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Original Article

Evaluation of *in vitro* Anti-Inflammatory Activity of Zolpidem

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ABSTRACT

Objectives: Zolpidem, a novel non-benzodiazepine sedative, has shown antioxidant, muscle relaxant, anti-convulsant and anti-anxiety properties. The Objective of our study is to evaluate it's *in vitro* anti-inflammatory activity. **Materials and Methods:** Three assays were done: Protein Denaturation, Erythrocyte Membrane Stabilization and Proteinase Inhibition Assays using standard techniques. **Results:** Zolpidem showed maximal inhibition of 88.34 ± 0.09%, 82.41 ± 0.41% and 79.20 ± 0.41% in the Protein Denaturation, Erythrocyte Membrane Stabilization and Proteinase Inhibition Assays. **Conclusion:** This shows that Zolpidem possesses excellent anti-inflammatory activity and untapped potential to be used in neurodegenerative diseases. Further *in vivo* studies could be done to further explore this activity.

Keywords: Erythrocyte Membrane Stabilization and Proteinase Inhibition, Protein Denaturation, Zolpidem.

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INTRODUCTION

Hypno-sedatives

Hypno-sedatives are a wide range of drugs that are commonly used for their calming or sleep-inducing property. They specifically activate the inhibitory $GABA_A$ receptors in the midbrain and limbic system leading to CNS (Central Nervous System) depression. Hypno-sedatives are principally divided into three main classes: Barbiturates, Benzodiazepines (BZD) and the non-benzodiazepine hypnotics (also known as the 'Z-compounds').

The Z compounds in popular use are Zopiclone, Zolpidem, Zaleplon, Etizolam and Eszopiclone. They bind selectively to the α_1 subtype of the central GABA receptors which is responsible for their effects on consciousness and memory. These compounds also show weak muscle relaxant, anti-convulsant and anti-anxiety effects as an unintended result of their central action (Durand *et al.*, 1992). The Z compounds have replaced BZD as the drug of choice for the treatment of non-refractory insomnia due to their low addictive potential and wide therapeutic index (Thénot *et al.*, 1988; Holm & Goa, 2000). Cardiac and respiratory suppression, poisoning and rebound insomnia on discontinuation as seen with barbiturates or BZDs are also reduced with Z compounds. In addition, their shorter duration of action translates to reduction in morning drowsiness and disorientation (Hoehns & Perry, 1993; Quera-Salva *et al.*, 1994). They are the drug of choice for sleep-maintenance insomnia (Langtry & Benfield, 1990).

Zolpidem

Zolpidem, with the chemical composition N, N-dimethyl-2-[6-methy-2-(4-methlyphenyl) imidazol[1,2-a] pyridine-3-yl] acetamide, is structurally an Imidazopyridine (Salvà & Costa, 1995). It is absorbed from the gastrointestinal tract and undergoes first pass hepatic metabolism to result in a bioavailability of 70%. Admission of the standard therapeutic dose produces a serum concentration between 30 µg/L and 300 µg/L. It is rapidly eliminated from breastmilk (Pons *et al.*, 1989). As a hypnotic, it shortens the latency and prolongs the duration of sleep. Although rare, Zolpidem has been known to have some effects on anterograde memory such as delayed free recall. It is reported to have caused an increase in apnea in predisposed individuals and hence is contraindicated in patients with obstructive sleep apnea.

Non-BZD hypnotic Zolpidem and BZDs, though chemically unrelated, have analogous sedative, hypnotic and amnesic properties. Several BZDs have been known to display anti-inflammatory and anti-oxidant action and hence the same can be suspected of the Z compounds. The antioxidant and antinociceptive effects of Z compounds especially that of Zolpidem and Zopiclone have been proved both *in vivo* and *in*

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silico (Bortoli *et al.*, 2019; Yousefsani *et al.*, 2020; Pick *et al.*, 2005). Its anti-inflammatory effect has not been explored. As of yet, there is no sound, well recognized, molecular pharmacological basis or clinical proof for the anti-inflammatory effects of Zolpidem. Unlike benzodiazepines, Zolpidem is neuroprotective and has been recorded to improve speech, cognition and motor function in human patients with severe brain injury (García-Santos *et al.*, 2004).

Inflammation which forms a part of most disease pathologies commonly involves enzyme activation, cytokine and chemokine release, chemotaxis, tissue collapse and repair. It is instrumental in the development of neurodegenerative diseases such as Alzheimer Disease (AD), Parkinson Disease (PD), Huntington Disease (HD), etc. This further raises the question of possible applications of Zolpidem as a neuroprotective agent in the above-mentioned diseases.

MATERIALS AND METHODS

The Institutional Ethics Committee (IEC) approval was obtained prior to the performance of the below mentioned procedures.

Materials

An undiluted sample of Zolpidem and the below mentioned reagents used in the study were procured via the institutional pharmacy and laboratory respectively from verified providers and standardized and accepted procedures were used to process the same.

Procedure

Three methods were used: Protein Denaturation, Erythrocyte Membrane Stabilization and Proteinase Inhibition Assays with Aspirin (Acetyl Salicylic acid) used as the control.

Protein denaturation assay

500 µL of 1% BSA (Bovine Serum Albumin) was taken and 100 µL of Zolpidem was added (Mizushima & Kobayashi, 1968). The mixture was then incubated for 10 min at 37°C following which the contents were warmed up in a water bath at 51°C for 20 min. The solution was cooled. Absorbance was estimated at 660 nm. This was repeated at different concentrations (3.90, 7.81, 9.62, 31.25, 62.5, 125, 250, 500, 1000 and 2000 µg/mL) of the test solution in triplicate (Image 1.1, 1.2, Figure 1) and compared with the positive control of corresponding concentration (Table 1). The inhibition of protein denaturation was estimated.

% of Inhibition = [(OD of test-OD of control)/OD of test] x 100

RBC membrane stabilization assay

Procedure

Blood was obtained from a healthy human volunteer with no history of consumption of drugs or materials with known anti-inflammatory activity for at least 2 weeks (Garcia loía-lópez *et al.*, 2021). This was mixed with same amount of Alsever's solution (composed of 2% dextrose, 0.7% sodium citrate, 0.5% citric acid and 0.4% NaCl). This mixture was placed in the centrifuge for 10 min at 3,000 rpm. It was then washed three times with saline. The erythrocyte layer was then extracted. Phosphate Buffer Saline (PBS) was added to dilute the solution to 10%. Following which 100 µL of the solution was added to 100 µL of the Zolpidem sample. This was heated at 56°C for 30 min, cooled and placed in the centrifuge at 2,000 rpm for 10 min. Post centrifugation, the absorbance of the clear supernatant was recorded at 560 nm. This process was repeated at different concentrations of the test sample (Zolpidem) and the standard (ASA) by varying the amount of solution used across 100, 200, 400 and 800 µg (Image 1.3, 1.4, Figure 2). Membrane stabilization percentage was calculated:

% of inhibition = (Abs of control-Abs of test sample / Abs of control) x 100

Proteinase inhibition assay (trypsin inhibition)

Procedure

Varying concentrations (100, 200, 400 and 800 µg) of Zolpidem (test sample) and ASA (standard) were taken along with 100 µL of 1% bovine serum albumin (Sakat *et al.*, 2010). After keeping in room temperature for 5 min, 250 µL of Trypsin was introduced to halt the reaction. This mixture was centrifuged. The absorbance was measured for the supernatant at 210 nm (Image 1.5, 1.6, Figure 3). The proteinase inhibition percentage was estimated.

% of inhibition = (Abs of control-Abs of test sample / Abs of control) x 100

Statistical analysis

The percentage of inhibition was expressed as mean ± standard deviation.

OBSERVATION

The assays evaluated the anti-inflammatory action of Zolpidem against a drug with well-known anti-inflammatory activity, Acetyl salicylic acid, at various key steps of the inflammatory pathway. As depicted graphically, both Zolpidem and ASA exhibit dose dependent anti-inflammatory property.

Albumin Denaturation Assay

Denaturation of proteins forms an important mechanism in the process of inflammation. Anti-inflammatory drugs such as ASA, Phenybutazone inhibit protein denaturation induced by heat. Zolpidem was also similarly effective in preventing albumin denaturation. It showed maximal inhibition at 800 g/mL (88.34 ± 0.09%). The IC_{50} of the Zolpidem and that of Aspirin was found to be was 23.14 µg/mL and 8.58 µg/mL respectively.

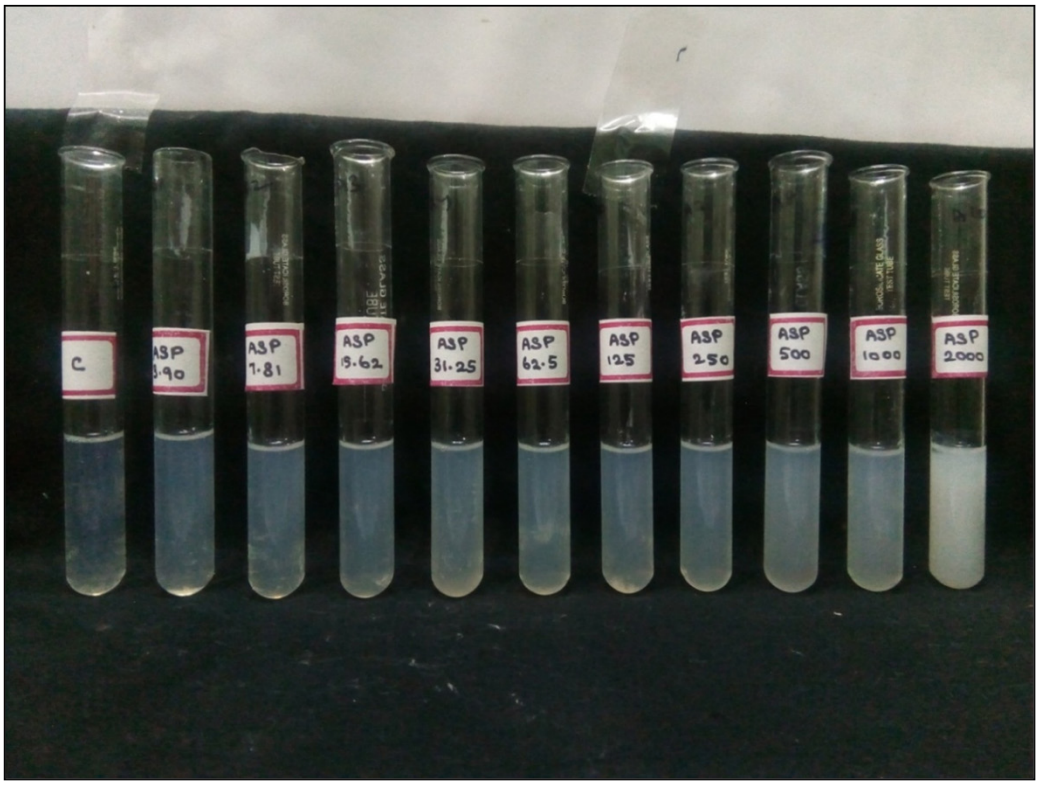

Image 1.1: Protein denaturation assay-Positive control model-Aspirin.

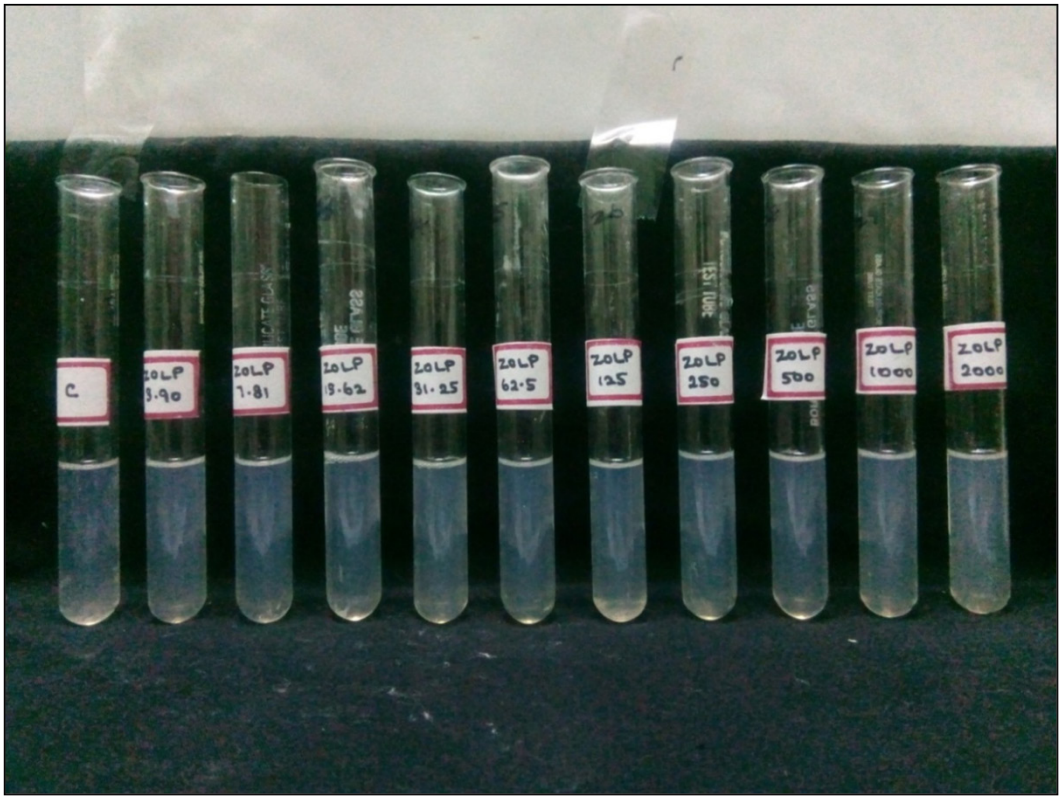

Image 1.2: Protein denaturation assay-Test solution–Zolpidem.

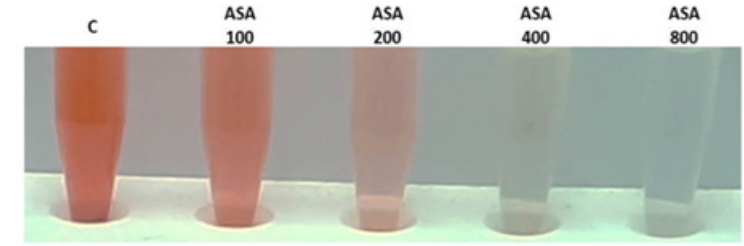

Image 1.3: RBC membrane stabilization assay-Positive control model-Aspirin.

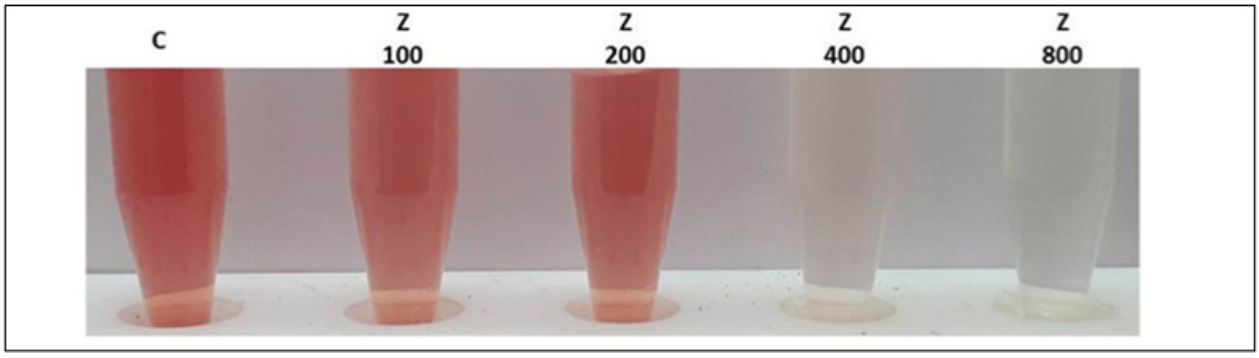

Image 1.4: RBC membrane stabilization assay-Test solution–Zolpidem.

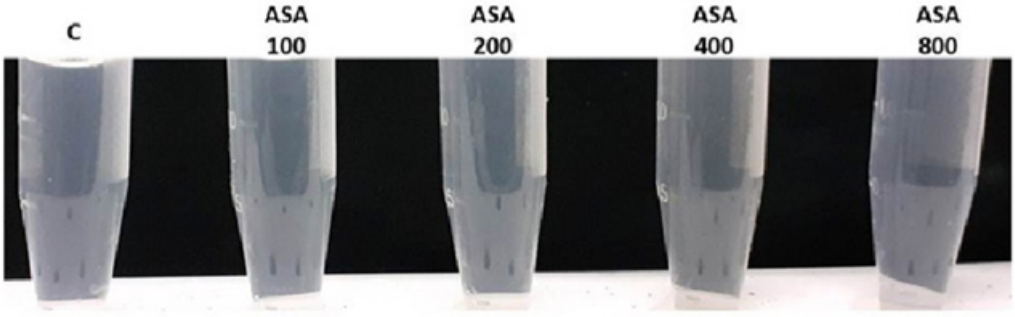

Image 1.5: Proteinase inhibition assay-Positive control model-Aspirin.

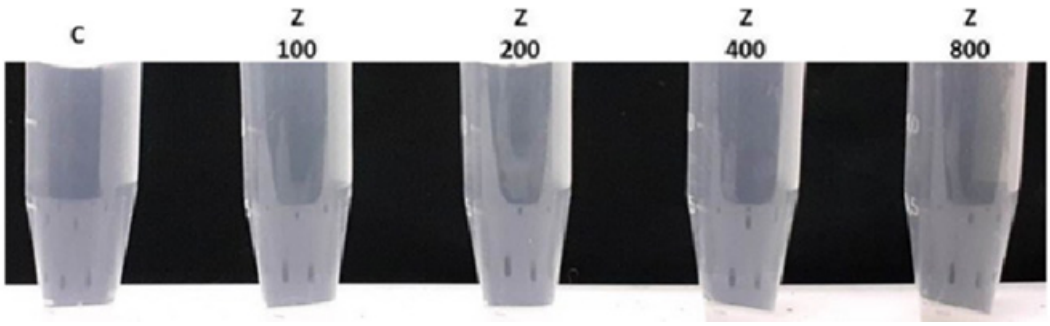

Image 1.6: Proteinase inhibition assay-Test solution-Zolpidem.

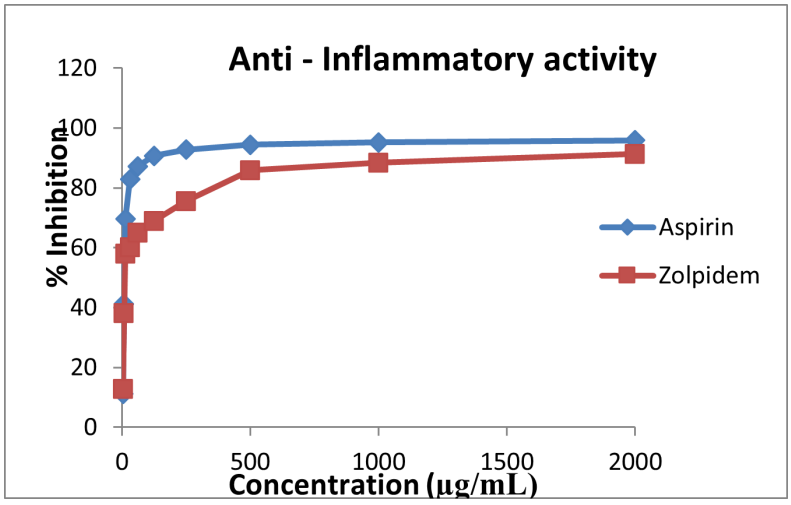

Figure 1: Protein denaturation assay.

Table 1: % Inhibition of Zolpidem in *in vitro* anti-inflammatory assays.

Test sample	Concentration	% inhibition		
		Albumin denaturation	RBC Membrane stabilization	Proteinase inhibition
Zolpidem	100	68.86±0.57	10.57±0.21	8.07±0.21
	200	75.50±0.38	23.95±0.63	19.38±0.63
	400	85.82±0.17	72.38±0.31	68.02±0.31
	800	88.34±0.09	82.41±0.41	79.20±0.41

RBC Membrane Stabilization Test

Ability of the compound to inhibit heat induced hemolysis is evaluated. Considering the shared characteristics between the RBC membrane and the lysosomal membrane, the property of RBC membrane stabilization also alludes to the stabilization of neutrophilic lysosomal membrane and hence the inhibition of the release of its contents at the site of inflammation. These lysosomal granules contain proteases, myeloperoxidases which worsen the damage. Maximum inhibition of 82.41 ± 0.41% was observed at 800 g/mL. The IC_{50} value of the Zolpidem and that of Aspirin was found to be was 303.34 µg/mL and 220.87 µg/mL respectively.

Proteinase Inhibition Test

Granulocytes contain a vast amount of serine proteases in their lysosomal granules. This causes tissue damage during the inflammation process. Highest amount of inhibition (79.20 ± 0.41%) was seen at 800 g/mL. Zolpidem showed an IC_{50} value of 334.38 µg/mL while Aspirin showed a value of 240.11 µg/mL.

Zolpidem exhibited dose dependent anti-inflammatory effect, increasing with dose in a sigmoidal fashion. Zolpidem has moderate anti-inflammatory activity at sub-therapeutic concentrations. At higher concentrations, it exhibits anti-inflammatory activity comparable to that of Aspirin.

DISCUSSION

The anti-oxidant activity of Zolpidem by free radical scavenging has been established in silico using the hydrogen atom transfer mechanism (Yousefsani *et al.*, 2020). A similar conclusion was reached in another study recording the role of Zolpidem in post-Cisplatin renal damage in mouse model where Zolpidem addition improved renal function (Hasanvand *et al.*, 2018).

As per the assays performed in this study, it is found that Zolpidem possesses anti-inflammatory properties mediated by the inhibition of protein denaturation, stabilization of the neutrophil lysosomal membrane, thus preventing the release of inflammatory mediators and by the inhibition of proteases which cause cellular protein damage during inflammation. It can hence be theorized that this retards the inflammation process and is cytoprotective. Proteinase plays an important role in inflammation and its inhibition protects the cells from damage (Das & Chatterjee, 1995). Membrane stabilization of the erythrocyte prevents the leakage of serum proteins and fluids (Yesmin *et al.*, 2020). Trypsin inhibition alleviated the oxidative stress and inflammation in Dextran sulfate sodium induced mice colitis (Jia *et al.*, 2022).

Inflammation forms the basis of several disease processes and is of current relevance in neurodegenerative diseases (Chen *et al.*, 2017). Regardless of etiology, the pathophysiology is common

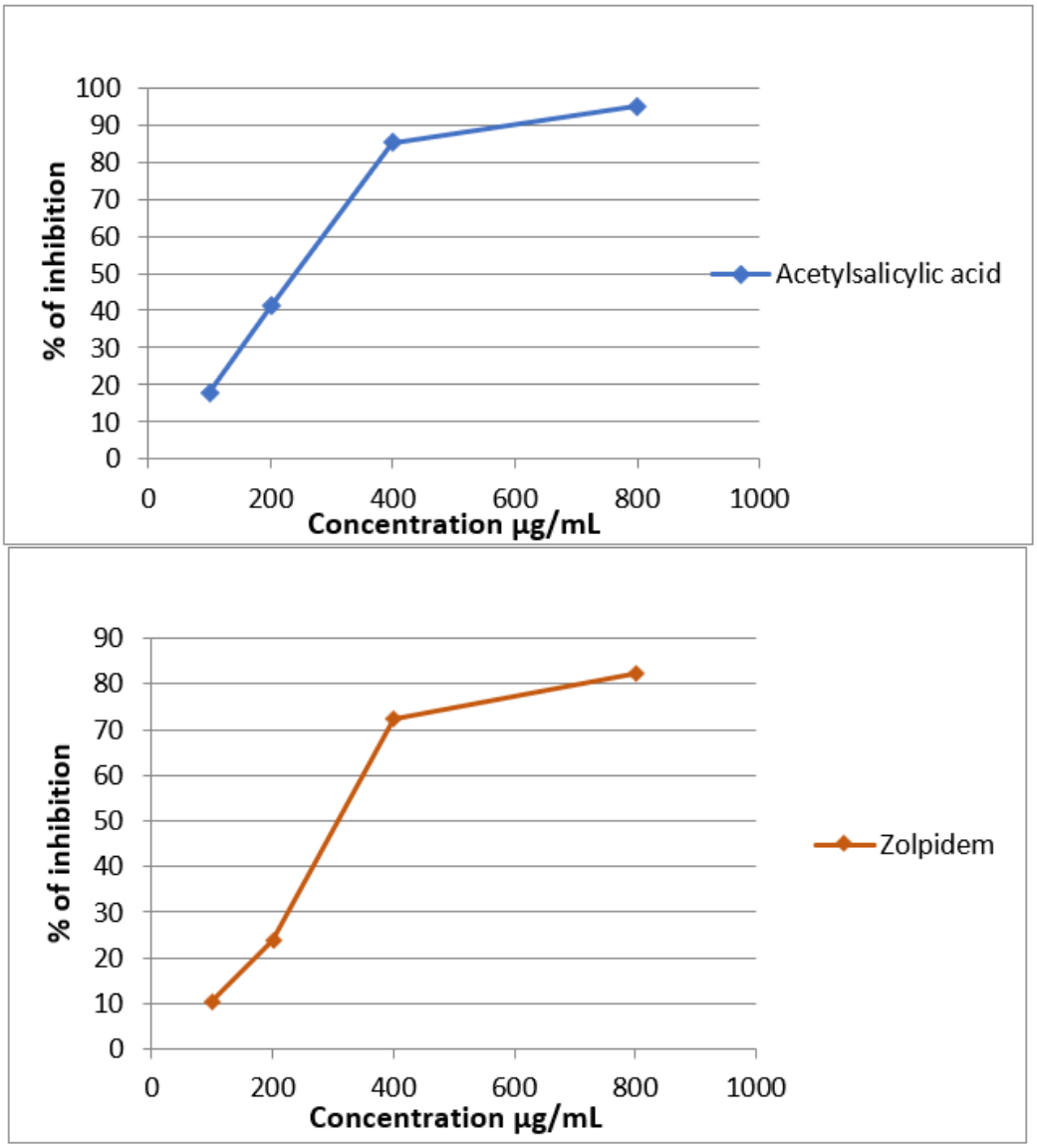

Figure 2: RBC Membrane Stabilization assay.

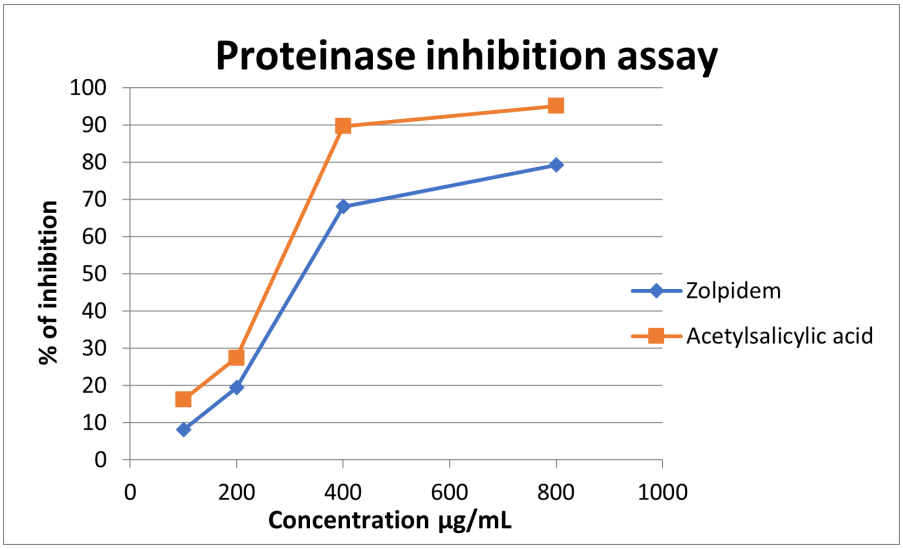

Figure 3: Proteinase Inhibition assay.

across most neurodegenerative diseases, it includes toxic damage and loss of neuronal function initiated by deposition of non-degradable protein products (Lamptey *et al.*, 2022; Ayeni *et al.*, 2022). The role of these inclusions (Aβ) in awakening of the inflammatory response from microglial cells and astrocytes, activation of inflammatory cascade and leading to oxidative injury by tau phosphorylation is well studied in case of AD (Lamptey *et al.*, 2022; Miao *et al.*, 2023).

NSAIDs have long been studied for their possible protective effects against neuronal injury in AD but its results are contradictory (Vlad *et al.*, 2008). The results of this study prove the effectiveness of Zolpidem at blocking key steps involved in the inflammatory pathways which form a part of the above-mentioned conditions. It is hence worth exploring Zolpidem as a possible adjunct in the treatment of neurodegenerative disorders with a stress on Alzheimer's disease (Bomalaski *et al.*, 2017).

CONCLUSION

This research was undertaken to analyze the anti-inflammatory effect of Zolpidem. Keeping in mind the previously identified neuroprotective and antinociceptive effects of Zolpidem, its newly identified anti-inflammatory capacity could be very beneficial in formulating cutting-edge protocols for neurodegenerative diseases which share a ubiquitous feature of chronic aberrant inflammation (like Alzheimer's disease). Zolpidem has selective action on the CNS and hence has great potential for use in several psychiatric and neurological disorders as an adjuvant in either slowing or arresting disease progression. Owing to the higher margin of safety and low day time sedation, it is safer for long term use in elder individuals at risk for neurodegenerative disorders. Understanding how Zolpidem fights inflammation is important for finding which diseases it would help in healing. Further investigations are required to find the active components and receptors involved in the same. As the pathways for the anti-inflammatory effect of the drug remain unknown, it may show possible immunosuppressive effects in clinical settings and hence may be contraindicated in immune-deficient or postoperative states.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethics approval was obtained from the Institutional Ethics Committee in 2019.

ABBREVIATIONS

AD: Alzheimer's Disease; **ASA:** Acetyl Salicylic Acid; **BZD:** Benzodiazepines; **CNS:** Central Nervous System; **GABA:** Gamma-Aminobutyric Acid; **HD:** Huntington Disease; **IC_{50}:** Half Maximal Inhibitory Concentration; **IEC:** Institutional Ethics Committee; **NSAID:** Non-Steroidal Anti-Inflammatory Drug; **PBS:** Phosphate Buffer Saline; **PD:** Parkinson Disease; **RBC:** Red Blood Cell.

AUTHORS CONTRIBUTIONS

All the authors contributed equally to this work. The authors confirm contribution to the paper as follows: study conception and design by RHEA SURANA L; Data analysis and interpretation of results by KAVITHA RAMASAMY; Draft Manuscript guidance and preparation by KARTHIKA K. All the authors reviewed the results and approved the final version of the manuscript.

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