

EVALUATION OF NEUROPROTECTIVE EFFECT OF SITAGLIPTIN IN STREPTOZOTOCIN INDUCED ALZHEIMER'S DISEASE IN WISTAR RATS

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ABSTRACT

Background: This study explores the neuroprotective potential of sitagliptin, an anti-diabetic drug, in managing Alzheimer's disease (AD) symptoms in a streptozotocin (STZ)-induced rat model. **Objective:** This research aimed to assess whether sitagliptin could improve cognitive function and reduce anxiety by modulating acetylcholinesterase (AChE) activity. **Methods:** Rats were divided into five groups: a control, a negative control with only STZ, a standard treatment group with STZ and Rivastigmine, and low- and high-dose sitagliptin groups (5 mg/kg and 10 mg/kg, respectively). Cognitive performance was evaluated using the elevated plus-maze test, which measures spatial memory and anxiety-like behaviors. **Results:** It was showed that both low and high doses of sitagliptin led to significant behavioral improvements, with the high dose producing effects comparable to Rivastigmine, the standard AD treatment. Notably, sitagliptin administration reduced AChE

activity, correlating with observed cognitive and anxiety-related improvements, suggesting its action may involve modulation of cholinergic pathways. These findings support sitagliptin's potential as an adjunct or alternative treatment for AD, especially considering its dose-dependent effects. **Conclusion:** The study underscores the need for further research into sitagliptin's broader neuroprotective mechanisms, particularly its impact on AD biomarkers

such as amyloid-beta and tau proteins. This research provides promising insights into the potential repositioning of sitagliptin for neurodegenerative disease management, highlighting its prospective benefits in AD treatment.

KEYWORDS: Sitagliptin, Alzheimer's Disease (AD), Streptozotocin, Neuroprotective, Acetylcholinesterase.

INTRODUCTION

Neurodegeneration is associated with synapse dysfunction, neuronal network impairment, and the accumulation of physiochemically altered protein variants in the brain.^[1-4] The most prevalent neurodegenerative disorders comprise Alzheimer's disease, Parkinson's disease, prion disease, Amyotrophic lateral sclerosis, motor neuron disease, Huntington's disease, spinal muscular atrophy, and spinocerebellar ataxia.^[5-9] Studies on the pathophysiology of Alzheimer's disease have revealed the significant impact of amyloid-beta (A) and tau protein buildup in the disease's advancement. A hallmark of Alzheimer's disease (AD) is the emergence of A- rich plaques in the brain, which are linked to neurofibrillary tangles (NFTs) made up of hyperphosphorylation.^[10-13] The formation of these plaques interferes with hippocampal circuits, hindering the consolidation of long-term memories. AD is marked by extensive neuronal death, abnormal synaptic connections, and deterioration of crucial neurotransmitter systems essential for cognitive functions, including memory. As a result, the initial clinical manifestation in early-stage Alzheimer's disease typically presents as specific memory deficits. Moreover, functions reliant on the hippocampus and medial temporal lobe, such as declarative episodic memory, are often compromised.^[14]

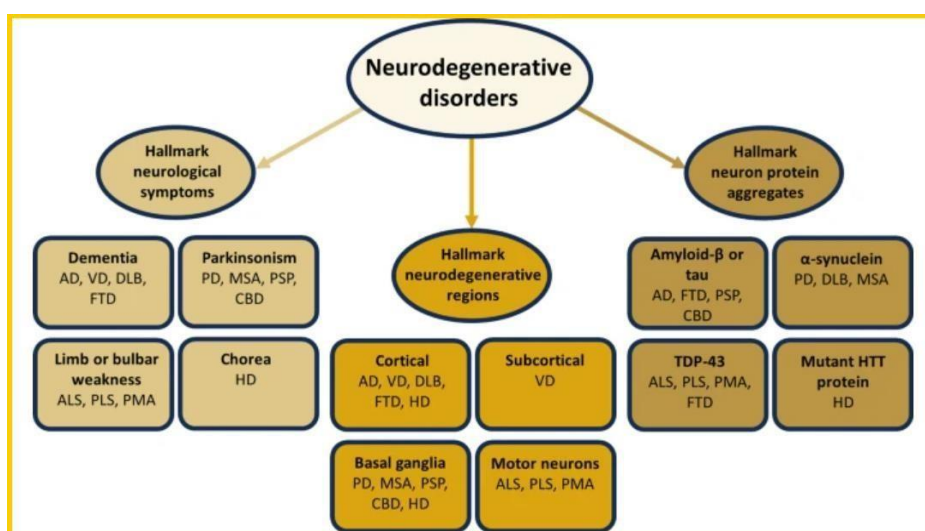


Fig. 1: Illustration of Type of Neurodegenerative Disorders

The prevalence of neurological conditions, including Alzheimer's disease (AD), has increased globally. AD is a complex neurodegenerative disorder responsible for 70% of dementia cases worldwide.^[15,16] Its primary pathophysiological features include amyloid beta plaques and intracellular neurofibrillary tangles.^[17] Cerebral amyloid angiopathy (CAA) is characterized by extracellular amyloid deposits in the brain parenchyma and blood vessels.^[18] Neurofibrillary tangles comprise intracellular paired helical filaments of hyperphosphorylated tau proteins, leading to synaptic and neuronal degeneration.^[19] AD mainly affects the association areas of the cerebral cortex and the hippocampus.^[20] A healthy brain has billions of neurons that process and transmit information via electrical and chemical signals, relaying messages between brain regions and from the brain to the body. AD disrupts this neuronal communication, causing cell death and functional loss.

Normal aging involves some brain atrophy without significant neuron loss. In contrast, AD causes extensive damage as neurons cease functioning, lose connections, and die. AD impairs the essential communication, metabolism, and repair processes of neurons and their networks. The disease typically begins by destroying neurons and connections in the entorhinal cortex and hippocampus, crucial for memory. It then affects parts of the cerebral cortex responsible for language, thought, and social interaction, eventually leading to deterioration in other brain regions. Over time, individuals with AD lose the ability to live and function independently, and the disease is ultimately fatal.

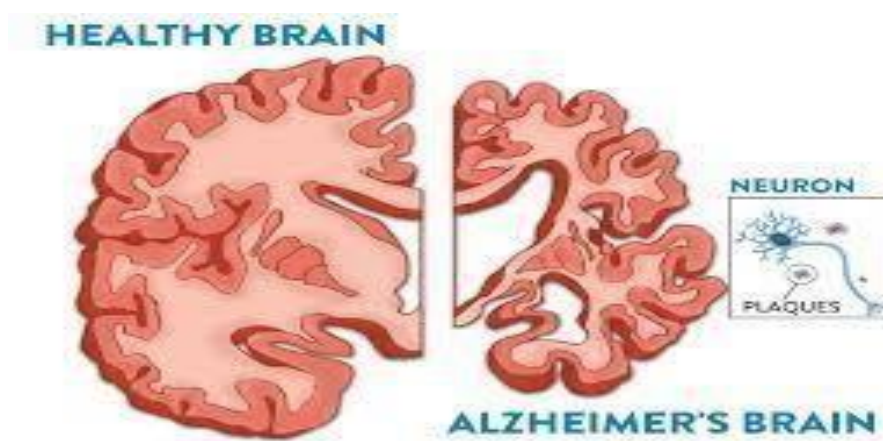


Fig. 2: Anatomy of the brain in Alzheimer's Disease.

Neurons in layer II of the entorhinal cortex, the subiculum, and the CA1 region of the hippocampus are most vulnerable to damage due to accumulating high levels of neurofibrillary tangles and early degeneration in initial disease stages.^[21] In Alzheimer's

disease, neuronal loss affects specific cortical and subcortical regions, including the trans-entorhinal and entorhinal areas, hippocampus, amygdala, medial septal nucleus, nuclei of the diagonal band of Broca, basal nucleus of Meynert, substantia nigra, locus ceruleus, midbrain, and pontine raphe nuclei.^[22] The main contributors to neuronal death are abnormal protein aggregations, such as hyperphosphorylated tau and insoluble beta-amyloid proteins, found both intracellularly and extracellularly.^[23]

As Alzheimer's disease advances, symptoms progressively worsen, including memory impairment, difficulties in planning and organizing, confusion, disorientation, altered sleep patterns, anxiety, hallucinations, delusions, paranoia, and reduced physical control. These symptoms intensify as the disease progresses.^[24]

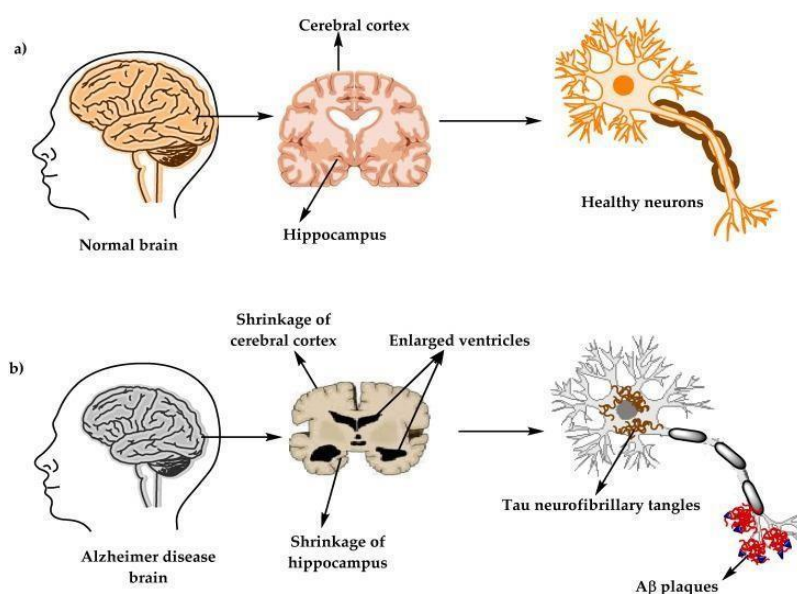


Fig. 3: The physiological structure of the brain and neurons in (a) healthy brain and (b) Alzheimer's disease (AD) brain.

The prevalence of Alzheimer's disease (AD) is influenced by numerous factors, including age, concurrent health conditions, genetic makeup, and educational background.^[25] Definitive diagnosis of AD requires post-mortem examination. While there is currently no cure for AD, ongoing research shows promise in developing methods for early detection and treatment.^[26]

Sitagliptin, a DPP-4 inhibitor (dipeptidyl peptidase-4 inhibitor), has gained significant attention primarily for its role in managing type 2 diabetes mellitus by enhancing incretin levels, particularly GLP-1 (glucagon-like peptide-1) and GIP (gastric inhibitory polypeptide). This results in improved insulin release, reduced glucagon secretion, slowed gastric

emptying, and improved glucose control. Notably, Sitagliptin's properties have also led to research into its potential neuroprotective effects, which has promising implications in treating neurodegenerative diseases such as Alzheimer's disease.^[27]

The overall study is designed to evaluate the Neuroprotective effect of Sitagliptin in Streptozotocin induced Alzheimer's disease in rats.

MATERIALS AND METHODS

Streptozotocin was procured from Samarth Life Sciences, sitagliptin was obtained from Zydus Cadila, and rivastigmine was procured from Cipla Limited, India. The analytical weighing balance (BL110S, Sartorius) and digital pH meter (Hanna Instruments) were used for accurate weighing of experimental materials and measurement of pH, respectively, during the study.

Animals Male Wistar rats (one year old weighing 450 ± 10 g) were used in this study. They were kept in the Animal House facility of Aditya Bangalore Institute of Pharmacy Education and Research, Bangalore, India. The animals were housed in a propylene cages in a room maintained at $25 \pm 2^\circ\text{C}$, 45-50% humidity and 12 h light/dark cycle with free access to standard rodent pellet diet and water. Experiments were conducted in accordance with the Animal Ethics Committee (No: 101/1611/CCSEA)

Induction Of Alzheimer's: Intracerebroventricular injection of streptozotocin (ICV-STZ) The animals were anaesthetized with chloral hydrates (400 mg/kg b Wt, i.p) and placed on stereotaxic frame (Dual manipulator model 51600, Stoelting Co-USA). The skin overlying the skull was incised to expose it and the coordinates for the lateral ventricle (Paxinos and Watson, 1986) were measured accurately as anterioposterior 0.8 mm, lateral 1.5 mm, and dorsoventral -4.0 mm relative to bregma and ventral from dura, with the tooth bar set at 0 mm. A burr hole was made on the skull by an automatic micro drilling machine attached on the stereotaxic apparatus. Through the hole, a 28-gauge Hamilton® syringe of 10 µl attached to another arm of the stereotaxic frame and piston of the syringe was lowered manually to infuse STZ into each lateral ventricle on either side. The lesion group received a bilateral ICV injection of STZ (3 mg/kg, b.wt in saline 5µl/ injection per site). The sham group underwent the same surgical procedures, and the same volume of saline was injected instead of STZ. After surgery, the rats were housed individually and had access to food and water.^[28,29]

Animal Grouping: The experimental animals were randomly divided into five groups, with eight animals in each group ($n = 8$). Group I served as the normal control and received normal saline. Group II served as the negative control and was administered streptozotocin (40 mg/kg body weight). Group III served as the standard treatment group and received streptozotocin followed by rivastigmine (6 mg/kg). Groups IV and V received streptozotocin followed by sitagliptin at doses of 10 mg/kg (high-dose group) and 5 mg/kg (low-dose group), respectively.

Passive avoidance test

The passive avoidance test was employed to assess short-term and long-term memory in rodents based on their natural aversion to brightly illuminated areas and preference for dark compartments. The apparatus consisted of a larger illuminated compartment and a smaller dark compartment interconnected by a plexiglass sliding door. The larger compartment was illuminated using a 15-W bulb, whereas the smaller compartment was devoid of any light source. The floor of the apparatus consisted of a stainless-steel grid. The floor of the dark compartment contained a 15×15 cm electrifiable grid connected to a constant-current stimulator, which was used to deliver the aversive foot shock during the training session. The test is carried out in three phases, Exploration test,^[30,31] Aversive stimulation delivery^[32] and Memory retention test.^[33]

Positive memory retention was indicated by the absence of entry into the dark compartment.

Parameters studied in this test

1. Latency to enter the dark compartment.
2. Total time spent in the dark compartment.
3. Number of entries to the dark compartment.



Fig. 4: Light/Dark Compartment Model.

Preparation of brain homogenate: Following the behavioral tests, animals were anesthetized and sacrificed by cervical decapitation. Whole-brain tissue was carefully dissected and thoroughly washed with ice cold phosphate buffer (pH 7.4). The hippocampus was identified and dissected. It was homogenized in phosphate buffer (10% w/v, pH 7.4). The obtained homogenate was centrifuged at 3000 rpm for 10 minutes at 4°C. The supernatant was then carefully separated and used for the estimation of acetylcholinesterase activity.^[34]

Histopathology of brain

Central nervous system has specialized structures compared to other tissues. These include normal components such as myelin sheaths and nerve processes. They can be detected using special staining methods. We use a general staining method such as Haematoxylin and Eosin method to give an overview of tissue structures, and several neurophysiological staining methods.^[35]

RESULTS

Table 1: Effect of Sitagliptin on Entries and Time Spent.

Group	Number of entries in open arm	Number of entries in closed arm	Distance travelled in open arm (m)	Distance travelled in closed arm (m)
Normal	3.4 ± 1.14	3.2 ± 0.836	2.59 ± 0.54	1.938 ± 0.5
Disease	9 ± 1.00 ***	9.4 ± 1.14***	2.33 ± 0.77	4.531 ± 0.5***
Standard	10 ± 1.00***	7 ± 1.00***	3.86 ± 0.87 **	3.16 ± 0.91*
Sitagliptin Low Dose	2.2 ± 0.83	1.4 ± 0.54	1.518 ± 0.2*	0.398 ± 0.21 **
Sitagliptin High Dose	8.4 ± 1.14***	6.4 ± 1.14***	3.87 ± 0.69 **	3.251 ± 0.79**

Mean ± SEM are expressed as mean ± SEM with significant levels of *** $p < 0.001$, ** $p < 0.01$, and * $p < 0.05$ compared to the normal control group.

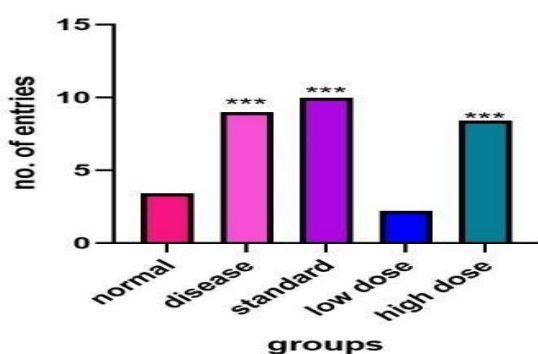


Fig. 5: Number of entries in open arm.

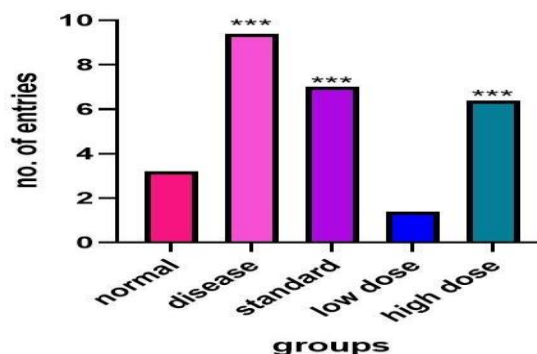


Fig. 6: Number of entries in open arm.

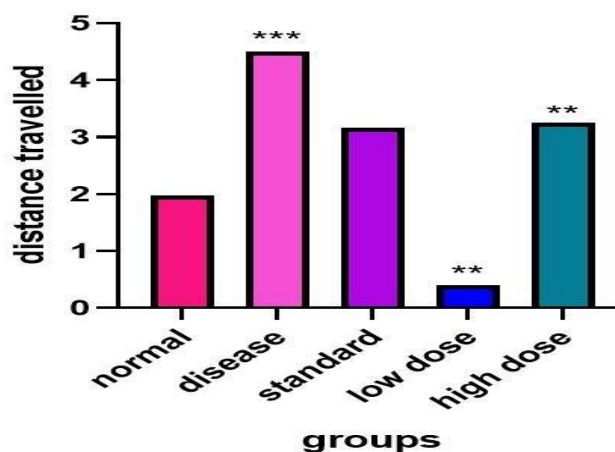
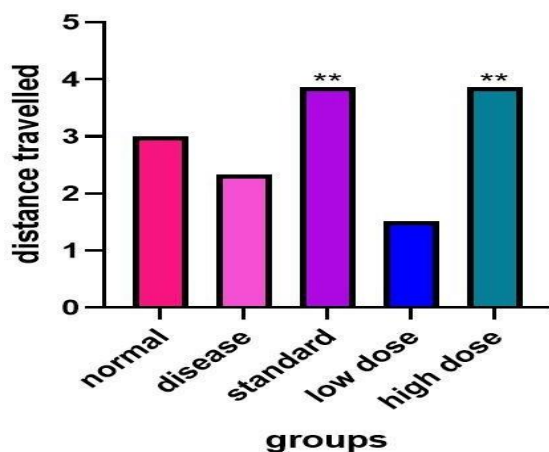


Fig. 7: Distance travelled in open arm. Fig. 8: Distance travelled in Closed arm.

Table 2: Effect of Ach Esterase in Brain Hippocampus.

Group	Ach Esterase in Brain Hippocampus
Normal	0.03 ± 1.14
Disease	0.07 ± 1.00 ***
Standard	0.05 ± 1.00***
Sitagliptin Low Dose	0.04 ± 0.83***
Sitagliptin High Dose	0.05 ± 1.14***

Mean ± SEM are expressed as mean ± SEM with significant levels of *** $p < 0.001$, ** $p < 0.01$, and * $p < 0.05$ compared to the normal control group.

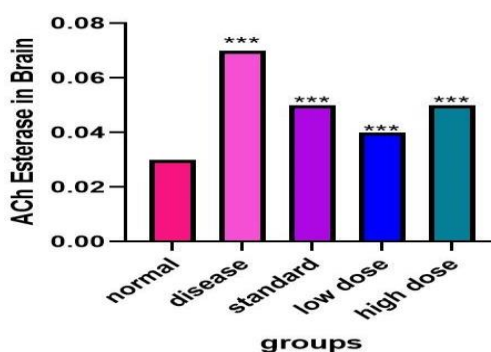


Fig. 9: Effect of Ach Esterase levels in the Brain.

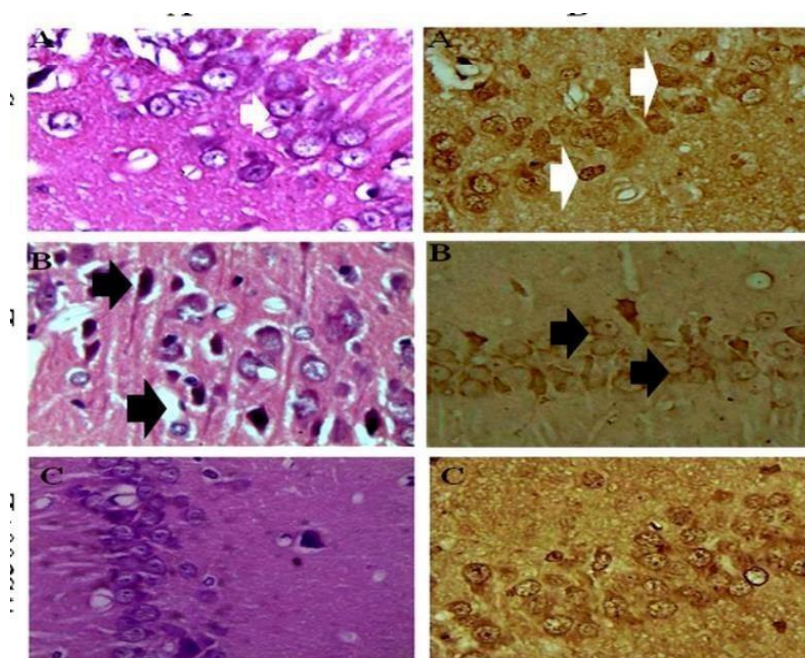


Fig. 10: Histopathological samples of Brain restoring the white matter and Neuronal Cells.

DISCUSSION

The current study investigated the effects of sitagliptin at 5 and 10 mg/kg in a streptozotocin (STZ)-induced model of Alzheimer's disease (AD)-like cognitive impairment in rats. STZ administration resulted in behavioral and biochemical changes, including decreased open-arm exploration, a preference for closed arms in the elevated plus-maze, and increased acetylcholinesterase (AChE) activity. These modifications are similar with the behavioral and cholinergic changes observed in STZ-induced animals with AD-like characteristics. However, because the elevated plus-maze examines anxiety-like behavior and exploratory activity, it should not be used as a direct measure of memory.

Treatment with sitagliptin enhanced the measured behavioral parameters when compared to the STZ group. Both doses boosted open-arm exploration while decreasing closed-arm preference, with the 10 mg/kg dose showing larger improvement than the 5 mg/kg dose. The behavioral effects of the larger dose were similar to those seen with the conventional medication rivastigmine.

Sitagliptin therapy similarly lowered AChE activity relative to the STZ group, with a greater reduction shown at 10 mg/kg. The decrease in AChE activity was accompanied by an improvement in the observed behavioral measures, showing a link between sitagliptin

administration and these positive effects in the current experimental paradigm. However, the current data do not support a direct causal link between decreased AChE activity and observed behavioral alterations.

In general, sitagliptin exhibited positive impacts on behavioral parameters and AChE activity in the STZ-induced AD-like model, with the 10 mg/kg dosage showing more pronounced effects compared to 5 mg/kg. These results encourage additional research on sitagliptin in experimental models of cognitive dysfunction. Subsequent research involving particular learning and memory assessments, alongside more biochemical and histopathological factors, is necessary to better define its possible neuroprotective effects.

CONCLUSION

This study showed positive effects of sitagliptin at doses of 5 and 10 mg/kg in a rat model of cognitive impairment resembling Alzheimer's, induced by streptozotocin. Both doses enhanced behavioral parameters and decreased AChE activity, with the 10 mg/kg dose demonstrating more significant effects and responses similar to those of rivastigmine. These results indicate that sitagliptin may be worth exploring further in studies on cognitive impairment. Nonetheless, the elevated plus-maze mainly evaluates anxiety-related and exploratory actions and cannot solely determine progress in learning and memory. Additionally, the research was constrained by the lack of extra AD-related biochemical and histopathological markers and the assessment of just two doses within a restricted experimental duration. Additional research involving targeted learning and memory assessments, extra AD-related indicators, dose refinement, and extended treatment durations is necessary to validate and define the possible neuroprotective properties of sitagliptin.

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