal injury [15, 26].

The dose-dependent elevation of acetylcholine levels observed in lead-treated rats is a particularly noteworthy finding. While seemingly paradoxical, rising ACh concentrations in the context of neurotoxicity may reflect inhibition of acetylcholinesterase (AChE), the enzyme responsible for ACh degradation at the synapse [9, 21]. Lead has been shown to inhibit AChE activity through direct binding to its catalytic site and through oxidative modification of the enzyme, leading to ACh accumulation [25, 9]. Paradoxically, elevated ACh, rather than improving cholinergic signaling, may trigger receptor desensitization, excitotoxicity, and further neuronal injury—a phenomenon analogous to organophosphate poisoning [25]. This interpretation is consistent with the concurrent finding of structural hippocampal damage and reinforces the view that cholinergic dysregulation is both a mediator and a marker of lead-induced neurotoxicity.

The hippocampal cholinergic system is critically involved in memory encoding and retrieval [9, 25]. Disruption of this system by lead, as evidenced in this study, has significant implications for cognitive function and may help explain the well-documented association between lead exposure and deficits in learning, memory, and executive function in both animal models and human populations [4, 22, 19]. The findings of this study are particularly relevant in the context of the growing recognition of heavy metal exposure as a modifiable risk factor for neurodegenerative diseases such as Alzheimer's disease [20, 23].

Some limitations of this study warrant acknowledgment. The 21-day exposure period, while sufficient to demonstrate subacute neurotoxicity, may not fully capture the trajectory of neurodegeneration or the potential for recovery. The exclusive use of male rats limits the generalizability of findings to female animals, which may respond differently to lead due to hormonal influences on metal metabolism and CNS vulnerability. In addition, hematological screening and broader biochemical enzyme analyses were not included in the present study; therefore, the systemic toxicological profile of lead exposure could not be fully characterized. Future studies

should incorporate female subjects, longer exposure durations, and assessments of hematological indices, biochemical enzyme markers, specific oxidative stress biomarkers, neuroinflammatory markers, and functional cognitive outcomes to build a more comprehensive picture of lead neurotoxicity.

CONCLUSION

This study provides experimental evidence that subacute oral exposure to lead acetate induces progressive, dose-dependent neurotoxic effects in the hippocampus of adult male Wistar rats. Lead acetate administration resulted in dose-related reductions in body weight, severe hippocampal histopathological changes including neuronal loss, pyknosis, and neuropil vacuolation, and significant elevation of brain acetylcholine levels suggestive of AChE inhibition. These findings collectively highlight the hippocampal pyramidal cell layer and the cholinergic neurotransmitter system as sensitive targets of lead neurotoxicity. The dose-response relationship established in this study reinforces the need for stringent regulation of human lead exposure and supports the continued investigation of therapeutic strategies aimed at mitigating lead-induced CNS damage.

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Ethical Considerations

All animal procedures were conducted in accordance with institutional and international guidelines for the care and use of laboratory animals, following review and approval by the Nile University of Nigeria Animal Care and Use Committee/Ethics Committee, Abuja, Federal Capital Territory, Nigeria (NILE/IAEC/2026/034). The animal trials were conducted at the Animal House Facility of the Department of Anatomy, College of Health Sciences, Nile University of Nigeria, Abuja, Federal Capital Territory, Nigeria.

Conflict of Interest: The authors declare no conflict of interest.

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