AMERICAN JOURNAL OF PHARMTECH RESEARCH
Journal home page: http://www.ajptr.com/

Herbal Drugs Used In Treatment Of Alzimer's Disease

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ABSTRACT

Alzheimer's disease (AD) is a progressive neurodegenerative disease leading to a gradual and irreversible loss of memory, linguistic skills, and perception of time and space, thinking, and behavior. Food habits, Dietary pattern has been presented as a contributor to the incidence of Alzheimer's. Alzheimer's disease is the most common cause of dementia worldwide, with the prevalence continuing to grow in part because of the aging world population. Some Food and Drug Administration-approved drugs are available for the treatment of Alzheimer's disease, the outcomes are often unsatisfactory, and there is a place for alternative medicine, in particular, herbal medicine. This paper reviews for the how food habits act as one cause for Alzheimer's disease & the clinical effects of a number of commonly used types of herbal remedies/medicines for the treatment of AD.

Keywords: Alzheimer's disease, dementia, amyloid, food habits, herbal remedies.

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Received 28 July 2020, Accepted 07 August 2020

Please cite this article as: Deore *et al*., Herbal Drugs Used In Treatment Of Alzimer's Disease. American Journal of PharmTech Research 2020.

INTRODUCTION

Alzheimer's disease (AD), first described by German psychiatrist Alois Alzheimer in 1906, is a progressive neurodegenerative disease characterized by cognitive deterioration together with behavioral disturbances and declining activities of daily living[1]. AD is the most common cause of senile dementia [2] with a prevalence estimated to be 1.6% in the USA [3]. The rate is expected to increase over time as the aging population grows in as much as more than 80% of AD patients are 65 years or older. Microscopically, AD is characterized by amyloid plaques and neurofibrillary tangles [4]. Alzheimer's disease (AD) is a disease of later life, affecting one in four people 85 years of age or over, and the incidence is expected to rise in the coming years, with 131.5 million estimated cases by 2050[5].

AD has to face two major challenges

The delay in the diagnosis and the lack of neuroprotective or curative pharmacological treatment. In fact, AD is recognized only in the late stage when cognitive symptoms appear, and currently approved drugs only provide modest and temporary relief for symptoms such as memory loss[6].Today, it is well accepted that a prodromal phase ranging from 10 to 20 years precedes the symptomatic state. During this long period, many biochemical changes occur in the brain, anticipating cognitive impairment. In this preclinical phase, preventive strategies, such as dietary modification and nutritional supplementation, might reduce the global burden of AD. One of the first links between dietary intake and incidence of AD is represented by a large prospective population-based cohort study (Rotterdam study) that reported an associated lower risk with the use of cholesterol-lowering statin drug [6] The association of dietary fats with plasma cholesterol levels is highly relevant because cholesterol is involved in both generation and deposition of Aβ[7]. Furthermore, the protein product of APOE-ε4, a recognized genetic risk factor for AD, is the principal cholesterol transporter in the brain. In fact, many epidemiologic data suggest that nutritional intake can influence the development and progression of AD [8].

Foods that May Increase the Risk of Alzheimer's

First, we know that an unhealthy diet can promote cognitive decline along with other health problems. Scientists have looked at what they call the "Western diet," a pattern rich in convenient, processed foods and rich in animal products. The typical western diet is high in total fat, saturated fat, cholesterol, sodium, and processed grains, large amounts of added sugar. This eating pattern has been linked to obesity, diabetes, and heart disease. A high intake of saturated and trans fats increase the body's levels of oxidative stress and inflammatory response, harmful processes which

contribute to the development of dementia. The take-away message? Cut down on total caloric intake, saturated and trans fats, and sugar [9].

Foods that May Decrease the Risk of Alzheimer's

Next, we know that a healthier diet can protect cognitive functioning while also improving other aspects of health. Most of the research on diet and Alzheimer's has focused on the Mediterranean diet (MD), an eating pattern that emphasizes healthy fats such as olive oil, fresh fruits and vegetables, legumes, nuts, and less dairy food, red meat, butter or margarine, sweetened beverages, and pastries. Moderate wine consumption is included in some, but not all, European and Middle Eastern populations who have adopted the MD [9].

Alzheimers disease is a neurodegenerative disorder characterized by progressive global deterioration in intellect, which affects memory, thought, learning, orientation, language, comprehension, and judgment, as well as behaviour and the ability to perform everyday activities. The major pathological hallmarks of this disease include accumulation of protein deposits in the brain as beta-amyloid (Aβ) plaques and neurofibrillary tangles [10]. A similar approach can be applied to nutritional and other lifestyle-related exposures, particularly for conditions, such as cancer or Alzheimer's disease, for which there may be a long latency period between exposure and disease manifestation and for which randomized controlled trials are impractical or are, for whatever reason, not rapidly forthcoming. Some have argued that the level of evidence required for making dietary recommendations for disease prevention may be different from that required for establishing the efficacy of medical treatments, such as pharmaceuticals [11].

Dietary-Advanced Glycation End Products (d-AGEs) and Cognitive Decline

During the processing of foods, the temperature, the duration of the heat treatment, and the food's water content can drive different biochemical reactions, transforming the original content. At high heat administered for a long period of time, we expect the loss of a high amount of water and the degradation of heat-sensitive micronutrients, such as vitamin C, folates, and thiamine. In addition, higher temperatures used for cooking induce a series of reactions that lead to the characteristic smell, taste, and colour of the dish. Those reactions are also involved in the formation of toxic secondary products known as advanced glycation end products (AGEs). AGEs are a heterogeneous group of compounds derived from a non-enzymatic glycation of free amino groups of proteins, lipids, or nucleic acids by reducing sugars and reactive aldehydes[12].They are also continuously formed in the body as a part of normal metabolism under hyperglycaemic and/or oxidative stress condition [12]. It is well known that AGEs derived from the diet can highly contribute to the body pool of AGEs and constitute a large amount of the total AGE serum content. Since the half-life of

AGEs is about double the average of a cell's life, their detrimental effects can persist for a long time, especially in "long-lived" cells like nerve and brain cells[13]. Their toxic effects are related to their ability to promote oxidative stress and inflammation by binding to cell surface receptors or cross-linking with body proteins, altering their structure and function [14,15].

The most studied AGE receptor is RAGE, a single transmembrane multiligand receptor that belongs to the immunoglobulin superfamily [16]. RAGE receptors are mainly expressed on vascular, endothelial, and smooth muscle cells and on monocyte/macrophage membranes [16], but also in microglia and astrocytes, as well as in neurons [17, 18]. Ligands of RAGE, apart from AGEs, include members of the S100 protein family, proteins of the high mobility group box-1 (HMGB1), prions, and amyloid-β peptides. RAGE is implicated in the pathogenesis of several chronic diseases, such as cardiovascular diseases, hypertension, and diabetes, which are risk factors for AD, suggesting it might be the molecular link that initiates a chronic positive feedback loop, ultimately leading to AD etiology [16].

The interaction of RAGE receptors with AGEs induces the activation of different intracellular cascades, which involve the nuclear factor kB (NF-κB) pathway and inflammatory mediators like tumour necrosis factor-α (TNF-α), interleukin-6, and C-reactive protein (CRP)[19].All of these pathways lead to increased oxidative stress and a proinflammatory status

Recently, different studies reported that an elevated serum level of AGEs is associated with a faster rate of cognitive decline [13, 20]. More specifically, increasing evidence in the literature suggests that AGEs could be implicated in the progression of Alzheimer's, Parkinson's disease, and cerebrovascular dementia. In particular, RAGE seems to be involved in AGE-induced oxidative stress and chronic subclinical inflammation in the AD brain [21]. In fact, RAGE is increased in the brains of AD patients and has a role in regulating the transport of beta-amyloid across the blood-brain barrier (BBB)[22]. In particular, RAGE was found to act as cell surface receptor for Aβ[22, 23]and promote the influx of circulating Aβ across BBB from blood to brain, which is antagonized by LRP-1-mediated efflux of Aβ [24,25]. The interaction of AGEs with their receptor (RAGE) activates also the proinflammatory pathway via NF-kB. The neuroinflammation induced by AGEs can establish a vicious circle, whereby the overregulation of RAGE potentially increases Aβ influx across the BBB, leading to an accumulation of Aβ in the brain [26]. Furthermore, in the last years, a newly role of RAGE is emerging in microglia activation. This can have some implication in AD pathogenesis [27]. In fact, the interaction of RAGE with Aβ in activated microglia can initiate a cascade of events, resulting in sustained generation of toxic mediators and, ultimately, exacerbating neuroinflammation and leading to neuronal loss [16].

Food, as both source of bioactive nutrients and reservoir for potential toxic compounds, can have a dual role in AD pathology. All these findings indicate that AGEs can be considered as dietary risk factors not yet recognized and important pathogenic mediators involved in AD. The discovery of natural or pharmacological AGE inhibitors and the adoption of an AGE-restricted diet might be further new challenges, in order to promote a healthy ageing status and prevent cognitive decline exacerbation [28].

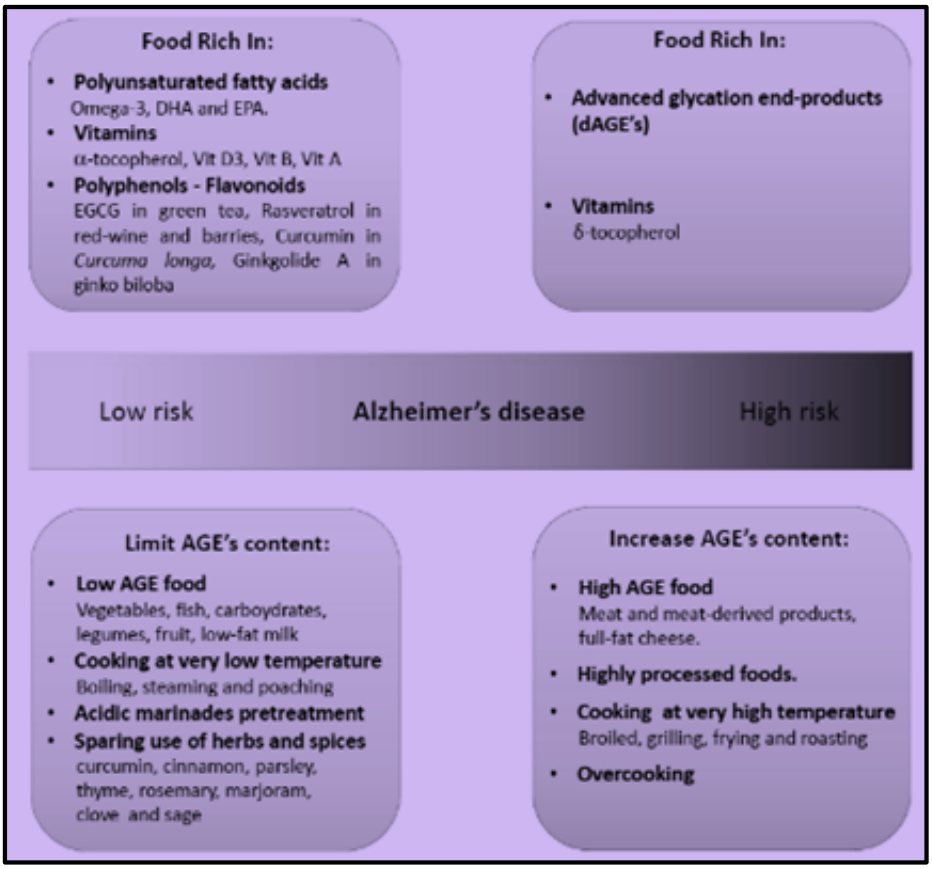


Figure 1: Involvement of Diet and cooking method in Alzheimer's disease prevention

In particular, the Dietary Approaches to Stop Hypertension (DASH) diet, originally developed as a plant-focused dietary plan against hypertension, considers high consumption of vegetables, fruits, lean meats, fish, nuts, legumes, poultry, whole grains, and low-fat dairy products, and lower intake of sodium, red meat, saturated fats, and sweets. In a randomized clinical trial (RCT), higher levels of accordance with the DASH diet conferred greater cognitive improvements in comparison to control subjects[29].The Mediterranean diet (MeDi) is another plant- focused dietary pattern with higher intake of vegetables, fruits, breads, other forms of cereals, nuts, potatoes, legumes, and seeds, with a low-to-moderate consumption of fish, poultry, red meat, and wine, while olive oil is the main source of monounsaturated fatty acids (MUFA). In several recent population-based studies, higher levels of accordance with a Mediterranean-type diet has been linked to slower

cognitive decline, reduced risk of AD, transition from mild cognitive impairment (MCI) to AD, and decreased mortality in AD patients[30].

Treatment: medicinal herbs to treat Alzheimer's disease:

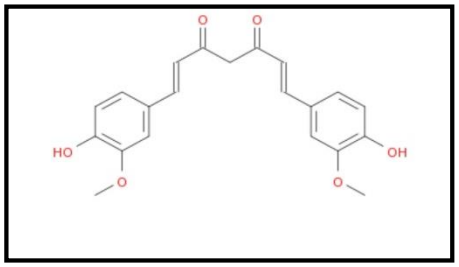
A number of scientific researchers have been carried out on medicinal herbs. Herbs have anti-inflammatory and antioxidant activities that may be used in the treatment of AD. Alzheimer's patients have an acetylcholine deficiency. Anti-inflammatory herbs may reduce inflammation of the brain tissue in Alzheimer's: German chamomile, Ginseng, licorice, turmeric, and white willow bark. Acetylcholine is a neurotransmitter that plays a key role in cognitive function and reasoning. The brains of those with mild-to-moderate Alzheimer's disease, a progressive type of dementia, have abnormally low acetylcholine concentrations. This means that any compound that enhances the cholinergic system in the brain may be useful in treating Alzheimer's disease and similar brain malfunctions. The herbs that inhibit Acetylcholinesterase (AchE) contain natural COX-2 inhibitors, also reported as medicinal herbs, for AD indication. [31]

Some ayurvedic herbs like Guduchi, Yashtimadhuk, Padma (Nelumbo nucifera), Vacha, Convolvulus pluricaulis, Shankhpushpi, Pancha-Tikta-Ghruta Gugguli, Amalaki, Musta Arjun, Amalaki, Ashwagandha, Galo Satva, Kutaj, and others are excellent herbs for slowing down the brain cell degeneration caused by Alzheimer's. They enhance the brain's ability to function, and therefore, provide stability when used consistently [31]

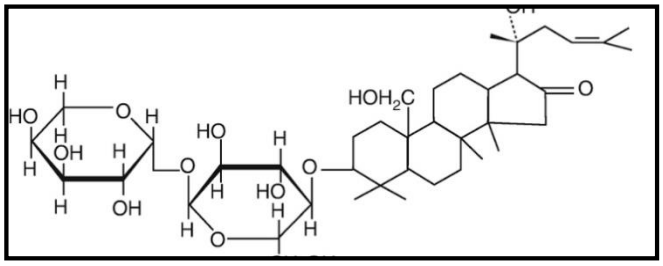
1) **Curcuma longa L. (Zingiberaceae)**

Curcuma longa (Turmeric, Harida) has been used as a source of Curcumin (diferuloylmethane), an orange-yellow component of turmeric or curry powder. Studies have proved that Curcumin has anti-inflammatory and antioxidant activities, and it helps in combating Alzheimer's disease (AD). Regular consumption of this herb helps in keeping the mind balanced [32] .The dose of curcumin can be reduced by making it to colon targeting [33].

Structure of curcumin:



Structure of Bacoposide



2) BacopamonnieraWettst. (Scrophulariaceae)

Goswami et al., evaluate the effect of Bacopa monnieri (Brahmi), associated with the Ayurveda system of medicine, on the cognitive functions in Alzheimer's disease patients, and conclude that it could be beneficial in these patients, but more study is needed [34].

3) Centellaasiatica L. (Umbelliferae)

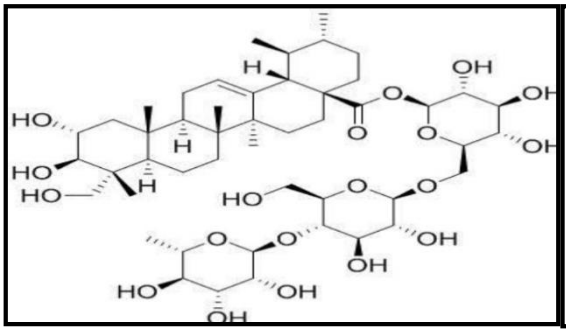
Extract from the leaves of Gotu Kola (Centella asiatica) has been used as an alternative medicine for memory improvement in the Indian Ayurvedic system of medicine for a long time [31].

4) Ginkgo biloba:-

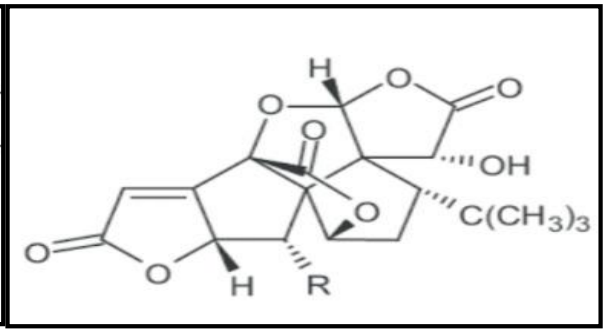
Ginkgo Biloba is the best known herb for Alzheimer's disease and its associated symptoms. In controlled clinical trials, using a placebo and control group, ginkgo biloba extracts showed therapeutic benefits in Alzheimer's, similar to prescription drugs such as Donepezil or Tactrin, with minimal undesirable side effect [35] .The chief chemical constituent of gingko biloba is gingkolides and it is a pertinent antioxidant, with neuroprotective and cholinergic activities that help in the management of AD. Ginkgo biloba improves protection against Aβ protein-induced oxidative damages (degrading hydrogen peroxide, preventing lipids from oxidation, and trapping the reactive oxygen species)[36]. Scientific studies have shown its promise on cognition-enhancement (booster), if used during the early stages of Alzheimer's disease. [37].

The ginkgo seeds contain a potentially toxic substance, ginkgotoxin (4-O-methoxypyridoxine), which has anti-vitamin B6 activity and inhibits GABA formation, which can potentially lead to convulsions and loss of consciousness.

Structure of Asiaticoside:-



Structure of Ginkgolide L :-



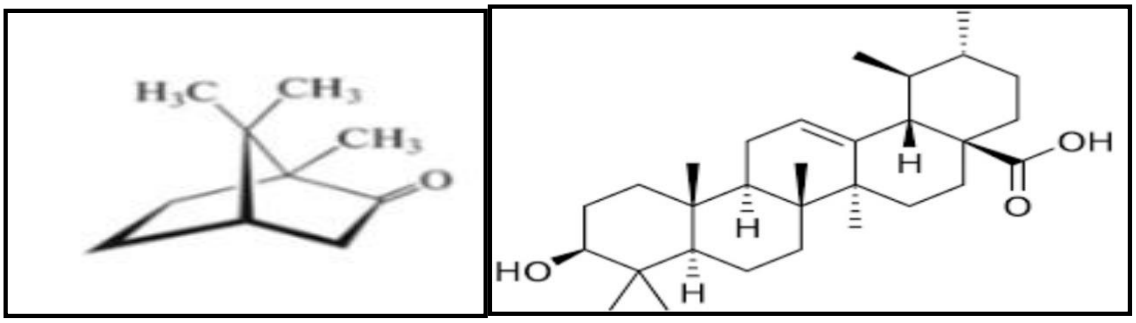
5) Salvia officinalis (Lamiaceae):-

Sage as it is more commonly referred for Alzheimer's disease treatment. It has been reported to assist the brain in the fight against AD. Sage contains the antioxidants carnosic acid and rosmarinic acid. These compounds are thought to protect the brain from oxidative damage [38].

Salvia officinalis has been used in herbal medicine for many centuries. After 4 months of treatment, salvia officinalis extract produced a significantly better outcome on cognitive functions

than placebo - as seen on the ADAS cognitive subscale and the Clinical Dementia Rating Sum of Boxes scale in patients with mild to moderate AD aged between 65 and 80 years [39].

Structure of Camphor: **Structure of Ursolic acid:**



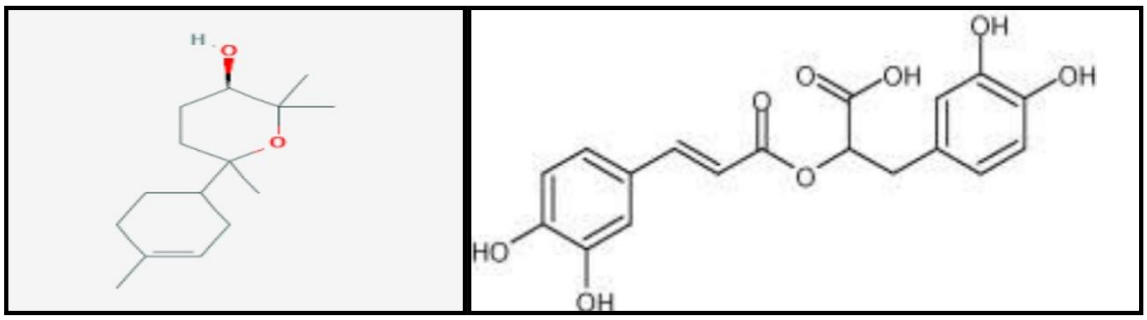
6) Rosmarinus officinalis (Lamiaceae)

Rosemary (Satapatrika) contains the following natural COX-2 inhibitors: Apigenin, carvacrol, eugenol, oleanolic acid, thymol, and ursolic acid. 'If a synthetic COX-2 inhibitor could prevent Alzheimer's disease, so could a natural COX-2 inhibitor,' according to Duke 2007. In addition, Rosemary contains nearly two dozen antioxidants and another dozen anti-inflammatory compounds. Some of the strongest antioxidant substances in the herb are carnosic acid and ferulic acid, which have even greater reported antioxidant activity than the widely common synthetic antioxidants butylated hydroxytoluene (BHT) and butylated hydroxyanisole (BHA) [40]. Rosemary can be used as a tea, in shampoo, or in bath water, because it can be absorbed through the skin [41].

7) Matricariarecutita (Asteraceae)

German Chamomile is having different compounds like, Chamazulene (27.80 %), β-pinene (7.93 %), 1,8- cineole (7.51 %), α-pinene (5.94 %), α-bisabolol (5.76 %) were found major compounds in Chamaemelum nobile which said to stimulate the brain, dispel weariness, calm the nerves, counteract insomnia, aid in digestion, break up mucus in the throat and lungs, and aid the immune system. Chamomile can relieve anxiety, and in higher doses, leads to drowsiness, according to the University of Maryland Medical Center [42].

Structure Alpha - bisabolol oxide A **Structure of Rosmarinicacid:-**



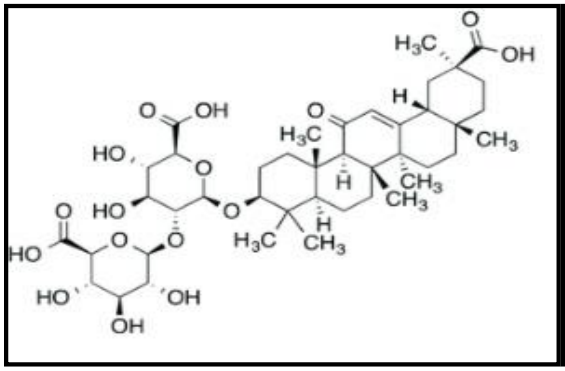
8) Melissa officinalis L. (Lamiaceae)

Historically, Melissa officinalis (lemon balm) was believed to sharpen memory. Lemon balm can also temporarily improve cognitive decline as well as improve the mood for Alzheimer's patients. Another study addressing the use of lemon balm for Alzheimer's disease, concluded that Melissa officinalis is one of several plants that may be useful in the prevention and treatment of Alzheimer's disease due to its ability to inhibit acetylcholinesterase and its antioxidant activity [42, 43].

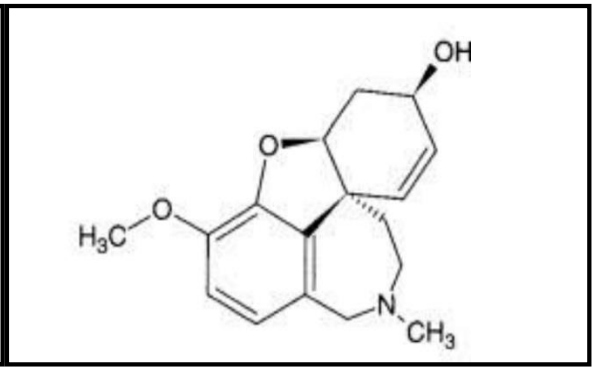
9) Glycyrrhizaglabra (Fabaceae)

Alzheimer's disease is characterized by neuronal loss and the presence of extracellular senile plaques, whose major constituent is amyloid-β peptide (Aβ). In one study scientists investigated the effects of a water extract of licorice (Yashti-madhuka) on Aβ25-35-induced apoptosis in PC12 cells. Results suggest that GWE exerts a protective effect against apoptotic neuronal cell death induced by Aβ fragments. Extract from the licorice root is reported to treat or even prevent brain cell death in diseases like Alzheimer's and its associated symptoms [44]. Liquorice contains 18β-glycyrrhizic acid (which is the major bioactive compound in the underground parts, which also called glycyrrhizic or glycyrrhizinic acid and a glycoside of glycyrrhetinic acid),

Structure of Glycyrrhizic acid:



Structure of Galanthamine:



10) Galanthusnivalis L. (Amaryllidaceae)

The chief chemical constituent of the Galanthus nivalis L. (common snowdrop) is Galanthamine, and this is an isoquinoline alkaloid. Acetylcholinesterase (AChE) inhibitors, which are also called 'anticholinesterase drugs', have been recently approved as a promising treatment approach for AD. Galanthamine has been found to be the long-acting and specific inhibitor of the AChE enzyme and to potentiate cholinergic nicotinic neurotransmission by allosterically modulating the nicotinic acetylcholine receptors, which may be of additional value in the treatment of AD[45,46].

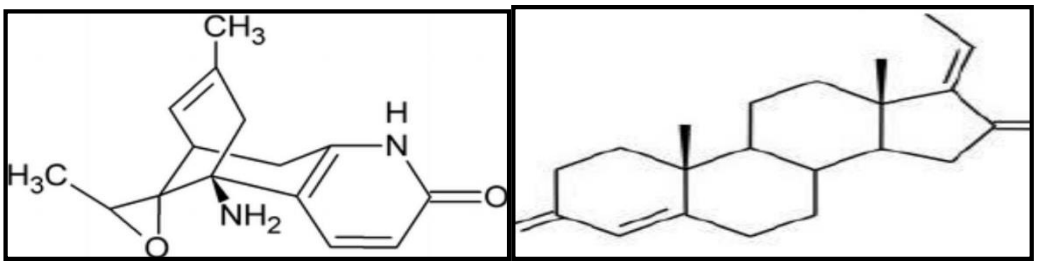
11) Huperzia serrata (Lycopodiaceae)

Huperzia serrata (Thunb. ex Murray) is one of the genera in the Huperziaceae family (syn.

Lycopodiaceae family). This genus, has been used for its memory-enhancing effect since ages in the Traditional Chinese Medicinal system (TCM), and is known to contain a large group of alkaloids called 'Lycopodium alkaloids'. Huperzine A, a novel Lycopodium alkaloid extracted from Huperzia serrata, is well known as a reversible, potent, and selective AChE inhibitor. It is also known as 'Qian Ceng Ta' in China, and Huperzine A has been used as a therapeutic agent for AD from centuries [47].

As reported by researchers, taking Huperzine-A leads to a significant improvement in memory, concentration, and the learning capacity. Research has also shown that Huperzine-A substantially reduces the abnormally high radical activity both in the brains of elderly animals as well as in the blood of Alzheimer's patients. An experimental study in monkeys has shown that it reverses scopolamine-induced amnesia, suggesting that it may benefit the cognitive problems in Alzheimer's patients or those with other cognitive disorders [48].

Structure of Huperzine- A: **Structure of Guggulsterone:**



12) Commiphorawhighitti (Burseraceae)

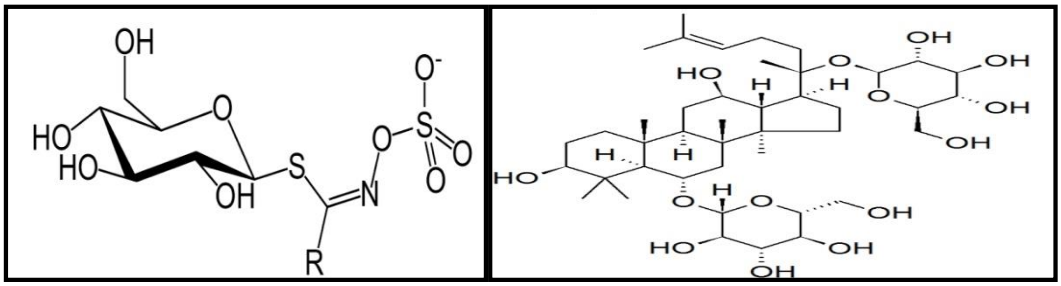
Commiphora whighitti (Guggulu), a plant resin, contains the major constituent of guggulipid, which is guggulsterone. The guggulipid has been seen to be a potential cognitive enhancer for improvement of memory in scopolamine-induced memory deficits [49].

Commiphora whighitti acts on impairment in learning and memory and decreased choline actyl transferase levels in hippocampus. However, Commiphora whighitti shows maximum effects on memory functions and the potential for dementia disorder [50].

13) Lipidium Meyenii Walp (Brassicaceae)

LipidiumMeyenii (maca), is known as Maca. Maca shows beneficial improvement in memory and learning. Black maca improves experimental memory impairment, induced by ovariectomy, due in part, to its antioxidant and AChE inhibitory activities. Results demonstrated that black maca can enhance learning and memory in OVX (ovariectomized) mice and this effect might be related, at least in part, to its ability to reduce LPO (Lipid peroxidation) and AChE in OVX mice[51].

Structure of Glucosinolate: **Structure of Ginsenoside:**



14) Panax Ginseng (Araliaceae)

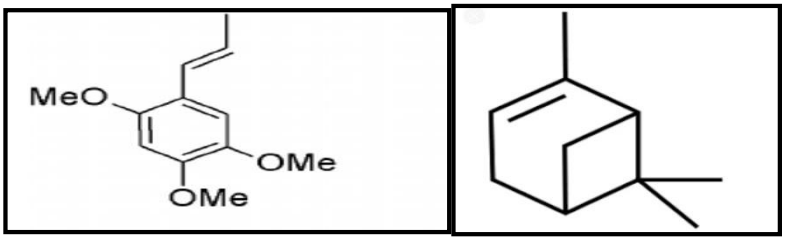
Panax Ginseng (Ren-shen) contains saponins protopanaxadiol, protopantriol, and oleanolic acid saponins that are reported to have memory-enhancing action for the learning impairment induced by scopolamine [49]. Ginseng grows in Northeastern Asia. The Ginseng root has been used in folk medicine in countries like China and Korea, for boosting Qi (energy), from ancient time. Ginseng has a history of medicinal use that goes back thousands of years. The ginseng extract has many uses, and claim to achieve and maintain both physical health and mental well-being [52].

Research has also suggested that ginseng is able to enhance the psychomotor and cognitive performance, and can benefit AD by improving the brain cholinergic function, reducing the level of AD, and repairing the damaged neuronal networks [42].

15) Acorus calamus L. (Araceae)

Acorus Calamus (Sweet flag)) (Araceae) possesses a beneficial memory enhancing property for memory impairment, learning performance, and behavior modification. Acorus Calamus inhibits the acetylcholinesterase (AChE). Acorus Calamus contains a majority of α-and β-asarone[49]. In the Ayurveda medicine system, Acorous Calamus has been used for the treatment of memory loss and its related symptoms. Acorus Calamus also shows anti-inflammatory, antioxidant, antispasmodic, cardiovascular hypolipidemic, immunosuppressive, cytoprotective, antidiarrheal, antimicrobial, and anthelmintic activities.

Structure of Alpha-asarone : **Structure of α-pinene:**



16) Angelica archangelica L.(umbelliferae)

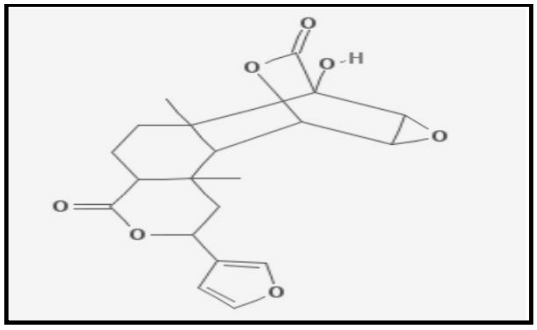
Angelica archangelica L., also known as Dudhachoraa (Laghu Coraka), contains several substances that have the same kind of activity as drugs used for Alzheimer's disease. These

substances do not cause the side effects observed with drugs, such as, nausea, stomach ache, insomnia, and so on. The same phytochemicals in Angelica archangelica can also increase blood flow in the brain. A study shows that chloromethane sub-fraction of a methanol extract inhibit AChE in-vitro [53,54].

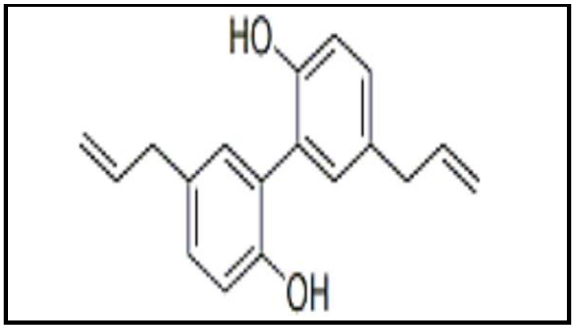
17) Tinosporacordifolia (Menispermaceae)

Tinospora Cordifolia (Guduchi) possesses a memory enhancing property for learning and memory in normal and memory-deficits animals. Tinospora Cordifolia's mechanism for cognitive enhancement is by immunostimulation and synthesis of acetylcholine, this supplementation of choline enhances the cognitive function [49].

Structure of tinosporide



Structure of magnonol:



18) Magnolia officinalis (Magnoliaceae)

The bark of Magnolia Officinalis (talauma) is used as a traditional memory enhancing agent in Chinese medicine for the treatment of neurosis, anxiety, stroke, and dementia. Magnolia Officinalis inhibits the memory impairment induced by scopolamine through the inhibition of AChE. The ethanolic extracts of M. officinalis, magnolol and honokiol, are reported to have antioxidant activity in vitro and in vivo [49, 54].

19) Collinsoniacanadensis (Lamiaceae)

Horsebalm (Monarda) has been reported to prevent the breakdown of acetylcholine. The chief chemical constituents of horsebalm are carvacol and thymol which are used for AD. Normally our body's protective blood-brain barrier helps prevent harmful substances in the blood from reaching the tissues of the brain. However, it can also prevent helpful medicines from reaching the brain. The horsebalm compounds seem to cross that great divide. [41]

20) Bertholettiaexcelsa (Lecythidaceae)

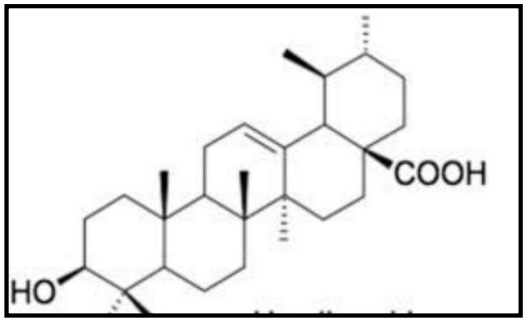
Although the name is Brazil Nuts, the most significant exporter of Brazil nuts is not Brazil, but Bolivia. In Brazil these nuts are called castanhas-do-Para [55].It has a high concentration of lecithin, which contains choline. Choline is a building block for acethycholine. These building blocks enhance the concentration of acethylcholine in AD patients. Other plants that contain good

amounts of lecithin are dandelion flowers, poppy seeds, soybeans, mung beans, horehound, ginseng, cowpeas, English peas, and lentils [41].

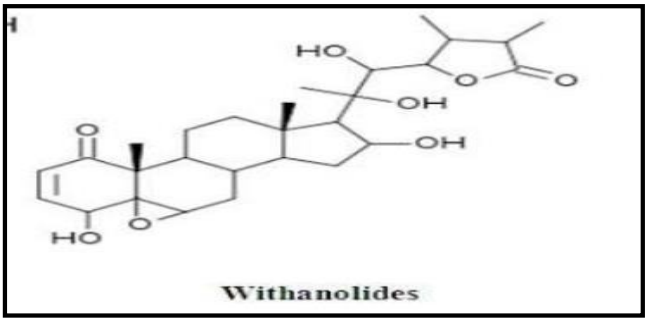
21) Urticadioica L. (Clusiaceae)

Stinging Nettle has been used for centuries to treat allergy symptoms, particularly hayfever, which is the most common allergy problem. It contains biologically active compounds that reduce inflammation. It contains the mineral boron that is reported to enhance the levels of estrogen, which is a hormone in the body, which can be beneficial in short-term memory. Stinging nettle has also been shown to elevate the mood in some Alzheimer's patients [41, 55].

Structure of Ursolic acid



Structure of withanolode:



22) Withaniasomnifera (Solanaceae)

Active glycowithanolides of Withania somnifera (Ashawgandha) have a significant antioxidant function, which is accomplished by increasing the activities of superoxide dismutase, catalase, and glutathione peroxidase [56]. Ashwagandha is also reported as a Nervine tonic that rejuvenates the cells and boosts energy. The assessment of cholinesterase inhibition was carried out by some scientists using a colorimetric method based on Ellman's reaction and demonstrated that the W. Somnifera extract significantly inhibited AChE in a concentration-dependent manner[57].

Structure of Withanolides:-

23) Ginseng

Panaxi ginseng's main active ingredient is panaxsaponin, which can enhance psychomotor and cognitive performance, and can benefit AD by improving brain cholinergic function, reducing the level of Aβ and repairing damaged neuronal networks [58]. The high-dose ginseng group showed statistically significant improvement on the Alzheimer Disease Assessment Scale (ADAS) and Clinical Dementia Rating [59]. The evidence for ginseng as a treatment of AD is thus scarce and inconclusive. Further rigorous trials seem warranted [60].

Structure of Ginsenosides:-

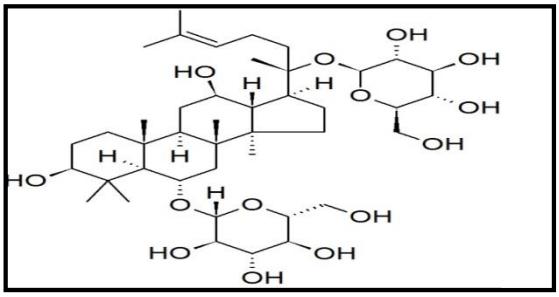


Figure 2: Treatment medicinal herbs to treat Alzheimer's disease

THE MIND DIET TO TREAT ALZHEIMER'S:

- Vegetables
- Nuts
- Berries
- Beans
- Whole grains
- Fish
- Olive oil

Green leafy vegetables:

The MIND diet recommends frequent servings of green leafy vegetables. Kale, spinach, broccoli, collards and other greens are packed with vitamins A and C and other nutrients. At least two servings a week can help, and researchers found six or more servings a week provide the greatest brain benefits. The Mediterranean and DASH diets do not specifically recommend these types of vegetables, but the MIND diet study found that including them in addition to other veggies made a difference in reducing the risk of Alzheimer's [60].

Other vegetables:

Like other diets focused on weight loss and heart health, the MIND diet emphasizes the importance of vegetables for brain health. The researchers recommend eating a salad and at least one other vegetable every day to reduce the risk of Alzheimer's [60].

Nuts:

Nuts are a good snack for brain health, according to the MIND diet study. Nuts contain healthy fats, fiber and antioxidants, and other studies have found they can help lower bad cholesterol and reduce the risk of heart disease. The MIND diet recommends eating nuts at least five times a week [60].

Berries:

Berries are the only fruit specifically recommended in the MIND diet. "Blueberries are one of the more potent foods in terms of protecting the brain," Morris said. She noted that strawberries have also shown benefits in past studies looking at the effect of food on cognitive function. The MIND diet recommends eating berries at least twice a week [60].

Beans:

If beans aren't a regular part of your diet, they should be. High in fiber and protein, and low in calories and fat, they also help keep your mind sharp as part of the MIND diet. The researchers recommend eating beans three times a week to help reduce the risk of Alzheimer's [60].

Whole grains:

Whole grains are a key component of the MIND diet. It recommends at least three servings a day [60].

Fish:

The MIND diet study found eating fish at least once a week helps protect brain function. However, there's no need to go overboard; unlike the Mediterranean diet, which recommends eating fish almost every day, the MIND diet says once a week is enough oil [60].

Olive oil:

Olive oil beat out other forms of cooking oil and fats in the MIND diet. The researchers found people who used olive oil as their primary oil at home saw greater protection against cognitive decline [60].

Figure 3: The Mind Diet to Treat Alzheimer's

CONCLUSION:

A healthy diet may have a profound impact on many possible risk factors for AD and cognitive decline, so influencing the onset and progression of these disorders. In particular, healthy dietary models as MeDi or DASH diet may have the potential of modifying some cognitive outcomes, and some recent prospective studies focusing on AD and dementia appeared to be really promising. In fact, higher levels of accordance with the MeDi has been linked to slower cognitive decline, reduced risk of AD, transition from MCI to AD, and decreased mortality in AD patients. Therefore, higher adherence to the MeDi may affect the risk of both MCI and AD, probably influencing also disease progression. However, cognitively intact individuals have increased chance to maintain healthy lifestyle and diet, while more or less mildly cognitively impaired individuals apparently do not. One important task is to test whether lifestyle and dietary interventions may improve cognition to the extent that the individuals in question regain the ability to permanently maintain healthy lifestyle and diet associated with cognitive improvement without intervention. Single herbs or formulations may be able to complement approved drugs for AD. No serious adverse events have been reported. The current evidence to support their use alone, however, is inconclusive or inadequate. The detailed study about herbs and their benefits in treatment of AD is needed to be studied currently.

ACKNOWLEDGEMENT

Authors wish to express their sincere thanks to Hon. Smt. Padmatai Shinde madam, President, Shriram Dnyan Shikshan Prasarak Mandal, Vaijapur, Dist-Aurangabad, MS, India-423701 and

Hon. Shri. Rajesh Shinde sir, President, Shriram Dnyan Shikshan Prasarak Mandal, Vaijapur, Dist-Aurangabad, MS, India-423701 for their constant encouragement and support.

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