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Original Article

Elucidating the Medhya Role of *Lashuna* (*Allium sativum* L) Through *in Silico* Method with Special Reference to Alzheimer's Disease

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ABSTRACT

Introduction: In Ayurveda, *Allium sativum* (*Lashuna*) has been described in great detail as a *Medhya* medication that may improve cognitive function. The current study used molecular docking and *in silico* network pharmacology to clarify the molecular mechanisms behind its neuroprotective function in Alzheimer's disease. **Materials and Methods:** *Allium sativum* phytochemicals were obtained from Dr. Duke's databases and IMPPAT 2.0. ADME and drug-likeness screening were then performed using SwissADME, BindingDB and UniProtKB were used for target prediction. GeneCards and the Human Protein Atlas provided disease-associated targets, and Venny 2.1.0 was used to find overlapping targets. **Results:** A total of 181 targets, or 34 bioactive phytochemicals, were found; 79 of these targets overlapped with Alzheimer's disease. Important hub genes, including as GRM1, GRM5, and GRIN2B, were identified by Protein-Protein Interaction (PPI) studies. Significant participation in glutamatergic synapses, PI3K/Akt signaling pathways, and neuroactive ligand-receptor interactions was revealed by KEGG pathway analysis. Strong target interactions were shown by substances like apigenin, kaempferol, and quercetin, according to network topology. Molecular docking studies showed that Mocetinostat had the highest binding affinity, particularly with GRM1 (-11.6 kcal/mol), followed by quercetin and kaempferol, suggesting strong ligand-target interactions. **Discussion:** Mechanistically, these compounds may exert neuroprotective effects through antioxidant, anti-inflammatory, anti-amyloidogenic, and epigenetic modulation pathways. In conclusion, *Allium sativum* demonstrates multi-targeted therapeutic potential against Alzheimer's disease through modulation of key genes and signaling pathways. **Conclusion:** However, further *in vitro, in vivo*, and clinical studies are required to validate these findings and establish its efficacy as a therapeutic agent.

Keywords: Alzheimer's Disease, *In Silico*, *Lashuna*, Molecular Docking.

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INTRODUCTION

Memory power is ultimate as it is needed for all the living beings to remember the day-to-day activities and to continue them without any flaw. Alzheimer and Dementia are two common diseases that are prevalent among the individuals. While the Dementia is an overall term for a particular group of symptoms such as difficulty in decision making, confusion, trouble memory, struggling to understand and express his/her thoughts. Alzheimer disease is one cause for dementia symptom. Dementia is a symptom in many different conditions, while Alzheimer is caused when there is an accumulation of Beta-amyloid protein, Protein tau and inflammatory changes which subsequently lead to inability in usage of glucose (Roach *et al*., 2025).

An estimated 50 million people are diagnosed with the disease around worldwide and it is estimated that by 2050 the number will rise up to 152 million. These people age is commonly 65 and above, but the development of the Beta-amyloid proteins starts averagely at the age of 22 years. Four stages of Alzheimer are currently classified and the major symptoms are absence of clinical signs, loss of concentration, trouble recognizing family members and may be bedridden as the stages of Alzheimer goes from Stage 1 to stage 4 (Breijyeh *et al*., 2020). Dementia is prevalent in 47 million people and this number may go up to 131million by 2050. Dementia is confirmed in a person when he/she has loss cognitive abilities such as language, visual and execution (Arvanitakis *et al*., 2019). Currently there is no permanent cure for the disease, the medications give symptomatic relief. Donepezil, Rivastigmine, NMDA antagonist, Lecanemab however decreases the symptoms but it produces severe side-effects such as nausea, tremors, hallucinations and adverse neurological events (Hafiz *et al*.,

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2023). Newer drugs with both safety and efficacy is essentially required for managing these diseases at least at the early stages of the disease.

Lashuna (Allium sativum L.) in *Ayurveda* is told as *Medhya* (Promotes intellect) by various *samhitas* such as *Charaka, Sushruta and Vagbhata Samhita.* Even *Nighantus* like *Bhava Prakash, Raja Nighantu* have also mentioned *Lashuna's* action as *Medhya* (Shaik *et al*., 2016). *Lashuna (Allium sativum L.)* has various pharmacological actions such as Anti-inflammatory, Anti-oxidant, Anti-Alzheimer and Neuro-protective activity mentioned through various in-vitor and *in vivo* researches (Okoro *et al*., 2023). These evidences collectively represent the action of the drug in the disease, the main gap left and unexplained is the mechanism of action. To clarify the role of the drug in the disease and to analyze the mechanism of action we thought of using *in silico* method to find out its mechanism.

MATERIALS AND METHODS

Ethical statement

Since the preparation, *in vitro* and *in silico* investigation doesn't involve any human or animal participants the permission from Institutional Ethical Committee and Institutional Animal Ethical Committee was not required.

Obtaining Phytochemicals and targets related to plant

The phytochemicals related to *Lashuna* were retrieved from IMPPAT 2.0 (Indian Medicinal Plants, Phytochemistry, and Therapeutics 2.0) and Dr. Duke's Phytochemical and Ethnobotanical Databases with the term "*Allium sativum*" and part used as *"Bulb"* (Lans *et al*., 2020; Mohanraj *et al*., 2018). The phytochemicals compound ID number was retrieved from Pub Chem database (Kim *et al*., 2016). Followed by this smilies related to phytochemicals were obtained from Swiss ADME, the phytochemicals were then filtered on the basis of Drug likeliness and Oral bio availability (Daina *et al*., 2017). With similarity score of $\geq$ 0.85 the target related to the drug were procured from Binding DB (Liu *et al*., 2007). The Gene ID of these targets were acquired from UniProt KB for further assessment (Apweiler *et al*., 2004).

Acquiring common targets

Gene cards and Human Protein Atlas (HPA) was used to acquire targets related to the disease. (Thul *et al*., 2018; Stelzer *et al*., 2016). "Alzheimer's" was the term used in the databases and the targets related to the disease were obtained. The Phyto-targets obtained and disease targets were uploaded in Venny 2.1.0 for finding the common targets related to disease and the plant. (https://bioinfogp.cnb.csic.es/tools/venny/).

Protein interaction, Hub genes and Pathway analysis

With medium confidence of 0.7 as FDR the common targets were put into STRING platform for interaction of the obtained genes was obtained (Szklarczyk *et al*., 2023). We chose the species as "*Homo sapiens*" and the PPI was obtained. The constructed network was then sent to Cytoscape 3.10.3 for further construction and with CytoHubba the top 10 genes were calculated (Chin *et al*., 2014; Shannon *et al*., 2003).

Pathway analysis

The KEGG pathway (Kyoto Encyclopedia of Genes and Genomes) was assessed on the basis of obtained genes from the STRING platform. Analysis of Top 10 pathways were done to interpret how the targets are involved in the disease pathway.

Network construction

The common targets, pathway and phytochemicals of the common targets were uploaded into the Cytoscape 3.10.3 to construct the Network. The topological examination was performed using the network analyser. The network was examined in terms of degree strength in order to determine the role and relationship between the active phytochemicals and the overlapping targets Alzheimer's.

Molecular Docking

The crystal structures of the top three compounds and the three most well-known target proteins were obtained from the RSCB PDB and Pub Chem databases, respectively (Berman *et al*., 2000). We used Biovia Discovery Studio to eliminate the hetero-atoms in order to stabilize the protein structures. Using the Auto dock tool 1.5.7, the hydrogen bond and Kollman charges were added (Morris *et al*., 2009). The docking technique was used to estimate the binding interaction of top target proteins and ligands, while Py Rx software was utilized to automatically identify binding sites (Dallakyan *et al*., 2015). The interaction with the best docking scores in 2D and 3D structures has been demonstrated using the Biovia Discovery Studio application.

RESULTS

Phytochemicals and Targets

538 phytochemicals were found for the term *"Allium sativum L."* in the IMPPAT and Dr. Duke's Phytochemical and Ethnobotanical Databases. Following the completion of the toxicity and ADME screening utilizing Swiss ADME, a total of 209 phytochemicals were discovered based on the bioavailability, oral absorption, and Lipinski 5 violation. Following the Smiles' screening with Binding DB, the targets were acquired using Uniprot KB, resulting in 181 targets for a total of 34 phytochemicals.

Disease targets and Overlapping targets analysis

8197 targets were acquired from Gene cards and HPA for Alzheimer's. The disease targets and Phyto-targets were uploaded into Venny 2.1.0 and the common targets of Alzheimer -79 targets were obtained. Figure1 shows the common targets of the disease and the plant.45.

PPI Network

With the common targets, both Protein networks were constructed with the help of STRING database. The 79 targets were uploaded into the database and the network was created, among the targets of the Alzheimer's GRIA2 target had the highest interaction with other targets while GRINB2 had the lowest interaction in the protein network. In the Cytohubba top 10 hub genes, GRM1, GRM5 and GRIN2B were the top 3 targets, with a score of 22500000000000 for GRM1 Figure 2 depicts the PPI interaction of the targets. The top 10 genes of the disease were with Cytohubba and it is illustrated in Figure 3.

Pathway analysis

With the overlapping targets of the disease and the plant, the pathway analysis was done. A total of 59 pathways were found to be present in the disease, among which Neuroactive ligand-receptor interaction had highest gene count of 22. The top 10 KEGG pathway was assessed and they were further taken for topological analysis. Few other pathways that were with highest gene count includes, Glutamatergic synapse, Alzheimer disease, cAMP signaling pathway and Amphetamine addiction with gene count of more than 10.

Statistical analysis

Since, network pharmacology and molecular docking techniques rely on computational predictions, database mining, and *in silico* simulations rather than experimental or quantitative comparative data requiring statistical validation, statistical analysis was not carried out in this study.

Topological analysis

The pathway - target, compound - target and plant - compound was uploaded into Cytoscape for topological assessment. 34 chemical compounds, 10 pathways and 79 targets were uploaded and the network was constructed. Quercetin, Glutamic acid, Apigenin and Kaempferol had the highest compound - target interaction while trigonelline, aspartic acid and phenylalanine had the lowest degree layout of 1 Figure 4 shows the topological analysis of the plant, chemical, pathway and target.

Molecular docking

After literature review of the compounds in the network, 5 phytochemicals were selected and then 3 targets with highest ranking were chosen for ligand - target interaction. Among the 5 phytochemicals, Mocetinostat was having the highest binding affinity towards GRM1 with a score of -11.6 kcal/mol and the lowest was for diallyl tetrasulfide towards GRM1 and GRIN2B with binding affinity of -3.5 kcal/mol. The average binding score was on a higher side for mocetinostat -9.6 kcal/mol while diallyl tetrasulfide had lowest of -3.6 kcal/mol Table 1.

DISCUSSION

Lashuna (Allium sativum) bulb in the classical ayurveda is having *Katu rasa* (pungent taste), *Katu vipaka* (pungent post digestive effect) and *ushna virya* (hot potency). The *katu rasa* with its *Bandaschinnati* (breaks the obstruction) and *Margavivarnotti* (clears the channels) will help in the Alzheimer's and it will also help in neuroprotection. *Lashuna*, because of its high concentration of organosulfur compounds including allicin, S-Allyl Cysteine

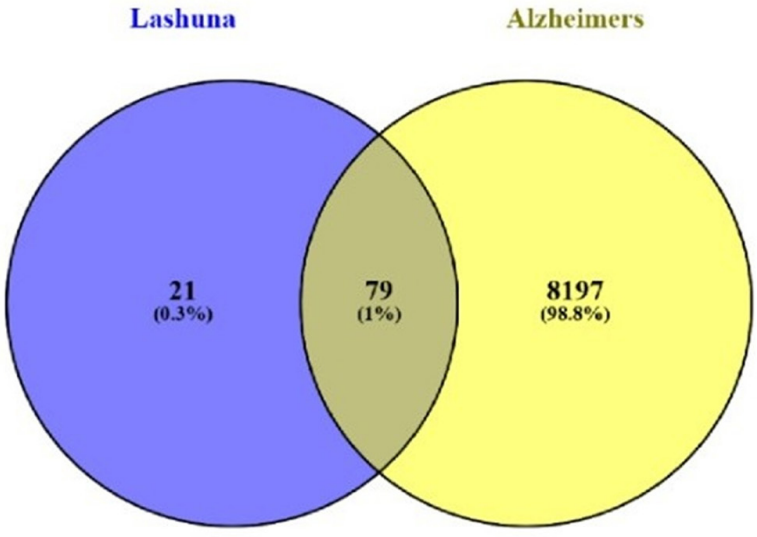


Figure 1: Overlapping targets of *Lashuna* (*Allium sativum* L.) and Alzheimer Disease Targets.

(SAC), and diallyl disulfide, *Allium sativum*, or *garlic*, has notable neuroprotective effects in Alzheimer's disease (Ray *et al.*, 2011). By scavenging reactive oxygen species and boosting endogenous antioxidant enzymes like glutathione peroxidase and superoxide dismutase, these phytochemicals exhibit strong antioxidant activity and lower oxidative stress, a crucial component of AD pathogenesis. By preventing Amyloid-β (Aβ) aggregation and lowering plaque formation while concurrently shielding neurons from Aβ-induced damage, garlic also exhibits anti-amyloidogenic properties (Borek *et al.*, 2001). So because of the phytochemicals and the ayurvedic pharmacology perspective it is suggested that *Lashuna* has properties that help in neuroprotection. ven in the *in silico* analysis, the results suggested that the phytochemicals have role on the disease.

Mocetinostat is a selective class I/ IV Histone Deacetylase (HDAC) inhibitor that exerts neuroprotective potential primarily through epigenetic modulation of gene expression. HDAC inhibition leads to histone acetylation, thereby activating transcription of neuroprotective genes, including those encoding Brain-Derived Neurotrophic Factor (BDNF) and Glial - Derieved Neurotrophic Factor (GDNF), which support neuronal survival, synaptic plasticity and cognitive function (Wu *et al.*, 2013). In neurodegenerative conditions such as Alzheimer's disease, HDAC inhibitors have been shown to reduce neuroinflammation by suppressing microglial activation and pro-inflammatory mediators, while also improving memory and neuronal resilience (Khathayer *et al.*, 2024). Furthermore, mocetinostat boosts anti-apoptotic processes and modulates important signalling pathways including PI3K/Akt, indicating its potential to prevent neuronal loss and reverse epigenetic dysregulation linked to Alzheimer's pathogenesis. Mocetinostat's class impact as an HDAC inhibitor makes it a prospective treatment candidate that targets the neuroinflammatory and epigenetic aspects of neurodegeneration, despite the paucity of direct Alzheimer's-specific trials.

Quercetin, a naturally occurring flavonoid, demonstrates significant neuroprotective effects in Alzheimer's disease through its multi-targeted pharmacological actions. It reduces oxidative stress by scavenging reactive oxygen species and restoring mitochondrial membrane potential, thereby preserving neuronal integrity (Khan *et al.*, 2020). Quercetin also inhibits amyloid - beta aggregation and fibril formation, decreases beta secretase

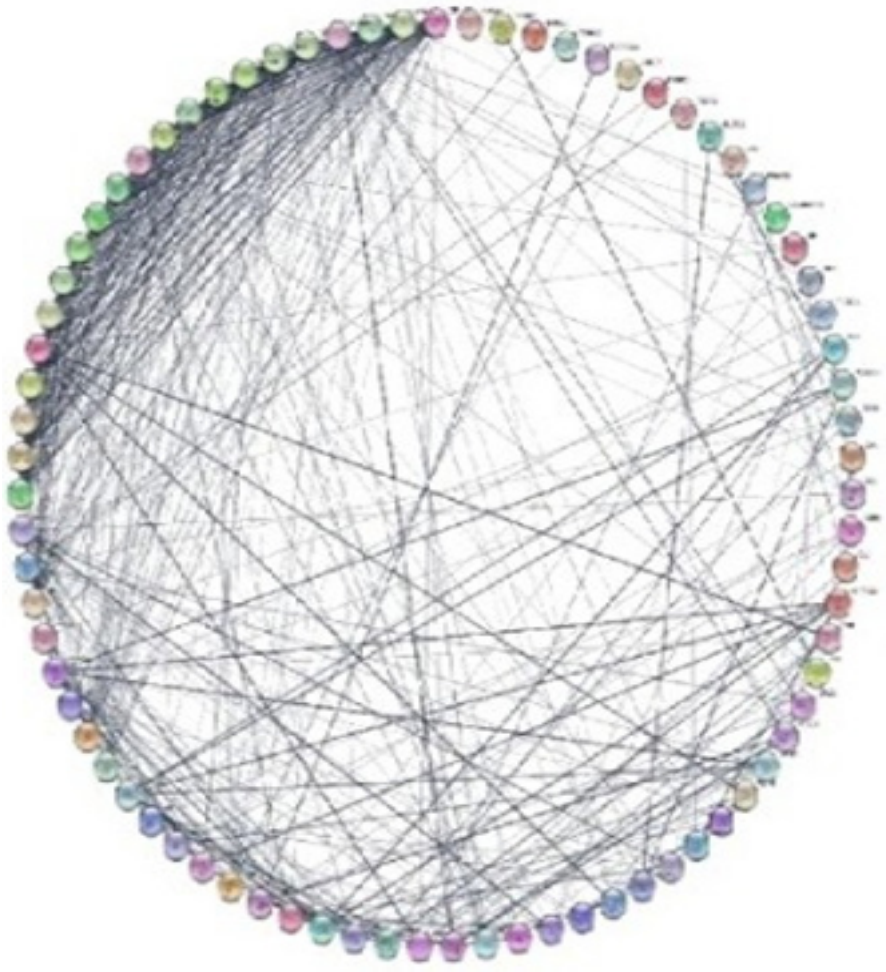


Figure 2: Protein Interaction of the Overlapping Targets.

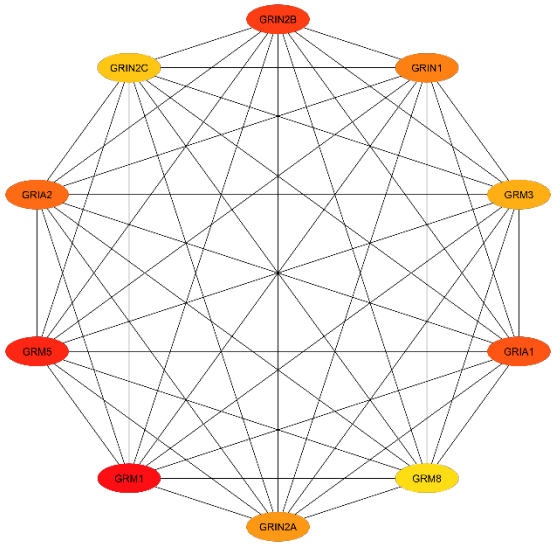


Figure 3: Top 10 Hub Genes of the Overlapping Targets.

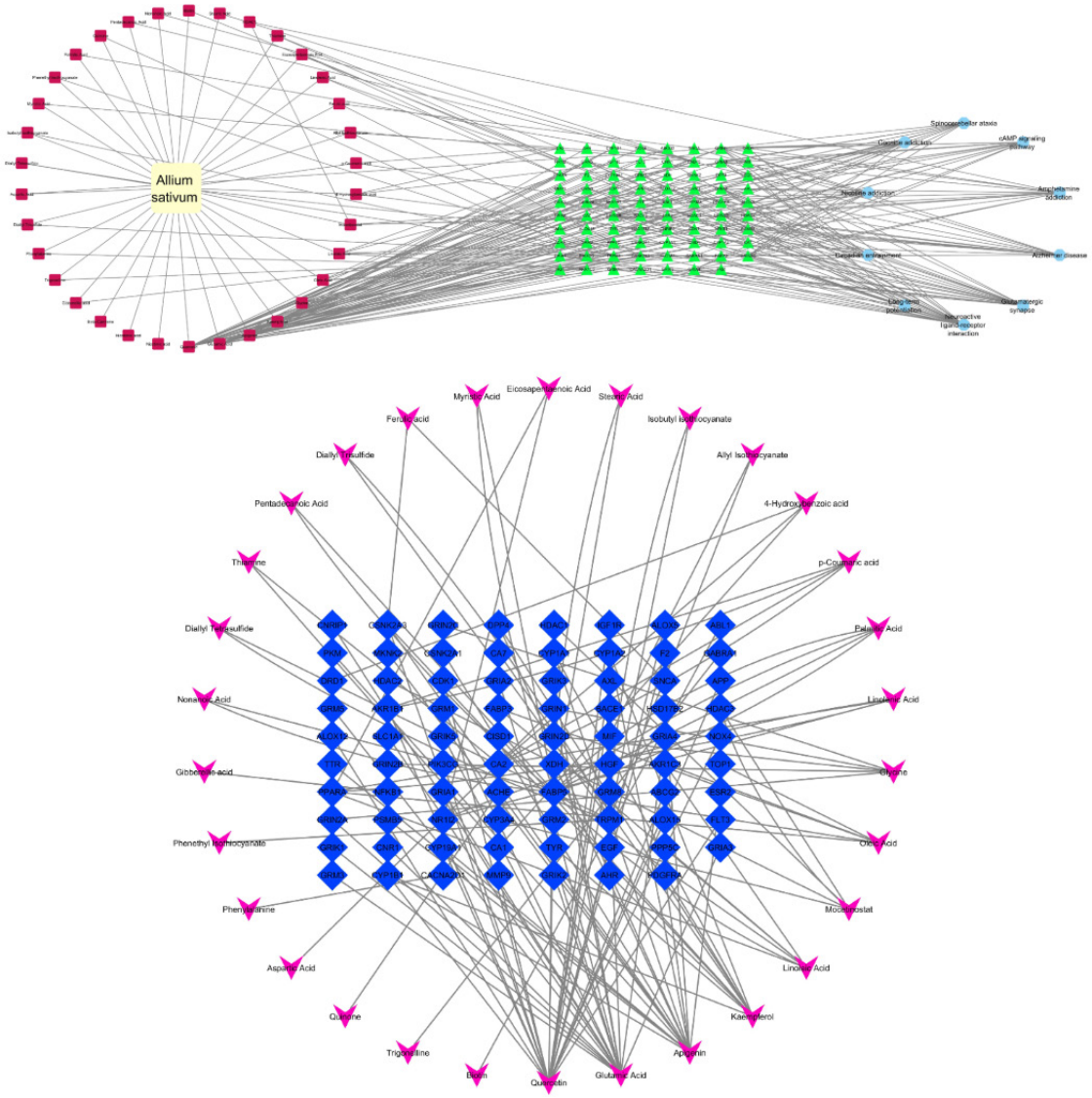


Figure 4: Topological analysis of Pathway-Target-Compound.

Table 1: Binding affinity of targets and ligand for "Alzheimer's".

Phytochemicals	Binding Affinity (kcal/mol)		
	GRM5	GRM1	GRIN2B
Quercetin	-7.4	-10.3	-8.3
Kaempferol	-7.9	-9.8	-7.6
Diallyl tetrasulfide	-3.9	-3.5	-3.5
Rivastigmine	-5.9	-6.4	-5.2
Mocetinostat	-8.8	-11.6	-8.6

activity and reduces tau protein hyperphosporylation, addressing key pathological hallmarks of Alzheimer's disease. Furthermore, it suppresses neuroinflammation via downregulation of NF-kB and pro-inflammaotry cytokines, enhances cholinergic transmission through acetylcholinesterase inhibition and activates pro-survival signalling pathways such as PI3K/ Akt (Adnan *et al.*, 2025). Quercetin has the potential to be a multi-mechanistic neuroprotective drug in the treatment of Alzheimer's disease because experimental studies have demonstrated that it enhances neuronal survival, lowers Aβ-induced toxicity, and promotes cognitive performance.

Dysregulation of the GRM1 gene, which codes for Metabotropic Glutamate Receptor-1 (mGluR1), contributes to the pathophysiology of Alzheimer's Disease (AD) and is important for synaptic transmission and neuronal survival. The G-protein coupled receptor mGluR1 influences learning, memory, and synaptic plasticity by modulating intracellular calcium signaling through the phospholipase C-IP_3 pathway (Nicoletti *et al.*, 2011). Overactivation of mGluR1 causes elevated intracellular Ca2+ levels, mitochondrial malfunction, and neuronal death in AD. Excitotoxicity is caused by excessive glutamatergic activity. Additionally, it increases cholinergic transmission by inhibiting acetylcholinesterase, decreases neuroinflammation by downregulating NF-κB and pro-inflammatory cytokines, and activates pro-survival signaling pathways including PI3K/ Akt (Ferreira *et al.*, 2016). Quercetin has the potential to be a multi-mechanistic neuroprotective drug in the treatment of Alzheimer's disease because experimental studies have demonstrated that it enhances neuronal survival, lowers Aβ-induced toxicity, and promotes cognitive performance.

A network of signaling cascades, including PI3K/Akt, MAPK, p53, Wnt/β-catenin, and apoptotic pathways, are included in the pathways in cancer. While these pathways are mostly linked to carcinogenesis, they also play important roles in the pathogenesis of Alzheimer's Disease (AD). In AD, their dysregulation frequently results in increased neuronal death and neurodegeneration, but in cancer, these pathways support unchecked cell proliferation, survival, and resistance to apoptosis (Arendt *et al.*, 2017). For example, aberrant MAPK pathway activation leads to oxidative stress and inflammation, while defective PI3K/Akt signaling in AD decreases neuronal survival and promotes tau hyperphosphorylation via GSK-3β activation. Similar to how p53-mediated apoptotic pathways are suppressed in cancer cells, they are increased in AD, resulting in neuronal death. Amyloid-β toxicity and synaptic dysfunction are caused by the downregulation of Wnt/β-catenin signaling, which is necessary for synaptic plasticity. These similar but inversely regulated systems point to a complicated relationship between AD and cancer, which is sometimes referred to as an inverse comorbidity. When molecular pathways that support cancer cell survival are dysregulated in the brain, they may cause neurodegeneration (Nudelman *et al.*, 2014).

Molecular docking of the phytochemicals especially the Mocetinostat had the highest binding affinity towards all the targets and moreover the neuroprotection activities and the mechanism of action on Alzheimer's also suggests us that this phytochemical has more effect on the disease. Meanwhile, quercetin also had equally good docking score against Alzheimer's targets, with -8.7 kcal/mol as average binding affinity.

Although, the phytochemicals, targets, pathways and the docking suggests that *Lashuna* has its activity on brain, there are few limitations in this study as the databases get updated continuously the phytochemicals and the targets can't be considered as the major evidence that the plant will work in the disease. Furthermore, *in vivo* and clinical evaluation on the plant is needed for making this plant as a single drug that will help in Alzheimer's.

CONCLUSION

Single drug for a single disease is the aim of Ayurvedic treatment known as "*Ekamoolika prayoga*". Although there is very few mentioning of single drug therapy in *Bruhatrayees* in ayurveda when compared to *Laghutrayees*, we need a greater number of single drugs for easier and better prescriptions. Hence, with the reference that *Lashuna* is mentioned as "*Medhya*" the study was performed using bio-informatics. Thus, by performing computational pharmacology we found out that *Lashuna's* phytochemical does have a role on Alzheimer's disease and neuroprotection. But further study on animals and human subjects are needed to validate the computational evidence.

ABBREVIATIONS

Aβ: Amyloid-beta; **AD:** Alzheimer's disease; **ADME:** Absorption, Distribution, Metabolism, and Excretion; **Akt:** Protein kinase B; **BDNF:** Brain-derived neurotrophic factor; **FDR:** False Discovery Rate; **GDNF:** Glial-derived neurotrophic factor; **HDAC:** Histone deacetylase; **HPA:** Human Protein Atlas; **IMPPAT:** Indian Medicinal Plants, Phytochemistry, and Therapeutics; **KEGG:** Kyoto Encyclopedia of Genes and Genomes; **MAPK:** Mitogen-activated protein kinase; **mGluR1:** Metabotropic glutamate receptor-1; **NF-κB:** Nuclear factor kappa B; **PI3K:** Phosphoinositide 3-kinase; **PPI:** Protein-protein interaction; **SAC:** S-allyl cysteine.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHOR CONTRIBUTION

Dr. Jigneshkumar Varu: Collection and/or assembly of data, Data analysis and interpretation, Writing the article, Critical revision of the article.

Dr. Dr Pradeep Shahajirao Shindhe: Research concept and design, Data analysis and interpretation, Final approval of the article.

Dr. Ramesh Shivappa Killedar: Data analysis and interpretation, Critical revision of the article.

Dr. Aniket Ughade: Collection and/or assembly of data.

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