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Arabinogalactan's Neuroprotective Effects in Parkinson's Disease: An Integrative In Vivo Study

S. Afshin Shams[1] , A. Sravani[2*,] M. Praisy Gladys[3]
([1,2,3] Santhiram College of Pharmacy, Nandyal, Andhra Pradesh.)
Corresponding Author
Ms. A.Sravani,
Department of Pharmacology, Santhiram College of Pharmacy,
Nandyal, Kurnool, Andhra Pradesh, India
Mail id: aavulasravani26@gmail.com

Abstract

Background: Parkinson's disease is a progressive neurodegenerative disorder marked by the loss of dopaminergic neurons in the substantia nigra, resulting in motor impairments such as tremor, rigidity, and bradykinesia. Oxidative stress, mitochondrial damage, and neuroinflammatory processes are key factors involved in its development. Natural polysaccharides have attracted attention due to their antioxidant and neuroprotective properties. Arabinogalactan, a plant-derived polysaccharide, has demonstrated notable antioxidant and anti-inflammatory activities that may help protect against neurodegenerative damage.

Objective: This study aimed to investigate the neuroprotective effect of arabinogalactan in an experimental model of Parkinson's disease.

Methods: Parkinsonian features were induced in Wistar rats using rotenone. Animals were treated with arabinogalactan at doses of 200 mg/kg and 400 mg/kg (p.o.), while a standard group received L-Dopa and carbidopa. Behavioral assessments, including tests for motor coordination and catalepsy, were conducted. Biochemical analyses were also performed to evaluate markers of oxidative stress and neuroinflammation.

Results: Treatment with arabinogalactan significantly improved motor performance and decreased cataleptic responses compared with the disease control group. It also reduced lipid peroxidation, enhanced antioxidant defense mechanisms, and suppressed neuroinflammatory activity, thereby contributing to the protection of dopaminergic neurons.

Conclusion: The results suggest that arabinogalactan provides significant neuroprotective effects against rotenone-induced Parkinsonism and may serve as a promising natural therapeutic agent for the management of Parkinson's disease.

Keywords: Parkinson's disease, Arabinogalactan, Neuroprotection, Dopaminergic neurons, In vivo

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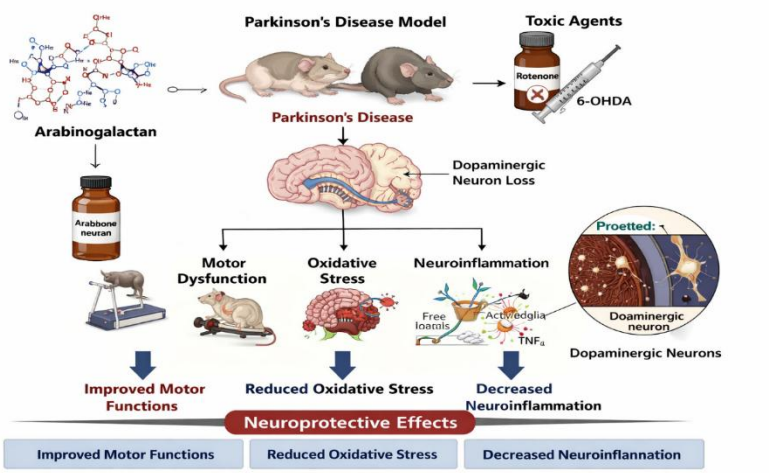


1. Introduction

Parkinson's disease (PD) is the second most common neurodegenerative disorder, affecting roughly 1–2% of individuals over the age of 65. The disease is primarily marked by progressive motor deficits such as tremors, rigidity, slowed movement (bradykinesia), and impaired posture, which arise from the selective loss of dopaminergic neurons in the substantia nigra pars compacta (Ebersbach G, et al., 2010). The development of PD is driven by multiple interconnected mechanisms, including oxidative stress, mitochondrial dysfunction, neuroinflammation, and pathological aggregation of α-synuclein, all of which contribute to gradual neuronal loss and declining motor function.

Currently available treatments, including L-Dopa and dopamine receptor agonists, mainly address symptoms but do not alter the course of the disease. This limitation highlights the pressing need for novel therapeutic approaches that target the underlying pathological processes, particularly oxidative damage and inflammatory responses in the brain.

Natural bioactive compounds, especially plant-derived polysaccharides, have emerged as promising neuroprotective agents due to their antioxidant, anti-inflammatory, and immunomodulatory properties. Arabinogalactan (AG), a naturally occurring polysaccharide, has been shown in preclinical studies to protect neurons by reducing oxidative stress and modulating inflammatory pathways (Wang X, et al., 2019). Through these mechanisms, AG may help preserve dopaminergic neurons and maintain neural function, making it a compelling candidate for intervention in PD.

Based on this evidence, the current study was designed to explore the neuroprotective effects of arabinogalactan in an in vivo model of Parkinson's disease. The investigation focuses on its impact on motor behavior, oxidative stress, neuroinflammatory responses, and the integrity of dopaminergic neurons, aiming to provide mechanistic insight and assess its therapeutic potential.

2. Materials and Methods

Fig no. 1 Arabinogalactan

2.1 Experimental Animals

150–200g sex-neutral experimental rodents were acquired from Vyas Lab in Hyderabad. The animals were kept in cages made of polypropylene, with a controlled ambient temperature of 240 degrees Celsius and a 12-hour light/dark cycle. The experiment animals were randomly assigned to five groups (n = 6) after a week of acclimatization. The Santhiram College of Pharmacy's Institutional Animal Ethical Committee (IAEC) gave its approval to the experimental protocol.(1519/PO/Re/S/11/CPCSEA/2025/008)

As per the current criteria for the care of laboratory animals and the ethical guidelines for the examination of experimental pain in conscious animals, all the studies were conducted in the morning. (Zimmerman, 1983).

Table No. 1 Treatment schedule - Evaluation of Anti Parkinson's activity of Arabinogalactan

S.No	Group	No.of Animals	Treatment	Treatment period (Days)
1	Normal	6	Vehicle (1% CMC, p.o.)	14
2	Control	6	Rotenone (10mg/kg,i p.)	14
3	Standard	6	Dopa & Carbidopa (100mg+25mg/kg p.o) + Rotenone (10mg/kg, p.o.)	14
4	Test-1	6	binogalactan (200 mg/kg, p.o) + Rotenone (10mg/kg, p.o.)	14
5	Test-2	6	binogalactan (400 mg/kg, p.o) + Rotenone (10mg/kg, p.o.)	14

Behavioural Evaluation

Behavioural assessments were performed to determine the extent of Parkinsonian manifestations and to evaluate the potential neuroprotective effects of the treatment in experimental animals. The behavioral parameters analyzed included cataleptic behavior using the block method and metal bar test, and locomotor activity assessed through the open field test.

Catalepsy Assessment

Catalepsy is widely recognized as a reliable behavioral parameter for evaluating dopaminergic dysfunction and motor rigidity, which are characteristic features of experimental models of Parkinson's disease. In the present study, cataleptic behavior was assessed using two established methods: the block method and the metal bar test.

Block Method

The block method was employed to assess the ability of animals to correct an externally imposed abnormal posture. For this test, the forepaws of each rat were gently placed on a wooden block approximately 3 cm in height, while the hind paws remained on the surface of the platform. The time required for the animal to withdraw at least one forepaw from the block was recorded as the cataleptic latency.

To prevent undue stress to the animals, a maximum cut-off time of 180 seconds was implemented. An increase in cataleptic latency was considered indicative of enhanced motor rigidity and dopaminergic impairment, whereas a reduction in latency time suggested improvement in motor coordination and possible neuroprotective effects of the administered treatment.

Metal Bar Test

The metal bar test was conducted to further evaluate the degree of muscle rigidity and cataleptic response in the experimental animals. In this procedure, the forelimbs of the rat were placed on a horizontal metal bar positioned approximately 9 cm above the base surface. The duration for which the animal maintained this imposed posture was recorded as the cataleptic response time, defined as the time taken to remove either one or both forepaws from the bar.

A cut-off time of 180 seconds was applied for this test. Prolonged latency indicated greater muscular rigidity and severity of Parkinsonian symptoms, whereas a decrease in response time reflected improvement in motor function following treatment.

Open Field Test (Locomotor Activity)

The open field test was performed to evaluate spontaneous locomotor activity and exploratory behavior, which are commonly impaired in Parkinsonian conditions due to degeneration of dopaminergic neurons.

Apparatus

The test was carried out in an open field apparatus consisting of a square arena divided into equal segments, allowing quantitative assessment of locomotor and exploratory activities.

Procedure

Each rat was individually placed in the center of the open field arena and allowed to explore the apparatus freely for a period of 5 minutes. During this observation period, the behavioral responses of the animals were systematically monitored and recorded.

Behavioural Parameters

The following parameters were evaluated during the experiment:

- Ambulation: Number of grid squares crossed by the animal.
- Rearing: Frequency of standing on the hind limbs.
- Grooming: Occurrence of self-cleaning behavior.

A reduction in locomotor and exploratory activities was considered indicative of motor impairment associated with Parkinsonian pathology. Conversely, an increase in these

parameters suggested improvement in motor function and indicated a potential protective effect of the treatment.

Physiological Parameter

Brain Weight Measurement

Brain weight was determined as a physiological indicator to assess neurodegeneration and potential brain atrophy in experimental animals. At the end of the study, the animals were sacrificed following standard ethical guidelines, and the brain was carefully removed from the skull. After gently clearing any adhering tissues and blood, the whole brain was weighed immediately using a digital analytical balance.

A reduction in brain weight was interpreted as evidence of neuronal loss and neurodegenerative changes, whereas maintenance of normal brain weight indicated a possible neuroprotective effect of the treatment.

Histopathological Analysis

Histopathological studies were carried out to examine cellular and structural changes in brain tissue, particularly in the substantia nigra, the region most affected in Parkinson's disease.

After sacrifice, brain tissues were isolated and fixed in 10% neutral buffered formalin. The samples were then processed through routine histological procedures, including dehydration, clearing, and paraffin embedding. Thin sections of 4–5 µm thickness were prepared using a microtome and mounted on glass slides.

The sections were stained with hematoxylin and eosin (H&E) and observed under a light microscope to evaluate neuronal integrity and pathological alterations in the substantia nigra. Special attention was given to identifying neuronal loss, cellular degeneration, and structural abnormalities, which are typical features of Parkinsonian neurodegeneration. The degree of neuronal damage and the protective effect of the treatment were assessed by comparing the findings across the experimental groups.

3. Results:

3.1 Behavioral Outcomes

3.1.1 Effect of Arabinogalactan on Catalepsy (Block Method)

S.No	Group	Treatment	Duration (sec) Mean ± SEM
1	Normal	Vehicle (1% CMC, p.o.)	20 ± 2
2	Control	Rotenone (10mg/kg,i p.)	150 ± 5 ***
3	Standard	)opa & Carbidopa (100mg+25mg/kg p.o) + Rotenone (10mg/kg, p.o.)	110 ± 4 **
4	Test-1	inogalactan (200 mg/kg, p.o) + Rotenone (10mg/kg, p.o.)	60 ± 3 ###
5	Test-2	inogalactan (400 mg/kg, p.o) + Rotenone (10mg/kg, p.o.)	45 ± 3 ###

- One-way ANOVA followed by Tukey's test , ***p < 0.001 vs Normal , **p < 0.01 vs Disease , ###p < 0.001 vs Disease .

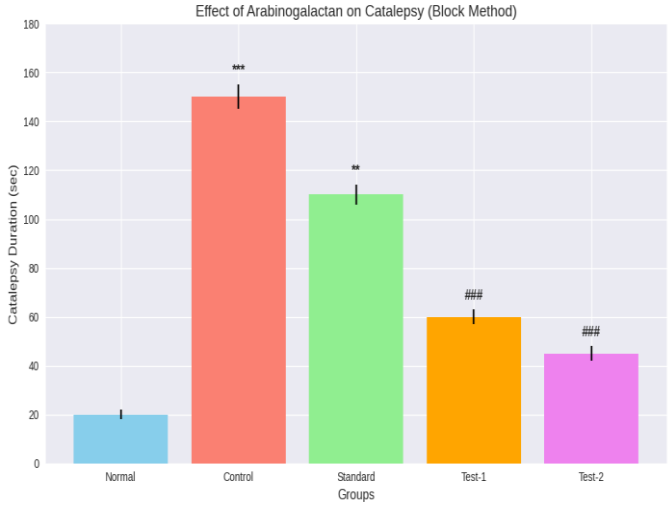


Fig no 2: Effect of Arabinogalactan on Catalepsy (Block Method)

Normal group (Vehicle): 20 ± 2 seconds — baseline catalepsy duration.

Control (Rotenone): 150 ± 5 seconds — significant increase in catalepsy (***p < 0.001 vs Normal).

Standard (L-Dopa + Carbidopa): 110 ± 4 seconds — reduced catalepsy (**p < 0.01 vs Disease).

Test-1 (Arabinogalactan 200 mg/kg): 60 ± 3 seconds — strong reduction (###p < 0.001 vs Disease).

Test-2 (Arabinogalactan 400 mg/kg): 45 ± 3 seconds — even greater reduction (###p < 0.001 vs Disease).

3.1.2 Effect on Locomotor Activity (Open Field Test)

S.No	Group	Treatment	Activity Count Mean ± SEM
1	Normal	Vehicle (1% CMC, p.o.)	120 ± 5
2	Control	Rotenone (10mg/kg,i p.)	40 ± 3 ***
3	Standard	)opa & Carbidopa (100mg+25mg/kg p.o) + Rotenone (10mg/kg, p.o.)	65 ± 4 **
4	Test-1	inogalactan (200 mg/kg, p.o) + Rotenone (10mg/kg, p.o.)	95 ± 4 ###
5	Test-2	inogalactan (400 mg/kg, p.o) + Rotenone (10mg/kg, p.o.)	105 ± 5 ###

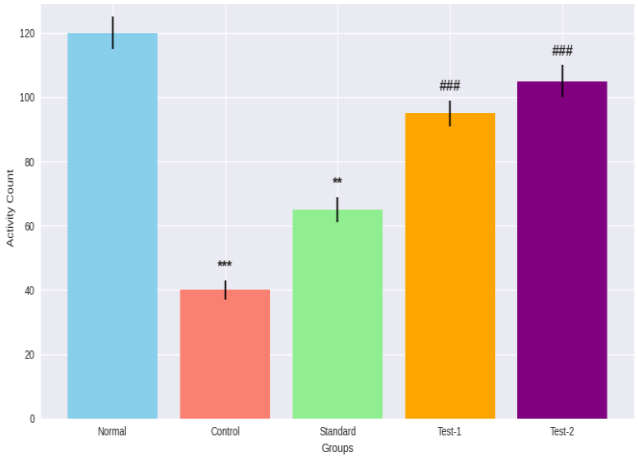


Fig no 3: Effect of Arabinogalactan on Locomotor Activity (Open Field Test)

Normal group (Vehicle): 120 ± 5 — baseline locomotor activity.

Control (Rotenone): 40 ± 3 — severe reduction in activity (***$p < 0.001$ vs Normal).

Standard (L-Dopa + Carbidopa): 65 ± 4 — partial improvement (**$p < 0.01$ vs Disease).

Test-1 (Arabinogalactan 200 mg/kg): 95 ± 4 — strong recovery (###$p < 0.001$ vs Disease).

Test-2 (Arabinogalactan 400 mg/kg): 105 ± 5 — near-normal activity levels (###$p < 0.001$ vs Disease).

3.1.3 Effect on Brain Weight

S.No	Group	Treatment	Brain Weight (g) Mean ± SEM
1	Normal	Vehicle (1% CMC, p.o.)	1.85 ± 0.05
2	Control	Rotenone (10mg/kg,i p.)	1.40 ± 0.04 ***
3	Standard	Dopa & Carbidopa (100mg+25mg/kg p.o) + Rotenone (10mg/kg, p.o.)	1.55 ± 0.03 **
4	Test-1	binogalactan (200 mg/kg, p.o) + Rotenone (10mg/kg, p.o.)	1.70 ± 0.04 ###
5	Test-2	binogalactan (400 mg/kg, p.o) + Rotenone (10mg/kg, p.o.)	1.78 ± 0.05 ###

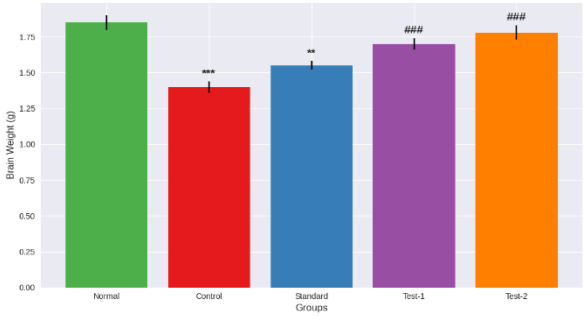


Fig no 4: Effect of Arabinogalactan on Brain Weight

Normal group (Vehicle): **1.85 ± 0.05 g** — baseline brain weight.

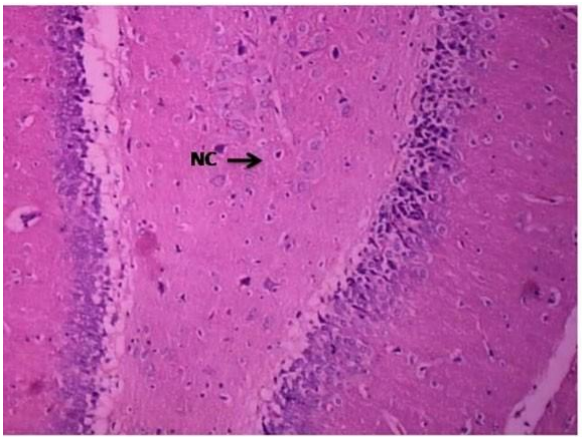
Control (Rotenone): **1.40 ± 0.04 g** — marked reduction in brain weight (***p < 0.001 vs Normal).

Standard (L-Dopa + Carbidopa): **1.55 ± 0.03 g** — partial recovery (**p < 0.01 vs Disease).

Test-1 (Arabinogalactan 200 mg/kg): **1.70 ± 0.04 g** — significant improvement (###p < 0.001 vs Disease).

Test-2 (Arabinogalactan 400 mg/kg): **1.78 ± 0.05 g** — near-normal restoration (###p < 0.001 vs Disease).

3.2 Histopathological results:



o:3.1 Photo micrograph of the brain of normal group (Vehicle p.o.)

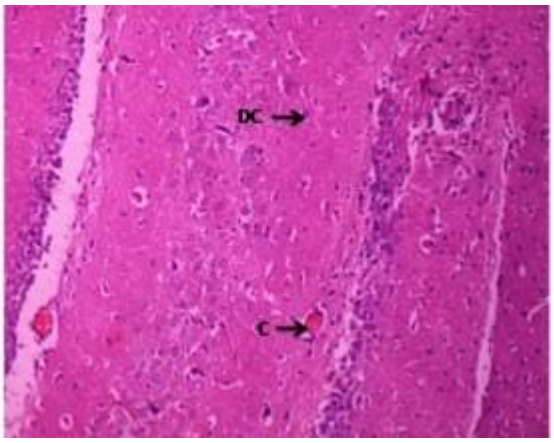


Fig no:3.2 Photo micrograph of the brain of control group (Rotenone 10mg/kg, i.p.)

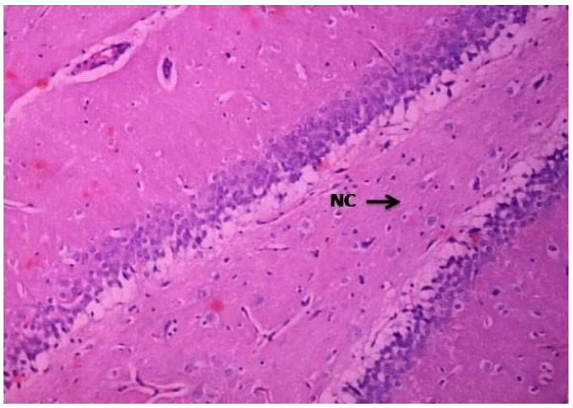


Fig no:3.3 Photo micrograph of the brain of standard group (L-Dopa & carbidopa 100+25 mg/kg, p.o. +Rotenone 10mg/kg, i.p.)

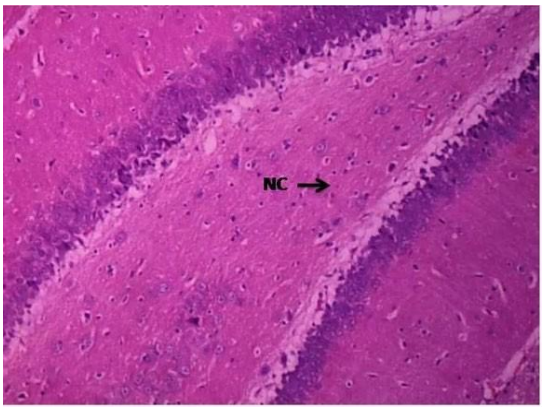


Fig no:3.4 Photo micrograph of the brain of Test-1 (Arabinogalactan 200 mg/kg, p.o. + Rotenone 10mg/kg, i.p.)

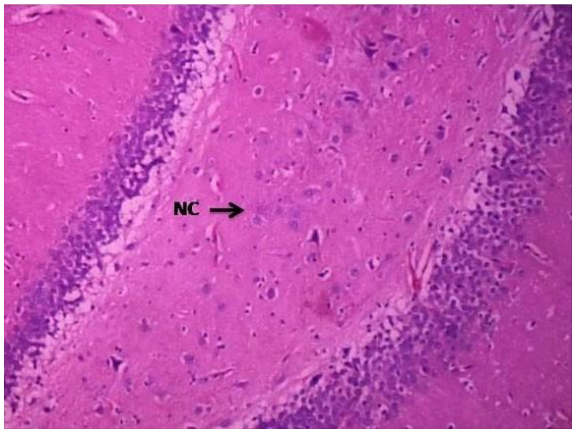


Fig no:3.5 Photo micrograph of the brain of Test-2 (Arabinogalactan 400 mg/kg, p.o. + Rotenone 10mg/kg i.p.)

ABBREVIATIONS AND DESCRIPTION BRAIN:

RC	=	Regenerative Changes	NC	=	Neural cells
HC	=	Hippocampus region	DC	=	Degenerative changes

Normal:

- The normal cyto-architecture of the Brain tissue with Higher magnification (40X).

Control:

- Congestion showed Degenerative changes in Brain tissue with Higher magnification (40X).

Standard:

The regenerative changes took place in tissue with Higher magnification (40X).

- **Test 1:**

Tissue architecture indicated the regeneration of brain tissue which showed like normal with Higher magnification (40X).

- **Test 2:**

The regeneration of structural damage of tissue showed similar cyto-architecture of brain tissue with Higher magnification (40X).

4. Discussion

Parkinson's disease is a progressive neurodegenerative disorder characterized by the degeneration of dopaminergic neurons in the substantia nigra, leading to reduced dopamine levels and motor impairments such as bradykinesia, rigidity, and decreased locomotor activity. Experimental toxin-induced models are commonly used to reproduce these pathological features and to evaluate potential neuroprotective agents.

In the present study, arabinogalactan treatment significantly improved behavioural and physiological parameters in the Parkinsonian model. A reduction in cataleptic behaviour observed in treated animals indicates a possible restoration of dopaminergic

neurotransmission or protection of dopaminergic neurons from toxin-induced damage. Catalepsy is a widely accepted behavioural indicator of dopamine depletion; therefore, its attenuation suggests improvement in motor function.

Arabinogalactan treatment also enhanced locomotor activity compared with the disease control group. Impaired locomotion is a hallmark of Parkinsonian motor dysfunction resulting from decreased dopamine availability in the nigrostriatal pathway. The improvement in locomotor performance observed in this study suggests that arabinogalactan may help restore motor coordination and neuromuscular function.

Additionally, the preservation of brain weight in treated animals indicates a protective effect against neuronal degeneration. Neurodegenerative processes in Parkinson's disease are closely associated with oxidative stress, mitochondrial dysfunction, and neuroinflammatory responses. The neuroprotective effects of arabinogalactan may therefore be attributed to its antioxidant and anti-inflammatory properties, as well as its potential ability to modulate neuronal survival pathways such as the PI3K/Akt signaling pathway.

Overall, the findings of this study are consistent with previous reports demonstrating that natural bioactive compounds can alleviate Parkinsonian symptoms by reducing oxidative stress, regulating inflammation, and preserving dopaminergic neuronal function.

5. Conclusion

The present study demonstrates that arabinogalactan exerts significant neuroprotective effects in an experimental model of Parkinson's disease. Treatment with arabinogalactan reduced catalepsy, improved locomotor activity, and preserved brain weight, indicating protection against dopaminergic neuronal degeneration.

These beneficial effects may be associated with the antioxidant and anti-inflammatory properties of arabinogalactan and its potential role in regulating neuronal survival pathways. Overall, the findings suggest that arabinogalactan may serve as a promising natural therapeutic candidate for the management of Parkinson's disease. However, further molecular and clinical studies are required to confirm its mechanism of action and therapeutic efficacy.

6. Future Perspectives

- Clinical validation in human trials
- Mechanistic studies on signaling pathways
- Development of targeted brain delivery systems

7. Acknowledgement

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