



Review Article

Exploring Alpinia Galanga As A Neuroprotective Agent: Role in Mitigating Alzheimer's Via Antioxidant and Anti-Microbial Activity

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Alzheimer's disease (AD) is a common neurodegenerative disorder that leads to memory loss and cognitive decline, predominantly affecting the elderly. Its pathophysiology includes mechanisms like amyloid-beta accumulation, tau protein aggregation, oxidative stress, and mitochondrial dysfunction. Current treatments, such as acetylcholinesterase inhibitors and NMDA receptor antagonists, offer symptomatic relief but do not halt disease progression. The rising global prevalence of AD calls for enhanced research into effective treatments. Notably, the plant *Alpinia galanga* exhibits significant potential against AD, with its rhizome extracts exhibiting antibacterial and antioxidant properties. The bioactive compounds in *Alpinia galanga* may provide neuroprotective effects by addressing oxidative stress, inhibiting A β aggregation, and restoring cholinergic function, indicating its therapeutic promise in AD management.

Keywords: *Alpinia galanga*, Antioxidant, Amyloid plaque, Oxidative stress, Bacterial infection.

INTRODUCTION

Alzheimer's disease (AD) is the most prevalent neurodegenerative disease that causes memory loss and progressive neurocognitive deterioration in the elderly. It is characterized by neocortical atrophy developing over decades, showing the increasing loss of synapses and neurons first described by Alois Alzheimer in 1907. However, the causes of AD are far from being unknown. Clinically, AD is characterized by a slowly progressive loss of memory and cognitive impairments. The decline in patients is mostly due to the aberrant accumulation of toxic protein fragments in the nervous system, namely amyloid-beta (A β) deposition within senile plaques, outside of neurons, and intracellular accumulation of microtubule-

associated Tau protein. A β is pointed to contribute to cell death by interfering with neuron-neuron communication at synapses, while neurofibrillary tangles of Tau restrict the passage of essential nutrients and other compounds inside neurons. Currently, 30 million people worldwide suffer from Alzheimer's dementia and the World Health organization projects that this number will triple over the next 20 years.¹ The cumulative incidence of Alzheimer dementia has been estimated to rise from about 5% by age 70 to 50% by age 90, making it a very common disease.² With the global population aging, the incidence of AD is rising, posing a growing threat to public health and necessitating preventive strategies and effective treatment.

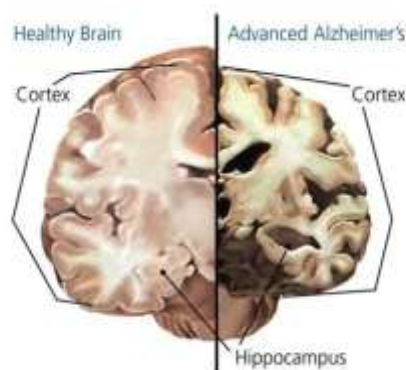


Fig 1 Normal brain and Alzheimer's brain

PATHOPHYSIOLOGY OF AD

1) Amyloid Hypothesis:

Amyloid beta ($A\beta$) is the prime suspect for driving pathology in Alzheimer's disease (AD) and, as such, have become the focus of therapeutic development. The amyloid precursor protein (APP) is a type I transmembrane glycoprotein containing 695–770 amino acids. It is regarded that abnormal proteolytic processing of APP leads to the generation of $A\beta$. In the non-amyloidogenic pathways, APP is cleaved by α - and γ -secretases. This α -secretase can cleave APP at residue L688, located in the middle of the $A\beta$ domain. α -secretase causes APP cleavage leading to

the formation of soluble APP alpha ($sAPP\alpha$) and a cell-membrane-bound C-terminal fragment 83 (CTF83). The generated CTF83 is cleaved by γ -secretase to produce AICD and a small p3 fragment. The second pathway is the amyloidogenic pathological pathway in which APP is cleaved to CTF- β 99 by β -secretase and then different lengths of $A\beta$ peptide, including $A\beta_{42}$ by γ -secretase. The $A\beta_{1-40}$ isoform is the most prevalent, followed by $A\beta_{1-42}$ which is hydrophobic in nature and aggregates at a faster rate than $A\beta_{1-40}$ which leads to aggregation and plaque formation will disrupt synapses and initiate cascade of toxic events, which ultimately leading to neuronal loss and death.⁴

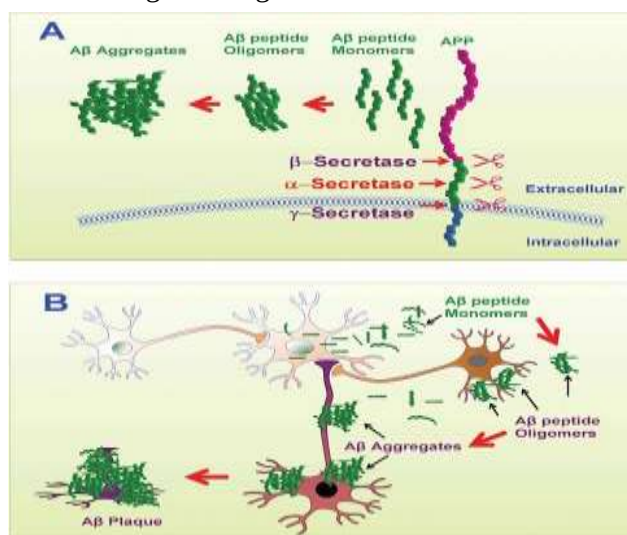


Fig 2 Amyloid Hypothesis

2) Tau Hypothesis:

The second typical morphologic symptom of AD is neurofibrillary tangles (NFT), which are formed in neuronal cytoplasm (especially in axoplasm) and are

insoluble filaments, consisting mainly of hyperphosphorylated tau protein (which is related to the microtubule-associated protein group). Impairments of tau protein functions result in destabilization and depolymerization of microtubules;

the degradation of microtubules causes a disconnection of intracellular transport in the cytoplasm, loss of synapses, and, ultimately, nerve cell death. Tau is predominantly found in brain and

peripheral nerves, tau protein expression has been detected in locations including salivary glands, breast tissue, cardiac myocytes.⁵

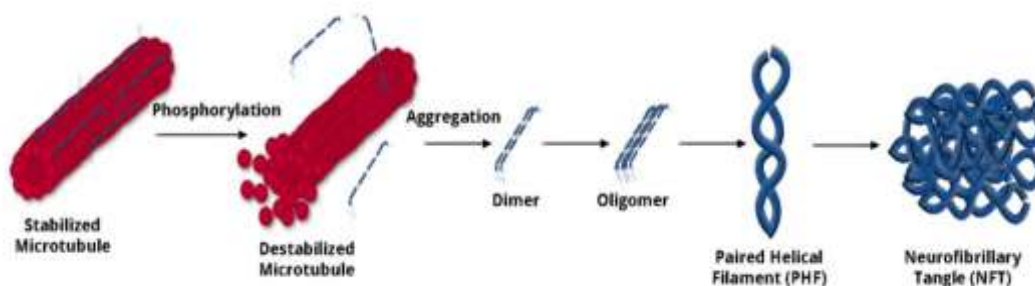


Fig 3 Tau Hypothesis

3) Cholinergic Hypothesis:

The cholinergic hypothesis has been proposed to describe the pathogenesis of AD earlier and is one of the currently and widely accepted hypotheses. Acetylcholine (ACh) is an important neurotransmitter used by cholinergic neurons for many critical physiological processes such as cognition and memory. Cholinergic neurons in the normal basal forebrain can synthesize a large amount of ACh. However, in the brains of patients with AD, a large number of cholinergic neurons are lost, and the activity of choline acetyltransferase is reduced, resulting in a significant reduction in the synthesis, storage, and release of ACh. Cholinergic neuronal damage is considered a critical pathological change that is correlated with cognitive impairment observed in AD. A β accumulation, hyperphosphorylation of tau protein, oxidative stress, inflammation, and metal imbalance cause cholinergic neuronal damage. The first drug approved for the treatment of AD was tacrine, a cholinesterase inhibitor. However, the drug was withdrawn from the market in 2012, as it resulted in severe side effects. Thus far, acetylcholinesterase (AChE) inhibitors are the most widely used drugs in the clinical treatment of AD.⁶

4) Oxidative Stress Hypothesis:

5) Oxidative stress is another common pathological feature of AD. Compared with other aerobic tissues and organs; neurons of the brain are more prone to oxidative stress due to several reasons. First, the brain, a component of the central nervous system, consumes

more oxygen, which leads to increased ROS production. Second, the intrinsic antioxidant defense of the neurons of the brain is weak because they have low levels of antioxidant enzymes or compounds, such as superoxide dismutase, peroxidase, and glutathione. Additionally, antioxidants in other body parts cannot easily cross the blood–brain barrier (BBB). Moreover, the cell membranes of neurons in the brain contain high levels of unsaturated fatty acids, which are prone to react with free radicals through peroxidation reactions. ROS accumulates in the neurons of the brain, causing neurotoxicity and neuronal synaptic dysfunction. In AD, A β aggregation, tau phosphorylation, metal ion accumulation, and inflammatory response can stimulate excessive production of ROS and damage the antioxidant defense ability, causing oxidative stress.⁷

5) New Pathway of AD- The Infectious Theory:

The bacterial infection hypothesis of AD was proposed in the 1990s, and it appears to be a plausible explanation for the induction and exacerbation of AD. Inhibition of *E. coli* is considered a potential neuroprotective strategy in Alzheimer's disease (AD) because bacterial components are associated with neuropathology and neuroinflammation. Currently, it is generally believed that AD is a disease caused by multiple factors that can work. The above hypotheses indicate different aspects of the pathogenesis of AD however; the exact mechanism is synergistically and form a feedback loop mechanism.⁸

Alpinia Galanga

Introduction:

Alpinia galanga is a perennial plant commonly known as “Greater galangal”. It is known by several synonyms such as *Amomum galangal*, *Maranta galangal*. It is known by several common names such

as Kulanjan in Hindi, Dhumarasmī in Kannada, Arattha in Malayalam. It is a ginger like shrub belonging to Zingiberaceae family. It is an aromatic; rhizomatous herb is widely distributed in India. In India it is traditionally used as nervine tonic and stimulant effect. It is also used as carminative, stomachic, disinfectant, and for inflammation. It is a ginger substitute for flavouring food.¹⁰



Fig 5: Alpinia Galanga

Cultivation for Alpinia galanga:

Alpinia galanga is Galangal is best suited for planting in shaded open areas in warmer climates as it's a tropical plant. This plant will rot easily when left exposed to cold, wet conditions. With adequate irrigation provisions, it can be grown up to an altitude of 1000 m. The soil should be rich in organic matter such as sandy loam. This type of soil provides the thick roots with space to grow while allowing water to properly drain. It is commonly propagated by rhizome splits. The Rhizomes should be planted in southern India April to May. It is best to harvest the remaining *Alpinia galanga* roots during the early winter to prevent rotting, one can leave a few hands, in the ground over the winter if one can heavily mulch the entire area to keep them warm and dry. *Alpinia galanga* can be stored in the vegetable drawer of a refrigerator for two to three weeks. The *Alpinia galanga* should be plastic wrapped preferably, wrap the root first in a damp cloth, then in a plastic bag. Galangal can be frozen without losing any of its flavour.

Botanical description:

The plant is a perennial herb. It grows up to a height about 5 feet. Leaves are oblong-lanceolate, tuberous

root, slightly aromatic. The rhizome is from 3.5-7.5 cm in length, and seldom more than 2 cm thick. The leaves are long, oblong-lanceolate, acute, glabrous, ligules are short and rounded.

Geographical distribution:

Alpinia galanga grows in many Asian countries such as India, Arabia, China, Sri Lanka, and Indonesia. It favours hot places exposed to extensive sunlight, but it can also grow in shrubs, forests, and open spaces. In India the plant is distributed in Himalaya and Southern region of Western Ghats and states of Kerala and Karnataka it is also present in Tamil Nadu and Andhra Pradesh.¹¹

Rhizome:

Alpinia galanga is an herb that can grow up to 3.5 cm, with underground rhizomes and minor adventitious roots. The rhizomes have a red-brown colour on the surface, while the inside of the rhizome is brown, orange. They are 2.5–10 cm long with a pseudo-stem that is erect and covered with leaves. Size and shape of the leaves are 3.8–11.5 cm, oblong-lanceolate, glabrous, and acute.



Fig 6 Galangal Rhizome

Traditional use:

The galangal rhizome is effectively used as a therapeutic treatment for various diseases, because it contains antibacterial, anti-fungal, anti-inflammatory, anti-hepatotoxic, antioxidant, immune modulator, anti-ulcerative, anti-tumour, and anti-allergic activities. It can be used to treat stomach pain, back pain, rheumatism, asthma, diabetes, heart disease, disorders of the liver, kidney disease, and to increase the appetite. Galangal rhizome can also be used as a substitute for antibiotics, disinfectants, and food seasonings.¹²

Anti-Oxidant Properties of Alpinia Galanga In Alzheimers Disease:

Several diseases in humans are linked to the build-up of free radicals. Antioxidants can scavenge free radicals and reduce their impact. As a result, the research for naturally occurring antioxidants of plant origin is critical. Antioxidants can convert free radicals into waste by-products that are excreted by the body. The H_2O_2 assay evaluates the ability of the plant extract to scavenge hydrogen peroxide, which is a harmful reactive oxygen species. Hydrogen peroxide can cause oxidative damage to cells and tissues. The assay measures the reduction in hydrogen peroxide concentration after exposure to the plant extract, indicating its antioxidant potential. The scavenging activity of natural antioxidants found in plant extracts against hydrogen peroxide (H_2O_2) has been widely tested by detecting the decrement of H_2O_2 in an incubation system containing H_2O_2 and the scavenger using the classical UV method at 230 nm.¹³

Anti-Bacterial Activity of Alpinia Galanga In Alzheimers Disease:

Alpinia galanga's potential antibacterial activity in Alzheimer's disease (AD) might stem from its ability to inhibit acetylcholinesterase (AChE), reduce oxidative stress, and modulate signaling pathways, ultimately contributing to neuroprotection. Galangal or greater galangal belongs to Zingiberaceae family that contain chemical compounds like alkaloids, glycosides, terpenoids, flavonoids, phenols and essential oil. *E. coli* bacteria are bacterial pathogens belong to Enterobacteriaceae family. The result of galangal rhizome extract, (*Alpinia galanga* L.) shows that the average result of growth inhibition *Escherichia coli* is 0,7028 cm. That the average inhibition diameter of essential oils in red galangal rhizome to *E. coli* are at its peak, with 50% concentration, and 18,8 mm.¹⁴

Phytoconstituents Of Alpinia Galanga:

1) Flavonoids:

Eg: Galangin, Quercetin, Myricetin, Kaempferol, Galangin 3-methyl ether, Quercetin 3-methyl ether.

Flavonoids have emerged as a promising leading molecule either alone or in association with other compounds for showing the effective plan and improvement as anti-AD drugs. Some constituents like Quercetin and Quercetin 3 – methyl ether has beneficial properties against general mechanisms of AD. The anti-Alzheimer's disease properties of quercetin include the inhibition of $A\beta$ aggregation and tau phosphorylation. Galangin provide a certain degree of protection against oxidative stress-related neurological disorders. Flavonoids combat oxidative stress through direct antioxidant effects, such as scavenging free radicals and reactive oxygen species

ROS. They also indirectly boost the bodies antioxidant defence by activating pathways that regulate the production of antioxidant enzymes, suppress inflammation cell against cell damage. Flavonoids have antibacterial properties that work by disrupting bacterial cells, inhibiting their growth, and interfering with key processes. They can damage cell walls and membranes, suppress nucleic acid and energy metabolism, reduce biofilm formation, and block virulence factors. This makes them promising candidates for developing new antibacterial treatments, especially in the fight against antibiotic-resistant bacteria.¹⁵

2) Phenols:

Eg: Gallic acid, Catechin, Quercetin, Catechol.

Phenols considered beneficial to the development of AD through several biological mechanisms. Phenols exhibit antibacterial activity against *E. coli* by damaging its cell membrane, disrupting its ability to regulate internal ions, and interfering with its genetic material. Specific compounds like protocatechuic acid (PCA) and certain phenolic acids show strong inhibitory effects, while others like procyanidins can reduce biofilm formation by interfering with the bacteria's ability to adhere to surfaces.¹⁶

Antibacterial Mechanisms of Phenols Against *E. Coli* Are:

- **Cell membrane damage:** Phenols can disrupt the cell membrane's permeability, leading to the leakage of essential intracellular components like potassium ions. This depolarization damages the cell's ability to maintain its internal environment.
- **DNA interference:** Some phenols can bind to the bacterial DNA, inhibiting its function and growth.
- **Biofilm inhibition:** Certain phenolic compounds, like those found in cranberries, can interfere with the initial steps of biofilm formation by preventing *E. coli* from adhering to surfaces, which reduces its ability to form a protective matrix.
- **Synergistic effects:** When combined with antibiotics, phenolic compounds can enhance the

antibiotic's effectiveness, potentially by making the cell membrane more permeable and allowing the antibiotic to enter more easily.

Antioxidant mechanism of phenols are:

- **Directly scavenge free radicals:** Phenols have the ability to neutralize reactive oxygen species (ROS) by donating electrons, which prevents them from damaging cells and other biomolecules.
- **Boost intrinsic defense systems:** They can enhance the activity of the body's natural antioxidant enzymes, such as superoxide dismutase (SOD) and glutathione peroxidase (GPX).
- **Inhibit inflammatory pathways:** Phenols can reduce inflammation by modulating inflammatory pathways and cytokine release, which is helpful in conditions like neurodegenerative diseases.
- **Inhibit ROS-producing enzymes:** Some phenolic compounds can inhibit enzymes that generate ROS, thereby reducing the overall production of damaging free radicals.¹⁶

3) Terpenes:

Eg: Galangal diterpenes, Disabolene, Cineole, Fencyl Acetate.

Terpenes have a strong anti-Alzheimer's activity and provide a very high neuroprotective potential by inhibiting acetylcholinesterase (AChE) and butyryl cholinesterase (BChE) activities, regulating neurotransmitter synthesis, release, and transmission, as well as suppressing β -amyloid peptide ($A\beta$), reducing the levels of reactive oxygen species (ROS)

Antibacterial activity against *E. coli*

- **Cell membrane disruption:** Terpenes' lipophilic nature allows them to interact with the lipid assembly of the cell membrane, increasing its permeability and causing it to break down. This leads to cell lysis and the release of intracellular components.

- **Cellular damage:** Some terpenes can cause degradation of cellular proteins, damage to cell walls, and inhibit processes like oxidative phosphorylation, which is crucial for cellular respiration.

Antioxidant effect of terpenes:

- **Oxidative stress:** Certain terpenes can generate reactive oxygen species (ROS), which cause oxidative stress within the bacterial cell, leading to lipid peroxidation and further membrane damage.
- **Direct ROS scavenging:** Terpenes can directly neutralize free radicals like superoxide. They achieve by acting as electron donors, which terminates chain reaction of lipid peroxidation & other oxidative damage.
- **Modulating endogenous systems:** Terpenes can indirectly boost the body's natural antioxidant defenses. They can increase the activity of enzymes like catalase, superoxide dismutase, and peroxidase, and elevate levels of the antioxidant glutathione.¹⁷

4) Essential Oils:

Eg: 1,8 Cineole, pinene, Terpineol, Methyl cinnamate.

Essential oils from certain plants possess several important biological activities. Among those activities, the antimicrobial activity of plant essential oils is one of the most interesting issues to be focused on because they might be useful as the natural antimicrobial alternatives to be used instead of the harmful chemical antimicrobial agents. Essential oils kill *E. coli* primarily by disrupting its cell membrane, leading to leakage of cellular contents and metabolic damage. Key mechanisms include increasing membrane permeability, altering membrane integrity, and causing cell deformation, which ultimately leads to cell death.

Antioxidant activity of essential oils:

- **Cell membrane disruption:** Hydrophobic components of essential oils interact with the lipid

bilayer of the cell membrane, causing it to lose its integrity. This results in increased permeability, which allows vital cytoplasmic materials to leak out.

- **Metabolic damage:** Membrane damage disrupts normal cell functions, leading to metabolic abnormalities. Treatments can reduce the intracellular ATP levels and cause significant deformation and leakage of cellular components like proteins and nucleic acids.
- **Changes in cell morphology:** *E. coli* cells treated with essential oils can become deformed or pitted, which is a visual indicator of membrane damage and cell death.

Antibacterial activity of essential oils:

- **Cell membrane damage:** The hydrophobic compounds in essential oils interact with and damage the lipids in the bacterial cell membrane, leading to increased permeability, leakage of intracellular contents, and cell death.
- **Protein structure alteration:** Some oils can alter the structure of proteins, which impairs normal cellular function and growth.
- **Metabolic disruption:** Essential oils can interfere with essential metabolic processes in *E. coli*, such as ribosomal assembly, and may affect pathways related to butyric acid, ascorbic acid, and aldehyde metabolism.¹⁸

5) Alkaloids:

Eg: Galantamine, Huperzine A, Berberine, Caffeine

Alkaloids are a class of naturally occurring organic nitrogen-containing compounds that are found primarily in plants, especially in certain families of flowering plants.

Antibacterial activity:

- **Cell membrane damage:** Many alkaloids directly damage the cell membrane of *E. coli*.

- **Modulating bacterial behaviour:** Sub-inhibitory concentrations of certain alkaloids can reduce bacterial motility (by decreasing flagellin expression), potentially helping to contain infection.
- **Enhancing antibiotic effectiveness:** Alkaloids can work in combination with existing antibiotics to enhance their activity against bacteria, which can be useful in treating infections that are resistant to standard drugs.

Antioxidant properties

- **ROS scavenging:** Alkaloids can directly neutralize damaging free radicals, such as hydroxyl radicals, which are linked to oxidative stress and diseases.
- **Metal chelation:** Some alkaloids can bind to metal ions, preventing them from catalysing the formation of free radicals.
- **Cellular pathways:** Alkaloids can work by modulating cellular signaling pathways, like Nrf2, which can activate the body's own antioxidant defenses.¹⁹

6) Stilbenes:

Eg: Pterostilbene, Pinosylvin, Piceatannol

Stilbenes exhibit many neuroprotective mechanisms, including antioxidant characteristics, antibacterial activities, anti-inflammatory effects, modulation of signaling pathways, and the capacity to hinder the production of amyloid proteins. They collect and remove free radicals and reactive oxygen species (ROS)

Antibacterial activity of stilbenes:

- **Disruption of cellular functions:** Stilbenes can interfere with bacterial energy metabolism by inhibiting the phosphotransferase system, which is crucial for carbohydrate transport and phosphorylation.
- **Cell membrane damage:** Some stilbenes can disrupt the cell membrane's structure and

function, leading to effects like intramembranous edema and depolarization.

Antioxidant activity of stilbenes:

- **Scavenging free radicals:** Stilbenes, such as resveratrol, neutralize harmful reactive oxygen species (ROS).
- **Reducing oxidative stress:** By combining these two mechanisms, stilbenes help prevent and reverse the cellular damage caused by oxidative stress.²⁰

7) Resveratrol:

Resveratrol is a polyphenolic compound found in a number of plants has been reported to possess antioxidant properties

Antibacterial activity of resveratrol:

- **Inhibits cell division:** Resveratrol can suppress the formation of the Z-ring, an essential structure for bacterial cell division, particularly in *E. coli*.
- **Interferes with energy production:** Resveratrol can inhibit the electron transport chain and ATP synthase, leading to reduced cellular energy production and proliferation.

Antioxidant activity of resveratrol:

- **Free radical scavenging:** Resveratrol directly neutralizes ROS and RNS by donating hydrogen atoms from its phenolic hydroxyl groups.
- **Inhibition of ROS production:** It inhibits the production of ROS by enzymes like NADPH oxidase, reducing their activity and expression.
- **Scavenging metal ions:** As a polyphenol, it chelates metal ions, preventing them from catalysing free radical formation, such as in the Fenton reaction.²¹

CONCLUSION

We ultimately concluded that Alzheimer's disease (AD) poses a serious problem because of its progressing nature and the ineffectiveness of the

available treatments. The need for safer and more efficient treatments is highlighted by the pathophysiology of AD. *Alpinia galanga*'s antibacterial and antioxidant qualities may hold promise for treating AD. In order to create innovative treatment approaches and enhance the quality of life for those afflicted by this debilitating illness, more clinical research on *Alpinia galanga* is essential.

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