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INVITRO AND INSILICO EVALUATION OF ANTIPARKINSON'S ACTIVITY OF ETHANOLIC FRUIT EXTRACT OF COCCINIA GRANDIS IVY GOURD

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Abstract:

Parkinsonism is a progressive neurological disorder associated with the degeneration of dopaminergic neurons, oxidative stress, and mitochondrial dysfunction. The present study aimed to evaluate the in vitro anti-parkinsonism potential of the ethanolic fruit extract of Coccinia grandis (ivy gourd). The fruits were collected, authenticated, dried, powdered, and extracted using ethanol by the maceration method. The percentage yield of the extract was found to be 19.84% w/w. Preliminary phytochemical screening was performed to identify the major phytochemical groups present in the extract. Molecular docking studies were carried out to evaluate the predicted binding interactions of selected phytoconstituents, including cucurbitacin-B, β-sitosterol, daucosterol, lupeol, β-amyrin acetate, and β-carotene, against Parkinsonism-associated target proteins SNCA, Parkin, and DJ-1. The in vitro neuroprotective activity was evaluated using the MTT assay in rotenone-induced SH-SY5Y neuroblastoma cells. The extract demonstrated a concentration-dependent increase in cell viability, from 53.32% at 6.25 µg/mL to 85.11% at 100 µg/mL, compared with 49.67% in the rotenone-treated group. Molecular docking also showed favourable predicted interactions between selected phytoconstituents and the target proteins. These findings provide preliminary evidence of the potential neuroprotective activity of Coccinia grandis fruit extract. Further mechanistic, toxicological, and in vivo studies are required to establish its therapeutic potential against Parkinsonism.

Keywords:*Coccinia grandis, Ivy gourd, Parkinsonism, Neuroprotective activity, Ethanolic fruit extract, MTT assay, Molecular docking, SH-SY5Y cells, Rotenone, Phytochemicals.*

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INTRODUCTION:

Parkinsonism is a progressive neurological syndrome characterized by motor symptoms such as bradykinesia, muscular rigidity, resting tremor, and postural instability, which significantly affect the daily activities and quality of life of affected individuals. It may also be associated with non-motor manifestations, including cognitive impairment, sleep disturbances, depression, pain, and autonomic dysfunction [1]. According to the World Health Organization (WHO), more than 8.5 million people were living with Parkinson's disease worldwide in 2019. During the same year, Parkinson's disease contributed to approximately 5.8 million disability-adjusted life years (DALYs), representing an 81% increase since 2000, and caused around 329,000 deaths, an increase of more than 100% since 2000. WHO also reported that the prevalence of Parkinson's disease had doubled over the preceding 25 years. These statistics highlight the increasing global burden of Parkinsonian disorders, which results in progressive disability, reduced independence, increased caregiving requirements, and considerable socioeconomic challenges [2]. Although currently available medications provide symptomatic relief, their use is associated with several limitations. Levodopa, the most effective medication for improving motor symptoms, may produce wearing-off phenomena, motor fluctuations, and dyskinesia during long-term treatment. Dopamine agonists may cause excessive sleepiness, hallucinations, nausea, and impulse-control disorders, while anticholinergic drugs may produce dry mouth, constipation, confusion, and memory impairment, particularly in elderly patients. Furthermore, existing medications primarily control symptoms rather than completely halting the underlying neurodegenerative process. The availability and affordability of essential medicines, including levodopa/carbidopa, also remain inadequate in many low- and middle-income countries. According to WHO, levodopa/carbidopa was consistently available at the primary-care level in only 34% of 110 surveyed countries, and none of the low-income countries reported consistent availability. These therapeutic and accessibility challenges emphasize the need for continued research into alternative and complementary treatment approaches [3].

Medicinal plants have gained considerable scientific attention due to their diverse phytochemical constituents and reported antioxidant, anti-inflammatory, and neuroprotective properties. Traditional medicinal systems have utilized numerous plants for managing neurological disorders, and their bioactive compounds may provide valuable leads for the development of novel therapeutic agents. Therefore, the investigation of medicinal plants represents a promising area of research in the search for safer, more accessible, and potentially effective approaches to managing Parkinsonian disorders [4].

Parkinsonism is essentially a clinical syndrome brought about by combining a number of motor symptoms that include tremors, muscle stiffness or rigidity, bradykinesia or slowness of movement, and postural instability. Such symptoms usually result from the dysfunction or degeneration of dopamine-producing neurons in a specific region of the brain called substantia nigra. Parkinsonism is closely associated with Parkinson's disease as the primary cause of the disease but can also be caused by several other diseases like multiple system atrophy, progressive supranuclear palsy, or due to drug-induced parkinsonism. The symptoms of the disease are generally so progressive that they seriously impair a person's ability to lead an everyday life and bring down a person's quality of life in general. There is no cure; therefore, the treatment objective has remained symptomatic relief in medication, such as with levodopa, physical therapy, and sometimes surgical intervention. Newer therapeutic manoeuvres for symptomatic relief as well as the possibility of checking the syndrome's course are now under study.[5]

ETIOLOGY OF PARKINSONISM

A wide variety of underlying conditions, medications, and environmental factors results in Parkinsonism. The most common cause includes:

A. Parkinson's Disease (PD): It is the chief cause of parkinsonism and accounts for almost 80%. Parkinson's disease, there is slow death of neurons in the substantia nigra that synthesizes dopamine which is a chemical playing a crucial role in controlling movements.

B. Drug-Induced Parkinsonism: Several drugs, including antipsychotic drugs and some antiemetic drugs, block the dopamine receptors or decrease concentration of dopamine and cause symptoms almost identical to those seen in parkinsonism. The effects often reverse by stopping the drug

C. Vascular Parkinsonism: This is most commonly the effect of small, multiple strokes to motor control areas. Symptoms can occur suddenly and tend to be less responsive to traditional treatments for Parkinsonism.

D. Traumatic Brain Injury (TBI): Repeated cerebral trauma, as commonly observed in contact sports or accidents, may impair areas of the brain, which control movement and hence lead to parkinsonism. Sometimes, this happens in various disorders, such as chronic traumatic encephalopathy (CTE).

E. Environmental Factors and Toxins: Exposure to specific toxic agents like pesticides and herbicides and some heavy metals has been said to have a predisposition to parkinsonism. MPTP is also an

impurity of some synthetic drugs that causes irreversible parkinsonism.

F. Genetic Causes: The majority of Parkinson's disease cases are idiopathic, but some carry genetic mutations attached with them. Some inherited forms of parkinsonism result from a small set of rare conditions such as Wilson's disease, caused by copper accumulated in the brain. The etiology of parkinsonism has to be established for proper management because the cause varies with the severity of the treatment applicable.[6]

SIGNS OF PARKINSONISM

A. Motor Symptoms: Bradykinesia, Muscle stiffness, Rigidity, Tremor in hands, Stooped position.

B. Non-Motor Symptoms: Memory loss, Mood changes, Sleep Disturbances, Drooling, Loss of Smell, Constipation and Gastrointestinal problems, Dysphagia, Unintentional weight loss.[7]

TYPES OF PARKINSONISM

A term for a group of disorders whose clinical features mimic those of Parkinson's disease (PD) but are of different etiology, progression, and response to treatment. Major categories include:

A. Idiopathic Parkinson's Disease (PD):

The most prevalent form of parkinsonism is that whose symptoms slowly deteriorate with time due to degeneration of the dopamine-producing neurons in the brain. It generally seems responsive to dopaminergic replacement therapies, such as levodopa.

B Drug-induced Parkinsonism:

Many drugs, particularly antipsychotics, anti-nausea drugs, and some calcium channel blockers, cause parkinsonism by blocking the dopamine receptor. Unlike parkinsonism, manifestations are reversed by the stopping of this drug.

C. Vascular Parkinsonism:

This condition arises due to many miniature strokes and decreased blood flows to other parts of the brain that control movement. It responds only in an often-limited way to standard treatments of Parkinson's.

D. Atypical Parkinsonian Disorders (Parkinson-Plus Syndromes)

These are neurodegenerative disorders. Similar in nature to Parkinson's, but differ in characteristics and accelerate faster. This condition is associated with manifestations of effects on different body systems: the motor system, autonomic regulation-such as blood pressure-and often, regulation over bladder function.

- PSP: It presents with difficulties in balance, saccadic eye movements-vertical being particularly affected-and tends to be more rapidly progressive.
- Corticobasal Degeneration (CBD): Usually presents asymmetrically with rigidity, cognitive alterations, and "alien limb" phenomena.
- Dementia with Lewy Bodies (DLB): An overlap of parkinsonism, cognitive decline, visual hallucinations, and fluctuation in alertness.

E. Genetic or Hereditary Parkinsonism: Other genetic mutations have been associated with parkinsonism, although less common, include Wilson's disease which is due to copper deposition and juvenile parkinsonism. These inherited variants sometimes symptomatize similarly to the onset of Parkinson's Disease but very often run their course or respond differently to treatment.

F. Post-Traumatic Parkinsonism: Most of the times also carry a past or a history of traumatic brain injury in athletes, which are recurrent cranial injuries. This may lead to a condition known as chronic traumatic encephalopathy, which shares symptoms with parkinsonism but is accompanied by other cognitive and behavioural changes.[8]

PLANT PROFILE

Coccinia grandis (L.) Voigt, commonly known as ivy gourd, is a fast-growing perennial climbing plant belonging to the family Cucurbitaceae, commonly known as the gourd, melon and pumpkin family. It is widely used as a food crop as well as in traditional medicine. The plant is native to East Africa and has spread widely throughout tropical regions of Asia, America and the Pacific. *Coccinia grandis* is characterized by its climbing habit, alternate heart-to pentagon-shaped leaves, large white star-shaped flowers, red ovoid fruits and succulent tuberous roots. The plant has attracted considerable attention because of its traditional medicinal importance and diverse pharmacological properties. Different parts of *Coccinia grandis* contain several bioactive constituents, including β-sitosterol, alkaloids, flavonoids, triterpenoids, saponins, lupeol and β-amyrin. Traditionally, the plant has been used for conditions such as diabetes, inflammation, infections and gastrointestinal disorders. The reviewed literature reports pharmacological activities including antidiabetic, antioxidant, anti-inflammatory, antimicrobial, antiulcer, hepatoprotective, hypoglycemic, antimalarial and antidyslipidemic activities, indicating its potential as an important medicinal plant.[9]



Figure 1: ***Coccinia grandis***

BOTANICAL DESCRIPTION

It can grow up to 3m (i.e., 9ft 10in). The roots are tuberous; stems are green along with simple axillary tendrils. Leaves are glabrous and pentagonal in shape with dentate margins measuring about 6.5 to 8.5 cm long and 7 to 8 cm wide. Flowers are monoecious, white, star-shaped about 4cm in diameter and contain 5 tubular petals, female and male flowers emerge at the axils on the petiole having 3 stamens. The fruit is the edible part of the plant; they are slimy in touch, pulpy and ovoid to an ellipsoid shape. Young fruits are green in colour and turn to scarlet red when ripen which ranges from 2.5 to 5 cm in length and 1.3 to 2.5 cm in diameter. Fruits contain numerous seeds with thickened margins measuring up to 6 to 7 mm in length. [10]

CHEMICAL CONSTITUENTS

Phytochemicals present herbal medicinal plants are responsible for eliciting pharmacological action. Nature and extent of phytochemicals present in the plants elaborate the medicinal properties. Phytochemical evaluation studies conducted on the plant extracts of *C. grandis*, reveals the presence and content of various components like alkaloids, saponins, reducing sugar alkaloids, steroids, tannins, ellagic acids, phenols, glycosides and triterpenoids that are present in the plants.

- Aerial part: Heptacosane, Cephalandrol, β -sitosterol, Alkaloids Cephalandrins A and B
- Fruits: β-Amyrin Acetate, Lupeol, Cucurbitacin B, Taraxerone, Taraxerol, β-carotene, Cucurbitacin-B, Lycopene, Cryptoxanthin, Xyloglucan, Carotenoids, β-sitosterol, Stigma-7-en-3-one, Daucosterol.
- Root: Resin, Alkaloids, Starch, Fatty Acids, Carbonic acid, Triterpenoid, Saponin Coccinoside, Flavonoid Glycoside, Lupeol, β-amyrin, β-sitosterol, Taraxerol. [10]

MEDICINAL USE [11]

- ❖ Antidiabetic
- ❖ Antioxidant
- ❖ Osteoarthritis
- ❖ Antihyperlipidemic
- ❖ Anti-diarrheal
- ❖ Anti-ulcer
- ❖ Cardiovascular diseases
- ❖ Anticancer
- ❖ Hepatoprotective

MATERIALS AND METHODS:

MATERIALS

- Ethanol
- Sodium hydroxide
- Nitric acid
- Wagner' reagent
- Glacial acetic acid
- Hydrochloric acid
- Fehling's solution A
- Fehling's solution B
- Chloroform
- Ferric chloride solution

METHODS

Collection and Authentication of Plant Materials

- The fruits were collected from certain areas of Thiruvananthapuram district Kerala, India, in the month of August. The plant material was identified and authenticated from the Department of Botany, NMC College Marthandam.
- Then, the sample was thoroughly washed with fresh water and air-dried at room temperature under shade for 15 days and pulverized into coarse powder with the aid of a mechanical grinder. The powdered material was stored in an airtight container.

Preparation of ethanolic fruit extract of *Coccinia grandis*

- About 10 grams of dried fruit powder were weighed using a weighing balance and placed under shade for 15 days and pulverized into coarse powder by the aid of mechanical grinder.
- In which, 500 mL of ethanol and powder was macerated with 3 L of 95% ethanol.
- The mixture is continuously shaken at room temperature.
- After 1 week, the extract was filtered with muslin cloth and the crude extract was concentrated to dryness under reduced pressure and controlled temperature and stored in airtight container for further use.
- The percentage yield of the extractive was calculated.[12]

Calculation of percentage yield

- The percentage yield of extract was calculated using the formula:
- **Percentage yield = (W_2 / W_1) × 100**
- Where,
- W_1 – Weight in grams of dried plant material.
- W_2 – Weight in grams of extracts obtained.

Percentage yield = (12.9/65.26) ×100
=19.84 %w/w

Primary phytochemical screening

Qualitative phytochemical screening was carried out for carbohydrate, tannins, steroids and terpenoids, flavonoids, amino acid, cardiac glycoside.

***IN SILICO* MOLECULAR DOCKING STUDIES**

Steps involved in docking study

Step 1: Ligand Preparation

Chemical structure of the compounds was obtained from the world's largest freely accessible chemistry

database, PubChem. Chemical structures of the compounds are downloaded in 3D format and converted to SDF format. Finally, the input format prepared as PDB ligand format and can be visualized in Open Babel.

Step 2: Protein Preparation

Target molecules such as SNCA, Parkin and DJ-1 can be determined by literature survey and respective PDB files such as 3Q26,5C1Z and 6M8Z were downloaded from Protein Data Bank. Then the protein is downloaded in crystal structure and after processing convert this to input format.

Step 3: Protein Processing

Protein structure processing is done by removal of water molecules, hydrogens, heteroatoms and bound ligand, if any.

Step 4: Docking Analysis

Docking analysis was done by 'CB-Dock 2' web server software. [13]

INVITRO STUDY OF COCCINIA GRANDIS IVY GOURD

MTT ASSAY

Procedure

Two-day-old confluent monolayers of cells were trypsinized, suspended in 10% growth medium, and seeded into a 96-well tissue culture plate at 100 µL cell suspension (5×10^3 cells/well) before incubation at 37°C in a humidified 5% CO_2 incubator. Stock solutions were prepared by dissolving 1 mg of sample in 1 mL DMEM with a cyclomixer and filtered through a 0.22 µm Millipore syringe filter to ensure sterility. To evaluate cytotoxicity, toxicity was induced with 10 µM Rotenone for 1 hour, after which freshly prepared test compounds were added in triplicates at concentrations of 25, 12.5, 6.25, 3.1, and 1.5 µg/mL alongside untreated control and Rotenone-alone treated wells. Morphological changes such as cell rounding, shrinkage, granulation, and vacuolization—were observed and captured after 24 hours using an inverted phase-contrast microscope (Olympus CKX41 with Optika Pro5 CCD camera). Following 24 hours of incubation, well contents were removed, and 30 µL of reconstituted MTT solution (15 mg Sigma M-5655 in 3 mL PBS, filter-sterilized) was added to each well, followed by 4 hours of incubation at 37°C in 5% CO_2. Finally, the supernatant was removed, 100 µL of DMSO was added to dissolve the formazan crystals, and absorbance was measured at 540 nm using a microplate reader to calculate the percentage cell viability using the formula. [14,15]

$$\textbf{Percentage viability (\%)} = \frac{\text{Mean OD Sample x 100}}{\text{Mean OD of control group}}$$

RESULT AND DISCUSSION:

Primary phytochemical screening

SL.NO	NAME OF THE CONSTITUENTS	NAME OF THE TEST	INFERENCE
1	Carbohydrates	Fehling's test	Positive
2	Tannins	Ferric chloride	Positive
3	Steroids and Triterpenoids	Salkowski test	Positive
4	Flavonoids	Alkaline reagent test	Positive
5	Amino acid	Xanthoproteic test	Positive
6	Cardiac glycosides	Keller Killiani test	positive

***IN SILICO* MOLECULAR DOCKING STUDIES**

Molecular docking study was performed to investigate the potential of selected phytoconstituents of *Coccinia grandis* against target proteins (SNCA, Parkin and DJ-1) associated with parkinsonism. The docking analysis provides information about the binding affinity, binding orientation, and possible interactions of the selected compounds with the active site of the target proteins. The selected phytoconstituents of *Coccinia grandis,* including Cucurbitacin-B, β-sitosterol, daucosterol, lupeol, β-Amyrin acetate, and β-Carotene.

Gene/Target	Compound	Docking Score (kcal/mol)
SNCA	Cucurbitacin B	−9.2
	β-Sitosterol	−9.4
	Daucosterol	−9.6
	Lupeol	−10.3
	β-Amyrin acetate	−10.3
	β-Carotene	−8.8
Parkin	Cucurbitacin B	−7.5

Gene/Target	Compound	Docking Score (kcal/mol)
	β-Sitosterol	−6.4
	Daucosterol	−7.0
	Lupeol	−6.2
	β-Amyrin acetate	−6.6
	β-Carotene	−7.2
DJ-1	Cucurbitacin B	−7.2
	β-Sitosterol	−6.2
	Daucosterol	−6.4
	Lupeol	−6.4
	β-Amyrin acetate	−6.6
	β-Carotene	−7.8

Table showing the binding affinity of the chemical constituents towards the target proteins

***IN VITRO* ANALYSIS OF *COCCINIA GRANDIS* 'IVY GOURD'**

***In vitro* neuroprotective effect determination by MTT assay**

At increasing concentrations, the ethanolic fruit extract of Coccinia grandis (ivy gourd) showed increased cell viability, indicating neuroprotective effects and potential anti-Parkinsonian activity.

Table shows the *in vitro* neuroprotective effect of *Coccinia grandis* in comparison with the standard drug Levodopa.

Graphical representation

Sample Concentration (µg/ml)			**OD I**		**OD II**	**OD III**	**Average Absorbance at 540nm**	**Percentage Viability**
CONTROL			0.6235	0.6122	0.6048	0.6135	100.00	
ROTENONE			0.292	0.3026	0.3195	0.3047	49.67	
Sample code-L-DOPA								
6.25			0.3854	0.3962	0.4122	0.3979	64.86	
12.5			0.5366	0.5281	0.5299	0.5315	86.64	
25			0.4752	0.4695	0.4663	0.4703	76.66	
50			0.2564	0.2461	0.2419	0.2481	40.45	
100	0.185	0.1962	0.1965	0.1926	31.39			
Sample Code-EFCG								
6.25	0.3362	0.3264	0.3188	0.3271	53.32			
12.5	0.4267	0.4155	0.409	0.4171	67.98			
25	0.4436	0.4529	0.4476	0.4480	73.03			
50	0.4906	0.4866	0.4763	0.4845	78.97			
100	0.5144	0.5297	0.5223	0.5221	85.11			

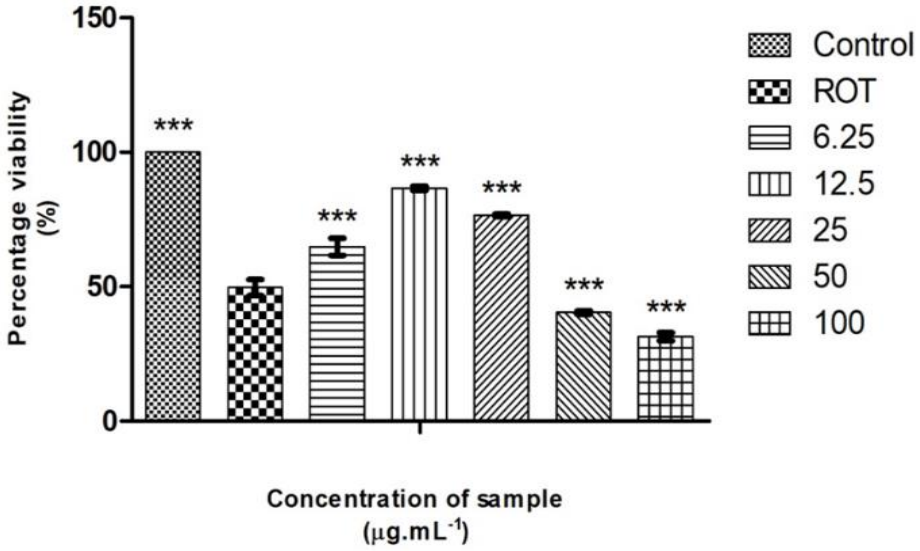


Figure 2:Graphical representation of LDOPA

Figure no 2: Graphical representation depicting the neuroprotective effect of LDOPA on rotenone induced SH-SY5Y cells by MTT assay- Along Y axis Percentage viability, Along X axis varied concentration of LDOPA All experiments were done in triplicates and results represented as Mean+/- SD. One-way ANOVA and Dunnett's test were performed to analyse data. ***p< 0.0001 compared to rotenone exposed group

Graphical representation

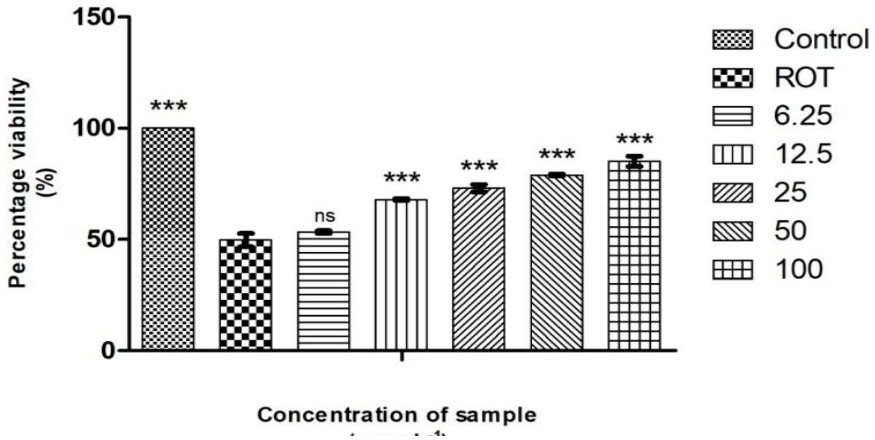


Figure 3: Graphical representation of sample

Figure no 3: Graphical representation depicting the neuroprotective effect of sample on rotenone induced SH-SY5Y cells by MTT assay- Along Y axis Percentage viability, Along X axis varied concentration of sample. All experiments were done in triplicates and results represented as Mean+/- SD. One-way ANOVA and Dunnett's test were performed to analyse data. ***p< 0.0001 compared to rotenone exposed group, ns – non significant compared to rotenone exposed cells

DISCUSSION:

Parkinsonism is a progressive neurological disorder characterized by the degeneration of dopaminergic neurons and a reduction in dopamine levels. Oxidative stress, mitochondrial dysfunction, neuroinflammation, and abnormal protein

accumulation play important roles in its development. The present study was conducted to evaluate the potential anti-parkinsonism activity of the ethanolic fruit extract of Coccinia grandis using preliminary phytochemical screening, molecular docking, and an in vitro MTT assay.

The ethanolic fruit extract was prepared by maceration and showed a percentage yield of 19.84% w/w. Preliminary phytochemical screening indicated the presence of amino acids, flavonoids, carbohydrates, alkaloids, tannins, steroids, terpenoids, and cardiac glycosides. These phytochemical groups may contribute to the biological properties of the extract and provide a basis for further investigation of its potential neuroprotective effects.

Molecular docking studies were performed using six selected phytoconstituents, namely cucurbitacin-B, β-sitosterol, daucosterol, lupeol, β-amyrin acetate, and β-carotene, against the Parkinsonism-associated target proteins SNCA, Parkin, and DJ-1. Among the selected compounds, lupeol and β-amyrin acetate showed the most favourable docking scores against SNCA, with values of −10.3 kcal/mol. Cucurbitacin-B showed the most favourable docking score against Parkin at −7.5 kcal/mol, while β-carotene showed the most favourable score against DJ-1 at −7.8 kcal/mol. These results suggest potential interactions between the selected phytoconstituents and the target proteins. However, docking scores alone cannot confirm biological activity or therapeutic effectiveness.

The in vitro neuroprotective activity of the extract was evaluated using rotenone-induced toxicity in SH-SY5Y neuroblastoma cells. Rotenone treatment reduced cell viability to 49.67% compared with the untreated control. Treatment with the ethanolic fruit extract increased cell viability from 53.32% at 6.25 µg/mL to 85.11% at 100 µg/mL. The highest tested concentration demonstrated the greatest cell viability among the extract-treated groups, suggesting a protective effect against rotenone-induced cellular damage under the experimental conditions.

The findings from preliminary phytochemical screening, molecular docking, and the MTT assay collectively support the potential neuroprotective activity of Coccinia grandis fruit extract. Nevertheless, the exact mechanism of action and the individual compounds responsible for the observed effect remain to be established. Further studies, including mechanistic investigations, toxicity evaluation, and in vivo experiments, are necessary to confirm its potential application in anti-parkinsonism drug development.

CONCLUSION:

The present study evaluated the in vitro anti-parkinsonism potential of the ethanolic fruit extract of Coccinia grandis (ivy gourd). The extract was obtained by maceration, with a percentage yield of 19.84% w/w. Preliminary phytochemical screening revealed the presence of several phytochemical groups, including flavonoids, alkaloids, tannins, steroids, terpenoids, and cardiac glycosides.

Molecular docking studies demonstrated favourable predicted interactions of selected phytoconstituents with the Parkinsonism-associated target proteins SNCA, Parkin, and DJ-1. Furthermore, the in vitro MTT assay showed an increase in SH-SY5Y cell viability from 53.32% at 6.25 µg/mL to 85.11% at 100 µg/mL in the extract-treated groups, compared with 49.67% in the rotenone-treated group.

Overall, these findings provide preliminary evidence that the ethanolic fruit extract of Coccinia grandis may possess neuroprotective activity against rotenone-induced cellular damage. The results indicate that this medicinal plant warrants further investigation as a potential source of compounds for anti-parkinsonism drug development. However, additional mechanistic, toxicological, and in vivo studies are required before its therapeutic efficacy and safety can be established.

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