SCIENTIFIC CULTURE
www.sci-cult.com

SCIENTIFIC CULTURE, Vol. 12, No. 4, (2026), pp. 6787-6794
Open Access. Online & Print

DOI: 10.5281/zenodo.12426698

MITIGATION OF LEAD-INDUCED NEUROTOXICITY BY EUGENOL: BIOCHEMICAL AND HISTOPATHOLOGICAL EVALUATION IN WISTAR RATS

Sushmita Tripathi[1], Durgesh Kumar[1], Anveshika Manoj[1], Shivani Pandey[1], Abbas Ali Mahdi1, R.K. Garg[2], Mohammad Kaleem Ahmad[1*]

[1]*Department of Biochemistry, King George's Medical University, Lucknow*
[2]*Department of Neurology, Era Medical University, Lucknow*

Received: 13/12/2025
Accepted: 18/03/2026

Corresponding Author: Mohammad Kaleem Ahmad (email)

ABSTRACT

Lead is a pervasive environmental toxicant that accumulates in neural tissue and disturbs redox equilibrium, resulting in progressive neuronal damage. Oxidative imbalance strategies for treatment or prevention of neurotoxic effects associated with exposure to lead were explored using eugenol in the chronic lead exposure Wistar rat model. Twenty-five adult male Wistar rats were divided into five groups (control, lead acetate 30 mg/kg, lead + Emblica officinalis (amla) extract (500 mg/kg), lead + eugenol 6 µg/kg, and lead + vitamin C 2 g/kg). Treatments were administered orally for 90 consecutive days. Blood and brain tissues were analysed for antioxidant enzyme activities and lipid peroxidation markers. Blood and brain lead concentrations were quantified using inductively coupled plasma–mass spectrometry. Histological assessment of the cerebral cortex and cerebellum was conducted following hematoxylin and eosin staining. Lead administration significantly increased tissue lead levels and a significant suppression of catalase, superoxide dismutase, glutathione peroxidase, and glutathione reductase activities, accompanied by increased malondialdehyde concentrations. Microscopic findings included neuronal shrinkage, nuclear condensation, cytoplasmic vacuolation, Purkinje cell depletion, and interstitial oedema. Eugenol markedly reduced lead burden, improved antioxidant defence status, and limited lipid peroxidation. Preservation of cortical and cerebellar architecture was evident in the eugenol-treated group, while vitamin C and amla extract showed comparatively modest protective effects. Eugenol conferred substantial neuroprotection by mitigating oxidative injury, lowering tissue lead accumulation, and maintaining neuronal structural integrity in chronically exposed rats, supporting its potential therapeutic relevance in heavy metal–associated brain toxicity.

KEYWORDS: Lead neurotoxicity, Eugenol, Oxidative stress, Antioxidant enzymes, Lipid peroxidation, Histopathology, Wistar rats, Heavy metal toxicity

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1 INTRODUCTION

Lead is a ubiquitous environmental toxin and has no known physiological role. It has well-documented negative effects even at low levels of exposure [1, 2]. Exposure to contaminated food, water, and industrial or environmental inhalants causes adverse health effects in humans and animals. These substances subsequently accumulate in bones and soft tissues [3–5]. Lead exposure is still a global public health problem, even though governments have tried to fix it [4, 8].

Lead selectively accumulates within neuron-supportive cells called astrocytes (and other neural cell types) within the central nervous system where it inhibits regulation between nerve and supportive cells thereby resulting in neuronal damage [6, 25]. Alterations in synaptic transmission, complications related to the release of neurotransmitters and problems associated with the operation of calcium-dependent signaling pathways all result in decreased function of neurons [9, 10]. Children are particularly susceptible to lead exposure, exhibiting cognitive and behavioral deficits even at minimal blood lead levels [5, 26]. Developmental exposure has been linked to enduring neurobehavioral deficits that persist into adulthood [27].

Lead exposure causes damage to the blood-brain barrier, cerebral edema, loss of neurons and reactive gliosis [11]. Oxidative stress has been identified as a primary mechanism related to lead neurotoxicity [11–14]. Lead increases the production of reactive oxygen species and lowers the body's natural defenses against them. This causes problems with mitochondria and membranes [12–14]. Oxidative injury that lasts for a long time can lead to lipid peroxidation, protein oxidation, DNA damage, and neuronal death by apoptosis [14–16].

Redox imbalance is an acknowledged factor in the pathogenesis of significant neurodegenerative disorders, such as Alzheimer's disease and Parkinson's disease [17–19, 28]. Exposure to metals in the environment has been suggested as a possible factor that changes the risk of neurodegeneration [26].

Chelation therapy using calcium disodium ethylenediaminetetraacetic acid (EDTA) or dimercaptosuccinic acid (DMSA) help remove heavy metals but do not completely reverse oxidative damage that has occurred in the neurons [20,21]. Therefore, there is growing interest in using antioxidant-based therapies in conjunction with other therapies [13,14,29]. Eugenol, a phenolic compound prevalent in clove oil and various aromatic flora, demonstrates significant antioxidant and anti-inflammatory effects [15–19]. Eugenol has been shown to scavenge free radicals; modulate redox-sensitive signal transduction (signaling pathways) and enhance the activity of the body's native antioxidant enzymes [15,16]. Further research corroborates its neuroprotective efficacy in models of oxidative stress and neuroinflammation [24].

As oxidative stress plays a significant role in lead-induced neuronal injury, the purpose of this study was to evaluate whether administration of eugenol would reduce the biochemical and histopathological changes occurring in the brain of Wistar rats that were chronically exposed to lead.

2 MATERIALS AND METHODS

Experimental Animals and Ethical Approval

Adult male Wistar rats (180–200 g) were procured from the Central Drug Research Institute, Lucknow. Animals were housed under standard laboratory conditions (12 h light/dark cycle, controlled temperature and humidity) with free access to food and water. All experimental procedures were approved by the Institutional Animal Ethics Committee, King George's Medical University (Approval No. 136/IAEC/2020), and were conducted in accordance with CPCSEA guidelines.

Experimental Design

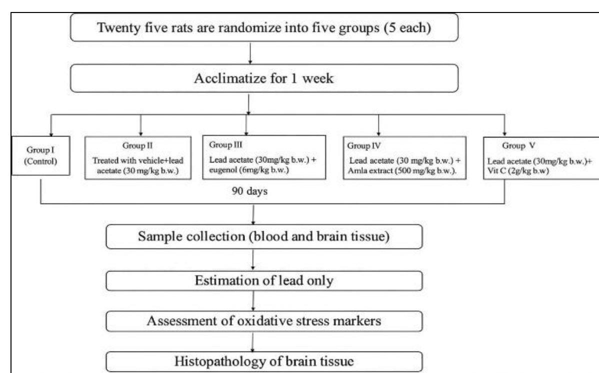

Fig 1 Study Design

Rats were randomly divided into five groups (n = 5 per group):

Group I: Control (distilled water)

Group II: Lead acetate (30 mg/kg/day)

Group III: Lead acetate (30 mg/kg/day) + eugenol (6 µg/kg/day)

Group IV: Lead acetate (30 mg/kg/day) + *Emblica officinalis* (amla) extract (500 mg/kg/day)

Group V: Lead acetate (30 mg/kg/day) + vitamin C (2 g/kg/day)

All treatments were administered orally for 90 consecutive days.

Sample Collection
At the end of the treatment period, animals were euthanized under ketamine–xylazine anesthesia. Blood samples were collected by cardiac puncture. Brains were removed, rinsed in ice-cold phosphate buffered saline (PBS), and processed for biochemical and histological analyses.

Lead Estimation
Lead concentrations in the biological samples were determined using inductively coupled plasma mass spectrometry (ICP-MS) by following standardized heavy metal analytical procedures [20].

Biochemical Assessment of Oxidative Stress
Catalase activity was assessed spectrophotometrically based on hydrogen peroxide decomposition [12]. Superoxide dismutase activity was determined by inhibition of superoxide radical–mediated reactions [13]. Glutathione Peroxidase and Glutathione Reductase were determined by using standardized enzymatic methods [14]. The extent of lipid peroxidation was determined by measuring the concentration of malondialdehyde as a function of thiobarbituric acid reactive substances (TBARS) [15,16].

Statistical Analysis
All experimental data were compiled and analyzed after completion of the study. Quantitative results are presented as mean ± standard error of the mean (SEM). Statistical comparisons among multiple experimental groups were performed using one-way analysis of variance (ANOVA) to evaluate overall group differences. When a statistically significant F-value was obtained, Tukey's post hoc multiple comparison test was applied to determine pairwise intergroup differences. The F-statistic and corresponding p-values were calculated and reported. A p-value of less than 0.05 was considered statistically significant. All statistical analyses were performed using GraphPad Prism version 5.1 (GraphPad Software Inc., San Diego, CA, USA).

3 RESULTS

Blood analysis revealed marked dysregulation of heavy metals in lead-exposed animals, and Tukey post-hoc analyses of the blood data, confirmed significant differences among groups. Lead levels increased dramatically in the lead group (22.48 ± 0.26 µg/dL) compared with controls (4.03 ± 0.64 µg/dL, p = 0.0006). Tukey testing further showed that Eugenol treatment significantly lowered lead accumulation (10.47 ± 0.60 µg/dL, L vs Eug p = 0.0295), while Amla (19.00 ± 0.13 µg/dL) and Vitamin C (13.16 ± 0.09 µg/dL) did not reach statistical significance. ANOVA revealed highly significant group differences, and Tukey analysis reinforced that Eugenol consistently provided stronger protection than Vitamin C or Amla across most parameters. [Table-1]

Table 1. Lead Concentration in Blood of Wistar Rats (n = 5 per group)

Group	Lead (µg/dL) (Mean ± SD)	F-statistic	p-value
Control	4.03 ± 0.64	F(4,25) = 7.65	**0.0007**
Lead	22.48 ± 0.26		
Lead + Eugenol	10.47 ± 0.60		
Lead + Amla	19.00 ± 0.13		
Lead + Vitamin C	13.16 ± 0.09		

Values are expressed as mean ± standard deviation (SD). Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc multiple comparison test.

Tissue analysis confirmed that lead heavily accumulates in both cerebrum and cerebellum, with Tukey post-hoc tests demonstrating highly significant group differences. Cerebral lead rose from 2.21 ± 0.12 µg/g in controls to 138.8 ± 3.24 µg/g in lead rats (p < 0.0001). Tukey analysis confirmed that Eugenol reduced cerebral accumulation significantly (28.12 ± 0.67 µg/g, L vs Eug p < 0.0001), with Amla (75.73 ± 3.87 µg/g) and Vitamin C (70.02 ± 6.25 µg/g) also showing significant but lesser protection (both p < 0.0001 vs lead). In the cerebellum, lead rose from 1.83 ± 0.04 µg/g to 75.91 ± 1.55 µg/g (p < 0.0001). Tukey post-hoc again confirmed a highly significant protective effect of Eugenol (22.08 ± 2.88 µg/g, p < 0.0001), with Amla (52.90 ± 1.39 µg/g, p < 0.0001) and Vitamin C (46.17 ± 5.05 µg/g, p < 0.0001) partially effective. ANOVA values (F = 1042 and 536.9, both p < 0.0001) confirmed robust inter-group differences, with Tukey confirming Eugenol as the superior neuroprotectant. **[Table-2]**

Table 2. Lead Accumulation in Brain Tissues (n = 5 per group)

Group	Cerebrum Lead (µg/g) (Mean ± SD)	F-statistic	p-value	Cerebellum Lead (µg/g) (Mean ± SD)	F-statistic	p-value
Control	2.21 ± 0.12	F(4,25) = 1042.0	**<0.0001**	1.83 ± 0.04	F(4,25) = 536.9	**<0.0001**
Lead	138.8 ± 3.24			75.91 ± 1.55		
Lead + Eugenol	28.12 ± 0.67			22.08 ± 2.88		
Lead + Amla	75.73 ± 3.87			52.90 ± 1.39		
Lead + Vitamin C	70.02 ± 6.25			46.17 ± 5.05		

Values are expressed as mean ± standard deviation (SD). Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc multiple comparison test.

Lead exposure induced systemic oxidative stress, confirmed by ANOVA and Tukey tests. The lead exposed group exhibited a significant reduction in catalase activity (212.4 ± 7.1 vs 240.9 ± 5.6 U/mg, $p < 0.0001$). **(Fig 2 (A))** Tukey showed significant recovery with Eugenol (238.8 ± 1.3 U/mg, $p = 0.0001$) but only borderline improvement with Vitamin C ($p = 0.0667$). SOD was severely reduced by lead (767.6 ± 228.3 vs 3166 ± 454.6 U/mg, $p < 0.0001$), with Tukey confirming highly significant restoration by Eugenol (2912 ± 618.5 U/mg, $p = 0.0002$) and Vitamin C (2812 ± 1154 U/mg, $p = 0.0004$), but not by Amla.**(Fig 2 (B))** GPx activity fell from 605.5 ± 159.6 to 167.1 ± 14.6 U/mg ($p = 0.0002$), with Tukey showing that Eugenol produced strong recovery (490.4 ± 171.1 U/mg, $p = 0.0052$), while Amla and Vitamin C were nonsignificant.**(Fig 2 (C))** GR activity dropped significantly with lead (121.0 ± 8.8 vs 268.8 ± 66.7 U/mg, $p = 0.0017$), but Tukey analysis revealed no treatment was able to restore it significantly. **(Fig 2 (D))** MDA increased from 1.92 ± 0.26 to 2.67 ± 0.56 nmol/mg ($p = 0.0107$), and Tukey analysis showed significant normalization with Eugenol (1.96 ± 0.08, $p = 0.016$) and Vitamin C (2.02 ± 0.15, $p = 0.0316$), but not with Amla. **(Fig 2E)** Thus, Tukey comparisons emphasized that Eugenol was most effective in reversing lead-induced oxidative stress markers in blood.

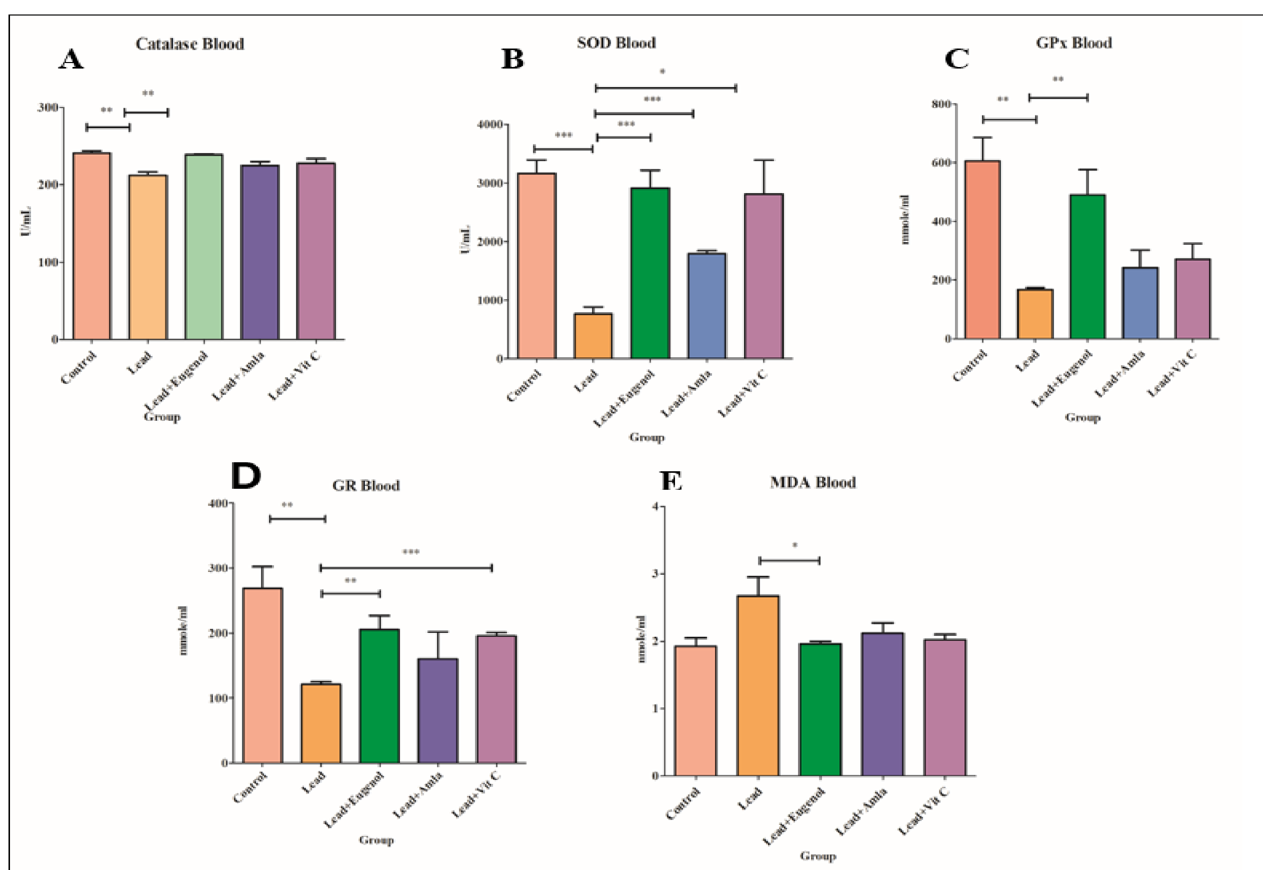

Figure 2. Blood oxidative stress biomarkers in control and treated groups.

Catalase activity (A), superoxide dismutase (SOD) activity (B), glutathione peroxidase (GPx) activity (C), glutathione reductase (GR) activity (D), and malondialdehyde (MDA) levels (E) measured in blood samples of control and experimental groups. Data are expressed as mean ± standard deviation (SD) (n = 5 per group). Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc multiple comparison test. *p=0.05, **p=0.01, and ***p=0.001 compared with the control group.

In brain tissues, oxidative stress was profound, with Tukey post-hoc tests confirming strong group differences. Catalase activity dropped sharply in lead rats (144.0 ± 9.7 vs 246.7 ± 8.5 U/mg, $p < 0.0001$), but Tukey showed complete restoration by Eugenol

(245.1 ± 1.1 U/mg, $p < 0.0001$).**(Fig 3 (A))** SOD activity fell drastically with lead (742.6 ± 332.4 vs 3307 ± 1913 U/mg, $p = 0.0034$), but Tukey demonstrated significant recovery by Eugenol (2679 ± 497.8 U/mg, $p = 0.0329$). **(Fig 3 (B))** GPx was markedly reduced (72.4 ± 10.6 vs 517.2 ± 62.4 U/mg, $p < 0.0001$), with Tukey showing highly significant recovery by Eugenol (350.2 ± 64.8 U/mg, $p < 0.0001$), while both Amla (203.2 ± 18.5 U/mg, $p = 0.0105$) and Vitamin C (232.8 ± 82.9 U/mg, $p = 0.0016$) also conferred partial benefits.**(Fig 3(C))** GR activity declined under lead (157.6 ± 24.6 vs 251.2 ± 11.9 U/mg, $p = 0.0007$), and Tukey confirmed significant improvement by Eugenol (235.7 ± 18.3 U/mg, $p = 0.0043$) and Vitamin C (220.9 ± 55.3 U/mg, $p = 0.0243$).**(Fig 3 (D))** Lipid peroxidation (MDA) was elevated in lead rats (2.45 ± 0.09 vs 1.88 ± 0.10 nmol/mg, $p = 0.0004$), with Tukey confirming strong normalization by Eugenol (1.95 ± 0.04, $p = 0.0017$) and Vitamin C (2.03 ± 0.32, $p = 0.0076$), but not by Amla.**(Fig 3 (E))** ANOVA and Tukey consistently established Eugenol as providing near-complete neurochemical normalization.

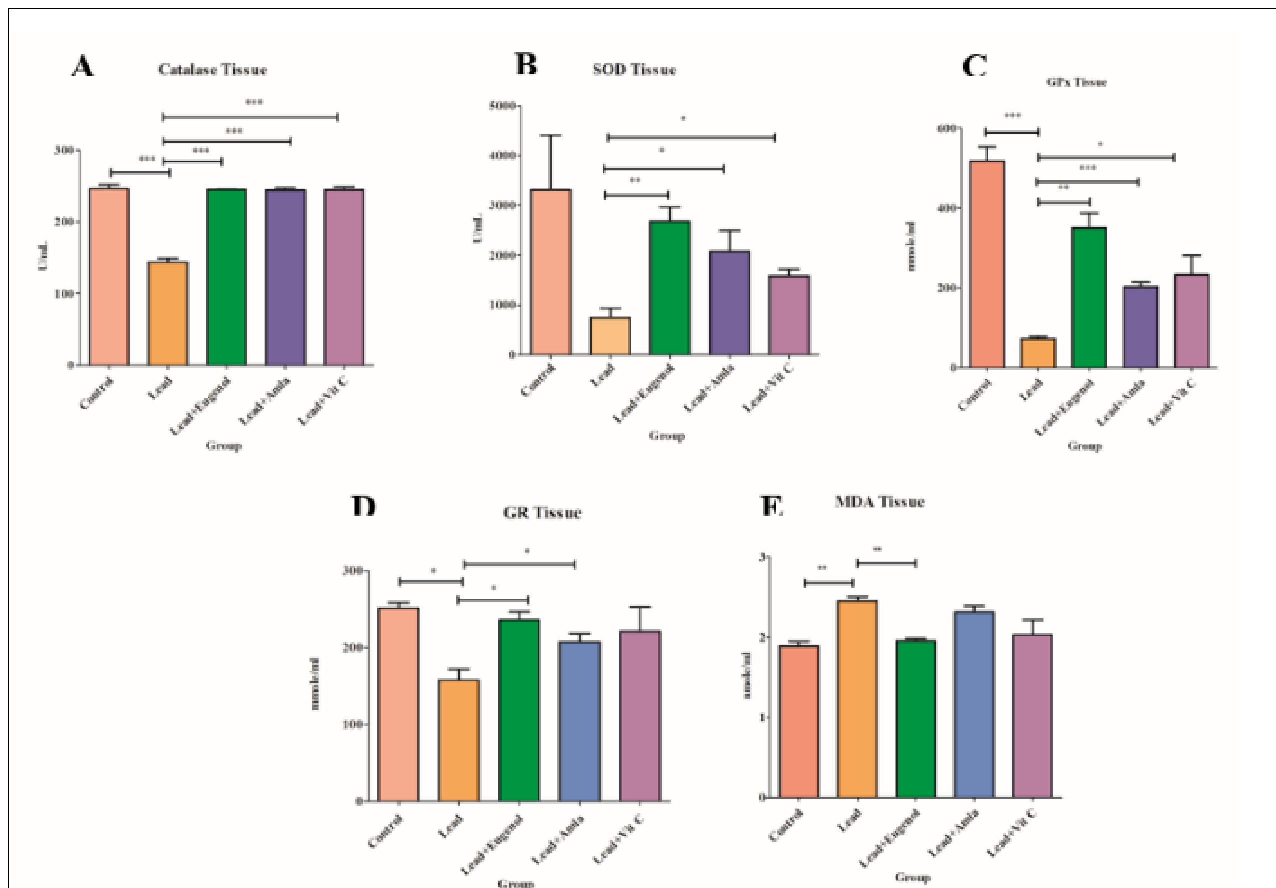

Figure 3. Brain oxidative stress biomarkers in control and treated groups.

Catalase activity (A), superoxide dismutase (SOD) activity (B), glutathione peroxidase (GPx)
activity (C), glutathione reductase (GR) activity (D), and malondialdehyde (MDA) levels (E)
measured in brain tissue of control and experimental groups. Data are expressed as mean ±
standard deviation (SD) (n = 5 per group). Statistical analysis was performed using one-way
analysis of variance (ANOVA) followed by Tukey's post hoc multiple comparison test. *p <
0.05, **p < 0.01, and ***p < 0.001 compared with the control group.

Histopathology corroborated biochemical findings, and Tukey significance patterns matched the protective trends. Control brains displayed intact neuronal and cerebellar structures. **(Fig 4)** Cerebral lesions (degenerative changes, nuclear pyknosis, vacuolation), together with Purkinje cell and cerebellar edema, were noted after lead exposure (Figure 4). Tukey's comparisons of the animals' histology reflected that Eugenol provided a greater degree of protection and had the closest to normal architecture with the least amount of vacuolation, while Amla showed moderate-to-slightly significant protective capability, and Vitamin C provided minimal-to-partially significant protective capability.

Thus, Tukey supported the conclusion that Eugenol was the best compound for preventing lead induced damage to the nervous system of lead exposed animal tissue (Figure 4).

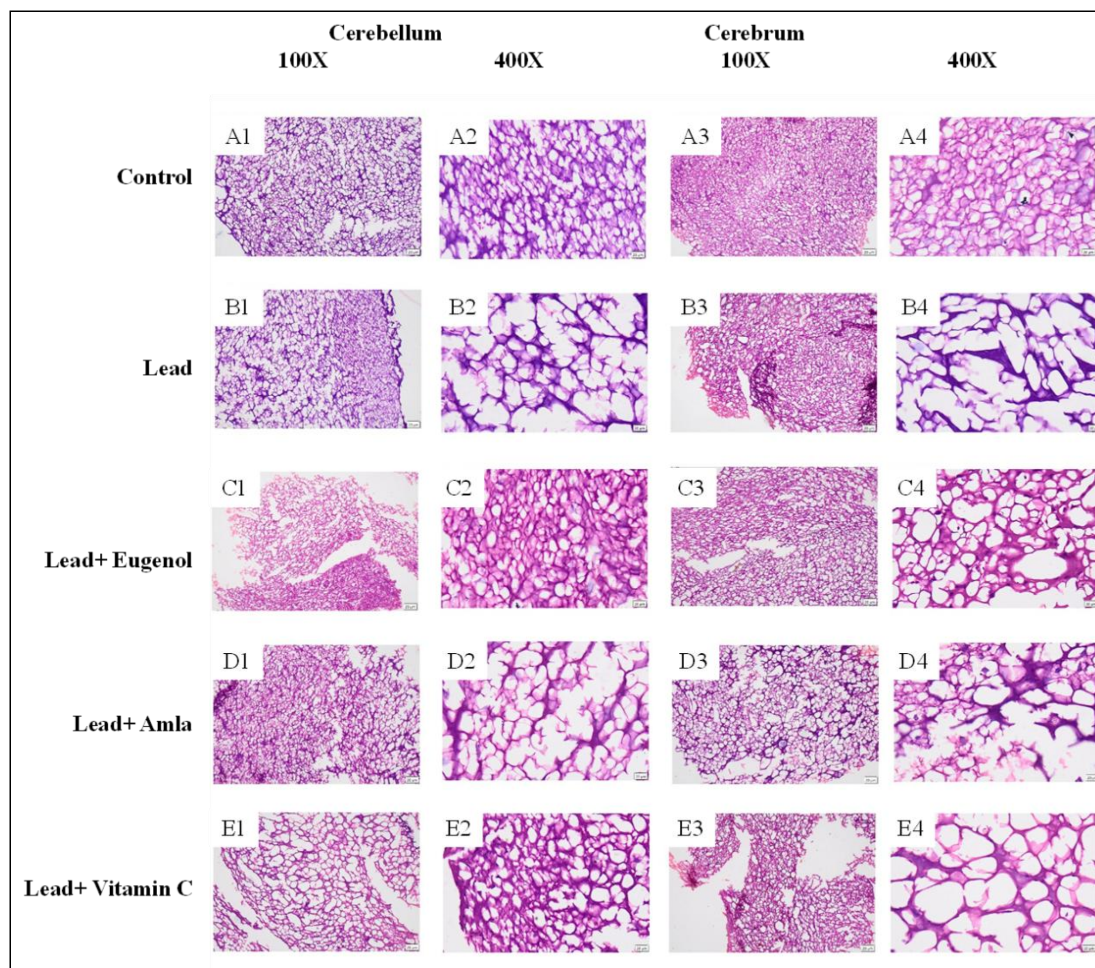

Figure 4: Hematoxylin and eosin (H&E) staining of cerebellum and cerebrum sections of rat brain (100× and 400×). Representative photomicrographs of cerebellum and cerebrum across groups. Control:(A1–A2) Normal cerebellar layers with preserved Purkinje cells; (A3–A4) intact cerebral architecture. Lead: (B1–B2) Cerebellar disorganization with Purkinje cell loss and vacuolation; (B3–B4) cerebral degeneration with nuclear pyknosis and structural distortion. Lead + Eugenol: (C1–C2) Improved cerebellar morphology with preserved Purkinje cells; (C3–C4) reduced cerebral damage and minimal vacuolation. Lead + Amla: (D1–D2) Moderate cerebellar recovery; (D3–D4) partial cerebral protection. Lead + Vitamin C: (E1–E2) Mild cerebellar improvement; (E3–E4) limited attenuation of cerebral alterations

4 DISCUSSION

The present study confirms that chronic lead exposure induces significant oxidative stress–mediated neurotoxicity, evidenced by increased cerebral lead accumulation, suppression of endogenous antioxidant enzymes, and enhanced lipid peroxidation. The data presented in this article are consistent with previous research findings indicating a neurotoxic affinity for lead, as well as a preferential accumulation of lead into glial cells [6, 25]. Reduced levels of catalase, superoxide dismutase, glutathione peroxidase and glutathione reductase activity combined with increased malondialdehyde concentrations highlight the role of oxidative damage as a primary mechanism for lead-related neuronal damage [12-16]. Other human and animal studies also demonstrate similar changes in biochemical markers due to developmental and adult exposure to lead [26,27].

Oxidative stress–driven neuronal damage is increasingly implicated in the pathogenesis of neurodegenerative disorders [17–19,28]. Environmental toxicants, including heavy metals, may therefore act as modifiers of long-term neurodegenerative risk [26]. Eugenol administration showed significant improvement compared to the

lead-treated group in terms of restoring antioxidant enzyme activity and decreasing blood and brain lipid peroxidation. These findings substantiate previous studies suggesting that eugenol increased antioxidant defense and decreased oxidative damage [15,16,24]. Additionally, the findings of this study provide further evidence that eugenol modulates redox-dependent signal transduction pathways and decreases neuroinflammation.

Chelation remains the primary method currently used for the treatment of lead exposure; however, it does not successfully overcome already developed oxidative injury [20, 21]. The findings of this investigation demonstrate the potential of adjunctive antioxidant therapy to provide additional neuroprotective and functional recovery benefits [13,14,29]. Evidence from histopathological preservation of eugenol treated animals further supports its neuroprotective potential, and ability to inhibit oxidative neuronal degeneration. The current data support the central role that oxidative decompensation has on lead-induced neurotoxicity and also suggest the possibility that phenolic antioxidants like eugenol may provide a therapeutic effect.

In this study, a controlled Wistar rat model was used to simulate experimental conditions. The primary objective of the study was the assessment of the lead burden, the markers for oxidative stress, and a histopathological examination. Future research trends that are designed to expand on molecular analytical techniques and treatment modalities for eugenol may elucidate the mechanisms and provide additional clinical data related to eugenol and lead-induced neurotoxicity

5 CONCLUSIONS

In Wistar rats, lead was found to cause significant neuronal damage and resulted in a high level of oxidative stress (increased free radical activity). The presence of excessive lipid peroxidation (damage to membranes) along with degeneration of neurons as seen on the microscope further validated that lead exhibited pronounced neurotoxicity. Of all treatments used in this study, the eugenol provided the most protective effect. Eugenol reduced lead accumulation in the brain, improved antioxidant capacity, as well as maintained the normal structure of the cortex and cerebellum. Vitamin C also provided some protection while amla provided little or no protection. Therefore, these findings suggest that eugenol has significant potential as an adjunctive approach for the treatment of neurotoxic effects associated with exposure to heavy metals

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