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Effect of *Zingiber officinale* Ethanol Extract on Neurological Indices of Male Wistar Albino Rats Induced with Inflammation

Edoga, Cyril Onyekachi[1*], Okoh, Emmanuel Chidera[1], Nebechi, Sylvia Chiamaka[2]

[1] Department of Applied Biology and Biotechnology, Enugu State University of Science and Technology

[2] Deparment of Nursing Science, University of Huddersfield, Queens Gate, West Yorkshire, England

Corresponding Author: Edoga, Cyril Onyekachi

Department of Applied Biology and Biotechnology, Enugu State University of Science and Technology

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Abstract: This study investigated the effect of *Zingiber officinale* ethanol extract on neurological indices in male Wistar albino rats induced with inflammation. The study was laid out in a Complete Randomized Experimental Design (CRED). A total of five groups were assessed: Group A (Blank Control), Group B (Negative Control), Group C (Standard Control), Group D (Low-Dose Treated Group), and Group E (High-Dose Treated Group). Inflammatory induction led to significant reductions ($p < 0.05$) in the concentrations of key neurohormones including dopamine, serotonin, acetylcholine, and epinephrine in the negative control group. Specifically, dopamine and serotonin levels were markedly decreased in Group B (449 ± 0.001 pg/ml and 1.60 ± 0.001 µg/L, respectively) compared to Group A (653 ± 0.001 pg/ml and $5.21 \pm$ 0.001 µg/L, respectively). Acetylcholine and epinephrine levels also declined significantly in the negative control group (5.16 ± 0.031 mm and 3.12 ± 0.004 pg/ml, respectively) relative to the blank control. Treatment with *Z. officinale* ethanol extract, particularly at the high dose (Group E), significantly restored neurohormonal levels. The high-dose group showed values statistically similar ($p > 0.05$) to the blank control in dopamine (638 ± 0.000 pg/ml), serotonin (5.12 ± 0.003 µg/L), and acetylcholine (11.08 ± 0.013 mm), while moderate improvements were observed in epinephrine levels (7.91 ± 0.023 pg/ml). The findings suggest that *Z. officinale* ethanol extract exerts neuroprotective effects by enhancing neurotransmitter levels suppressed during inflammation, with the high dose showing the most significant efficacy.

Keywords: *inflammation, Zingiber officinale, dopamine, serotonin, acetylcholine, and epinephrine.*

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Introduction

Background to the Study

Inflammation is a basic biological response to harmful stimuli, such as pathogens, damaged cells, or irritants. While acute inflammation serves as a protective mechanism, chronic inflammation can lead to various pathological conditions, including neurodegenerative diseases like Alzheimer's and Parkinson's diseases (Ebert *et al*., 2019). The central nervous system (CNS) is particularly vulnerable to inflammatory processes, which can disrupt neurotransmitter systems and lead to cognitive and behavioral impairments. Neuro-inflammation, characterized by the activation of microglia and astrocytes, results in the release of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukins (IL-1β, IL-6), contributing to neuronal damage and synaptic dysfunction (Di-Sabato *et al*., 2016). This inflammatory milieu adversely affects neurotransmitter systems, including dopamine, serotonin, acetylcholine, and epinephrine, which are crucial for maintaining CNS homeostasis. Dopamine is involved in motor control and reward mechanisms; serotonin regulates mood and cognition; acetylcholine is essential for learning and memory; and epinephrine modulates stress responses (Seo *et al*., 2019). Alterations in the levels of these neurotransmitters are associated with various neurological disorders. For instance, decreased dopamine levels are linked to Parkinson's disease, while reduced serotonin and acetylcholine levels are associated with depression and Alzheimer's disease, respectively.

Pharmacological management of neuro-inflammation and neurotransmitter imbalance remains challenging. Conventional medications like non-steroidal anti-inflammatory drugs (NSAIDs), anticholinergics, dopamine agonists, and selective serotonin reuptake inhibitors (SSRIs) often come with significant side effects, limited efficacy over time, and do not halt disease progression (Zhou *et al*., 2020). Moreover, the high cost and limited accessibility of synthetic drugs in many low-resource settings have further necessitated the exploration of alternative therapeutic agents, particularly from natural sources (Akinyemi *et al*., 2021). *Zingiber officinale*, is a medicinal plant widely used for its anti-inflammatory, antioxidant, and neuroprotective properties. Studies have shown that *Zingiber officinale* extracts can modulate neurotransmitter levels and protect against neuroinflammation and oxidative stress. The ethanolic extract of *Zingiber officinale* contains bioactive compounds like gingerols and shogaols, which have been reported to exert beneficial effects on the CNS (Sahardi and Makpol, 2019).

Balance

Neuro-inflammation plays a central role in the pathogenesis of various neurological and neurodegenerative disorders, including Parkinson's disease, Alzheimer's disease, depression, multiple sclerosis, and traumatic brain injury (Almutairi *et al*., 2021). These conditions are often associated with dysregulation of key neurotransmitters such as dopamine, serotonin, acetylcholine, and epinephrine, leading to behavioral, cognitive, and motor impairments (Frost *et al*., 2020). The inflammation-induced alterations in neurotransmitter metabolism and signaling pathways contribute significantly to the clinical manifestations and progression of these disorders (Johnson *et al*., 2021).

Despite the growing interest in medicinal plants, there remains a substantial gap in empirical data concerning their efficacy and mechanisms in the modulation of neuro-inflammation and neurotransmitter pathways. *Zingiber officinale*, a traditionally used plant with well-documented anti-inflammatory and antioxidant properties, has shown potential neuroprotective effects in various in vitro and in vivo studies (Ghasemzadeh *et al*., 2022). However, there is limited research specifically examining its effects on central neurotransmitters under inflammatory conditions.

This lack of comprehensive data poses a barrier to the scientific validation and potential clinical application of ginger-based therapies. There is, therefore, a pressing need for well-structured experimental studies to investigate the neurochemical and neuroprotective responses to *Zingiber officinale* extracts in animal models exposed to inflammation. Such studies can clarify whether and how ginger influences neurotransmitter levels during neuro-inflammatory stress.

Justification of the Study

The increasing burden of neurodegenerative diseases and neuropsychiatric conditions linked to inflammation and neurotransmitter imbalance underscores the urgency for effective and safer therapeutic alternatives. According to the World Health Organization (WHO), neurological disorders account for over 6% of the global burden of disease, and inflammation is a key pathological mechanism in many of these disorders (WHO, 2022). There is a growing recognition of the potential benefits of plant-derived bioactive compounds in mitigating neuro-inflammation and restoring neurotransmitter balance (Pohl and Kong Thoo Lin, 2018).

Zingiber officinale has been widely used in traditional medicine across Asia and Africa and is known for its rich phytochemical composition, including gingerols, shogaols, paradols, and zingerone. These compounds exhibit strong antioxidant and anti-inflammatory activities that may play crucial roles in modulating neuroinflammatory cascades and preserving neuronal function (Sharma *et al*., 2021). Some preliminary studies have indicated that ginger extracts may influence brain-derived neurotrophic factor (BDNF), inhibit pro-inflammatory cytokines, and reduce oxidative stress—all of which are associated with neurotransmitter regulation (Hasanpour-Dehkordi *et al*., 2019).

However, while these findings are promising, there is a significant gap in literature focusing on the direct impact of *Zingiber officinale* ethanol extract on key neurotransmitters in the context of induced inflammation. Knowing the modulatory effects of ginger on these neurotransmitters is essential, considering their pivotal roles in neural transmission, emotion, learning, and stress response (Kaur *et al*., 2020). Additionally, this study will make use of male Wistar albino rats as an experimental model, which are well-validated in neuro-pharmacological and behavioral studies due to their consistent physiological responses and high translational value. The findings from this research could serve as a stepping stone for future clinical applications and pave the way for natural, accessible treatments with minimal side effects, especially in developing countries where synthetic drugs may be inaccessible or cost-prohibitive (Kaur *et al*., 2020).

Aim of the Study

The primary aim of this study was to investigate the effect of *Zingiber officinale* ethanol extract on neurological indices of male Wistar albino rats induced with inflammation.

Objectives of the study

The objectives of this study were to:

- Determine the effect of *Zingiber officinale* ethanol extract on dopamine level of male Wistar albino rats induced with inflammation.
- Investigate the effect of *Zingiber officinale* ethanol extract on serotonin level of male Wistar albino rats induced with inflammation.
- Determine the effect of *Zingiber officinale* ethanol extract on acetylcholine level of male Wistar albino rats induced with inflammation.
- Determine the effect of *Zingiber officinale* ethanol extract on epinephrine level of male Wistar albino rats induced with inflammation.

Materials and Methods

Plant Collection and Identification

Fresh rhizomes of *Zingiber officinale* were purchased from Nkpokiti Market, Enugu State. The plant material was authenticated by Prof. C. S. Eze in the Department of Applied Biology and Biotechnology at Enugu state University of Science and Technology.

Preparation of Ethanol Extract of *Zingiber officinale*

The rhizomes which weighed 76.4 grams were washed thoroughly with distilled water to remove dirt and dried using analytical oven 105°C. The dried rhizomes were ground into a fine powder using a mechanical grinder. The powdered sample was placed in a soxhlet apparatus, and 300 mL of pure ethanol was used as the solvent for extraction (Saha *et al.*, 2021). The soxhlet extraction process was carried out for 5 hours. The mixtutre was filtered using Whatman No. 1 filter paper. The filtrate was concentrated under reduced pressure using a rotary evaporator at 40°C to yield the crude ethanol extract.

Experimental Animals

A total of thirty (30) healthy male Wistar albino rats weighing 10–220 g were obtained from University of Nigeria Enugu Campus (UNEC) Animal House. The animals were housed in plastic cages under standard conditions of 12-hour light/dark cycle, temperature (25°C). Rats were acclimatized for three weeks before the commencement of the experiment and had standard rat feed and clean water.

Induction of Inflammation

Inflammation was induced by intraperitoneal injection of 0.5 ml of egg albumin solution and in the hind paw region. The inflammation peaked within 3-6 hours. The paw thickness was monitored using a Vernier caliper to confirm the presence and resolution of inflammation.

Experimental Design

The study was laid on a Complete Randomized Experimental Design (CRED), and the animals were sampled and grouped into 5 comprising six rats:

- Group A (Blank Control): were neither induced nor treated, but retained feed and, water *ad libitum*
- Group B (Negative Control): induced with concentrated egg albumin, but received no treatment.
- Group C (Standard Control): induced with egg albumin + treated with standard inflammatory drug (ibuprofen).
- Group D (Low Dose *Zingiber officinale* Extract): induced with egg albumin + treated with 50 mg/kg body weight of *Zingiber officinale* extract.
- Group F (High Dose *Zingiber officinale* Extract): induced with egg albumin + treated with 200 mg/kg body weight of *Zingiber officinale* extract.

Treatments were administered orally (via intubation) once daily for 3 consecutive weeks following egg albumin induction.

Blood Sample Collection

At the end of the treatment period, the rats were anesthetized using chloroform. Blood samples were collected via ocular puncture into plain bottles for biochemical analysis.

Biochemical Analyses

Neurotransmitter levels are typically measured using high-performance liquid chromatography (HPLC) with electrochemical detection or enzyme-linked immunosorbent assay (ELISA) kits specific to dopamine, serotonin, acetylcholine, and epinephrine (Anwar *et al*., 2020). These assays provide sensitive and accurate quantification of neurotransmitter concentrations in brain homogenates. Additionally, protein content normalization using the Bradford method ensures consistent comparison across samples.

This methodological approach enables researchers to evaluate the neuroprotective and anti-inflammatory efficacy of *Zingiber officinale* in modulating neurotransmitter levels disrupted by inflammation.

Statistical Analysis

All the statistical analysis was processed using the Statistical Package of Social Science (SPSS) for the window (version 21). The values of the measured parameters were expressed as mean ± SEM. One-way Analysis of Variance (1-way ANOVA) was used to determine the effect of inflammation and *Zingiber officinale* ethanol extract on the parameters studied and the difference between means were separated using Duncan's multiple range test. Test for significance was at 0.05 probability level.

Results

Dopamine Level

Group A (Blank Control) and Group E (High-Dose Treated Group) showed the highest dopamine concentrations (653 ± 0.001 pg/ml and 638 ± 0.000 pg/ml, respectively), showing no significant difference between them ($p > 0.05$). Conversely, Group B (Negative Control), which received the toxicant without treatment, exhibited the lowest dopamine level (449 ± 0.001 pg/ml), showing a statistically significant reduction ($p < 0.05$) compared to all other groups. Group C (Standard Control) and Group D (Low-Dose Treated Group) had intermediate dopamine levels (558 ± 0.001 pg/ml and 581 ± 0.004 pg/ml, respectively), with no significant difference between them ($p > 0.05$), but a significant difference when compared with Groups A, B, and E ($p < 0.05$). These findings suggest that the administration of the high-dose treatment was effective in restoring dopamine levels close to normal, comparable to the blank control, while the low dose and standard treatment produced moderate improvement (Table 1).

Table 1: Effect of Zingiber officinale ethanol extract on dopamine (pg/ml) of male wistar albino rats induced with inflammation

GROUPS	Dopamine (pg/ml)
A (Blank Control)	653 ± 0.001^{a}
B (Negative Control)	449 ± 0.001^{b}
C (Standard Control)	558 ± 0.001^{c}
D (Low-Dose Treated Group)	581 ± 0.004^{c}
E (High-Dose Treated Group)	638 ± 0.000^{a}

The values are expressed as (mean ± SEM)

Mean values with different letters as superscript are significantly different (p<0.05)

Serotonine Level

Group A (Blank Control) recorded the highest serotonin concentration (5.21 ± 0.001 µg/L), closely followed by Group E (High-Dose Treated Group) with 5.12 ± 0.003 µg/L. Both groups showed no significant difference between them ($p > 0.05$). This suggests that the high-dose treatment effectively restored serotonin levels to near-normal values. In contrast, Group B (Negative Control), which was exposed to the inflammation without any

treatment, exhibited a significantly reduced serotonin level (1.60 ± 0.001 µg/L), reflecting a significant difference ($p < 0.05$) when compared to all other groups. Group C (Standard Control) and Group D (Low-Dose Treated Group) showed moderate serotonin levels of 3.04 ± 0.001 µg/L and 3.45 ± 0.002 µg/L, respectively, showing no significant difference between them ($p > 0.05$), but a significant difference compared to Groups A, B, and E ($p < 0.05$). Overall, these results imply that the high-dose treatment was the most effective in restoring serotonin levels, while the low-dose and standard treatments showed partial improvements (Table 2).

Table 2: Effect of Zingiber officinale ethanol extract on serotonine (µg/L) of male wistar albino rats induced with inflammation

GROUPS	Serotonine (µg/L)
A (Blank Control)	5.21 ± 0.001^{a}
B (Negative Control)	1.60 ± 0.001^{b}
C (Standard Control)	3.04 ± 0.001^{c}
D (Low-Dose Treated Group)	3.45 ± 0.002^{c}
E (High-Dose Treated Group)	5.12 ± 0.003^{a}

The values are expressed as (mean ± SEM)

Mean values with different letters as superscript are significantly different (p<0.05)

Acetylcholine Level

Group A (Blank Control) recorded the highest acetylcholine concentration (11.21 ± 0.012 mm), closely followed by Group E (High-Dose Treated Group) with a value of 11.08 ± 0.013 mm. Both groups indicated no significant difference between them ($p > 0.05$), suggesting that the high-dose treatment effectively restored acetylcholine levels to near-normal values. In contrast, Group B (Negative Control), which received no treatment after exposure to the toxicant, showed a significantly lower acetylcholine level (5.16 ± 0.031 mm). reflecting a significant reduction in acetylcholine concentration compared to all other groups ($p < 0.05$). Group C (Standard Control) and Group D (Low-Dose Treated Group) showed intermediate values (7.26 ± 0.031 mm and 8.69 ± 0.012 mm, respectively) showing no significant difference between them ($p > 0.05$), but significantly difference when compared to Groups A, B, and E ($p < 0.05$). These findings imply that the high-dose treatment was most effective in reversing the acetylcholine-depleting effects of the toxicant, while the standard and low-dose treatments offered moderate restoration (Table 3).

Table 3: Effect of Zingiber officinale ethanol extract on acetylcholine (mm) of male wistar albino rats induced with inflammation

GROUPS	Acetylcholine (mm)
A (Blank Control)	11.21 ± 0.012^{a}
B (Negative Control)	5.16 ± 0.031^{b}
C (Standard Control)	7.26 ± 0.031^{c}
D (Low-Dose Treated Group)	8.69 ± 0.012^{c}
E (High-Dose Treated Group)	11.08 ± 0.013^{a}

The values are expressed as (mean ± SEM)

Mean values with different letters as superscript are significantly different (p<0.05)

Epinephrine Level

Group A (Blank Control) recorded the highest epinephrine level (10.46 ± 0.003 pg/ml). Group B (Negative Control), which was exposed to inflammation without any treatment, exhibited the lowest epinephrine concentration (3.12 ± 0.004 pg/ml), indicating a significant reduction in epinephrine levels compared to all other groups ($p < 0.05$). Groups C (Standard Control), D (Low-Dose Treated Group), and E (High-Dose Treated Group) showed moderate increases in epinephrine levels (6.72 ± 0.004 pg/ml, 7.03 ± 0.003 pg/ml, and 7.91 ± 0.023 pg/ml, respectively) showing no significant difference among the three treatment groups ($p > 0.05$), although they are significantly different from both the blank and negative control groups ($p < 0.05$). These findings suggest that while none of the treatment groups completely restored epinephrine to normal levels, the high-dose treatment provided the greatest improvement, followed by the low-dose and standard control treatments (Table 4).

Table 4: Effect of Zingiber officinale ethanol extract on epinephrine (pg/ml) of male wistar albino rats induced with inflammation

GROUPS	Epinephrine (pg/ml)
A (Blank Control)	10.46 ± 0.003^{a}
B (Negative Control)	3.12 ± 0.004^{b}
C (Standard Control)	6.72 ± 0.004^{c}
D (Low-Dose Treated Group)	7.03 ± 0.003^{c}
E (High-Dose Treated Group)	7.91 ± 0.023^{c}

The values are expressed as (mean ± SEM)

Mean values with different letters as superscript are significantly different ($p<0.05$)

Discussion, Conclusion, and Recommendations

Discussion

This study evaluated the neuroprotective effects of *Zingiber officinale* ethanol extract on key neurological indices, dopamine, serotonin, acetylcholine, and epinephrine in male Wistar albino rats subjected to inflammation. Inflammation significantly impaired neurological function, evidenced by the marked reduction in all four neurohormones in the negative control group. However, treatment with *Z. officinale*, especially at high doses, restored these indices near normal levels.

The findings aligned with those of Kumari *et al*. (2021), who reported that *Zingiber officinale* extract exhibited significant neuroprotective and anti-inflammatory properties by modulating neurotransmitter activities in lipopolysaccharide-induced neuroinflammation models. Similarly, Sharma and Bhatia (2020) demonstrated that *Z. officinale* extract reversed reductions in dopamine and serotonin concentrations in rats exposed to chronic stress, a condition that mimics the neurochemical imbalances observed during systemic inflammation. These parallels affirm the ability of *Z. officinale* to modulate neurochemical pathways disrupted during inflammatory responses.

In the present study, the high-dose treatment group exhibited dopamine and serotonin levels statistically similar to the blank control, suggesting near-complete neurochemical restoration. This agrees with findings by Ahmad *et al*. (2022), who reported dose-dependent recovery of neurotransmitters with *Z. officinale* administration in rats with chemically induced neurotoxicity. Moreover, acetylcholine and epinephrine levels were also improved significantly in treated groups, especially in the high-dose group, supporting previous reports by Yildiz and Kiziltunc (2019), who found that *Z. officinale* extract preserved cholinergic function and reduced oxidative damage in brain tissues.

The observed increases in neurotransmitter concentrations in the low-dose and standard treatment groups were modest but statistically significant compared to the negative control. This further supports the dose-dependent efficacy of *Z. officinale* in modulating neuroinflammation. Research by Ekpo *et al*. (2020) has similarly shown that suboptimal doses of *Z. officinale* may offer partial protection by activating antioxidant and anti-inflammatory pathways, although higher doses yield superior therapeutic outcomes.

This study showed that the high-dose treatment was most effective in reversing the acetylcholine-depleting effects of the toxicant, while the standard and low-dose treatments offered moderate restoration. This finding was consistent with a study by Olusanya *et al*. (2022), which demonstrated that *Z. officinale* infusion, in combination with *Vernonia amygdalina*, significantly improved brain redox status and reduced acetylcholinesterase (AChE) activity in Wistar rats exposed to toxicant stress, thereby enhancing acetylcholine availability. Similarly, Adebayo and Mensah (2024) reported that aqueous *Z. officinale* extract mitigated dichlorvos-induced oxidative stress and improved cholinergic function in a dose-dependent manner, with higher doses showing greater efficacy.

The present study suggests that while none of the treatment groups completely restored epinephrine to normal levels, the high-dose treatment provided the greatest improvement, followed by the low-dose and standard control treatments, which aligned with the study by Al-Yahya and colleagues (2019) who showed that rats induced with inflammation exhibited marked decreases in epinephrine across multiple brain regions; subsequent administration of *Z. officinale* extract resulted in significant increases in epinephrine. Though not necessarily back to baseline.

Conclusion

This study has inferred that ethanol extract of *Zingiber officinale* exerts significant neuroprotective effects on male Wistar albino rats exposed to inflammatory conditions. The extract, particularly at high doses, effectively restored dopamine, serotonin, acetylcholine, and epinephrine levels to near-normal concentrations. These findings highlight the therapeutic potential of *Z. officinale* in managing inflammation-induced neurochemical disturbances.

Recommendations

We recommend that *Zingiber officinale* extract should be produced in the pharmaceutical industries as tablets which will help cure nausea, digestive issues etc. and further research should explore the long-term safety and efficacy of *Zingiber officinale* in chronic neuroinflammatory conditions.

References

1. Adebayo, T. and Mensah, K. (2024). Ginger (*Zingiber officinale*) attenuates dichlorvos-induced neurotoxicity in

Wistar rats: Biochemical and neurotransmitter outcomes. *Basic and Applied Zoology*, 75: 15.

2. Ahmad, F., Khan, A., Rehman, A. and Mahmood, Z. (2022). Protective effects of *Zingiber officinale* against neurotoxicity: A biochemical and histological evaluation. *Journal of Herbal Medicine*, 33: 100570.
3. Akinyemi, A. J., Adebiyi, A. O. and Oguntibeju, O. O. (2021). Natural products and their derivatives with anti-neuroinflammatory potential in the treatment of neurodegenerative diseases: A review of recent literature. *Oxidative Medicine and Cellular Longevity*, 2021: 9914861.
4. Almutairi, M. M., Gong, C., Xu, Y., Gans, J. H., Alhazmi, H. A. and Yaseen, A. (2021). Inflammatory and oxidative stress markers in neurodegenerative diseases: A focus on Alzheimer's disease and Parkinson's disease. *Journal of Inflammation Research*, 14: 875–886.
5. Al-Yahya, M. A., Hassan, A. M. and Abdel-Rahman, H. A. (2019). Neuroprotective evaluation of extract of ginger (*Zingiber officinale*) root in monosodium glutamate–induced toxicity in different brain areas of male albino rats. *Indian Journal of Pharmacology*, 51(2): 102–110.
6. Anwar, M. M., El-Shahat, A. E. and Sayed, A. A. (2020). Ginger extract protects against neuroinflammation and brain damage induced by lipopolysaccharide in rats. *Journal of Functional Foods*, *65*: 103736.
7. DiSabato, D. J., Quan, N. and Godbout, J. P. (2016). Neuroinflammation: The devil is in the details. *Journal of Neurochemistry*, 139(2): 136–153.
8. Ebert, S. E., Jensen, P., Ozenne, B., Armand, S., Svarer, C. and Stenbaek, D. S., (2019). Molecular imaging of neuroinflammation in patients after mild traumatic brain injury: a longitudinal 123I-CLINDE SPECT study. *European Journal of Neurology*, 26(8): 1054–1061.
9. Ekpo, K. E., Edet, O. E. and Udo, A. O. (2020). Antioxidant and anti-inflammatory potential of *Zingiber officinale* in mitigating neurotoxic effects in rats. *Journal of Evidence-Based Integrative Medicine*, 25: 251.
10. Ghasemzadeh, A., Jaafar, H. Z. and Rahmat, A. (2022). Ginger and its bioactive compounds: Role in prevention and treatment of inflammation and neurodegenerative disorders. *Journal of Functional Foods*, 89: 104942.
11. Hasanpour-Dehkordi, A., Solati, K. and Salehi-Tali, S. (2019). The effect of ginger on inflammatory diseases and its mechanisms: A literature review. *Complementary Therapies in Medicine*, 45: 43–49.
12. Johnson, D. E., O'Connell, T. M. and Zhang, S. (2021). Metabolic reprogramming in inflammatory diseases. *Annual Review of Pathology: Mechanisms of Disease*, 16: 311–338.
13. Kaur, T., Sharma, S. and Mantha, A. K. (2020). Phytochemicals targeting epigenetic regulators: Novel avenues for neuroprotective effects in neurodegenerative disorders. *Frontiers in Pharmacology*, 11: 590057.
14. Kumari, R., Yadav, S. and Verma, A. (2021). Neuroprotective role of ginger extract in LPS-induced neuroinflammation: Behavioral and biochemical evidence. *Neurochemical Research*, 46(5):1139–1149.
15. Olusanya, O., Adewale, O. and Nwankwo, C. (2022). *Zingiber officinale* and *Vernonia amygdalina* infusions improve brain redox status and acetylcholinesterase parameters in Wistar rats. *Toxicology Reports*, 9: 1–10.
16. Pohl, F. and Kong Thoo Lin, P. (2018). The potential use of plant natural products and plant extracts with antioxidant properties for the prevention/treatment of neurodegenerative diseases: In vitro, in vivo and clinical trials. *Molecules*, 23(12): 3283.
17. Saha, P., Bose, R. and Ray, S. (2021). Neuroprotective effects of *Zingiber officinale* extract in inflammation-induced cognitive dysfunction: A study in animal models. *Journal of Ethnopharmacology*, 266: 113406.
18. Seo, D.-Y., Heo, J.-W., Ko, J. R. and Kwak, H.-B. (2019). Exercise and Neuroinflammation in Health and Disease. *International Neurourology Journal*, 23(S2): S82–S92.
19. Sharma, N. and Bhatia, P. (2020). Ginger modulates stress-induced neurochemical alterations in rats. *Biomedicine & Pharmacotherapy*, 125: 109925.
20. World Health Organization. (2022). *Neurological disorders: Public health challenges*.
21. Yildiz, G. and Kiziltunc, A. (2019). Ginger extract preserves cholinergic function and attenuates oxidative stress in neurodegenerative models. *Journal of Food Biochemistry*, 43(12): e13052.
22. Zhou, H., Deng, Y., Xie, Q., Lin, Y., Gong, Y. and Deng, H. (2020). Anti-inflammatory and antioxidant activities of gingerol and its derivatives: A review. *Frontiers in Pharmacology*, 11: 572977.
23. Zhou, Y., Peng, Z., Seven, E. S. and Leblanc, R. M. (2020). Crossing the blood-brain barrier with nanoparticles. *Journal of Controlled Release*, 328: 723–737.
24. Piserchia, O. (2025). Phishing Website Detection through Machine Learning Algorithms: A Comparative Analysis. IRASS Journal of Multidisciplinary Studies, 2(12), 11-29.
25. Nwashili, O. G., Abiodun, K. D., Amosu, O. & Oghoghorie, O. (2025). The Product Manager's Role in AI Security: Preventing Data Leaks and Model Manipulation in Consumer Applications. IRASS Journal of Multidisciplinary Studies, 2(12), 30-35.
26. Lopez, A. R., Oforiwaa, P., Achiampong, A. A., Adoba, P. & Acquah, R. (2025). PREVALENCE AND FACTORS ASSOCIATED WITH HEPATORENAL SYNDROME AMONG PATIENTS ATTENDING MEDYLIFE HEALTHCARE HOSPITAL, GHANA. IRASS Journal of Multidisciplinary Studies, 2(12),36-50.