Journal of Madhesh Institute of Health Sciences 2025;1(1):42-45

OPEN ACCESS

REVIEW ARTICLE

Neuroprotective potential of green tea polyphenols in age-related dementia: a review

Ram Chandra Shah[1,*]

[1]*Associate Professor, Department of Pharmacology, Madhesh Institute of Health Sciences, Janakpurdham, Nepal*

Article Information

Received: 12 Jun, 2025

Accepted: 28 Jun, 2025

Published: 30 Jun, 2025

Key words: Age-related; Dementia; Green tea; Review

ABSTRACT

The clinical manifestations of dementia are brought on by the deterioration of particular cognitive areas, particularly those linked to memory dementia. Nowadays, dementia is a severe issue that requires quick care. The avoidance dementia may be affected by dietary variables. Polyphenols are the main active components of tea. Epicatechin-3-gallate, epicatechin, gallocatechins, and gallocatechins gallate are the main polyphenolic components of green tea, which are referred to as catechins. The amyloid cascade hypothesis states that naturally existing Aβ monomers form insoluble fibrils through a nucleation-dependent process, which are then deposited as plaques in the brain. Focused on green tea and carried out a thorough review of observational studies that examined the connection between green tea consumption and dementia. The publications that were recovered were then examined to determine whether they included original studies assessing a connection between drinking green tea and dementia, Alzheimer's disease, mild cognitive impairment, or cognitive impairment. These results seem to support the hypothesis that consuming green tea may reduce the risk of dementia, Alzheimer's disease, moderate cognitive impairment, or cognitive impairment. More results from well designed and carried out cohort studies are required to provide conclusive evidence. Recent research demonstrating the protective effects of tea polyphenols against dementia is reviewed in this article and summarized.

Nutritional aspects in brain aging and neurodegeneration:

Dementia has grown to be a significant social problem that needs to be addressed and prevented immediately. There are over 50 million dementia sufferers globally, and 10 million new cases are reported each year. It is anticipated that there would be 82 million dementia sufferers by 2030 and 152 million by 2050.[1] Given that age is the greatest known risk factor for dementia, this sharp rise is caused by the aging population. In terms of the expenses of informal care as well as direct medical and social care, dementia has substantial social and economic impacts. The prevention of dementia may be influenced by dietary factors, and beverages are thought to be helpful because their consumption is more acceptable and does not significantly alter other dietary practices. One of the most popular drinks in the world is tea. Tea consumption, which includes tea polyphenols and caffeine, may be significant.[2,3] The most popular beverage in the world is tea. In Asia, tea is the most popular beverage aside from water, and Chinese and Japanese people have been drinking it for generations. It has a wealth of pharmacologically active compounds that have been linked to a number of health advantages. Depending on the level of fermentation, there are three main types of tea: oolong, black, and green.[4] Different species, seasons, leaves, climates, and horticulture techniques all affect the composition of tea. Among the main active ingredients in teas are polyphenols.[5] The primary polyphenolic components found in green tea are called catechins, and they comprise epicatechin-3-gallate, epicatechin, gallocatechins, and gallocatechin gallate. The most common and researched catechin in green tea is EGCG.[6,7] It's possible that dietary modifications could help prevent AD. Plant-based polyphenol-containing beverages have been suggested as a natural supplemental treatment to reduce AD symptoms.[8]

Citation: *Shah RC. Neuroprotective potential of green tea polyphenols in age-related dementia: a review.Journal of Madhesh Institute of Health Sciences. 2025;1(1):42-5.*

***Correspondence:** Dr. Ram Chandra Shah, Associate Professor, Department of Pharmacology, Madhesh Institute of Health Sciences, Janakpurdham, Nepal.*

Email: rampharmashah@gmail.com

Green tea epidemiological and clinical studies: Cognitive impairment

Tea drinking has been linked by numerous epidemiological studies to a lower incidence of AD and other neurological illnesses. The action-based memory of individuals with AD dementia was evaluated using the process of making a cup of tea.[9,10] A community-based complete geriatric assessment conducted in Japan with 1003 Japanese citizens 70 years of age or older revealed that a reduced prevalence of cognitive impairment (CoI) was linked to higher green tea drinking.[11] The greatest class of flavonoids found in green tea, the naturally occurring antioxidant polyphenolic catechins, may meet the criteria for a potential neuroprotective medication due to their wide range of pharmacological actions.[12,13] Compared to black tea, green tea extract has about four times more of the catechin fraction and is especially rich in flavanols called catechins, which make about 30–40% of the dry weight of the leaves. According to the amyloid cascade hypothesis, naturally occurring Aβ monomers combine through a nucleation-dependent process to create insoluble fibrils, which are then deposited in the brain as plaques. AD is characterized by the self-assembly of Aβ into neurotoxic oligomers and fibrillar aggregates. Amyloid fibrils misfold, aggregate, and build up in the brain in an insoluble state as a result of AD. To protect cells from Aβ-mediated neurotoxicity, green tea polyphenols (GTPs) such as (−)-epigallocatechin gallate (EGCG), (+)-catechin (C) and (−)-epicatechin (EC), myricetin, quercetin, and kaempferol can destabilize preformed fAβ and dose-dependently inhibit the formation of Aβ fibrils (fAβ) from fresh Aβ (1–40) and Aβ (1–42).[14,15] In neuronal cell oxidative stress, Aβ peptides are involved on both sides. The production of Aβ by reactive oxygen species (ROS) promotes oxidative stress and neuronal damage. Usually, antioxidants and free radical scavengers slow down this process.[14] Tea catechins are a class of natural antioxidants that scavenge reactive oxygen species (ROS) and have protective effects against Aβ-induced neuronal death. Following a 48-hour exposure to Aβ, one study found significant hippocampus neuronal damage along with elevations in malondialdehyde (MDA) levels and caspase activity.[16] Green and black tea extracts including rutin, EGCG, and L-theanine (a unique amide present in tea leaves) demonstrated protective effects against mitochondrial dysfunction, a very early stage in the pathophysiology of AD. Therefore, treatments that focus on enhanced mitochondrial function may be helpful. Aβ contributes to the development and progression of AD by producing ROS, which causes mitochondrial dysfunction and synaptic deficits.[16,17]

Neuroprotective studies with green tea:

a. Preclinical animal data: EGCG has been shown to significantly increase longevity in Caenorhabditis elegans under stress and to reduce cognitive deficits, antioxidant enzyme decrease, and apoptotic parameters in D-galactose-induced mice. Additionally, EGCG has been demonstrated to prevent brain inflammation, neuronal damage in experimental autoimmune encephalomyelitis, and cerebral ischemia/reperfusion injuries, as well as to alleviate age-related cognitive decline.[18,19] In rat models of middle cerebral artery occlusion (MCAO), the impact of EGCG against neuronal damage was examined. Following MCAO, EGCG treatment decreased the neurological function score, preserved nerve cells, suppressed neuronal apoptosis, and decreased the amount of brain injury indicators and oxidative stress injury.[20]

b. Neuronal cell culture studies: The neurotoxins 6-OHDA and 1-methyl-4-phenylpyridinium (MPP+) caused neuronal cell death in human neuroblastoma (NB) SH-SY5Y cells. EGCG shielded rat pheochromocytoma (PC12) cells and native hippocampus neurons from Aβ-induced toxicity. By preventing cytosolic calcium increase, catechin and EC have more recently been demonstrated to shield cultured rat cortical neurons from Aβ-induced damage.[21,22]

Mechanism of neuroprotective action of green tea polyphenol EGCG:

The anti-amyloid mechanisms of these bioactive compounds include: a. APP cleavage inhibition through the regulation of related enzyme activity b. prevention of Aβ-induced membrane damage and protein misfolding c. mitigation of Aβ-induced oxidative stress d. suppression of Aβ oligomer aggregation e. regulation of signalling pathways involving Aβ generation f. reduction of Aβ-induced mitochondrial dysfunction, and g. inhibition of TAU protein hyperphosphorylation.

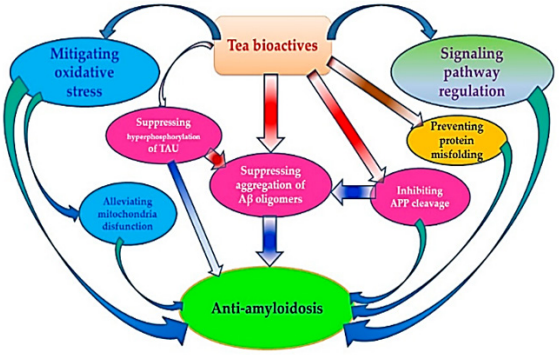

Figure 1: Anti-amyloidosis effects of tea[23]

Before conclusively supporting a connection between tea drinking and the prevention of an AD cure, more research will be needed. More clinical research is specifically required to help elucidate contradictory epidemiological findings.[24,25]

Summary and future prespectives

Data from animal studies and human epidemiology indicate that tea consumption may lower the risk of dementia. Specifically, (-)-epigallocatechin-3-gallate (EGCG), the primary catechin polyphenol ingredient in tea, has demonstrated neuroprotective effects in a variety of cellular and animal models of neurological diseases.[26] Through α-secretase activation and unfolded peptide disruption, EGCG also prevents Aβ aggregation in animal models. EGCG significantly increases the cleavage of α-C-terminal APP fragments and raises the N-terminal of the soluble APP-α, the result of APP cleavage.

Additionally, it prevents the production of Aβ oligomers by adhering to the protein directly or perhaps by interacting with a protein chaperone.[27]

Before definitive support, a connection between tea drinking and preventing dementia in old age, more research is needed.[28] More clinical research is specifically required in order to assist elucidate contradictory epidemiological findings. Low bioavailability, dosage variations between in vitro and in vivo testing, poor stability of tea's bioactive components, and conversion of bioactivities in the gastrointestinal tract are some of the factors contributing to inconsistencies.[20,29] Research on these parameters in depth will be important to bridge the gap between clinical applications and in vitro investigations.

CONFLICT OF INTEREST: None

FINANCIAL DISCLOSURE: None

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