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

F. Mohammed Ghassan

*Corresponding Author. Department of animal production Technologies, College of Agricultural Technique, Northern Technical University, Mosul, Iraq. E-mail address: ghassanalubaidy1961@ntu.edu.iq

G. Mohammed Alaa

Department of Pharmacy Technique, Northern Technical University, Mosul, Iraq. E-mail address: alaaghanim2a@ntu.edu.iq

M. Ali Saad

Department of physiology, biochemistry and pharmacology, Veterinary Medicine College, University of Mosul, Mosul, Iraq. E-mail address: saadvet1974@uomosul.edu.iq

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 principal pathways 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).

Moreover, Cd alters the physiological steps of constitutive antioxidant enzymes, blocking brain metabolism and interfering to its neurotoxicity profile (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).

Alpinia galanga (commonly known as galangal) originated in Asia and grew in different countries thereafter. Galangal has been used in traditional medicine for diseases, including rheumatism, inflammation, diabetes, and neurological disorders (Thapa et al., 2023). The root of *A. galangal* contains biomolecules, such as, terpenoids, flavonoids, phenolic acids, saponins, and essential oils. The major active compounds include 1,8-cineole, kaempferol, and galangal acetate (Van et al., 2021). A 50% ethanolic A. galangal extract demonstrated excellent antioxidant impacts compared to essential oils (Rahman et al., 2024; Rajendiran et al., 2018). Antioxidant effects have also been reported in extracts involving 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 condition 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.

After 30-day experimental period elapsed, 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 conducted, and brain tissues samples were removed, rinsed in normal saline, and instantly kept at –80 °C for further analyses including RT-qPCR, RNA extraction, and oxidative stress assessment.

RNA extraction and quantification: According to manufacturer instructions using RNA extraction kit (Addbio, Korea), RNA was extracted from processed samples. The RNA purity was estimated using a NanoDrop spectrophotometer (ThermoFisher Scientific, China) by loading a drop of two NanoDrop spectrophotometer, and concentrations recorded. RNA was normalized to 25 ng/μL using the dilution equation ($C_1V_1 = C_2V_2$).

Gene expression analysis: According to manufacturer instructions, quantitative real-time PCR (qRT-PCR) based on Addbio SYBR Green Master (Rox) kit was used to determine the expression of target genes using StepOnePlus Real-Time PCR machine optimized to the 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 determined by $2^{-\Delta\Delta Ct}$ method (Livak & Schmittgen, 2001), versus housekeeping gene *glucose-6-phosphate dehydrogenase* (G6PD) as the reference gene (Mustafa & Alchalabi, 2022).

Table 1. The designed primer sequences and the amplicon sizes for antioxidant genes

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 detect the redox activity as per manufacturer instructions, the brain specimen was measured by ELISA kits for malondialdehyde (MDA) and glutathione peroxidase (GSH-Px) specified for rats (MyBioSource, USA).

Statistical analysis: The results analysis was conducted using SPSS (V26, IBM Corp., USA). One-way analysis of variance (ANOVA) test and post hoc Scheffé test for multiple comparisons used to highlight the different group. Data expressed as mean ± standard deviation (SD) and different group considered at $p < 0.05$.

Results

Brain tissue sample has reflected that malondialdehyde (MDA) level was elevated ($p = 0.001$) in cadmium-challenged group (1.6364 ± 0.01) versus control group (0.7247 ± 0.005). However, rats treated with standardized Alpinia galangal (AG) extract showed MDA levels nearly similar 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).

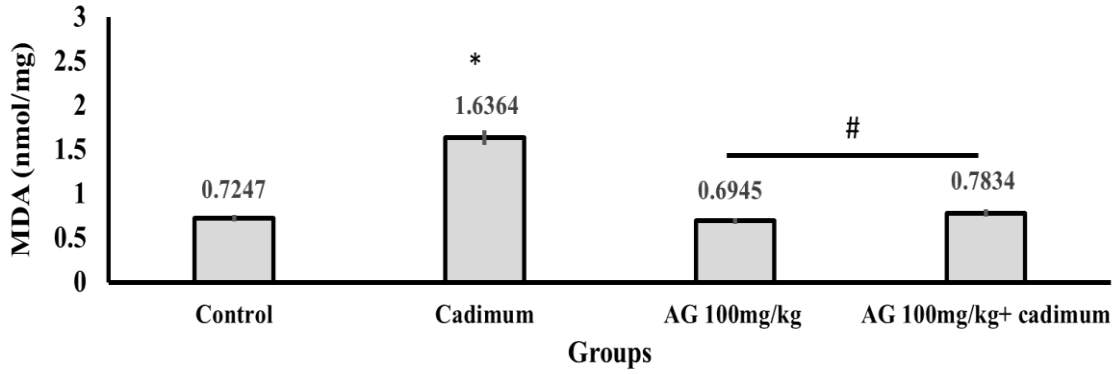

Figure 1. MDA levels in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root; MDA = Malondialdehyde

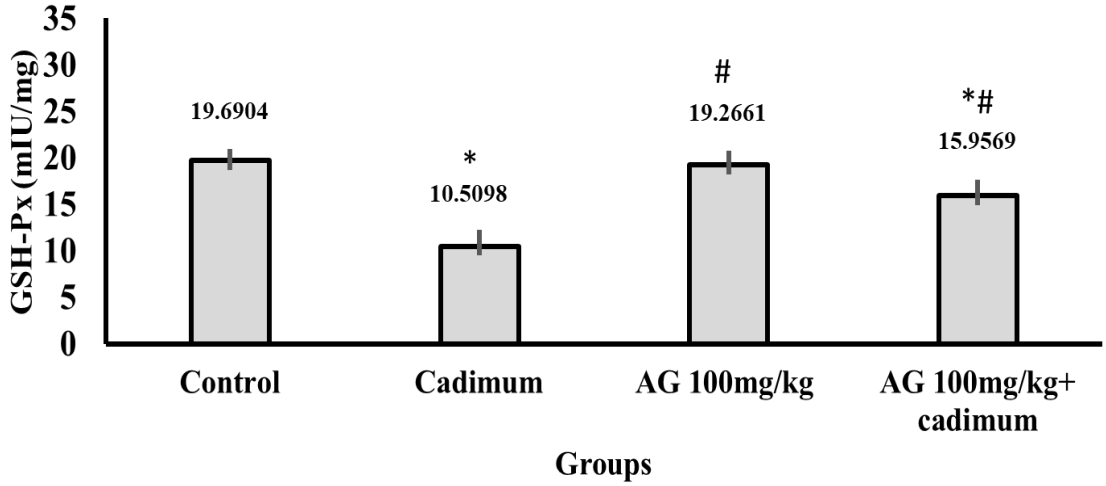

Figure 2. GSH-Px levels in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root; GSH-Px = Glutathione peroxidase

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

Being a heavy metal (HM), cadmium (Cd)-induced neurological defects is growing alarming due to its capacity to impact on the brain. Cadmium is drained into the surrounding environment from industrial companies. As it poisons the air, water, and soil, it carries a risk to society health. Due to its extended biological half-life, Cd build up in the neural tissues including brain, raising fears concerning its impact on the nervous system. Since Cd can enter neurons, it upregulates the generation of reactive oxygen species (ROS) and impairs the antioxidant defenses of those neurons (Rezaei et al., 2024).

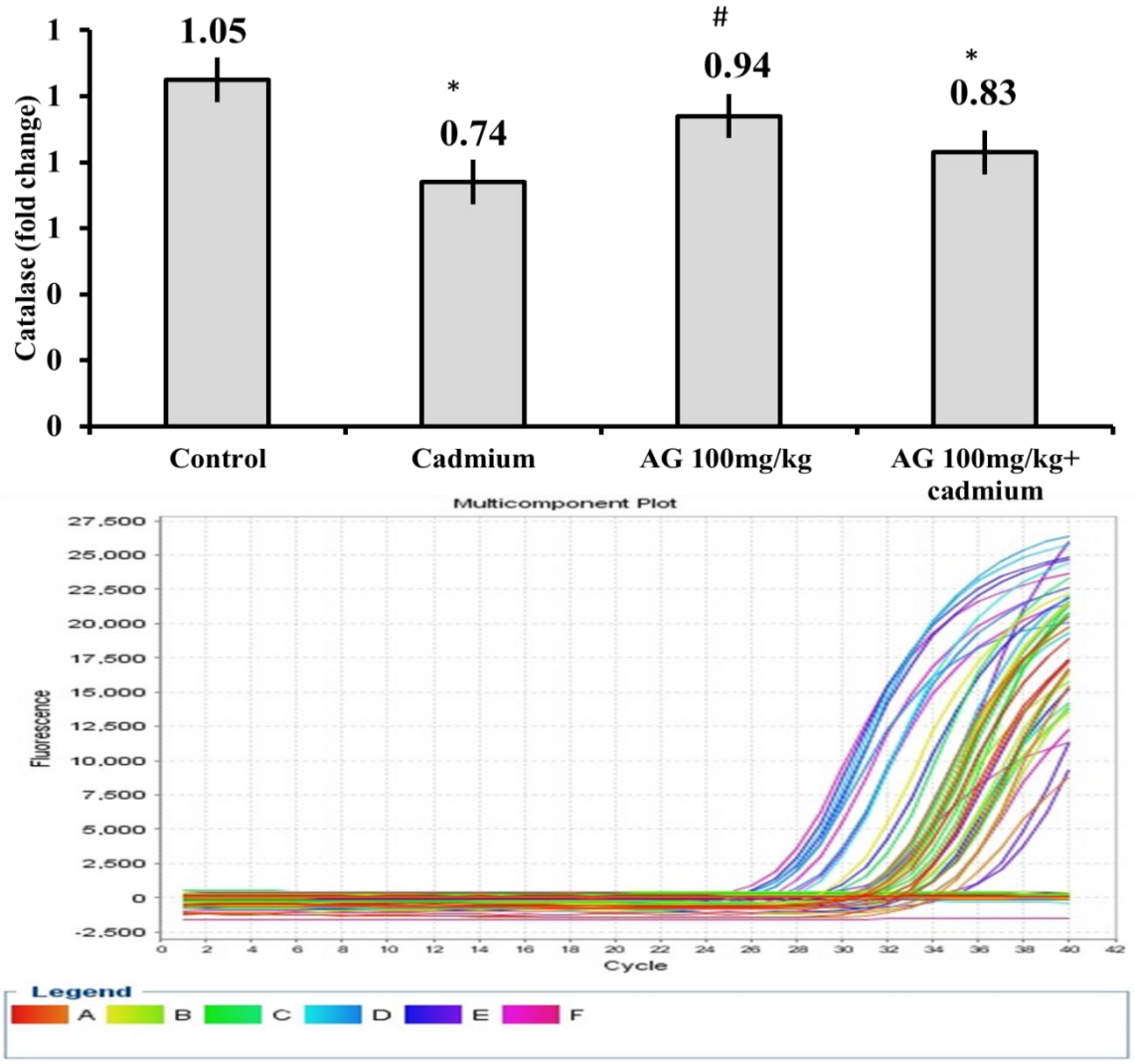

Figure 3. Fold change in catalase expression in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root

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).

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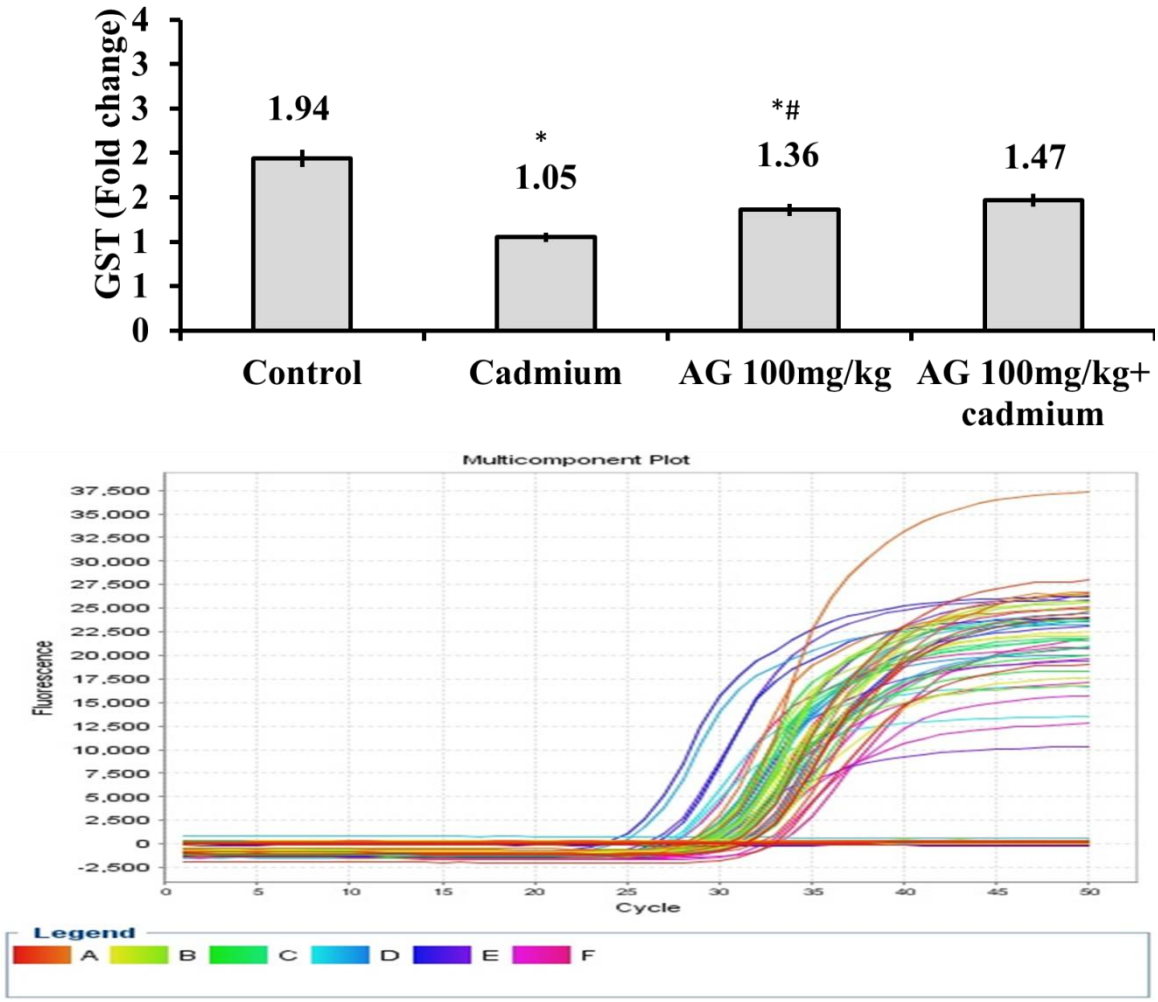

Figure 4. Fold change in GST expression in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root; GST = Glutathione S-transferase

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).

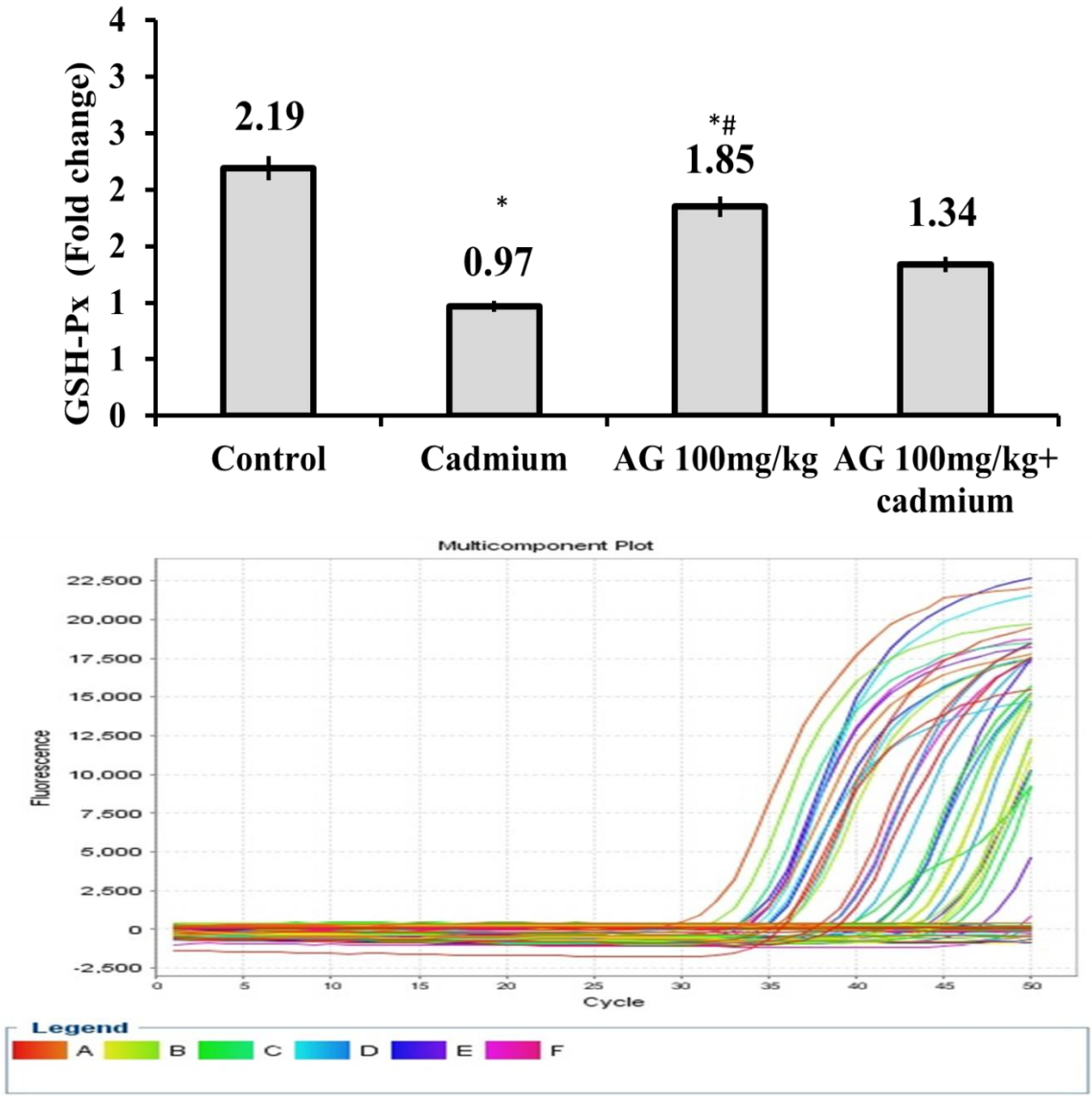

Figure 5. Fold change in GSH-Px expression in experimental groups. Data are expressed as mean ± SD. * indicates significant difference from control; # indicates significant difference from cadmium group ($p < 0.05$). AG = Alpinia galangal root; GSH-Px = Glutathione peroxidase

The dysregulated redox balance alters the oxidant/antioxidant equilibrium, induces excessive oxidative stress, and defects the cellular components, such as, DNA, proteins, and lipids biomolecules. In the present study, we highlighted that Cd exposure lowered GSH-Px concentrations. These findings might be explained in the context of extensive harnessing of enzymatic antioxidants during Cd-induced oxidative stress, reflecting that the brain's antioxidant

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efficiency was compromised. Nevetheless, reversing Cd-toxicity with 100 mg/kg of AG showed improved MDA and GSH-Px concentrations in brain tissues by the end of the experiment. Perhaps this could be related to the antioxidant properties of *Alpinia galanga* roots. These results are congruent with Aziz et al. (2024) and Tian et al. (2022), who have demonstrated that AG extract holds antioxidant efficiency alongside strong radical removing capacity in different tissues and cell cultures. Galangal extract can extensively improve cognitive capability in diabetic rats, mitigating hippocampal degenerative damage, and apply preventive or therapeutic effects on diabetic encephalopathy (Abd Rahman et al., 2024).

Cadmium has a negative impact on endogenous antioxidant genes by down-regulating them at the subcellular level in brain tissues sample as indicated by transcriptomic analyses performed on brain tissues from rats exposed to either cadmium and cadmium + AG. Moreover, supplementing AG to Cd-exposed rats decreased the biological effect of Cd on genes that control antioxidants in brain tissues, reflecting that AG root constitutes of active ingredients that change fundamental genetic pathways, principally those belong to antioxidant defense. This is in agreement with Yu et al. (2016), who reported that AG galangal extract can mitigate pathological processes in the hippocampus, improving the cognitive function in diabetic rats, and apply prophylactic or therapeutic effects on diabetic encephalopathy by boosting antioxidant subcellular ability in brain tissues. Furthermore, our results congruent with Srivastava et al. (2017), who have documented that galangal positively impacts cognitive enactment, especially when used together with coffee as it reduces the drop in caffeine levels and withstands attention for up to three hours.

Conclusions: Based on the outcome of the present study, 0.5 ppm of Cd-exposure was highly toxic and caused severe biological defects. However, up-regulating endogenous enzymatic antioxidant genes in brain tissues, the antioxidant capacity of *A. galanga* root extract can block these cadmium bioeffects by interfering with oxidant and antioxidant mechanisms and shielding the brain from significant damage caused by Cd 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.

References

Abd Rahman, I. Z., Adam, S. H., Hamid, A. A., Mokhtar, M. H., Mustafar, R., Kashim, M. I. A. M., Febriza, A., & Mansor, N. I. (2024). Potential Neuroprotective Effects of Alpinia officinarum Hance (Galangal): A Review. *Nutrients*, *16*(19). https://doi.org/10.3390/nu16193378

Afifi, O., & Embaby, A. (2016). Histological Study on the Protective Role of Ascorbic Acid on Cadmium-Induced Cerebral Cortical Neurotoxicity in Adult Male Albino Rats. *Journal of Microscopy and Ultrastructure*, *4*(1), 36. https://doi.org/10.1016/J.JMAU.2015.10.001

Al-Hashem, F. H., Bashir, S. O., Dawood, A. F., Aboonq, M. S., Bin-Jaliah, I., Al-Garni, A. M., & Morsy, M. D. (2024). Vanillylacetone attenuates cadmium chloride-induced hippocampal damage and memory loss through the upregulation of nuclear factor erythroid 2-related factor 2 gene and protein expression. *Neural Regeneration Research*, *19*(12), 2750–2759. https://doi.org/10.4103/1673-5374.391300

Aziz, I. M., Alfuraydi, A. A., Almarfadi, O. M., Aboul-Soud, M. A. M., Alshememry, A. K., Alsaleh, A. N., & Almajhdi, F. N. (2024). Phytochemical analysis, antioxidant, anticancer,

and antibacterial potential of Alpinia galanga (L.) rhizome. *Heliyon*, *10*(17), e37196. https://doi.org/10.1016/J.HELIYON.2024.E37196

Branca, J. J. V., Fiorillo, C., Carrino, D., Paternostro, F., Taddei, N., Gulisano, M., Pacini, A., & Becatti, M. (2020). Cadmium-induced oxidative stress: Focus on the central nervous system. *Antioxidants*, *9*(6), 1–21. https://doi.org/10.3390/antiox9060492

Dodelet-Devillers, A., Zullian, C., Beaudry, F., Gourdon, J., Chevrette, J., Hélie, P., & Vachon, P. (2016). Physiological and pharmacokinetic effects of multilevel caging on Sprague Dawley rats under ketamine-xylazine anaesthesia. *Experimental Animals*, *65*(4), 383–392. https://doi.org/10.1538/EXPANIM.16-0026

El-kott, A. F., Abd-Lateif, A. E. K. M., Khalifa, H. S., Morsy, K., Ibrahim, E. H., Bin-Jumah, M., Abdel-Daim, M. M., & Aleya, L. (2020). Kaempferol protects against cadmium chloride-induced hippocampal damage and memory deficits by activation of silent information regulator 1 and inhibition of poly (ADP-Ribose) polymerase-1. *Science of The Total Environment*, *728*, 138832. https://doi.org/10.1016/J.SCITOTENV.2020.138832

El-Tarras, A. E. S., Attia, H. F., Soliman, M. M., El Awady, M. A., & Amin, A. A. (2016). Neuroprotective effect of grape seed extract against cadmium toxicity in male albino rats. *International Journal of Immunopathology and Pharmacology*, *29*(3), 398–407. https://doi.org/10.1177/0394632016651447

Genchi, G., Sinicropi, M. S., Lauria, G., Carocci, A., & Catalano, A. (2020). The Effects of Cadmium Toxicity. *International Journal of Environmental Research and Public Health*, *17*(11), 3782. https://doi.org/10.3390/ijerph17113782

Hazrat, A., Ezzat, K., & Ilahi Ikram. (2019). Environmental Chemistry and Ecotoxicology of Hazardous Heavy Metals: Environmental Persistence, Toxicity, and Bioaccumulation. *Journal of Chemistry*, *2019*(Cd), 1–14. https://doi.org/10.1155/2019/6730305

Hung, N. H., Quan, P. M., Satyal, P., Dai, D. N., Hoa, V. V., Huy, N. G., Giang, L. D., Ha, N. T., Huong, L. T., Hien, V. T., & Setzer, W. N. (2022). Acetylcholinesterase Inhibitory Activities of Essential Oils from Vietnamese Traditional Medicinal Plants. *Molecules*, *27*(20), 7092. https://doi.org/10.3390/molecules27207092

Juntachote, T., & Berghofer, E. (2005). Antioxidative properties and stability of ethanolic extracts of Holy basil and Galangal. *Food Chemistry*, *92*(2), 193–202. https://doi.org/10.1016/J.FOODCHEM.2004.04.044

Kapil, L., Kumar, V., Kaur, S., Sharma, D., Singh, C., & Singh, A. (2024). Role of Autophagy and Mitophagy in Neurodegenerative Disorders. *CNS & Neurological Disorders - Drug Targets*, *23*(3), 367–383. https://doi.org/10.2174/1871527322666230327092855

Kojima-Yuasa, A., & Matsui-Yuasa, I. (2020). Pharmacological Effects of 1′-Acetoxychavicol Acetate, a Major Constituent in the Rhizomes of Alpinia galanga and Alpinia conchigera. *Journal of Medicinal Food*, *23*(5), 465–475. https://doi.org/10.1089/JMF.2019.4490

Livak, K. J., & Schmittgen, T. D. (2001). Analysis of Relative Gene Expression Data Using Real-Time Quantitative PCR and the 2−ΔΔCT Method. *Methods*, *25*(4), 402–408. https://doi.org/10.1006/METH.2001.1262

Mishra, J., Bhatti, G. K., Sehrawat, A., Singh, C., Singh, A., Reddy, A. P., Reddy, P. H., & Bhatti, J. S. (2022). Modulating autophagy and mitophagy as a promising therapeutic approach in neurodegenerative disorders. *Life Sciences*, *311*, 121153. https://doi.org/10.1016/J.LFS.2022.121153

Mustafa, W., & Alchalabi, A. S. (2022). Heat shock protein and antioxidant enzyme gene expression and fold changes in bone marrow samples from healthy rats. *Journal of Education and Science*, *31*(3), 165–175. https://scispace.com/papers/heat-shock-protein-and-antioxidant-enzyme-gene-expression-2f0w38s4

Rahman, I. Z. A., & , Siti Hajar Adam , Adila A. Hamid , Mohd Helmy Mokhtar , Ruslinda Mustafar , Mohd Izhar Ariff Mohd Kashim, A. F. and N. I. M. (2024). Potential Neuroprotective Effects of Alpinia officinarum Hance (Galangal): A Review. *Nutrients*, *16*(19), 3378. https://doi.org/10.3390/nu16193378

Rajendiran, V., Natarajan, V., & Devaraj, S. N. (2018). Anti-inflammatory activity of Alpinia officinarum hance on rat colon inflammation and tissue damage in DSS induced acute and chronic colitis models. *Food Science and Human Wellness*, *7*(4), 273–281. https://doi.org/10.1016/J.FSHW.2018.10.004

Ranjan, N., Shweta, S., & Kumari, M. (2022). In vitro screening for Acetylcholinesterase Inhibition and Antioxidant activity of selected Medicinal Plants. *International Journal of Agricultural and Applied Sciences*, *3*(2), 114–119. https://doi.org/10.52804/ijaas2022.3221

Rezaei, K., Mastali, G., Abbasgholinejad, E., Bafrani, M. A., Shahmohammadi, A., Sadri, Z., & Zahed, M. A. (2024). Cadmium neurotoxicity: Insights into behavioral effect and

neurodegenerative diseases. *Chemosphere*, *364*, 143180. https://doi.org/10.1016/J.CHEMOSPHERE.2024.143180

Satarug, S., Garrett, S. H., Sens, M. A., & Sens, D. A. (2010). Cadmium, environmental exposure, and health outcomes. *Environmental Health Perspectives*, *118*(2), 182–190. https://doi.org/10.1289/EHP.0901234

Srivastava, S., Mennemeier, M., & Pimple, S. (2017). Effect of Alpinia galanga on Mental Alertness and Sustained Attention With or Without Caffeine: A Randomized Placebo-Controlled Study. *Journal of the American College of Nutrition*, *36*(8), 631–639. https://doi.org/10.1080/07315724.2017.1342576

Thapa, R., Afzal, O., Alfawaz Altamimi, A. S., Goyal, A., Almalki, W. H., Alzarea, S. I., Kazmi, I., Jakhmola, V., Singh, S. K., Dua, K., Gilhotra, R., & Gupta, G. (2023). Galangin as an inflammatory response modulator: An updated overview and therapeutic potential. *Chemico-Biological Interactions*, *378*, 110482. https://doi.org/10.1016/J.CBI.2023.110482

Tian, Y., Jia, X., Wang, Q., Lu, T., Deng, G., Tian, M., & Zhou, Y. (2022). Antioxidant, Antibacterial, Enzyme Inhibitory, and Anticancer Activities and Chemical Composition of Alpinia galanga Flower Essential Oil. *Pharmaceuticals*, *15*(9). https://doi.org/10.3390/PH15091069

Unsal, V., Dalkiran, T., Çiçek, M., & Kölükçü, E. (2020). The Role of Natural Antioxidants Against Reactive Oxygen Species Produced by Cadmium Toxicity: A Review. *Advanced Pharmaceutical Bulletin*, *10*(2), 184. https://doi.org/10.34172/APB.2020.023

Van, H. T., Thang, T. D., Luu, T. N., & Doan, V. D. (2021). An overview of the chemical composition and biological activities of essential oils from Alpinia genus (Zingiberaceae). *RSC Advances*, *11*(60), 37767–37783. https://doi.org/10.1039/D1RA07370B

Villalón-García, I., Povea-Cabello, S., Álvarez-Córdoba, M., Talaverón-Rey, M., Suárez-Rivero, J., Suárez-Carrillo, A., Munuera-Cabeza, M., Reche-López, D., Cilleros-Holgado, P., Piñero-Pérez, R., & Sánchez-Alcázar, J. (2023). Vicious cycle of lipid peroxidation and iron accumulation in neurodegeneration. *Neural Regeneration Research*, *18*(6), 1196. https://doi.org/10.4103/1673-5374.358614

Wang, B., & Du, Y. (2013). Cadmium and its neurotoxic effects. *Oxidative Medicine and Cellular Longevity*. https://doi.org/10.1155/2013/898034

Yu, D.-R., Wang, T., Ji, L.-P., Fang, X.-Y., Y, X.-N., & Liu, Q.-B. (2016). Effects of galangal extract on cognitive dysfunction and nerve pathological change in rats with diabetic encephalopathy. *Journal of Hainan Medical University*, *22*(17), 1–5. https://www.ingentaconnect.com/content/doaj/10071237/2016/00000022/00000017/art00001

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

ف. محمد غسان

*نویسنده مسئول: گروه فناوری‌های تولیدات دامی، دانشکده فناوری کشاورزی، دانشگاه فنی شمال، موصل، عراق.
ایمیل: ghassanalubaidy1961@ntu.edu.iq

گ. محمد علاء

گروه فناوری داروسازی، دانشگاه فنی شمال، موصل، عراق.
ایمیل: alaaghanim2a@ntu.edu.iq

م. علی سعد

گروه فیزیولوژی، بیوشیمی و فارماکولوژی، دانشکده دامپزشکی، دانشگاه موصل، موصل، عراق.
ایمیل: saadvet1974@uomosul.edu.iq

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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 (1.5±10.5098) و گروه کادمیوم + عصاره (1.5±15.9569) 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 ($1.47 \pm 0.09, p < 0.05$). 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 ($2.19 \pm 0.15, p < 0.05$) 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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