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A Study on Anti-Inflammatory Activity of Moringa Oleifera Leaf Extract using Carrageenan-Induced Paw Edema and Chronic Inflammation Models in Swiss Albino Mice

Sanjay Kumar Srivastva[1], Anjana Devi[2], Gulshan Kumar[3]
[1,2] Department of Pharmacy, Career Point University, Hamirpur, Himachal Pradesh-176041, India
[3]Division of Botany, Department of Bio-Sciences, Career Point University, Hamirpur, Himachal Pradesh-176041, India
[3]Corresponding Author: **Dr. Gulshan Kumar**
[3]Corresponding Email ID: Gulshan.botany@cpuh.edu.in

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ABSTRACT

Inflammation is a complex biological response associated with numerous acute and chronic disorders, including neurodegenerative diseases. The present study investigates the anti-inflammatory potential of *Moringa oleifera* leaf extract using both acute and chronic experimental models. The evaluation was carried out using carrageenan-induced paw edema (acute inflammation), cotton pellet-induced granuloma, and formaldehyde-induced paw edema (chronic inflammation) in Swiss albino mice. Animals were treated with graded doses of *Moringa oleifera* extract (100, 200, 300, and 400 mg/kg), while standard drugs such as diclofenac sodium and indomethacin served as positive controls. Paw edema was measured using a digital plethysmometer, and percentage inhibition of inflammation was calculated. Biochemical analyses included the estimation of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6), nitric oxide levels, prostaglandin E_2, and oxidative stress markers such as malondialdehyde, superoxide dismutase, catalase, and glutathione. Additionally, molecular markers associated with neuroinflammation, including NF-κB, COX-2, and iNOS expression, were assessed. The extract demonstrated significant dose-dependent inhibition of inflammation across all models, comparable to standard drugs.The findings suggest that *Moringa oleifera* possesses potent anti-inflammatory and antioxidant properties, supporting its potential therapeutic application in inflammatory and neurodegenerative disorders

Keywords: Anti-inflammatory activity; Carrageenan-induced edema; Cytokines; *Moringa oleifera*; Oxidative stress

INTRODUCTION

Inflammation is a fundamental physiological response to injury, infection, or tissue damage, involving a cascade of biochemical and cellular events aimed at restoring homeostasis (Medzhitov, 2008). While acute inflammation is protective, chronic inflammation is implicated in the pathogenesis of several diseases, including arthritis, cardiovascular disorders, diabetes, and neurodegenerative conditions such as Alzheimer's disease (Nathan and Ding, 2010; Furman et al., 2019). The inflammatory response is mediated by a network of cytokines, chemokines, prostaglandins, and reactive oxygen species (ROS), which contribute to tissue damage when dysregulated (Mittal et al., 2014). Key mediators such as tumor necrosis factor-alpha (TNF-α), interleukins (IL-1β, IL-6), nitric oxide (NO), and prostaglandin E_2 (PGE_2) play pivotal roles in amplifying inflammation (Dinarello, 2011). In addition, oxidative stress resulting from excessive ROS production further exacerbates inflammatory responses and contributes to cellular damage (Reuter et al., 2010). Non-steroidal anti-inflammatory drugs (NSAIDs), including diclofenac and indomethacin, are commonly used to manage inflammation but are associated

with adverse effects such as gastrointestinal toxicity and renal complications (Wallace, 2008). This has led to increasing interest in plant-based therapeutics with fewer side effects and multiple mechanisms of action (Pan et al., 2014). *Moringa oleifera*, commonly known as the drumstick tree, is a medicinal plant widely used in traditional medicine for its diverse pharmacological properties, including anti-inflammatory, antioxidant, antimicrobial, and neuroprotective activities (Anwar et al., 2007; Leone et al., 2015). The leaves are rich in bioactive compounds such as flavonoids, phenolic acids, vitamins, and isothiocyanates, which contribute to its therapeutic potential (Sreelatha and Padma, 2009; Vergara-Jimenez et al., 2017). Previous studies have demonstrated that *Moringa oleifera* extracts can inhibit inflammatory mediators and reduce oxidative stress in various experimental models (Mahajan and Mehta, 2010; Sudha et al., 2010). However, comprehensive evaluation using both acute and chronic inflammation models, along with molecular and biochemical markers, remains limited. The carrageenan-induced paw edema model is widely used to study acute inflammation due to its biphasic response involving histamine, serotonin, and prostaglandins (Winter et al., 1962). Chronic models such as cotton pellet granuloma and formaldehyde-induced inflammation provide insights into proliferative and sustained inflammatory processes (Swingle and Shideman, 1972). Therefore, the present study aims to evaluate the anti-inflammatory efficacy of *Moringa oleifera* leaf extract using multiple experimental models and to investigate its effects on inflammatory mediators, oxidative stress parameters, and neuroinflammatory biomarkers.

MATERIALS AND METHODS

Experimental Design: The present study was designed to evaluate the anti-inflammatory potential of *Moringa oleifera* leaf extract using both acute and chronic experimental models. Acute inflammation was assessed using the carrageenan-induced paw edema model, while chronic inflammation was evaluated through cotton pellet-induced granuloma and formaldehyde-induced paw edema models. In addition, biochemical and molecular parameters associated with inflammation, oxidative stress, and neuroinflammation were analyzed to provide mechanistic insights (Winter et al., 1962; Swingle and Shideman, 1972).

Experimental Animals: Healthy Swiss albino mice (25–30 g) of either sex were procured and used in the study. Animals were housed in polypropylene cages under standard laboratory conditions (temperature 22 ± 2°C, relative humidity 55 ± 5%, and 12 h light/dark cycle). The animals were acclimatized for one week prior to experimentation to minimize stress-induced variability. Standard pellet diet and water were provided ad libitum, except for a 12-hour fasting period prior to drug administration, during which water remained available (OECD, 2001).

Ethical Approval and Regulatory Compliance: All experimental procedures were conducted following approval from the Institutional Animal Ethics Committee (IAEC) and in accordance with CPCSEA guidelines, Government of India. The study adhered to ARRIVE guidelines and the principles of the 3Rs (Replacement, Reduction, and Refinement) to ensure ethical use of laboratory animals (Kilkenny et al., 2010; CPCSEA, 2018).

Chemicals and Reagents: Carrageenan (λ-type), formaldehyde, diclofenac sodium, and indomethacin were obtained from standard laboratory suppliers. Pro-inflammatory cytokine ELISA kits (TNF-α, IL-1β, IL-6, CRP) and biochemical assay kits were procured from certified manufacturers. Griess reagent was used for nitric oxide estimation. All chemicals and reagents used in the study were of analytical grade.

Preparation of *Moringa oleifera* Leaf Extract: Fresh leaves of *Moringa oleifera* were collected, shade-dried, and powdered. The powdered material was subjected to Soxhlet extraction using ethanol as a solvent. The extract was concentrated under reduced pressure using a rotary evaporator and stored at 4°C until use. For administration, the extract was suspended in 0.5% carboxymethyl cellulose (CMC) to ensure uniform dosing. Fresh suspensions were prepared daily (Anwar et al., 2007; Sreelatha and Padma, 2009).

Dose Selection and Treatment Regimen: Based on previous pharmacological studies, four dose levels of *Moringa oleifera* extract (100, 200, 300, and 400 mg/kg body weight) were selected. The extract and standard drugs (diclofenac sodium: 10 mg/kg; indomethacin: 10 mg/kg) were administered orally. A vehicle control group receiving 0.5% CMC was included for comparison (Mahajan and Mehta, 2010).

Preparation of Carrageenan Solution: A 1% w/v solution of λ-carrageenan was prepared in sterile normal saline. The solution was heated to approximately 60°C with continuous stirring until complete dissolution and allowed to cool to room

temperature before use. The solution was freshly prepared on the day of the experiment to maintain stability (Winter et al., 1962).

Carrageenan-Induced Paw Edema Model (Acute Inflammation): Acute inflammation was induced using the carrageenan-induced paw edema model as described by Winter et al. (1962). Animals were divided into five groups (n = 6 per group):

- Group I: Normal control (saline)
- Group II: Carrageenan control
- Group III: Diclofenac sodium (10 mg/kg)
- Group IV: *Moringa oleifera* extract (100 mg/kg)
- Group V: *Moringa oleifera* extract (300 mg/kg)

One hour after oral administration of treatments, 0.05 mL of 1% carrageenan solution was injected into the subplantar region of the right hind paw. Paw volume was measured at 0, 1, 2, 3, 4, and 5 hours using a digital plethysmometer.

Edema volume was calculated as:

$\Delta V = Vt - V0$

Percentage inhibition of edema was calculated using:

$\% \text{ Inhibition} = [(\Delta V\text{control} - \Delta V\text{treated}) / \Delta V\text{control}] \times 100$

This model reflects biphasic inflammation involving histamine, serotonin (early phase), and prostaglandins (late phase) (Di Rosa et al., 1971).

Cotton Pellet-Induced Granuloma Model (Chronic Inflammation): Sterile cotton pellets (10 ± 1 mg) were implanted subcutaneously in the dorsal region of anesthetized mice. Animals were treated daily for 7 days with extract or standard drug. On day 8, pellets were removed, dried at 60°C, and weighed. The increase in dry weight of the pellet indicated granuloma formation, reflecting chronic inflammation (Swingle and Shideman, 1972).

Formaldehyde-Induced Paw Edema Model: Chronic inflammation was also induced by injecting 0.1 mL of 2% formaldehyde into the subplantar region on days 1 and 3. Paw thickness was measured periodically, and treatment was administered daily for 10 days. This model mimics arthritis-like inflammatory conditions (Brownlee, 1950).

Biochemical Analysis-Cytokine Estimation

Levels of TNF-α, IL-1β, IL-6, and CRP were quantified using ELISA kits following manufacturer protocols (Dinarello, 2011).

Nitric Oxide Estimation: Nitric oxide levels were determined by measuring nitrite concentration using the Griess reagent method (Green et al., 1982).

Prostaglandin E_2 (PGE_2): PGE_2 levels were estimated using ELISA or radioimmunoassay methods (Funk, 2001).

Oxidative Stress Markers: Malondialdehyde (MDA) levels were measured using the TBARS assay, while antioxidant enzymes such as superoxide dismutase (SOD), catalase, and reduced glutathione (GSH) were estimated using standard biochemical methods (Ohkawa et al., 1979; Aebi, 1984).

Myeloperoxidase (MPO) Activity: MPO activity was measured spectrophotometrically as an indicator of neutrophil infiltration into inflamed tissues (Bradley et al., 1982).

Brain Tissue Analysis: Brain tissues (hippocampus and cortex) were isolated after perfusion with saline. Protein expression of NF-κB, COX-2, and iNOS was analyzed using Western blot techniques. Markers of neurodegeneration, including amyloid-beta and tau phosphorylation, were also assessed (Heneka et al., 2015).

Statistical Analysis: Data were expressed as mean ± standard error of the mean (SEM). Statistical analysis was performed using one-way ANOVA followed by Bonferroni's post hoc test. A p-value < 0.05 was considered statistically significant (Motulsky, 2014).

RESULTS

Effect on Carrageenan-Induced Paw Edema (Acute Inflammation): Administration of *Moringa oleifera* leaf extract produced a dose-dependent reduction in paw edema volume compared to the carrageenan control group. The maximum edema was observed at 3 h in the carrageenan control, confirming the typical biphasic inflammatory response.

Table 1. Effect of *Moringa oleifera* on Paw Edema Volume (mL)

Group	0 h	1 h	2 h	3 h	4 h	5 h
Normal Control	0.21±0.01	0.22±0.01	0.22±0.01	0.23±0.01	0.22±0.01	0.22±0.01
Carrageenan Control	0.22±0.01	0.45±0.02	0.68±0.03	0.82±0.04	0.76±0.03	0.65±0.02
Diclofenac (10 mg/kg)	0.21±0.01	0.32±0.02	0.42±0.02	0.46±0.02	0.40±0.02	0.35±0.02
MO Extract (100 mg/kg)	0.22±0.01	0.38±0.02	0.55±0.03	0.62±0.03	0.58±0.02	0.50±0.02
MO Extract (300 mg/kg)	0.21±0.01	0.34±0.02	0.47±0.02	0.52±0.02	0.46±0.02	0.40±0.02

Percentage Inhibition at 3 h (Peak Edema)

Group	% Inhibition
Diclofenac	43.9%
MO (100 mg/kg)	24.4%
MO (300 mg/kg)	36.6%

Statistical analysis revealed that both doses of *Moringa oleifera* extract significantly reduced edema ($p < 0.05$), with the higher dose showing comparable efficacy to diclofenac.

Effect on Cotton Pellet-Induced Granuloma (Chronic Inflammation)

Group	Dry Weight (mg)	% Inhibition
Control	42.5 ± 2.1	—
Diclofenac	25.2 ± 1.8	40.7%
MO (100 mg/kg)	34.8 ± 2.0	18.1%
MO (300 mg/kg)	28.6 ± 1.7	32.7%

Moringa oleifera significantly reduced granuloma formation, indicating suppression of proliferative inflammation.

Effect on Formaldehyde-Induced Paw Edema

Moringa oleifera extract markedly reduced paw thickness over the treatment period, particularly at higher doses, demonstrating effectiveness in chronic inflammatory conditions.

Effect on Pro-Inflammatory Cytokines

Parameter	Control	Carrageenan	Diclofenac	MO (300 mg/kg)
TNF-α (pg/mL)	28 ± 3	82 ± 5	40 ± 4	48 ± 4
IL-1β (pg/mL)	18 ± 2	65 ± 4	30 ± 3	36 ± 3
IL-6 (pg/mL)	22 ± 3	75 ± 5	35 ± 4	42 ± 3

The extract significantly reduced cytokine levels ($p < 0.05$), confirming anti-inflammatory activity.

Effect on Oxidative Stress Markers

Parameter	Control	Carrageenan	Diclofenac	MO (300 mg/kg)
MDA (nmol/mg protein)	2.1	5.8	3.0	3.5
SOD (U/mg protein)	8.5	4.2	7.6	7.0
Catalase (U/mg protein)	6.8	3.1	5.9	5.4
GSH (µmol/g tissue)	7.2	3.8	6.5	6.0

The extract significantly restored antioxidant levels and reduced lipid peroxidation.

MPO and Nitric Oxide Levels

MPO activity and nitric oxide levels were significantly elevated in the carrageenan group and were reduced by *Moringa oleifera*, indicating decreased neutrophil infiltration and nitrosative stress.

Neuroinflammatory Markers

Western blot analysis revealed downregulation of NF-κB, COX-2, and iNOS expression in extract-treated groups, suggesting neuroprotective potential.

DISCUSSION

The present study demonstrates that *Moringa oleifera* leaf extract exerts significant anti-inflammatory effects in both acute and chronic experimental models. The carrageenan-induced paw edema model is widely recognized for evaluating acute inflammation, characterized by an early phase mediated by histamine and serotonin and a late phase involving prostaglandins (Winter et al., 1962; Di Rosa et al., 1971). The observed reduction in paw edema indicates that the extract effectively modulates both phases of inflammation. The inhibition of edema, particularly during the late phase, suggests suppression of prostaglandin synthesis, which aligns with the mechanism of action of standard NSAIDs such as diclofenac (Funk, 2001). The comparable efficacy of the higher dose of *Moringa oleifera* to diclofenac highlights its therapeutic potential. In the cotton pellet granuloma model, the extract significantly reduced granuloma formation, indicating its effectiveness in inhibiting proliferative and chronic inflammatory responses (Swingle and Shideman, 1972). Similarly, the formaldehyde-induced paw edema model further confirmed its role in mitigating chronic inflammation, which is relevant to conditions such as arthritis. The reduction in pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) observed in this study supports the immunomodulatory role of *Moringa oleifera*. These cytokines are key mediators in inflammatory pathways and are known to amplify tissue damage when overexpressed (Dinarello, 2011). The suppression of these markers suggests that the extract interferes with cytokine signaling pathways. Oxidative stress plays a critical role in the progression of inflammation, and the significant decrease in MDA levels along with restoration of antioxidant enzymes (SOD, catalase, GSH) indicates strong antioxidant activity of the extract (Mittal et al., 2014). This dual anti-inflammatory and antioxidant effect enhances its therapeutic relevance.

Furthermore, the observed reduction in MPO activity and nitric oxide levels indicates decreased neutrophil infiltration and nitrosative stress, which are critical contributors to inflammatory damage (Green et al., 1982; Bradley et al., 1982). At the molecular level, downregulation of NF-κB, COX-2, and iNOS expression suggests that *Moringa oleifera* modulates key signaling pathways involved in inflammation and neurodegeneration (Heneka et al., 2015). NF-κB is a central regulator of inflammatory gene expression, and its inhibition is associated with reduced production of inflammatory mediators. The pharmacological effects observed in this study can be attributed to the presence of bioactive compounds such as flavonoids, phenolics, and isothiocyanates in *Moringa oleifera*, which are known to exhibit anti-inflammatory and antioxidant properties (Anwar et al., 2007; Sreelatha and Padma, 2009). Overall, the findings provide strong evidence that *Moringa oleifera* leaf extract possesses significant anti-inflammatory activity through multiple mechanisms, including inhibition of inflammatory mediators, reduction of oxidative stress, and modulation of molecular signaling pathways (Figure-1).

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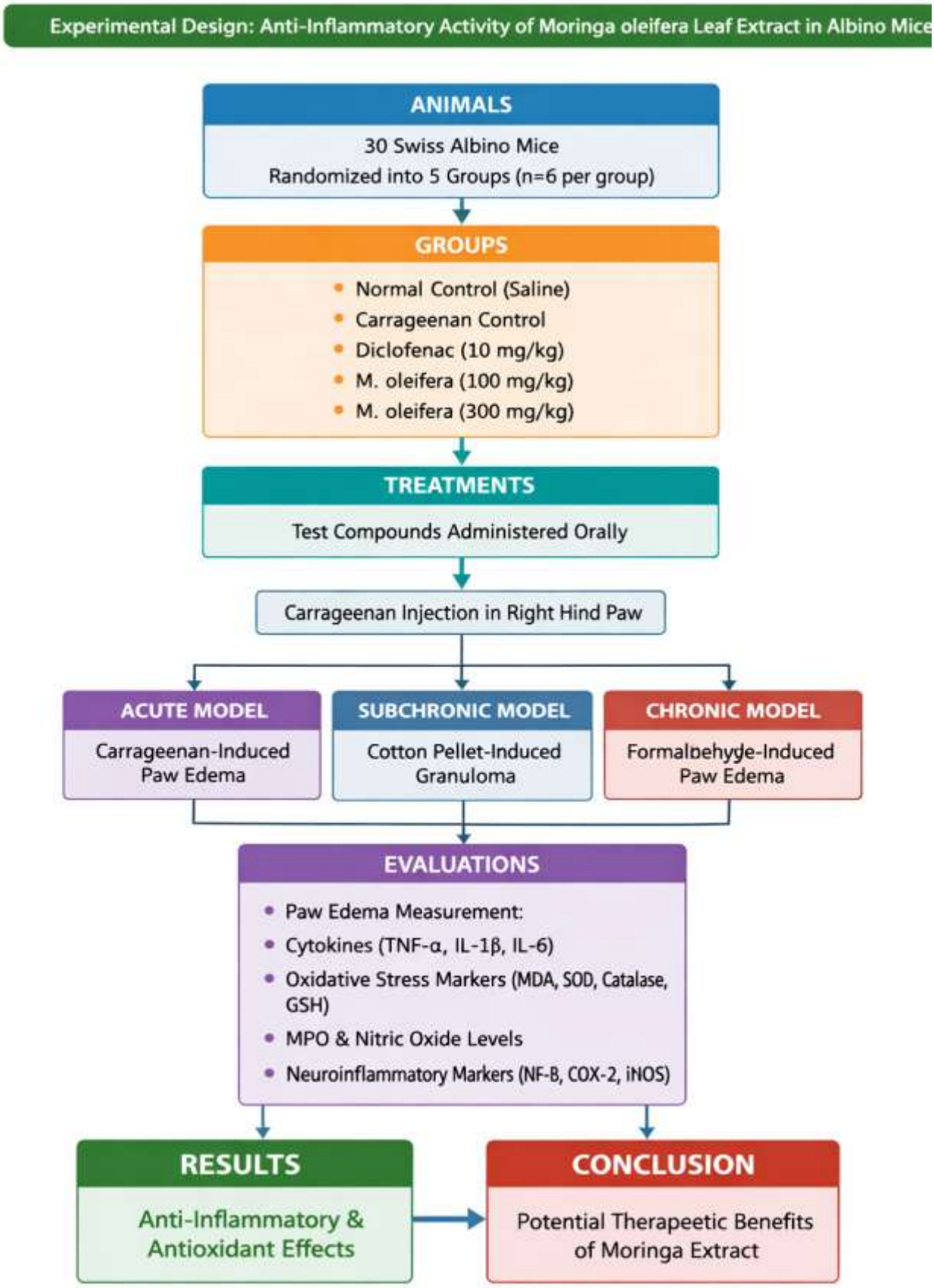

CONCLUSION

The present investigation demonstrates that *Moringa oleifera* leaf extract possesses significant and dose-dependent anti-inflammatory activity across both acute and chronic experimental models. In the carrageenan-induced paw edema model, the extract effectively attenuated edema formation, particularly during the prostaglandin-mediated late phase, indicating inhibition of key inflammatory mediators. Its efficacy in the cotton pellet-induced granuloma and formaldehyde-induced paw edema models further confirms its role in suppressing chronic and proliferative phases of inflammation. The extract also markedly reduced levels of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6, while restoring antioxidant defenses including SOD, catalase, and glutathione, alongside reducing lipid peroxidation. These findings highlight a dual mechanism involving both anti-inflammatory and antioxidant pathways. Moreover, downregulation of molecular markers such as NF-κB, COX-2, and iNOS suggests that the extract modulates key signaling cascades associated with inflammation and neurodegeneration. Collectively, the results provide strong experimental evidence supporting the therapeutic potential of *Moringa oleifera* as a natural, multi-target anti-inflammatory agent with possible neuroprotective benefits. Based on the findings of this study, *Moringa oleifera* leaf extract holds promise for development as a safer alternative or adjunct to conventional non-steroidal anti-inflammatory drugs. However, further studies are required to isolate and characterize the specific bioactive compounds responsible for the observed pharmacological effects. Detailed pharmacokinetic and toxicological evaluations should also be conducted to establish safety profiles for long-term use. Future research should focus on elucidating precise molecular mechanisms through advanced techniques such as gene expression profiling and proteomics. Additionally, well-designed clinical trials are essential to validate the efficacy and safety of *Moringa oleifera* in human populations. Exploration of its role in managing neuroinflammatory and neurodegenerative disorders could open new therapeutic avenues. Standardization of extract preparation and dosage regimens is also recommended to ensure reproducibility and facilitate its translation into clinical applications.

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