International Journal of Innovation and Research in Educational Sciences
Volume 5, Issue 6, ISSN (Online) : 2349–5219

Preventive Role of Spice Condiments in Alzheimer's Disease

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Date of publication (dd/mm/yyyy): 06/11/2018

Abstract **– Alzheimer's disease (AD) is a primary degenerative disease, characterized by memory loss, unusual behavior and a declining cognitive function. Main pathological culprits are extracellular deposition of beta amyloid-Aβ and intracellular accumulation of tau protein and some risk factors also. Study objective is to investigate the spice condiments which were used plentiful by our ancestors whether these have the potentiality to ameliorate the AD pathology. Ten, highly consumed Sri-Lankan spice condiments had been scientifica- -lly shown their potentiality to ameliorate AD pathology in different ways. Those are: *Cinnamomun zylanicum* (True cinnamon), *Cuminum cyminum* (Cumin), *Curcuma longa* L (Turmeric), *Myristica fragrans (Nutmeg), Piper nigrum (Black pepper), Caryophyllus aromaticus* (Clove), *Elettaria cardamomum* (Cardamom), *Zingiber officinale* (Ginger), *Trigonella foenum graecum* (Fenugreek) and *Coriandrum sativum* (Coriander). The review concluded that Cinnamon, Curcumin, Pepper, Clove and Zinger are involved in reversing the main pathological changes of AD while others are ameliorating the risk factors of AD.**

Keywords **– Alzheimer's Disease, Spice Condiments, Beta Amyloid and Tau Protein.**

I. INTRODUCTION

Alzheimer's disease (AD) is an age-associated, irreversible, primary degenerative brain disease of unknown etiology that is characterized by severe memory loss, unusual behavior, personality changes, and a decline in cognitive functions. Behavioural changes are common, wandering, agitation and aggression. Emotional symptoms like anxiety and phobia contribute significantly to the clinical profile in AD [1], [2]. AD is insidious onset and followed by gradual deterioration and then death in about ten years. In the early stage disease shows a loss of short-term memory, inability to learn new information, mood swings, and difficulty in finding words, forgetting names, and losing items. Further frustration, hostility, and irritability are common emotional features exhibited by patients with AD. In severe cases, patient's memory becomes completely lost, and sense of time and place disappears and patient become totally dependent upon others and eventually requires comprehensive care. Thus, AD presents a considerable problem in patient manageme--nt as well [3]. Sometimes AD involves people in their 40s and 50s, but is mainly a disease of old age. Based on clinical evaluations, 13% of persons over 65 years and 45% of those over 85 have AD [4].

It is believed that therapeutic intervention that could postpone the onset or progression of AD would dramatically reduce the number of cases in future. There is no cure for Alzheimer's exists, and only a few medicines are now available to treat the AD, which show limited effectiveness. In fact, a new path shows and several scientific studies have described the use of various herbs and their bio-active constituents for the treatment of Alzheimer's disease. The exact mechanism of their action is still not clear, however, phytochemical studies of the different parts of the plants have shown the presence of thousands of many valuable phytochemical compounds, such as lignans, flavonoids, tannins, polyphenols, triterpenes, sterols, and alkaloids, that show a broad spectrum of pharmacological activities, including anti-inflammatory, anti-amyloidogenic, anti-cholinesterase, antioxidant effects etc in reversing AD pathology. Interestingly, spice condiments also a group of plant origin which involve in reversing AD pathology.

Spice condiments may be dried seeds, root, bark or other vegetables matter, used to add flavor and colour to a dish. We also had a belief that the spice condiments are added to a dish as flavoring and colouring agents. In contrast to this belief, there are scientific evidences that the spice condiments are having wide varieties of phytochemicals and bio activities for maintaining the health and relieving pathological process. Our ancestors used plenty of variety of spice condiments in varies amounts in daily consuming curries and lived healthily, but only one or two little amount of spice condiments are being used to prepare a curry, now. Then, in Sri-Lanka around 7-8 spice condiments together with scraped coconut grind on the grinding stone prepared as a paste of these spice condiments and added to curries.

Aim of this review is to discover the contribution of daily using ten spice condiments on the health benefits of AD. The review summarizes the information from journal articles concerning the phytochemistry, biological, and cellular activities and clinical applications of these condiments on AD. This review would scientifically prove the value and contribution given by the spice condiment in reverse AD pathology.

II. METHODOLOGY

Initiate to investigate the preventive role of spice condiments on AD pathology, the pathogenesis of the disease should be understood and therefore, the pathogenesis of AD was composed from the e-books. Existing phytochemicals and bioactivities of the spice condiments which have shown promise in reversing the AD pathology were filtered from the journal articles. Botanical name or English name of the each different condiment combining with the term Alzheimer's Diseases were used as the search term. PubMed, Google scholar, Science Direct data bases were used to filter the journal articles up to August 2018.

III. RESULTS AND DISCUSSION

3.1 *Pathogenesis of AD*

AD is driven by two processes: extracellular deposition of beta amyloid-Aβ and intracellular accumulation of tau protein. Both these compounds are insoluble. Aβ is the main component of senile plaques and tau is the component of neurofibrillary tangles. These two mechani- -sms lead to neurodegeneration and causes the cell death by means of a process called 'apoptosis' or 'programmed cell death' [5]. Aβ deposition is specific for AD and is thought to be primary. Tau accumulation is also seen in other degenerative diseases and is thought to be secondary.

3.1.1 *Extracellular deposition of Beta Amyloid (Aβ)*

Aβ is a 36 to 43 amino acid peptide, which is part of a larger protein, the Amyloid Precursor Protein (APP)**.** APP is a transmembrane protein, made by neurons and other brain cells. It is also found in extra-neural tissues and is especially abundant in platelets. Its function is unknown. The Aβ residue includes part of the transmembrane domain of APP and is derived from cleavage of APP by the enzymes β- and **γ**-secretase. Aβ monomers and oligo- -mers are further degraded by other enzymes. Defective in clearance of Aβ from aberrant cleavage of APP and other mechanisms results in the accumulation. Aβ monomers polymerize initially into soluble oligomers and then into larger insoluble fragments such as Aβ - 42, which precipitate as amyloid fibrils **[4].** These form "amyloid plaques" are thought to contribute to the nerve cell death that causes Alzheimer's symptoms.

3.1.2 *Intracellular Accumulation of Tau Protein*

Under normal conditions tau protein is responsible for the assembly of "microtubules" in brain cells. These microtubules form the "skeleton" of the cells. If the protein does not bind properly to the microtubule it tends to clump together. This is known as tangles and these are insoluble fibers and deposits in the neuronal body and prevent neurons from functioning well. These deposits interfere with cellular functions by displacing organelles. By distorting the spacing of microtubules, they impair the axonal transport thus affecting the nutrition of axon terminals and dendrites. No mutations of the tau gene occur in AD. Abnormal tau first appears in the entorhinal cortex, then in the hippocampus, and at later stages in association cortex. Recent observations in transgenic mice suggest that the spread of the pathology to anatomically linked areas occurs by passage of abnormal tau across synapses [4].

3.1.4 *Contributing or Risk factors of AD*

Chronic cellular damage from free radicals, excitotoxicity, nonenzymatic glycation of proteins, and other factors contributes to the loss of neurons and synapses that is associated with old age and aggravates the pathology of AD. Other than Aβ and tau, neuro-inflammation is the most important factor involved in the pathogenesis of AD.

The effects of neuro-inflammation are mediated by activated microglial cells which are a source of cytokines and a potent generator of free radicals. Free radicals are atoms or groups of atoms with an odd (unpaired) number of electrons and can be formed when oxygen interacts with certain molecules. The main danger comes when they react with important cellular components such as DNA, or the cell membrane. This leads the cell to poor in its function or death of cell. To prevent free radical damage the body has a defense system of antioxidants. Antioxida- -nts are molecules which can safely interact with free radicals and terminate the chain reaction before vital molecules are damaged. This process is accelerated in AD by the action of Aβ and activated microglia, also a source of free radicals. Type 2 diabetes is a risk factor for AD. AD patients have low levels of insulin and insulin resistance in the brain. These changes impair energy metabolism in neurons and adversely affect signaling pathways dependent on insulin and its receptors. Furthermore, nonenzymatic glycation of proteins produces neurotoxic derivatives that aggravate oxidative damage. Increased levels of homocysteine (also a risk factor for stroke) and decreased dietary folate potentiate these neurotoxic effects. Homocysteine increases with advanci- -ng age and is elevated in persons with polymorphisms of 5, 10-methylenetetrahydrofolate reductase (MTHFR), an important enzyme involved in folate metabolism.

Cholinergic neurotransmitter systems are widely distributed in the human brain and play a main role in regulating many activities, including memory, learning, attention, and behavior. Normal cholinergic function depends on the rapid hydrolysis of the neurotransmitter acetylcholine (ACh) by cholinesterases [6]. The brain of mammals contains two major forms of cholinesterases: acetylcholinesterase (AChE) and butyrylcholinesterase (BuChE). The two forms differ genetically, structurally, and for their kinetics. In human brain, BuChE is found in neurons and glial cells, as well as in neuritic plaques and tangles in AD patients. Under normal condition AChE is many folds higher than that of butyrylcholinesterase (BChE) whereas, AChE activity decreases progressively in the brain of AD patients, BuChE activity shows some increase [7]. Therefore, cholinesterase inhibitors are currently the only approved therapy for the symptomatic treatment of AD.

In AD, the brain shows a loss of cholinergic neurons in the basal forebrain, decreased acetylcholine (Ach) levels, and a decrease in the acetylcholine synthesizing enzyme choline acetyltransferase in the cerebral cortex [4]. In the normal physiological view acetylcholine (ACh) is an organic chemical that functions in the brain and body, as a neurotransmitter (a chemical message released by nerve cells to send signals to other cells) and is essential for processing memory and learning. Cholinesterase is an enzyme that rapidly breaks down the neurotransmitter acetylcholine, which is vital for the transmission of nerve impulses. ACh is decreased in both concentration and function in patients with Alzheimer's disease. By any means, if can reduce the amount of cholinesterase breakdown, it would be a symptomatic relief for AD patients. Anticholinesterases are a class of drugs that decrease breakdown of acetylcholine and can be used in conditions whereby there is an apparent lack of this messenger transmission in AD.

Although other neurotransmitter systems (noradrenalin, serotonin, somatostatin and other peptides) are also deficient in AD, the cognitive impairment correlates best with the loss of cholinergic input [4].

3.2 *How the Spices Condiments Contribute Against*

different Pathogenesis of AD?

The ten spice condiments that have been widely used for thousands of years in household spices were analyzed in this study. The review reveals the results of these ten spice condiments show its protective role in different ways of pathogenesis of AD. The condiments are as follows *Cinnamomun zylanicum* (True cinnamon), *Cuminum cyminum* (Cumin), *Curcuma longa* L (Turmeric), *Myristi- -ca fragrans (Nutmeg), Piper nigrum L (Black pepper), Caryophyllus aromaticus* Linn (Clove), *Elettaria cardam- -omum* Maton (Cardamom), *Zingiber officinale* (Ginger), *Trigonella foenum graecum* (Fenugreek) and *Coriandrum sativum* Linn (Coriender).

3.2.1 *Cinnamomun zylanicum (Cinnamon)*

Cinnamomun Zylanicum, is known as Cinnamon in English. It is belonging to the family Lauraceae and mostly stem bark is used in curries as food additive due to its pleasant aroma. Aa the parts of the cinnamon tree including the bark, leaves, flowers, fruits and roots, has some medicinal or culinary use. Hence, in native Ayurvedic and in Unani medicine Cinnamon is considered as a remedy for respiratory, digestive and gynaecological ailments.

3.2.1.1 *Reversing Potentiality of the AD Pathology by Cinnamomun Zylanicum*

A study with an aqueous extract of Ceylon cinnamon (*C. zeylanicum*) or true cinnamon had found to inhibit tau aggregation and filament formation and reverse back the pathology of AD. This study had revealed that the aqueous extract of *C. zeylanicum C. zeylanicum* promotes complete disassembly of recombinant tau filaments and cause considerable alteration of the morphology of paired-helical filaments isolated from AD brain [8]. George & Graves revealed in their studies that two compounds found in cinnamon, cinnamaldehyde and epicatechin are showing some promise in the effort to fight the AD. The use of cinnamaldehyde, the compound responsible for the bright, sweet smell of cinnamon, has proven effective in preventing the tau knots. By protecting tau from oxidative stress, the compound, an oil, could inhibit the protein's aggregation. To do this, cinnamaldehyde binds to two residues of an amino acid called cysteine on the tau protein. The cysteine residues are vulnerable to modifications, a factor that contributes to the development of Alzheimer's. Graves further said to protect from sunburn we wear hat as like cinnamaldehyde is like a cap, it can protect the tau protein by binding to its vulnerable cysteine residues [9].

Another study had revealed that an oral Administration of Cinnamon extract reduces β-Amyloid oligomerization and corrects cognitive impairment in Alzheimer's disease in animal models. The research identified that cinnamon extract markedly inhibits the formation of toxic Aβ oligomers and prevents the toxicity of Aβ on neuronal PC12 cells. Further, in the same study it is said that the oral administration of cinnamon extract to an aggressive AD transgenic mice model led to marked decrease in Aβ oligomers, reduction of plaques and improvement in cognitive behavior [10].

3.2.2 *Cuminum cyminum (Cumin)*

Cuminum cyminum known as cumin in English and is belonging to the family Umbelliferae. The word cumin was derived from Latin cuminum, which itself was derived from Greek (kyminon) [11]. Native of the plant is from the Mediterranean area, may be Egypt and Syria. However, it has been cultivated extensively in India, China, U.S.A, Malta and Sicily but now, the major production of cumin was coming from India. The seeds are used in curries as food additive due to its pleasant aroma and taste. Seeds contain a volatile oil which is composed of the hydrocarbon cymol, an oxygenated oil called cuminal, acymene and other terpenes. In Unani system of medicine, the fruits of *Cuminum cyminum* were used as stomachic, carminative, astringent and emmenagogue. It is useful for dyapepsia, chronic diarrhea and bilious nausea in pregnant women. It increase the milk secretion soon after child birth [12]. It is useful for the treatment of corneal opacities, ulcers, boils, styes and to relieve cough and inflammation [13].

3.2.2.1 *Reversing Potentiality of the AD Pathology by Cuminum Cyminum*

In a recent study had revealed that aqueous extract of *Cuminum cyminum* seed acts as potent inhibitor of BChE. Note: Ellman's assay was used to study the cholinesterase inhibition in-vitro [14]. In another study an aqueous extract of *Cumininum cyminum* had been tested for in vitro acetylcholinesterase inhibitory activity based on the same Ellman's method. In that study *C. cyminum* had shown maximum inhibition of 76.90 ± 0.003 % in an aqueous extract at 50 µg / ml final concentration. Also, at lower concentration (12.5µg/ml) and higher concentration (25µg/ml) *C. cyminum* exhibited competitive and mixed mode of inhibition respectively. These results illustrates the fact that *C. cyminum* can acts as an inhibitor of AChE and helpful in enhancing memory and other cognitive functions of brain [15].

A study had done to find memory-enhancing activity of the extract of *Cuminum cyminum* by conditioned avoidance response (CAR) using Cook's pole climbing apparatus in normal and scopolamine-induced amnestic rats. The activities involve acquisition, retention, and retrieval was measured using CAR. The acquisition was quicker in the extract-treated rats (100, 200, and 300 mg/kg body weight) in comparison with control, indicating significant anti-stress effect by the extract. The amnesia was less in treated group showing better retention and recovery than control group. Finally this study had revealed that cumin extract possess anti-stress, antioxidant, and memory-enhancing activities [16].

3.2 3 *Curcuma Longa Linn (Turmeric)*

Curcuma longa Linn is known as curcumin or turmeric is a rhizome. Usually the primary and secondary rhizomes are dug up and dried or boiled and dried. Turmeric is used as a curry powder and has an aromatic odour and a warm nature and slight bitter in taste. A native of Southern Asia and now largely cultivated in India, Ceylon, China, Java and other tropical countries. Turmeric is used as an integral component of almost all dishes. It is used as a preservative, food additive or food colouring agent. It has been used as a main ingredient in all traditional systems of medicine from time immemorial. Internally, it is given as an anthelmintic and blood disorders. It has been given diarrhea, dysentery, flatulence, colic, jaundice, amenorrho- -ea etc. In Ceylon

paste is applied on urticarial, boils, sprains and internally for rheumatism, bronchial ailments and snake bites [12].

3.2.3.1 *Reversing Potentiality of the AD Pathology by Curcuma Longa*

A study found that curcumin help to improve the condition of behavioral and psychological symptoms of dementia (BPSD). The study further said that three patients had exhibited irritability, agitation, anxiety and apathy two patients suffer from urinary incontinence and wonderings. After intervention of turmeric powder capsules for 12 weeks they had started recovering from these symptoms. This had decreased significantly in both acuity of symptoms and burden of caregivers. Mini-Mental State Examination (MMSE) score had been gone up five points, from 12/30 to 17/30. Finally, this study concluded that the turmeric treatment had given a significant improvement of the behavioral symptoms in the AD [17].

A study had revealed that the levels of Aβ in AD mice that were given low doses of curcumin were decreased by around 40% in comparison to those that were not treated with curcumin [18]. Curcumin can cross the blood brain barrier as it possess a lipophilic nature. It is said that accumulation of small fibers called Aβ fibrils known as Aβ plaques are one of the most prominent pathological features of AD. Therefore, inhibition of A-beta generation, prevention of A-beta fibril formation, destabilization of pre-formed A-beta would be an attractive therapeutic strategy for the treatment of AD. An *in vitro* study had showed that curcumin inhibits aggregation as well as disaggregates to form fibrillar A-beta 40. A study used high technique demonstrated by mulita-photon microscopy revealed that curcumin reduces the existing senile plaques [19]. A review focused clearly that curcumin orally or its metabolites had shown the inhibition of Aβ deposition, Aβ oligomerization and tau phosphorylation in the brain of AD animal model including behavioural improvement [20]. AD pathology says that β-amyloid precursor cleavage enzyme (BACE-1) is a key enzyme that responsible for amyloid plaque production. Another study had done to assess anti- β-BACE -1 and behavioral activities of curcuminoids obtained from rhizomes of *Cur- -cuma longa*, diarylalkyls curcumin (CCN), demethoxycu- -rcumin (DMCCN) and Bisdemethoxy curcumin (BDMCCN) against AD *Drosophila melanogaster* models. Feeding and climbing activity, lifespan, and morphostructural changes in fly eyes also were evaluated. Finally, above study concluded that curcuminoids could be a potential therapeutics or lead molecules for prevention or treatment of AD [21].

A study had been conducted to examine the effect of ex--tract of *Curcuma longa* L on $H2O2^{-}$ induced toxicity in rat pheochromocytoma cell line PC12 by measuring cell lesion, level of lipid peroxidation and antioxidant enzyme activities. Cells were exposed 30 minutes to H2O2 (150 μM). A marked decrease in cell survival, activities of glutathione peroxidase and catalase as well as increased production of malondialdehyde (MDA) were found. Study revealed that pretreatment of the cells with extract of *Curcuma longa L* (0.5–10 μg/ml) prior to H2O2 exposure significantly elevated the cell survival, antioxidant enzyme activities and decreased the level of MDA. Further, it was understood that the protective effects of these radical scavengers reducing intracellular $O2_{\bullet} -$ on neuronal cell death [22]. It is to say that Curcuma *longa* L protect the neural death by many mechanisms.

3.2.4 *Myristica Fragrans Houttuyn (Nutmeg)*

Myristica fragrans is a small evergreen tree native to Eastern Moluccas and other Indian Islands, Amboyna, Borneo etc. it is now cultivated in Ceylon, Malaya, Philippines, West Indies and South America. This exotic tropical tree is unique in that it produces not just one, but two spices. One spice is nutmeg, the egg-shaped, brown-gray seed inside the fruit which has carminative and stomachic properties. Other spice is mace; red, lacy covering that surrounds the seed. Mace is dried, ground into a yellow-orange powder and used in both medicinally and sweet and savory dishes [23].

3.2.4.1 *Reversing Potentiality of the AD Pathology by Myristica Fragrans (Nutmeg)*

Anticholinesterases can be used in AD whereby there is an apparent lack of ACh messenger transmission. A study had revealed that 13 chemical compounds were isolated and identified from the seeds of *Myristica fragrans and showed anti-cholinesterase activity. From these 13 compounds, 3 had shown* the most effective activity ((7S)-8'-(4'-hydroxy-3'-methoxyphenyl)-7-hydroxypropyl] benze ne-2, 4-diol : (8R,8'S) -7'- (3', 4'-methylenedioxyphenyl) - 8, 8' - dimethyl - 7 - (3, 4 - dihydroxyphenyl) - butane : malabaricone C) [24].

Further another research had discovered that a compound in both nutmeg and mace known as macelignan, a polyphenol (plant-based antioxidant) that's found only in *Myristica fragrans*. It had also been shown that it possess a neuroprotective activity and use for AD [25]. In addition, memory boosting ability of nutmeg was invented in several studies in different ways. One study had designed to invent on memory boosting and regaining [26] while the other study had designed to invent on learning and memory [27] by the oral administration of extract of *Myristica fragrans* seed in Wistar albino rats. Both the studies revealed that the memory booting effect had seen clearly by the oral administration of the extract of *Myristica fragrans* seed. Added to this, another research had revealed that volatile oil of *Myristica fragrans* increase the levels of monoamine neurotransmitters in rats and gave the insight to its neuroprotective effect [28].

A study further revealed that methanolic and hydroalcoholic extracts of Myristica fragrans exhibit significant and paramount hydroxyl reducing capacity respectively in a dose dependent manner. This *in vitro* study concluded that the methanolic and hydroalcoholic Myristica fragrans extracts possess significant antioxidant activity and provide a scientific rationale for the traditional use of this plant [29]. As it possess an potent antioxidant it is a therapeutic effect on AD pathology.

3.2.5 *Piper Nigrum* L *(Black pepper)*

Common name is Black pepper belongs to the family Pip--eraceae. A climbing perennial indigenous to South Asia but extensively cultivated over the islands of Malay, West Indies and South America. Contain alkaloids such as β -

methyl – pyrroline, piperidine and piperovatine etc. Internally pepper is a stomachic, carminative and induces secretion of bile. It is an antidote for shell fish and mushroom poisoning. Externally it is good for rheumatism [23].

3.2.5.1 *Reversing Potentiality of the AD Pathology by Piper Nigrum L*

An in vitro and in vivo study had done on extract of *Piper nigrum* L. fruits for analyze the possible memory-enhancing and antioxidant properties. The memory-enhancing effects of the the extract of *Piper nigrum* L. fruits were studied by in vivo (Y-maze and radial arm-maze tasks) approaches. Methanolic extract of *Piper nigrum* L. fruits (50 and 100 mg/kg, orally, for 21 days) were given in amyloid beta (1–42) rat model of Alzheimer's disease. It was concluded in this study that administration of the extract of *Piper nigrum* L. fruit significantly improved memory performance and exhibited antioxidant potential [30].

Another AD induced rat model study had done to analyze the effect of *Piper nigrum* L for the prevention of alzheimer's associated histopathological, biochemical and behaviour changes. In this study animals were randomized into 4 groups each 6 rats in a group. Propylene glycol (1 ml / day) was gives to a group and named as Control group. Group 2 known as AD control group was given aluminium chloride 17mg / kg body weight for 2 months. 3rd and 4th groups were known as Test groups were given 20mg/kg/day and 200mg/kg/day *Piper nigrum* L, respectively and aluminium chloride 17mg/kg body weight for two months. At the end all rats were sacrificed and were send for histopathological and biochemical examination. Finally this study discovered that *Piper nigrum* L significantly improved learning and memory deficits associated with aluminium chloride and also showed the anticholinesterase activity with prevention of nerve degeneration. Also, histopathological report revealed that there was marked decrease in amyloidal plaque formation in rats brain who were pretreated with *Piper nigrum* L [31].

Many studies had discovered that methenolic extract of *Piper nigrum* L improves memory impairment by decreasing brain oxidative stress [32], piperine as the main component responsible for the anxiolytic and antidepress- -ant effects in Aβ rat model of AD. Further is had been reported that piperine inhibited monoamine oxidase activity, increased monoamine neurotransmitters levels in mouse models of behaviour disorders [33]. In addition, *Piper nigrum* fruits have the ability to protect against neurotoxicity and this effect could be related to its antioxi--dant activity [32].

A study had designed to investigate that the extract of *Piper nigrum* L in ameliorating neuro-inflammation. Adult male Sprague-Dawley rats models were used in groups for this study. Many bio markers such as levels of brain acetylcholine (Ach), serum and brain acetylcholinesterase (AchE) activity, C-reactive protein (CRP), total nuclear factor kappa-B (NF-κB), and monocyte chemoattractant protein-1 (MCP-1) were estimated to judge the study. The results revealed that *Piper nigrum* L methanolic extract have potent anti-inflammatory effects against neuro- infla--mmation characterizing AD.

3.2.6 *Caryophyllus Aromaticus Linn/ S*yzygium Aromaticum *(Clove)*

Common English name is clove. It is native of Moluccas, five islets lying off the coast of the largest Island of Dilolo, North-east of Celebes. Also it is grown in Sumatra, Brazil and most West Indian Islands. In Ceylon, it is cultivated in the mid country, upto an elevation of 2,000 feet. It is a small tree about 10 -13 feet height with horizontally spreading slender branches forming a dense pyramidal crown. Unexpanded flower buds are used medicinally and culinary purposes. Cloves are carminativ- -e, stomachic, stimulant. It cures colic; relieve chest ailments and allays thirst in children suffering from worms and indigestion. Further, clove oil or rubbed clove applied over the decay part of tooth in toothache [23].

3.2.6.1 *Reversing Potentiality of the AD Pathology by Caryophyllus Aromaticus/ S*yzygium *aromaticum* Linn

A study had done to investigate the neuroprotective mechanisms of clove oil in intracerebroventricular (icv)-colchicine induced cognitive dysfunction in rats. Colchicine icv administration formed impaired cognitive performance in Morris water maze (MWM) leads oxidative stress, raised AChE level, caused neuro-inflammation and mitochondrial dysfunction. The study discovered that treatment with clove oil (0.05ml/kg and 0.1ml/ kg) and minocycline (25 and 50mg/ kg) alone improved cognitive performance significantly with evidenced by reduced transfer latency and increased time spent in target quadrant (TSTQ) in MWM task, reduced AChE activity etc as compared to icv-colchicine treatment. This study had concluded that the major neuroprotective effect of clove oil due to its mitochondrial restoring and anti-oxidant properties along with a microglial inhibitory mechanism [34]. Proliferation and activation of microglia in the brain, concentrated around amyloid plaques, is a noticeable feature of AD [35].

To add this, another study had revealed that S*yzygium aromaticum* was capable to scavenge ROS and elevate the percentage of anti-oxidant enzymes. Further it said that also it activated and elevated the level of Sirtuin and downregulated γ-secretase level [36]. Sirtuin (SIRT1), one of the seven mammalian proteins of the sirtuin family, has recently been shown to attenuate amyloidogenic processi- -ng of amyloid-β protein precursor (APP) in cell culture studies *in vitro* and in transgenic mouse models of Alzheimer's disease [37].

A study had done on methanol extract of clove, its oil a--nd eugenol to determine the anti-cholinesterase activity. AChE and BChE inhibition study revealed that eugenol possess better inhibition of the enzymes than extract and oil. Also, it revealed that extract, oil and eugenol showed better inhibition of AChE than BChE. These evidences conclude that clove is potential to anti-cholinesterase agent for the management of cognitive ailments like Alzheimer's disease [38].

Cognitive function had been investigated in mice by using oral administration of clove powder with diet in three doses (400,800, 1600 mg / Kg) for seven days. The learning

and memory parameters were assessed. Clove showed significant improvement in the memory in young and aged animals and significant reduction in the brain cholinesterase activity in young mice (50.5 %) than aged mice (21.25 %) at the dose of 800 mg / Kg. Hence with this evidence the study conclude that clove has potentiality of the management of AD [39].

A study had done on worm (*Caenorhabditis elegans)* model and aim to evaluate effect of clove oil on aging and age-related neurodegenerative Alzheimer's disease. According to the authors view this model had been used due to its genetic similarity with human. Hence, at the conclusion it was said that Clove oil was found to improve health span and lifespan of *C. elegans* in dose-dependent manner [40].

3.2.7 *Elettaria Cardamomum Maton (Cardamom)*

Common name of *Elettaria cardamomum* Maton is known as cardamom. Occurs in Southern India especially near the Malabar coast and frequently cultivated in Ceylon. It's a perennial herb with a fleshy, branching, annulated rootstock. The fruit is an aromatic, carminative, stimulant, stomachic and emmenagogue. It is internally given for liver and uterine diseases. Seeds are diuretic [12].

3.2.7.1 *Reversing potentiality of the AD pathology by Elettaria cardamomum*

A study had been designed to assess the extract of *Elettaria cardamomum* fruit on learning and memory, brain cholinesterase levels and associated altered brain oxidative stress markers in scopolamine treated mice. Pre-treatment with *Elettaria cardamomum* extract (500 and 1000 mg/kg) for 15 days intervention significantly reversed Scopolamine induced amnesia with evidence. This study concluded that *Elettaria cardamomum* shows promise as a natural memory booster in scopolamine induced amnesia also it possess anti-cholinesterase and anti-oxidant activity that protects against oxidative stress and neurodegeneration [41].

3.2.8 *Zingiber Officinale (Ginger)*

Common name of *Zingiber officinale* is ginger. It is a native of Pacific Island. It is commonly grown in all vegetable gardens in Ceylon. It's a perennial herb with large, solid, tough horizontal rhizome. Rhizomes are used for medicinal and culinary purposes. Both fresh and dry forms are used. They are stomachic, carminative, stimulant, diaphoretic, sialogogue and digestive. Extremely valuable for all the bowel and respiratory disorders and useful in cold, coughs and fevers [12].

3.2.8.1 *Reversing Potentiality of the AD Pathology by Zingiber Officinale*

In a study dry ginger extract had been evaluated for multi focal anti-Alzheimer's coverage. The study covered that the antioxidant activity, cholinesterase inhibition, anti-amyloidogenic potential and neuroprotective properties of methanolic extract of dry ginger (GE). In the study it was understood that GE expressed high antioxidant activity evident by DPPH and FRAP assay. Increased cholinestera--se inhibitory activity found by Ellman's assay and mainly GE increased the cell survival against amyloid beta (Abeta) induced toxicity in primary adult rat hippocampal cell culture. Hence, these findings suggest that methanolic GE influences multiple therapeutic molecular targets of AD [42]. Some other studies also evident that ginger intervention showed an improvement of the morphological structure of the brain tissue with disappearance of most amyloid plaques with improvement in the learning and memory [43], [44]. Further in another study also revealed that ginger oil and the extract of ginger significantly ameliorates the neuroinflammation and apoptosis characte--rizing Alzheimer's disease in the rat model due to their anticholinesterase activity and antiapoptotic potential besides the anti-inflammatory effect [44].

3.2.9 *Trigonella Foenum Graecum (Fenugreek)*

Common English name of *Trigonella foenum graecum* is fenugreek. It occurs in India extending through Persia and Abyssinia to the Mediterranean regions. It is an annual herb 30 – 60 cm high with an erect, slightly branched, cylindrical, hollow smooth pubescent stem. Leave are alternate. It is much cultivated in central Europe, India and Egypt. The seeds and leaves are used for medicinal and culinary purposes. Seeds are carminative and aphrodisiac. They are used for dyspepsia, diarrhea, dysentery and rheumatism [45].

3.2.9.1 *Reversing Potentiality of the AD Pathology by Trigonella Foenum Graecum*

A study had discovered that fenugreek saponins (FS) decreased the ROS generation and DNA damage in AD-induced rats compared with control rats. Further in the same study FS increased the AChEI and apoptosis activities. The results suggested that the ability of fenugreek saponin to inhibit AD due to its increase AChE inhibition activity and which might be attributed to increase the antioxidants in this herb [46].

3.2.10 *Coriandrum Sativum Linn (Coriander)*

Common name of *Coriandrum sativum* is coriander. It's a native to Palestine, Syria, Mesopotamia and Greece but it is now extensively cultivated throughout India and Ceylon. It is a glabrous herb 15 – 45 cm in height emitting disagreeable odour when rubbed. Mostly fruits of this herb are used. It is a refrigerant, diuretic, tonic ad aphrodisiac. The oil is useful for flatulent colic, rheumatism and neuralgia. The infusion of the fruit is given for dyspepsia, sore throat, catarrh and bilious complaints [12].

3.2.10.1 *Reversing Potentiality of the AD Pathology by Coriandrum Sativum*

It had discovered in a research that inhalation of coriander volatile oil increased anxiolytic-antidepressant-like behaviors and decreased oxidative status in beta-amyloid (1-42) rat model of Alzheimer's disease [47]. In another study had revealed that *Coriandrum sativum* leaves appears to be a promising candidate for improving memory, and it would be worthwhile to explore the potential of this plant in the management of Alzheimer patients [48].

IV. CONCLUSION

The present review concludes that out of these ten spice condiments the contribution of cinnamon is the best of all to reverse the important two main pathologies of AD, such as to promotes complete disassembly of recombinant tau filaments and to reduces β-Amyloid oligomerization and inhibits the formation of toxic Aβ oligomers and to prevents

the toxicity of Aβ on neuronal PC12 cells. Next to that, Curcumin, Pepper, Clove and Ginger contribute to reverse one of the pathologies of AD that inhibits the formation of toxic Aβ oligomers. While Pepper and Ginger reduces the neuro-inflammation most of all the spices in different combination possesses anticholinesterase, antioxidant, increase monoamine neuro-transmitters and showed improve cognitive performance as multi therapeutic target of AD. Summary of this review has been shown in the below given table 1.

Usages of spice condiments should be increased and improved by the new generation which are not only the food additives but those have many curing potentialities. Our ancestors' pattern of eating is the classic examples on the usage of spice condiments.

Table 1 Multi potential therapeutic targets on AD pathology by Spice condiments

	Reduce Aβ	Disassembly of tau	Reduce neuro-inflammation	Anti-oxidant	Anti-cholinesterase	Increase monoamine neurotransmitters	Improve cognitive performance
Cinnamon	✓	✓		✓			
Cumin				✓	✓		✓
Turmeric	✓			✓			
Nutmeg				✓		✓	✓
Pepper	✓		✓			✓	✓
Clove	✓			✓	✓		✓
Cardamom				✓	✓		✓
Ginger	✓		✓		✓		
Fenugreek				✓	✓		
Coriander							✓

ACKNOWLEDGEMENT

I thank Dr MHM Nazeem, Senior Lecturer for his tremendous help in writing this review. This review study does not receive any grant from funding agencies in the public, commercial or not-for-profit sector.

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