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Protective effect of ethanolic extract of galangal root against cadmium-induced neurotoxicity in a rat model

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Abstract

Objective

Cadmium is a toxic heavy metal with detrimental effects on various biological systems. Herbal extracts, due to their antioxidant properties, may help mitigate such toxicity. This study aimed to evaluate the protective effect of *Alpinia galanga* (galangal) root ethanolic extract against cadmium-induced neurotoxicity in a rat model.

Materials and Methods

Forty male rats were randomly assigned to four groups (n = 10 per group). Group 1 (control) received normal drinking water and diet for 30 days. Group 2 was administered 100 mg/kg of *A. galanga* hydroalcoholic extract orally. Group 3 received 0.5 ppm cadmium chloride ($CdCl_2$) in drinking water. Group 4 received both 0.5 ppm $CdCl_2$ in water and 100 mg/kg *A. galanga* extract orally for 30 days.

Results

Cadmium exposure significantly increased brain malondialdehyde (MDA) levels (1.6364 ± 0.01) compared to the control group (0.7247 ± 0.005, $p = 0.001$), while MDA levels in the *A. galanga*-

treated rats were comparable to controls. Glutathione peroxidase (GSH-Px) levels were significantly reduced in the cadmium group (10.5098 ± 1.5) and the cadmium + extract group (15.9569 ± 1.5) compared to controls ($p = 0.023$). Gene expression analysis showed a significant downregulation of catalase in the cadmium (0.74 ± 0.2) and cadmium + extract (0.83 ± 0.19) groups relative to the control (1.05 ± 0.25) and extract-only (0.94 ± 0.21) groups ($p < 0.05$). Glutathione S-transferase (GST) expression was also significantly reduced in the cadmium group (1.05 ± 0.05) compared to the control (1.94 ± 0.1), but co-administration with *A. galanga* restored GST levels (1.47 ± 0.09, $p < 0.05$). A similar trend was observed for GSH-Px expression, which decreased significantly in the cadmium group (0.97 ± 0.05) and improved with *A. galanga* treatment (1.34 ± 0.12) compared to the control (2.19 ± 0.15, $p < 0.05$).

Conclusions

These findings demonstrate that cadmium exerts neurotoxic effects through oxidative stress mechanisms, and that *Alpinia galanga* root extract possesses significant antioxidant properties capable of mitigating cadmium-induced neurotoxicity in rats.

Keywords: *Alpinia galanga*, antioxidants, cadmium, neurotoxicity, oxidative stress

Paper Type: Research Paper.

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Introduction

Cadmium (Cd) is a heavy metal and environmental contaminant that accumulates in biological tissues, including the brain. In experimental models, Cd exposure at doses over 700 times higher than control levels results in markedly elevated cadmium concentrations in brain tissue. Among the different cadmium compounds, cadmium salts represented as some of the most

hazardous environmental pollutants due to their persistence and non-degradability. Cadmium pollution through the food chain persists to carry a growing general public health concern (Genchi et al., 2020). Cadmium can reach the body mainly through the ingesting of polluted cereals, vegetables, and other food products, leading to continuous, low-level exposure (Satarug et al., 2010). Chronic cadmium exposure has been joined with lethal impacts in multiple organs, including the brain, immune system, hematopoietic tissue, and cardiovascular system (Hazrat et al., 2019). Cd-associated neurotoxicity is mostly upsetting and has been linked with the interruption of normal neurochemical pathways and the development of different neurological disorders. One of the principle pathway by which cadmium employs its neurotoxic effects is through the initiation of oxidative stress. It has been demonstrated that cadmium intensifies the generation of free radicals, enhancing lipid peroxidation (LPO) and disrupting cellular membranes (Wang & Du, 2013). Cadmium has also been increasingly joined with the generation of reactive oxygen species (ROS) and diminution of antioxidant defenses. Mitochondrial deficts resulting from cadmium exposure directs to weakend mitochondrial membrane potential and a substantial diminution in intracellular glutathione levels, further aggravating oxidative stress in brain cells (El-Tarras et al., 2016). Brain tissue is exceptionally liable to lipid peroxidation due to its high oxygen consumption, plenty of polyunsaturated fatty acids, moderately depleted antioxidant defenses, and perhaps the presence of transition metals such as aluminum and nickel in certain brain regions (Unsal et al., 2020).

Furthermore, cadmium alters the physiological pathways of endogenous antioxidant enzymes, hindering brain metabolism and participating to its neurotoxicity (Afifi & Embaby, 2016). By advancing free radical generation and interrupting the antioxidant defense system, cadmium impaiirs lipid structure and negatively impacts membrane-bound enzymes. Its ability to traverse the blood–brain barrier and build up in neural tissue is a serious factor triggering its neurotoxic potential (Branca et al., 2020).

Among the medicinal plants with known antioxidant properties, *Alpinia galanga* (commonly known as galangal) has enticed scientific interest. Native to Asia and cultivated in many developing countries including Indonesia, galangal has long been used in traditional medicine to treat different conditions, such as rheumatism, inflammation, diabetes, and neurological disorders (Thapa et al., 2023). The rhizome of *A. galanga* is heavy in biomolecules, such as, terpenoids, flavonoids, phenolic acids, saponins, and essential oils. Key active constituents include 1,8-cineole, kaempferol, and galangal acetate (Van et al., 2021). The antioxidant potential of *A. galanga* has been shown in different studies. A 50% ethanolic extract demonstrated superior antioxidant activity contrasted to water extracts and essential oils, as assessed by assays, such as, oxygen radical absorbance capacity (ORAC) and 2,2-diphenyl-1-picrylhydrazyl (DPPH) radical

scavenging (Rahman et al., 2024; Rajendiran et al., 2018). The highest ORAC and DPPH values were demonstrated in ethanolic extracts. Antioxidant impacts has also been establishd in extracts comprising 1-acetoxychavicol acetate and its derivatives (Juntachote & Berghofer, 2005). Additional studies on methanolic extracts have estimated total phenolic content, metal ion chelation, reducing power, and β-carotene bleaching capacity (Kojima-Yuasa & Matsui-Yuasa, 2020). Both aqueous and ethanolic extracts of *A. galanga* have shown powerful antioxidant efficacy (Hung et al., 2022; Ranjan et al., 2022). Although the phytochemical components, pharmacological efficacy, and safety profile of *A. galanga* have been considerably studied, its neuroprotective influences, predominantly in the profile of central nervous system deficits, such as, Parkinson's disease, depression, epilepsy, and cerebral ischemia continue underexplored. Therefore, the present study aims to investigate the neuroprotective potential of *Alpinia galanga* root ethanolic extract against cadmium-induced neurotoxicity in a rat model.

Material and methods

Preparation of *A. galanga* root extract: The roots of *Alpinia galanga* were procured from local markets in Mosul, Iraq. At the Central Research Laboratory (CRL) of the Northern Technical University (NTU), Mosul, the roots were cleaned, air-dried, and ground into a fine powder. A total of 500 g of powdered root was subjected to Soxhlet extraction with 2 L of 50% ethanol for 4 hours to obtain the hydroalcoholic extract. The resulting solution was filtered, and the filtrate was concentrated under reduced pressure at 55 °C. The final extract yielded a dry weight of 18.5% (w/w) and was stored at 4 °C until use.

Animals and experimental protocol: Male albino rats (180–230 g, 8 weeks old) were used in this study. The animals were acclematized in a standard conditions with a 12-hour light/dark cycle, temperature of 22 ± 2 °C, relative humidity of 60±5%, and had free access to water and standard pellet feed. The animals were housed for one week before to the commence of the experiment.

Forty rats were randomly allocated into four groups (n = 10 per group) as follows: Group 1 (Control) received standard diet and drinking water, group 2 (Cadmium) received 0.5 ppm cadmium chloride ($CdCl_2$) freshly prepared in drinking water, administered daily for 30 days (El-Kott et al., 2020), group 3 (AG) received 100 mg/kg of *A. galanga* hydroalcoholic extract orally once daily for 30 days, and group 4 (Cadmium + AG) received both 0.5 ppm $CdCl_2$ in drinking water and 100 mg/kg of *A. galanga* extract orally for 30 days.

At the end of the 30-day experimental period, rats were anesthetized via intraperitoneal injection of ketamine (80 mg/kg, UK) and xylazine (5 mg/kg, Australia) (Dodelet-Devillers et al.,

2016). After anesthesia, cervical dislocation was performed, and brain tissues were dissected, rinsed in cold phosphate-buffered saline (PBS), and immediately kept at –80 °C for downstream analyses including RT-qPCR, RNA extraction, and oxidative stress assessment.

RNA extraction and quantification: Total RNA was extracted from brain tissues using an RNA extraction kit (Addbio, Korea), following the manufacturer's protocol. RNA concentration and purity were measured using a NanoDrop spectrophotometer (ThermoFisher Scientific, China). One microliter of each RNA sample was loaded on the pedestal for quantification, and concentrations were recorded in ng/µL. To prevent thermal degradation, RNA samples were kept on ice during measurements. Between each sample, the NanoDrop pedestal was cleaned with distilled water and dried. RNA concentrations were normalized to 25 ng/µL using the dilution equation ($C_1V_1 = C_2V_2$).

Gene expression analysis: Quantitative real-time PCR (qRT-PCR) was performed to assess the expression levels of selected antioxidant enzyme genes. SYBR Green-based detection was conducted using the Addbio SYBR Green Master (Rox) kit, following the manufacturer's instructions. PCR reactions were carried out on a StepOnePlus Real-Time PCR System with the following thermal cycling conditions: initial denaturation at 95 °C for 3 minutes, followed by 45 cycles of denaturation at 95 °C for 1 minute, annealing at 60 °C for 1 minute, and extension at 72 °C for 1 minute.

Relative gene expression was calculated using the $2^{-}\Delta\Delta Ct$ method (Livak & Schmittgen, 2001), with *glucose-6-phosphate dehydrogenase* (G6PD) as the reference gene (Mustafa & Alchalabi, 2022).

Table 1. Primer sequences and amplicon sizes for antioxidant genes analyzed via RT-qPCR

Amplicon Size (bp)	Primer Sequence (5'–3')	Gene Name	Accession Number
240	Sense: TGCAGCAGCTGTCCTCTATG Antisense: ACTTCAGCTTTGCGCTCATT	G6PD	AC094668.10
185	Sense: CAGCGACCAGATGAAGCA Antisense: GGTCAGGACATCGGGTTTC	Catalase	AH004967.2
270	Sense: TGTTACAACCCCGACTTTGA Antisense: TCTTCTCAGGGATGGTCTTCA	GST	AC097845.8
300	Sense: CGACATCGAACCCGATATAGA Antisense: ATGCCTTAGGGGTTGCTAAGG	GSH-Px	AC128721.4

Biochemical analysis: To assess oxidative stress, brain tissue samples were analyzed using rat-specific ELISA kits for malondialdehyde (MDA) and glutathione peroxidase (GSH-Px) (MyBioSource, USA), following the manufacturers' protocols.

Statistical analysis: Data were analyzed using one-way analysis of variance (ANOVA), followed by Scheffé's post hoc test for multiple comparisons, using SPSS version 26 (IBM Corp., USA). Results were expressed as mean ± standard error (SE), and differences were considered statistically significant at $p < 0.05$.

Results

Analysis revealed that brain malondialdehyde (MDA) content was significantly elevated in cadmium-exposed rats (1.6364 ± 0.01) compared to control rats (0.7247 ± 0.005; $p = 0.001$). However, rats treated with the Alpinia galangal (AG) hydroalcoholic extract exhibited MDA levels comparable to those in the control group (Figure 1).

Similarly, brain glutathione peroxidase (GSH-Px) concentrations were markedly reduced in cadmium-exposed rats (10.5098 ± 1.5) and in the group co-treated with cadmium and AG extract (15.9569 ± 1.5), in comparison with control rats ($p = 0.023$). Notably, AG extract alone significantly elevated GSH-Px levels relative to the cadmium-only group ($p = 0.046$) (Figure 2).

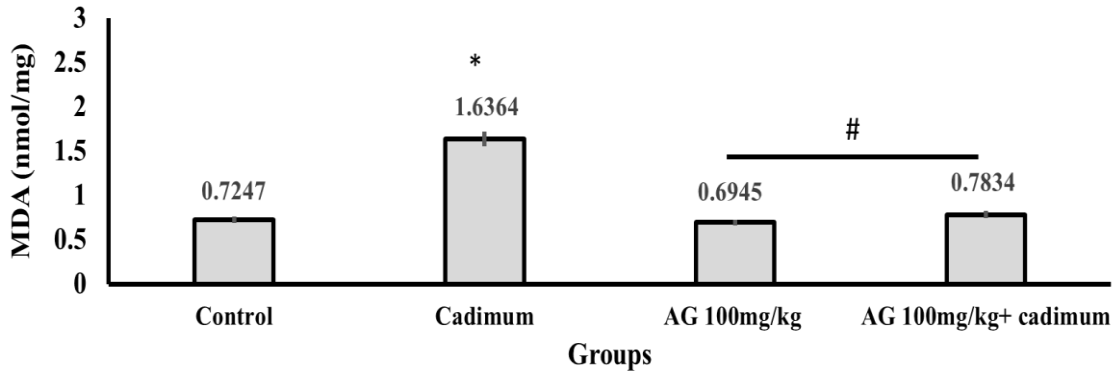

Figure 1. MDA levels in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root; MDA = Malondialdehyde

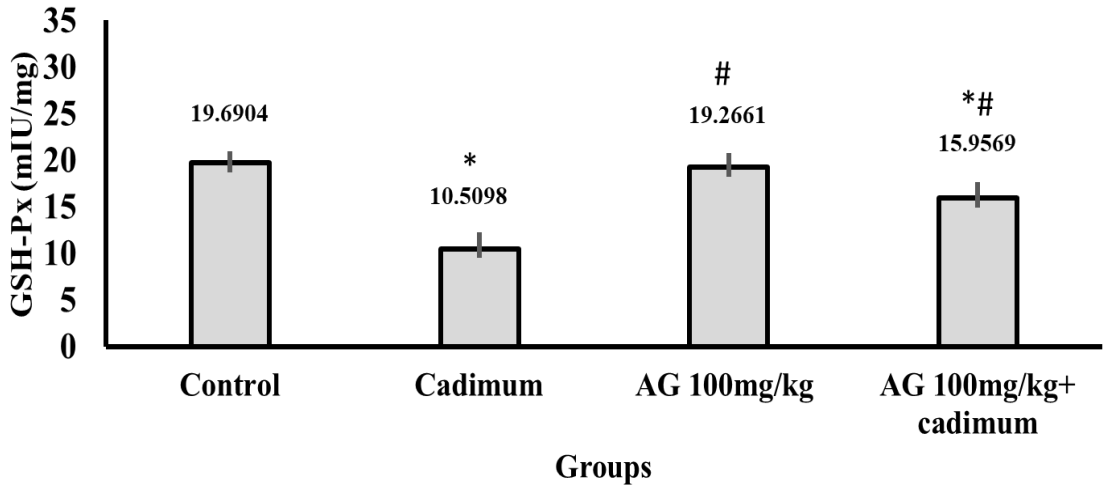

Figure 2. GSH-Px levels in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root; GSH-Px = Glutathione peroxidase

Gene expression analysis showed that catalase mRNA was significantly downregulated in both the cadmium-only group (0.74 ± 0.20) and the cadmium + AG group (0.83 ± 0.19) when compared to the control group (1.05 ± 0.25) and the AG-only group (0.94 ± 0.21) ($p = 0.001$). Nevertheless, treatment with AG extract significantly upregulated catalase expression relative to the cadmium-only group ($p = 0.036$ for AG alone; $p = 0.021$ for AG + cadmium) (Figure 3). The expression of glutathione S-transferase (GST) was significantly reduced in cadmium-exposed rats (1.05 ± 0.05) compared to the control group (1.94 ± 0.10; $p < 0.05$). Co-treatment with AG extract restored GST expression to 1.47 ± 0.09, which was significantly higher than in the cadmium-only group ($p < 0.05$) (Figure 4). Similarly, GSH-Px gene expression was significantly reduced in cadmium-treated rats (0.97 ± 0.05) compared to the control group (2.19 ± 0.15; $p < 0.05$). However, co-administration of AG extract with cadmium significantly increased GSH-Px expression to 1.34 ± 0.12 ($p < 0.05$) (Figure 5).

Discussion

Concern over cadmium (Cd)-induced neuropathy is growing due to its potential impact on the brain. Cadmium, a heavy metal (HM), is discharged into the environment through several industries. As it pollutes the air, water, and soil, it poses a threat to community health. Because of its extended biological half-life, Cd accumulates in the brain and other neural tissues, raising concerns about its effects on the nervous system. Since Cd can penetrate neurons, it increases the

formation of reactive oxygen species (ROS) and weakens the antioxidant defenses of those neurons (Rezaei et al., 2024).

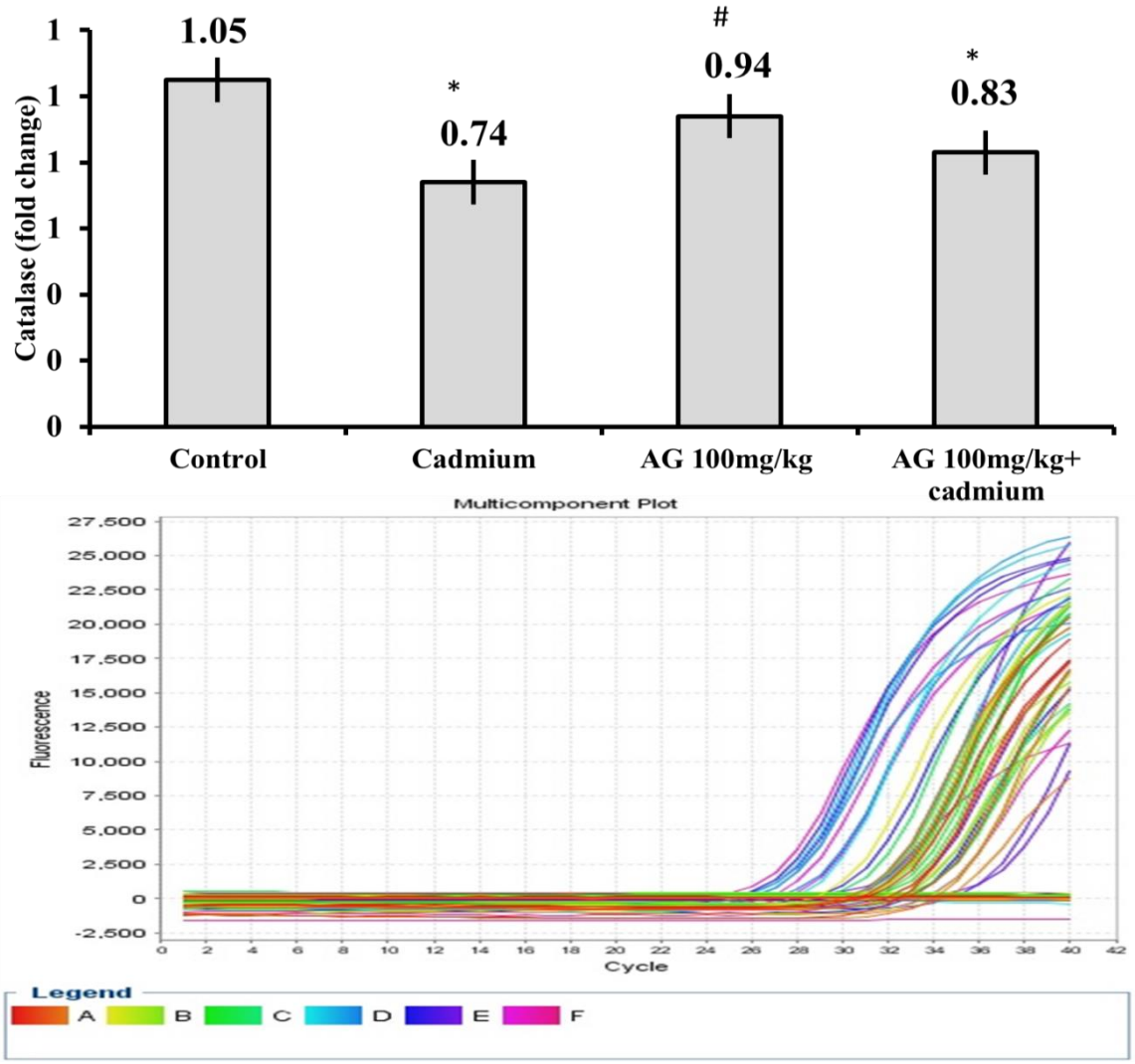

Figure 3. Fold change in catalase expression in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root

Exposure to 0.5 ppm cadmium results in elevated MDA concentrations in rat brain samples, due to the activation of free radical formation, which in turn attacks lipid molecules within brain tissue, leading to lipid peroxidation of the cellular components of the CNS (Al-Hashem et al., 2024).

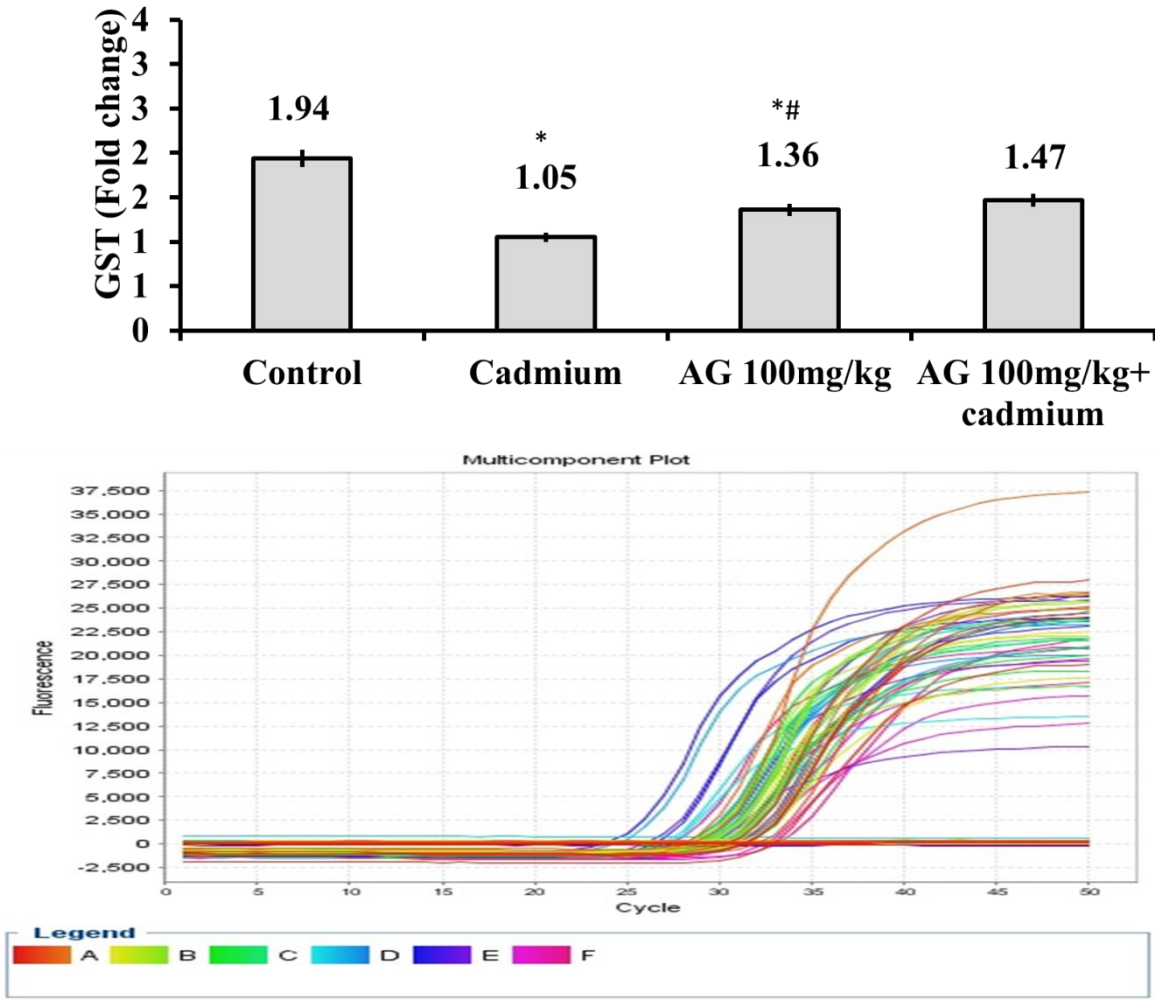

Figure 4. Fold change in GST expression in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root; GST = Glutathione S-transferase

Cadmium-induced lipid peroxidation is possibly the result of free radical-mediated oxidation via lipid peroxidation, which can damage both lipid molecules in biological membranes and low-density lipoproteins through a chain mechanism (Kapil et al., 2024; Villalón-García et al., 2023). Overall, besides phospholipid damage, growing free radical species may readily interact with membrane proteins and promote lipid–protein and protein–protein crosslinking, which leads to weakened membrane integrity and impaired function of the associated proteins (Mishra et al., 2022).

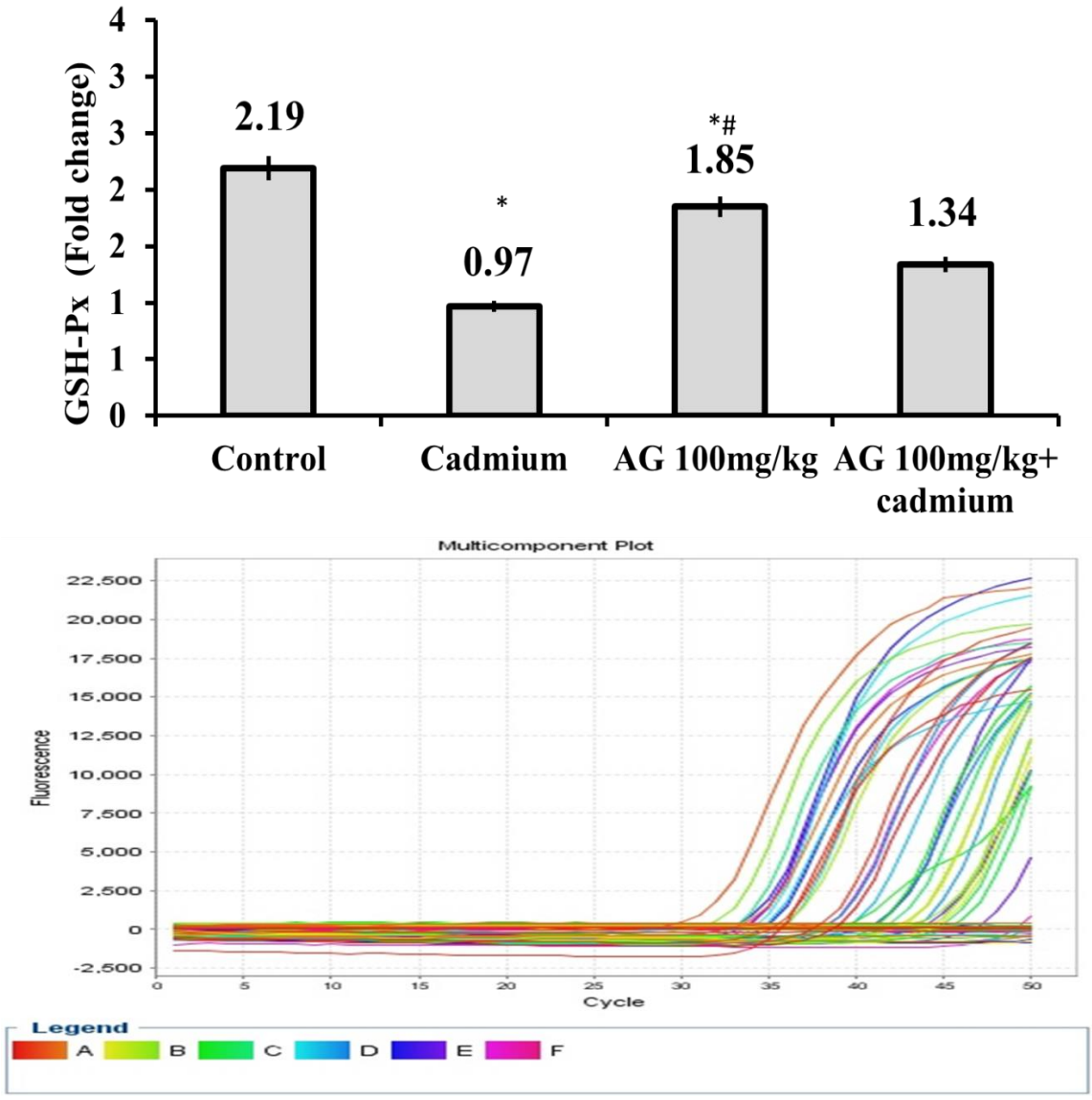

Figure 5. Fold change in GSH-Px expression in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root; GSH-Px = Glutathione peroxidase

An imbalance of free radicals and antioxidants alters the redox equilibrium, induces excessive oxidative stress, and harms cellular components such as DNA, proteins, and lipids. In the experiment, we reported that cadmium exposure lowered GSH-Px levels. This result could be linked to the excessive utilization of enzymatic antioxidants during cadmium-induced oxidative stress, suggesting that the brain's antioxidant capacity was depleted. Fortunately, treating

cadmium-toxic rats with 100 mg/kg of AG resulted in improved MDA and GSH-Px levels in brain tissues by the end of the trial. This could be attributed to the antioxidant properties of *Alpinia galanga* roots. These findings are consistent with Aziz et al. (2024) and Tian et al. (2022), who showed that AG extract possesses antioxidant properties as well as strong radical scavenging ability in various tissues and cell cultures. Galangal extract can considerably improve cognitive capacity in diabetic rats, minimize hippocampal degenerative alterations, and exert preventive or therapeutic effects on diabetic encephalopathy (Abd Rahman et al., 2024).

Cadmium has a detrimental effect on enzymatic antioxidant genes by downregulating them at the cellular level in brain tissues, according to transcriptomic analyses conducted on brain tissues from rats treated with cadmium and cadmium + AG. Additionally, administering AG to cadmium-exposed rats decreased the biological effect of cadmium on genes that regulate antioxidants in brain tissues, suggesting that AG root contains active components that influence essential genetic pathways, particularly those related to antioxidant defense. This is in line with Yu et al. (2016), who found that AG galangal extract can reduce pathological alterations in the hippocampus, improve cognitive function in diabetic rats, and exert preventative or therapeutic effects on diabetic encephalopathy by enhancing antioxidant cellular capacity in brain tissues. Furthermore, our findings align with Srivastava et al. (2017), who demonstrated that galangal positively influences cognitive performance—especially when used alongside coffee—as it reduces the drop in caffeine levels and sustains attention for up to three hours.

Conclusions: According to the study, 0.5 ppm of cadmium chloride exposure was extremely harmful and caused serious biological effects. However, by upregulating certain enzymatic antioxidant genes in brain tissues, the antioxidant capacity of *A. galanga* root extract can prevent these cadmium bioeffects by interfering with oxidant and antioxidant mechanisms and shielding the brain from significant damage caused by cadmium toxicity in a rat model.

Author Contributions

Conceptualization: SMA, AGM; Data curation: GFM; Formal analysis: AGM; Funding acquisition: GFM, AGM; Investigation: GFM, AGM; Methodology: GFM, AGM; Project administration: SMA, AGM; Software: GFM; Resources: AGM; Supervision: SMA, AGM; Validation: SMA, AGM; Visualization: SMA, AGM; Writing – original draft: SMA, GFM, AGM; Writing – review & editing: SMA, AGM, GFM. All authors contributed equally to the conceptualization of the article and the writing of the original and subsequent drafts.

Data Availability Statement

Data is available on request from the authors.

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Ethical Considerations

The study was approved by the Ethics Committee of the University of ABCD (Ethical code: IR.UT.RES.2024.500). The authors affirm that no data fabrication, falsification, plagiarism, or misconduct occurred.

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Conflict of Interest

The authors declare no conflict of interest.

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اثر محافظتی عصاره اتانولی ریشه خولنجان در برابر نوروتوکسیسیته القاشده توسط کادمیوم در مدل موش صحرایی

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چکیده

هدف: کادمیوم یک فلز سنگین سمی است که اثرات مخربی بر سامانه‌های زیستی مختلف دارد. عصاره‌های گیاهی به دلیل داشتن خواص آنتی‌اکسیدانی می‌توانند در کاهش این سمیت مؤثر باشند. هدف از این مطالعه، ارزیابی اثر محافظتی عصاره اتانولی ریشه خولنجان (Alpinia galanga) در برابر نوروتوکسیسیته ناشی از کادمیوم در یک مدل موش صحرایی بود.

مواد و روش‌ها: چهل موش نر به‌صورت تصادفی به چهار گروه (هر گروه ۱۰ عدد) تقسیم شدند. گروه ۱ (کنترل) به‌مدت ۳۰ روز آب آشامیدنی و غذای معمولی دریافت کرد. گروه ۲، عصاره هیدروالکلی خولنجان با دوز ۱۰۰ میلی‌گرم به‌ازای هر کیلوگرم به‌صورت خوراکی دریافت کرد. گروه ۳، آب آشامیدنی حاوی ۰/۵ پی‌پی‌ام کلرید کادمیوم ($CdCl_2$) دریافت کرد. گروه ۴، ترکیبی از $CdCl_2$ (۰/۵ پی‌پی‌ام در آب) و عصاره خولنجان (۱۰۰ میلی‌گرم/کیلوگرم به‌صورت خوراکی) به‌مدت ۳۰ روز دریافت کرد..

نتایج: در معرض قرار گرفتن با کادمیوم باعث افزایش معنی‌دار سطح مالون‌دی‌آلدئید (MDA) در مغز (1.6364±0.01) نسبت به گروه کنترل (0.7247 ± 0.005, $p = 0.001$) شد، در حالی که سطح MDA در موش‌های درمان‌شده با خولنجان مشابه

گروه کنترل بود. سطح گلوتاتیون پراکسیداز (GSH-Px) در گروه کادمیوم (10.5098±1.5) و گروه کادمیوم + عصاره (15.9569±1.5) به‌طور معنی‌داری کمتر از کنترل بود (p = 0.023). تحلیل بیان ژن کاهش معنی‌داری در بیان کاتالاز را در گروه‌های کادمیوم (0.2 ± 0.74) و کادمیوم + عصاره (0.19 ± 0.83) نسبت به گروه کنترل (0.25 ± 1.05) و عصاره تنها (0.21 ± 0.94) نشان داد ($p < 0.05$). بیان گلوتاتیون-S ترانسفراز (GST) نیز در گروه کادمیوم (0.05 ± 1.05) نسبت به کنترل (0.1 ± 1.94) به‌طور معنی‌داری کاهش یافت، اما با مصرف همزمان خولنجان سطح آن بهبود یافت ($p <$ 0.05, 0.09 ± 1.47). روند مشابهی برای بیان GSH-Px مشاهده شد که در گروه کادمیوم (0.05 ± 0.97) کاهش یافت و با درمان با خولنجان (0.12 ± 1.34) در مقایسه با کنترل ($p < 0.05$, 0.15 ± 2.19) بهبود یافت.

نتیجه‌گیری: یافته‌ها نشان می‌دهند که کادمیوم از طریق مکانیسم‌های استرس اکسیداتیو اثرات نوروتوکسیک اعمال می‌کند و عصاره ریشه خولنجان دارای خواص آنتی‌اکسیدانی قابل‌توجهی است که می‌تواند این اثرات نوروتوکسیک را کاهش دهد.

کلمات کلیدی: آنتی‌اکسیدان، استرس اکسیداتیو، خولنجان(Alpinia galanga) ، کادمیوم، نوروتوکسیسیته

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