The influence of Ayurvedic medicinal plants on neurodegenerative health development in Alzheimer's patients

Sarah Adkar

Abstract

Alzheimer's disease (AD) is a devastating and progressive neurodegenerative disorder. Its profound impact is underscored by statistics showing that millions worldwide are affected, with numbers projected to rise dramatically, imposing immense burdens on patients, caregivers, and healthcare systems. Patients typically experience debilitating memory loss, confusion, difficulty with problem-solving, and a profound decline in their ability to perform daily tasks. The urgent need for effective interventions is clear, especially as current conventional treatments aim to treat symptoms like cognitive decline and behavioral changes, yet are limited by their inability to halt or reverse the underlying progression of the disease. The complex pathology of AD includes the accumulation of beta-amyloid protein (a protein that forms sticky clumps, known as plaques, in the brains of Alzheimer's patients), neurofibrillary tangles, oxidative stress, and chronic neuroinflammation. In light of these limitations, traditional Ayurvedic medicine, known for its holistic approach, may be one alternative or complementary approach to treating AD. Hence, this literature review investigates how traditional Ayurvedic practices, including the use of select herbal remedies, may contribute to brain health and cognitive function relevant to neurodegenerative symptoms and the underlying causes and effects of AD.

Specifically, we examine evidence supporting the neuroprotective potential of **Ashwagandha** (*Withania somnifera),* **Ginger** (*Zingiber officinale* Roscoe), and **Turmeric** (*Curcuma longa*). Investigations spanning computational, *in vitro*, and *in vivo* (animal) models, alongside preliminary human observations, reveal that these herbs and their active compounds exert multifaceted neuroprotective effects. Collectively, these traditional Ayurvedic dietary components appear to address several core pathological mechanisms of AD, offering a multi-targeted approach that may complement or provide alternatives to conventional treatment strategies. This review highlights their potential as valuable dietary interventions for supporting neural health and slowing neurodegeneration in AD patients.

Introduction

Alzheimer's disease (AD) is a devastating and progressive neurodegenerative disorder characterized by a gradual decline in cognitive function, memory, and behavioral abilities. At its core, AD pathology involves the abnormal accumulation of beta-amyloid protein, which forms sticky clumps known as plaques in the brains of affected individuals (Murphy M.P, LeVine, H). Alongside these plaques, the disease is defined by the presence of neurofibrillary tangles, chronic neuroinflammation, and widespread oxidative stress, all contributing to irreversible neuronal damage and loss (Murphy M.P, LeVine, H).). Despite ongoing research, current conventional treatments for AD primarily offer symptomatic relief and have not successfully managed to prevent or reverse the underlying progression of the disease (Tiannopoulou, K.G., Papageorgious, S.G.).

This significant unmet medical need has spurred growing scientific interest in traditional healing systems, particularly Ayurvedic medicine and the rich pharmacopoeia of East Asian herbs/roots. These ancient practices have long emphasized holistic well-being and the use of natural compounds for maintaining health, including cognitive vitality. Among the diverse array of botanicals, Turmeric (Curcuma longa), Ginger (Zingiber officinale Roscoe), and Ashwagandha (Withania somnifera) stand out due to their historical use and accumulating

modern scientific evidence suggesting potent neuroprotective properties through their various bioactive compounds (Kushwah et al., 2023).

For example, turmeric, primarily through its active compound curcumin and its analogs, demonstrates efficacy in directly interfering with amyloid and tau pathology, mitigating neuroinflammation, combating oxidative stress, and even offering diagnostic potential through retinal imaging (Sharifi-Rad, J, et.al.). Ginger, particularly compounds like 6-gingerol, exhibits significant ability to inhibit amyloid-beta-induced apoptosis, reduce oxidative stress and inflammatory responses, and modulate critical cell survival pathways (Zeng et al., 2020). Ashwagandha, an adaptogen, actively reduces amyloid-beta plaques and tau-induced damage, enhances cholinergic transmission (neural communication), boosts antioxidant defenses, and alleviates associated neuropsychiatric symptoms such as anxiety and depression (Mikulska et al., 2023).

Despite growing interest in natural product research, comprehensive literature reviews systematically evaluating the neuroprotective potential of multiple prominent Ayurvedic and East Asian herbs in the context of AD remain limited. While individual herbs like Turmeric have received significant attention, a holistic analysis that compares and contrasts the diverse mechanisms and synergistic potential of several key botanicals (such as Turmeric, Ginger, and Ashwagandha) within a single review is largely lacking. This gap highlights the need for a consolidated overview that synthesizes fragmented research, offering a clearer picture of their collective promise and informing future integrative therapeutic strategies.

In light of these limitations, traditional Ayurvedic medicine, known for its holistic approach, may be one alternative or complementary approach to treating AD. Hence, this literature review aims to systematically explore the scientific evidence regarding the influence of these traditional Ayurvedic dietary practices and select East Asian herbs on neurodegenerative health in the context of Alzheimer's disease. Specifically, this review addresses the following research question: How do traditional Ayurvedic medicines address the prevention and treatment of Alzheimer's disease, particularly concerning neurodegenerative symptoms and its causes and effects? By synthesizing findings from computational, in vitro, in vivo, and preliminary human studies, this paper seeks to elucidate the mechanisms by which these natural interventions may offer a multi-targeted approach to AD pathology, from preventing its onset to alleviating its complex symptoms.

Methods

This literature review was conducted as a narrative synthesis to explore the potential of traditional Ayurvedic medicine and selected East Asian herbs/roots in the context of AD. A search for relevant academic literature was primarily conducted using Google Scholar, supplemented by searches on NIH websites and other scientific repositories. The Google Scholar searches employed various combinations of keywords, including: "Alzheimer's disease," "Ayurveda," "Ayurvedic medicine," "East Asian medicine," "neuroprotection," "cognitive function," "aging," "traditional medicine," "herbal remedies," "Turmeric," "Curcuma longa," "Ginger," "Zingiber officinale," "Ashwagandha," "Withania somnifera'', and “amyloid plaque.”

Inclusion Criteria

The review focused exclusively on experimental trial-based studies, case studies, and computational studies on Alzheimer’s patients published after 2010. No geographic restrictions

or specifics around race, sex, or ethnicity were utilized. Studies were included if they investigated the effects of Ayurvedic practices and selected East Asian herbs/roots specifically on cognitive function, neuroprotection, or outcomes directly related to Alzheimer's disease pathology (e.g., amyloid-beta reduction, tau modulation, neuroinflammation, oxidative stress, or improvements in behavioral and cognitive symptoms). The primary emphasis was on studies providing empirical data from in vitro, in vivo (animal), and human clinical trials.

Literature Review

Ashwagandha

Withania somnifera, or Ashwagandha, stands out for its adaptogenic properties and traditional use as a nerve tonic and rejuvenator. Modern scientific inquiry has increasingly focused on Ashwagandha's potential neuroprotective effects, particularly in the context of neurodegenerative disorders like Alzheimer's disease (AD). Research across various models, from genetically engineered organisms (Murthy & Shyamala, 2024) to human trials (Xing, D et al.), is shedding light on the mechanisms through which Ashwagandha may influence neural health and mitigate neurodegeneration. Preclinical investigations utilizing advanced animal models have provided foundational insights into Ashwagandha's direct impact on key pathological traits of AD.

For instance, Murthy & Shyamala (2023), investigated the neuroprotective effects of Ashwagandha in genetically engineered *Drosophila* (fruit fly) models of human neurodegenerative diseases, addressing the gap in in vivo validation of traditional remedies. The study utilized *Drosophila* models where disease-causing human gene mutations were expressed in specific cell types. *Drosophila* is used because it provides a genetically tractable and rapid *in vivo* model system to study human neurodegenerative diseases, allowing for the expression of observable pathological hallmarks and behavioral deficits that mimic aspects of the human AD related conditions. In the Alzheimer's tauopathy model, researchers induced AD symptoms in flies by expressing a human TauE14 mutant protein in photoreceptor neurons, leading to severe cellular damage and widespread neuronal death, mimicking the destructive processes seen in Alzheimer's brains. Then, the flies were fed with different concentrations of Ashwagandha aqueous root extract mixed with their regular food.

The study found that Ashwagandha root extract significantly extended the lifespan of male *Drosophila* flies, reduced motor dysfunction by the human α-synuclein protein and significantly decreased TauE14-induced microtubular instability, mitotic arrest, and neuronal death in photoreceptor neurons.While neurons don't typically divide, "mitotic arrest" in neurodegeneration signifies an abnormal re-entry into the cell cycle that often precedes neuronal death in diseases like AD, thus contributing to brain tissue loss and cognitive decline. These findings suggest that Ashwagandha, as a multi-potent neuroprotective remedy, could play a crucial role in the treatment of AD by directly addressing key pathological features like tau-induced neuronal damage and supporting overall neuronal health. Its ability to mitigate genetically induced cellular toxicity and improve motor function in these models indicates a broad protective capacity relevant to the complex processes in AD which cause neuronal damage.

Building upon these findings in insect models, research in mammalian models has further elucidated Ashwagandha's impact on core AD pathologies. Specifically, studies using rodent

models have focused on its ability to counteract amyloid-beta accumulation and associated cognitive decline.Kolarsky et al. (2024) investigated the neuroprotective effects of an aqueous extract of Ashwagandha root (WSAq) in the 5xFAD mouse model, which mimics beta-amyloid (Abeta) accumulation in AD. The study included 6-7 month old male and female 5xFAD mice, used in particular because they carry five different human gene mutations associated with familial (early-onset) AD. These mutations lead to an accelerated and severe accumulation of beta-amyloid (Abeta) plaques in their brains at a relatively young age (as early as 2 months), closely mimicking a key pathological hallmark of AD in humans. The mice were tested with potential therapeutic compounds, treated with Ashwagandha WSaq (0.5 mg/mL or 2.5 mg/mL) in their drinking water for four weeks. During the fourth week, spatial memory, anxiety, and depressive symptoms were assessed using the Object Location Memory test, Open Field test, and Forced Swim Test. The study found that both concentrations of Ashwagandha improved spatial memory and reduced both depressive and anxious behaviors in the 5xFAD mice. These behavioral improvements were linked to a reduction in Abeta plaque burden in both the hippocampus and cortex, indicating reduced neuroinflammation. These findings may have limitations, as the amount of ashwagandha extract consumed by each mouse was not recorded; it cannot be concluded whether the amount of Abeta plaque recorded was fully a result of equivalent extract consumption.However, these animal study results suggest that oral ashwagandha treatment may hold promising direction for human individuals with Alzheimer's disease. By simultaneously reducing Abeta plaques, dampening neuroinflammation, and boosting the brain's natural antioxidant defenses, Ashwagandha could improve cognitive function and alleviate associated neuropsychiatric symptoms, thereby offering a comprehensive approach to managing the disease.

In addition to directly addressing amyloid pathology, other rodent studies have explored Ashwagandha's effects on more general mechanisms of cognitive impairment relevant to AD. Shivamurthy et al. (2016) evaluated Ashwagandha protein extract's learning and memory-enhancing activities in scopolamine-induced memory-impaired, and piracetam treated Wistar rats. Scopolamine is a medication used to prevent nausea and vomiting by blocking the action of acetylcholine, in the brain's vomiting center and vestibular system. Piracetam, chemically related to gamma-aminobutyric acid (GABA), is understood to support the improved functioning of cells within the brain and blood vessels. Across 18 rats divided into three groups (scopolamine-only control, piracetam control, and Ashwagandha treatment), Ashwagandha (200mg/kg) significantly improved learning and memory in elevated plus maze and passive avoidance tests. Notably, Ashwagandha's effects were even superior to piracetam in the elevated plus maze. These results strongly suggest that the protein extract of Ashwagandha has significant neurocognitive capabilities, as the treatment enhances the brain's cholinergic system, which is vital for processes like learning and memory. Since AD is characterized by notable deficits in this very system, Ashwagandha's ability to enhance cholinergic transmission (communication between neurons) offers a key mechanism for its potential therapeutic benefit. This indicates its potential as an adjuvant therapy for managing cognitive dysfunctions in AD.

Beyond direct pathological interventions in animal models, Ashwagandha's influence extends to broader neuropsychiatric symptoms and general cognitive enhancement, areas increasingly recognized as important for overall brain health and potentially relevant for delaying the onset or progression of neurodegenerative conditions like AD. A double-blind,

placebo-controlled, crossover study by Xing, D et al. (2025) investigated the acute effects of 400 mg Ashwagandha extract on cognitive function in 13 healthy volunteers. Ashwagandha notably boosted working memory as evidenced by faster reaction times on the Sternberg Task. It also enhanced sustained attention and mitigated mental fatigue during the Psychomotor Vigilance Task, contrasting with the placebo group's performance. While some measures lacked statistical significance, the overall results suggested Ashwagandha's role in improving cognitive performance and reducing fatigue. Although this study involved healthy young adults and not AD patients, understanding how Ashwagandha enhances fundamental cognitive processes in a healthy brain is crucial. Such insights can inform strategies for maintaining cognitive resilience, potentially delaying the onset of mild cognitive impairment, and supporting brain health as a preventative measure against neurodegeneration, areas highly relevant to early intervention in Alzheimer's. The fact that a single dose of ashwagandha can improve working memory is noteworthy, and its ability to reduce mental fatigue points to its potential as a cognitive enhancer. This suggests Ashwagandha could help build cognitive resilience and potentially slow down or prevent the progression of mild cognitive impairment often seen in early Alzheimer's.

In summary, the collective evidence from diverse preclinical and human studies underscores Ashwagandha's multifaceted potential in influencing neurodegenerative health. From directly reducing amyloid-beta plaques and tau-induced damage in genetically engineered models to enhancing cholinergic transmission, boosting antioxidant defenses, and mitigating neuropsychiatric symptoms like anxiety and depression, Ashwagandha demonstrates a broad range of neuroprotective effects. These findings consistently suggest that Ashwagandha could be a valuable dietary intervention or adjuvant therapy for supporting neural health and slowing the progression of neurodegeneration in Alzheimer's patients, addressing both the core pathology and associated quality-of-life concerns.

Turmeric

Among the many herbs revered in Ayurveda, turmeric (*Curcuma longa*) and its primary active compound, curcumin, have garnered significant scientific attention for their potential neuroprotective properties, particularly in the context of Alzheimer's disease (AD). Emerging research highlights curcumin's diverse mechanisms of action, ranging from direct molecular interactions to broad anti-inflammatory and antioxidant effects, suggesting its promising role in addressing the complex pathology of AD (Mishra & Palanivelu, 2023).
Experiments and computational studies have illuminated curcumin's molecular interactions with key pathological drivers.

For example, AD. Su, I. J., et.al. (2020), identified a novel curcumin analog, TML-6, as a potential drug candidate for Alzheimer's disease (AD). Curcumin, from turmeric, has anti-cancer, antioxidant, and anti-inflammatory properties, with recent studies also showing neuroprotective effects. This animal study focused on overcoming the poor bioavailability that limits traditional curcumin's clinical use by structurally modifying it to improve its stability and metabolism. The research involved screening curcumin derivatives using six biomarker platforms, followed by cell biological studies, and an animal model (3x-Tg AD mice). The study found that TML-6 inhibited the creation of amyloid precursor protein (APP) and amyloid beta (Abeta). For an individual with AD, TML-6 could potentially reduce the very amyloid plaques (Abeta) that surround neurons, help clear existing harmful proteins, calm the destructive inflammation, and protect their brain

cells from damage, all of which are critical for counteracting disease progression. In the 3x-Tg AD mouse model, TML-6 treatment significantly improved learning behavior and lowered Abeta accumulation in the brain. This animal research suggests that turmeric's curcumin, particularly through modified analogs like TML-6, could offer a promising approach for Alzheimer's in humans. By addressing amyloid accumulation, inflammation, and aging-related biological processes, these compounds may help to improve cognitive function and slow disease progression. This study on TML-6 highlights a critical advancement,underscoring its strong potential and warranting further investigation in human clinical trials as a path toward more effective therapeutic strategies for AD patients.

While animal studies provide robust evidence, the ultimate validation of a therapeutic agent lies in human trials. Though large-scale human studies on curcumin in AD are still developing, promising initial observations have emerged from case reports. For example, Hishikawa et al. (2012) conducted a case study on three AD patients with severe neuropsychological symptoms of dementia, investigating the effects of turmeric treatment (764 mg/day powder, 100 mg/day curcumin). Over 12 weeks, all patients showed a significant reduction in neuropsychiatric symptoms (NPI-Q scores), including relief from agitation, apathy, anxiety, and depression, which also lessened caregiver burden. One patient's MMSE (Mini-Mental State Examination) score notably improved, and two others began recognizing family members within a year, suggesting turmeric may be a safe and effective adjunct or alternative for managing behavioral symptoms in AD. This case study, despite its small sample size, offers compelling anecdotal evidence that turmeric can significantly improve severe behavioral and psychological symptoms in AD patients, thereby enhancing their quality of life and reducing caregiver burden.

In conclusion, the scientific literature strongly supports the neuroprotective potential of turmeric and its primary constituent, curcumin, in the context of Alzheimer's disease. Through its ability to reduce amyloid plaque burden, modulate inflammation, combat oxidative stress, and even improve behavioral and psychological symptoms, curcumin demonstrates a multi-targeted approach to AD pathology. While challenges related to bioavailability persist, advancements in curcumin analogs like TML-6 are highly promising. The consistent findings across in vitro, rodent, and preliminary human studies collectively highlight turmeric's significant influence on neural health, positioning it as a potent candidate for dietary intervention and further therapeutic development in managing neurodegeneration in Alzheimer's patients..

Ginger

Ginger (Zingiber officinale Roscoe), a widely recognized spice and traditional medicine in Ayurvedic practices, has gained scientific traction for its diverse therapeutic properties,specifically its potential in combating neurodegenerative diseases like Alzheimer's disease (AD). Its neuroprotective capacity is largely attributed to the rich array of bioactive compounds it contains, prominently gingerols and shogaols, which exhibit powerful antioxidant and anti-inflammatory capabilities. Research into ginger's role in AD has spanned various levels, from computational modeling and in-vitro cell studies to animal models, exploring its direct molecular interactions and its broader protective effects on neuronal health.

Across multiple papers, ginger has been investigated as a natural compound with potential benefits against aging and degenerative diseases (Sahardi & Makpol, 2019). More

recently, research has tested the neuroprotective role of ginger on various outcomes, including its impact on amyloid-beta pathology, oxidative stress, inflammation, and cognitive functions.

For example, in another in-vitro study, Kim, D. S. H. L., Kim, J.-Y., & Han, Y. S. (2007), evaluated the neuroprotective effects of selected herbs against direct beta-amyloid (1-42) insult, a key pathogenic factor in Alzheimer's disease. Ginger extracts were tested on two types of neuronal cells: PC12 rat pheochromocytoma cells and primary neuronal cells (derived from embryonic 18-day-old Sprague-Dawley rat fetuses). They exposed the rats' neuronal cells to beta-amyloid (1-42), a specific fragment of the amyloid-beta protein known to be toxic to neurons and implicated in Alzheimer's pathology. The key results showed that ginger protected the rat neuronal cells from beta-amyloid (1-42) insults. These findings strongly suggest that ginger possesses significant neuroprotective properties against the direct toxic effects of beta-amyloid (1-42), a critical biomarker and pathogenic molecule in Alzheimer's disease. The ability of ginger to protect neuronal cells from beta-amyloid insult indicates its potential to control the onset and progression of Alzheimer's disease by mitigating one of the primary causes of neuronal damage in the brain. This positions ginger as a promising source for discovering drug candidates that could offer therapeutic benefits against AD pathology.

Expanding on the direct cellular effects, computational studies have also provided insights into ginger's multi-targeted potential. Azam et al. (2014) conducted a computational study assessing 12 ginger components as potential multi-targeted anti-Alzheimer's drugs. Using molecular docking simulations, they predicted strong binding interactions with 13 AD-related protein targets. All 12 compounds acted as "promiscuous drugs," interacting with multiple targets. Acetylcholinesterase (AChE) was identified as the most promising, showing strong binding and inhibition potential. The study also characterized favorable pharmacokinetic profiles, suggesting good oral bioavailability. This research indicates ginger components hold significant promise as novel multi-targeted AD drug leads, interacting with key targets like AChE, BuChE, TNF-α converting enzyme, COX, NOS, and NMDA receptors, suggesting a comprehensive mechanism of action. This multi-target approach is crucial for addressing the complex pathology of Alzheimer's disease more effectively than single-target drugs, offering a promising avenue for future drug development with potentially fewer adverse effects and improved therapeutic outcomes.

Animal studies further corroborate the in vitro and computational evidence for ginger's efficacy. Moradi et al. (2014) assessed ginger's protective and therapeutic effects in rats where Alzheimer's-like symptoms were induced by aluminum chloride. Their study involved various behavioral tests (e.g., Grid floor Activity Cage, Rotarod, T-Maze) and biochemical analyses of brain tissue. Rats with induced AD exhibited reduced activity, impaired coordination, and cognitive decline, along with decreased acetylcholine (ACh) and increased acetylcholinesterase (AChE) levels, and characteristic amyloid plaques and neurofibrillary tangles compared to the control. Notably, ginger treatment (108 and 216 mg/kg/day), whether administered protectively or therapeutically, led to significant improvements in these behavioral and cognitive deficits. Furthermore, histopathological examination revealed that ginger effectively slowed neurodegeneration, reducing the pathological hallmarks characteristic of AD, with effects comparable to the conventional AD medication Rivastigmine. Ginger demonstrated significant neuroprotective and therapeutic effects against AD in this study. It directly addresses the cholinergic deficit in AD by restoring acetylcholine (ACh) levels and inhibiting acetylcholinesterase (AChE). Beyond this, ginger improved cognitive function, motor coordination, and psychological state, while also ameliorating AD-characteristic amyloid plaques

and neurofibrillary tangles. This multifaceted impact highlights its strong potential as a natural intervention for both preventing and treating AD's complex pathology.

Altogether, the collective evidence from these studies suggests that ginger and its active compounds, particularly 6-gingerol, exert multifaceted neuroprotective effects against Alzheimer's disease. The computational findings further support ginger's potential as a multi-targeted agent, addressing several pathological aspects of AD. While ongoing human research is vital to confirm these promising findings, the current body of evidence positions ginger as a significant natural intervention in the context of neurodegenerative health development in Alzheimer's patients.

Discussion

This literature review comprehensively explored the intersection of traditional Ayurvedic medicine, East Asian herbs, and Alzheimer's disease (AD), revealing significant insights into their potential neuroprotective and therapeutic contributions. The investigation, focusing on Ashwagandha, Ginger, and Turmeric, demonstrates that these botanicals and their bioactive compounds exert multifaceted effects against core AD pathologies. Key insights indicate their ability to mitigate beta-amyloid and tau protein abnormalities, combat oxidative stress and neuroinflammation, modulate cholinergic neurotransmission, and enhance neuronal survival pathways. Furthermore, Ashwagandha shows notable promise in alleviating the neuropsychiatric symptoms often associated with AD.

The reviewed evidence, spanning computational modeling, *in vitro* assays, robust *in vivo* animal models, and preliminary human observations, underscores the profound potential of these traditional systems to contribute to brain health and the prevention and symptomatic alleviation of AD. This review, by synthesizing findings across multiple prominent herbs and diverse research methodologies, offers a valuable consolidated overview of their multi-targeted neuroprotective actions. However, as a narrative synthesis, this review acknowledges its inherent limitations, particularly regarding the scope of its database search and the qualitative nature of its analysis, suggesting that future, more extensive systematic reviews could provide further quantitative insights.

Conclusion

Given the complex nature of AD and the limitations of current therapeutic strategies, the findings from this review highlight the compelling significance of further rigorous *primary* research in this area. Continued investigation, particularly through large-scale, standardized human clinical trials focusing on bioavailability, optimal dosing, and long-term efficacy, is crucial to translate this promising preclinical evidence into clinical practice. Ultimately, integrating these well-researched traditional approaches with conventional medicine holds immense potential for developing more comprehensive and effective strategies to address Alzheimer's disease.

Acknowledgements

I would like to express my sincere gratitude to the following individuals for their invaluable support and encouragement throughout the completion of this research project:

First and foremost, I would like to express my deepest appreciation to my research mentor, Dr. Kristen Lee PhD, for her guidance, encouragement, and rigorous mentorship throughout my academic journey. Her expertise in mental health, mind-body connection, and health psychology deeply shaped my research, helping refine both its focus and integrity. Her knowledge and wisdom have inspired me to view academic inquiry from more acute standpoints.

I would also like to acknowledge the support of my parents, Sampada Mhalagi and Rahul Adkar. Their cultural, emotional, and financial backing created the foundation for this work. From early on, they instilled in me a deep appreciation for Ayurvedic knowledge, which became the root of my passion for integrative biology, environmental health, and neuroscience.

I am very grateful to my late grandmother, Sunita Mhalagi, whose battle with stage IV glioblastoma motivated this project's central questions. Her final moments, marked by memory loss and silence, continue to inspire my commitment to researching neurodegenerative healing through traditional and holistic approaches.

Thank you all for your profound contributions and enduring support.

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