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Formulation and Evaluation of Rivastigmine and Curcumin Microemulsion for Alzheimer's Diseases

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Abstract: *Alzheimer's disease is the most common type of neurodegenerative dementia and causes health problems for patients and their families. Rivastigmine is a potent, slowly reversible, noncompetitive carbamate cholinesterase inhibitor approved for the treatment of mild Alzheimer's disease. A randomized, double-blind, placebo-controlled study lasting up to 6 months showed that rivastigmine was more effective than placebo on measures of cognition and general functioning. Anecdotal but growing evidence suggests that beneficial effects may last up to 5 years, extend across multiple stages of Alzheimer's disease, and occur across cognitive domains such as activities of daily living and behavioral symptoms.*
Alzheimer's disease. Evidence from controlled studies also supports the use of rivastigmine in the treatment of cognitive and behavioral disorders such as Alzheimer's disease, dementia with Lewy bodies, and Parkinson's disease dementia associated with vascular involvement. Early and continued use of rivastigmine in the treatment of Alzheimer's disease may lead to the best results
The most serious side effects of rivastigmine are moderate cholinergic gastrointestinal events; This can be reduced by slow dosing and administration with a full meal. Clinical dosage is 6-12 mg/day twice daily, with higher doses providing greater benefit.
Worldwide, Alzheimer's disease (AD) is the most common multifactorial neurodegenerative disease affecting the elderly. Today, many drugs, including curcumin, are used in the treatment of AD. Curcumin is the main component of turmeric and is effective in preventing or treating AD. Over the past few years, research in the scientific community has focused in optimizing curcumin's therapeutic properties and improving its pharmacokinetic properties. This review includes literature from 2009 to 2019 on curcumin analogues, derivatives, and compounds and their therapeutic, preventive, and diagnostic properties in AD.
Recent advances in this field suggest that phenolic hydroxyl groups may be anti-amyloidogenic. The phenylmethoxy group appears to contribute to the inhibition of amyloid-β peptide (Aβ42) and amyloid precursor protein (APP), and hydrophobic interactions also play an important role. Additionally, the flexibility of the linker is important to prevent Aβ aggregation. The inhibitory activity of these derivatives increases with the expansion of the aromatic ring. The important role of curcumin-based drugs has been demonstrated in clinical studies. Keto-enol tautomerism appears to be a novel change in the formation of amyloid binders. Molecular docking results, (Q)SAR, and in vitro and in vivo assays to evaluate structure and chemical properties are relevant to specific activity. Therefore, information obtained from existing studies should enable the design and development of multipurpose curcumin analogs, derivatives, or curcumin hybrids that could be useful drugs and clinical tools in the diagnosis and treatment of AD.

Keywords: Alzheimer's disease, rivastigmine, curcumin, sign and symptoms, mode of action

I. INTRODUCTION

Alzheimer's disease

This disease is named after its researcher Alois Alzheimer. In 1906 Dr. Alzheimer discovered abnormalities in the brain tissue of a patient dying of dementia. The patient experiences memory loss, speech problems and behavioral disorders. After his death, he found abnormal amounts of toxic proteins (now called amyloid plaques) and tangled fibers (now called neurofibrillary tangles) in his brain. Plaques and tangles in the brain are two important features of AD. The third is the loss of connections between neurons in the brain.

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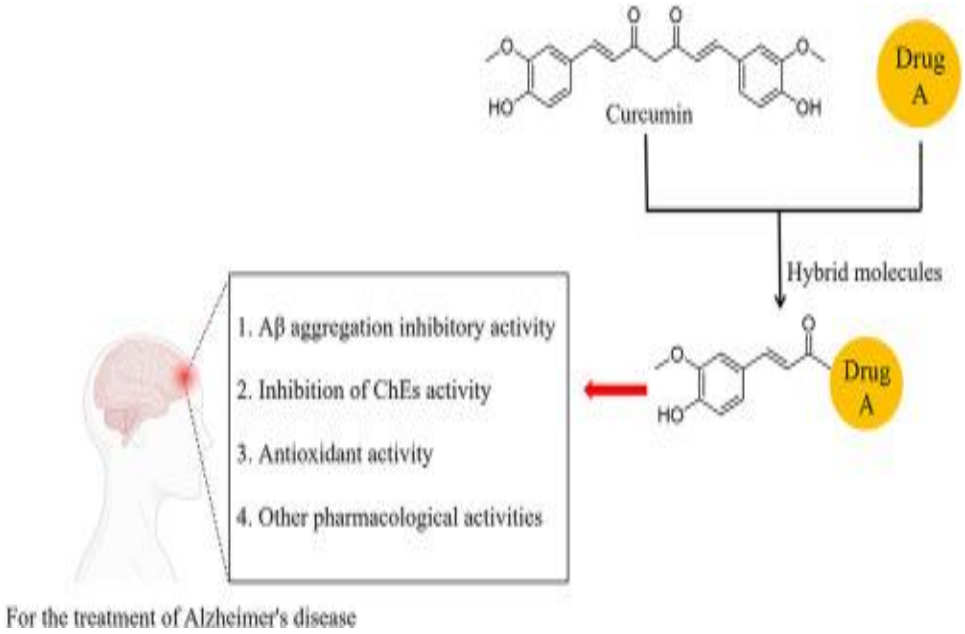

AD, the most common type of dementia (amnesia that occurs with aging), is an irreversible brain disease that gradually steals a person's mind, impairing their memory and ability to think, ultimately leading to the ability to do good. Simplicity of activities. According to reports, there are two types of AD patients: type 1 AD patients (early onset, before age 60) and type 2 AD patients (late onset, after age 60). The first symptoms in Alzheimer's patients usually appear after the age of 60.

Alzheimer's disease (AD) is the most common cause of dementia in the elderly, accounting for 60% to 80% of all deme ntia cases . Its clinical features are cognitive impairment and its neuropathological symptoms are amyloid plaques and neurofibrillary tangles (NFTs) . The "World Alzheimer's Disease Report 2023" published by the International Alzheim er's Disease Report shows that the number of people with dementia worldwide is expected to increase from 55 million i n 2019 to 139 million in 2050 as the population ages. However, few drugs are available to treat AD. FDA approved medications include aducanumab, donepezil, galantamine, rivastigmine, and memantine. Unfortunately, these drugs only relieve the symptoms of AD but do not cure it . Moreover, the basic principles of AD are still unclear and e xisting drugs can be used to modify the main target of AD. In recent years, scientists have conducted indepth research o n the pathogenesis of AD and have put forward many theories.

AD, as a neurodegenerative disease, has a multifactorial pathogenesis. Scientists call the Aβ cascade hypothesis, Tau hy pothesis, cholinergic hypothesis, etc. he calls it. They have proposed several hypotheses, including the following:

The Aβ cascade hypothesis states that amyloidβ (Aβ) is produced by protease cleavage of the amyloid precursor protei. (APP) APP is cleaved by β-secretase and γ-secretase complexes, resulting in the release of Aβ peptides of different lengths. Among Aβ peptides, Aβ40 is the most abundant form but is less pathogenic; Aβ42 is the main component of amyloid plaques and is considered the most toxic . Previous studies have shown that Aβ may promote the development of AD through its direct neurotoxic and indirect e ffects, including mitochondrial dysfunction, inducing neuroinflammation, and oxidative stress.

Tau is the most abundant microtubule-associated protein. Its major physiological functions include promoting the polymerization of tubulin into microtubules, maintaining the stability of microtubules, and promoting neurite outgrowth. Hyperphosphorylation of Tau leads to a de crease in microtubule binding affinity, leading to Tau aggregation and the appearance of NFTs Pathogenic bacteria hav e been shown to disrupt microtubule assembly, disrupt axonal transport, impair cell function, and ultimately cause cell d eath.

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Recent studies have shown that curcumin exerts antioxidant properties through direct and indirect antioxidant pathways . The direct antioxidant capacity of curcumin is mainly reflected in its ability to scavenge free radicals due to two pheno lic hydroxyl and βdiketone moieties . In various in vitro antioxidant tests, curcumin has been shown to have a potent sc avenging ability against many free radicals compared to Trolox and alpha-
tocopherol. Additionally, curcumin's metal ion chelating ability helps reduce free radicals . On the other hand, curcumin exerts indirect antioxidant effects by upregulating antioxidant enzymes such as heme oxygenase 1 (HO-
1), superoxide dismutase (SOD), catalase and related endogenous antioxidants. Synthetic enzymes, especially glutathio ne (GSH)

Signs and symptoms-

Memory problems are one of the first symptoms of AD. Conversely, some people have more memory problems than th eir peers. This is called amnestic mild cognitive impairment (MCI). People with MCI can have this disease, although th e symptoms are not as severe as in people with AD.

Other changes may also indicate the early stages of AD. For example, there are hearing problems and cognitive proble ms. The findings may provide tools to help detect Alzheimer's disease early, track the disease, and monitor response to t reatment. AD is characterized by various stages and progresses from mild to severe. In the final stage, plaques and tangl es spread throughout the brain and brain tissue decreases. People with severe AD cannot communicate and are depende nt on others for care. Finally, Alzheimer's patients may spend most of their time sleeping as the body begins to shut do wn.

Neuropathology

Neuropathological processes involve neuronal loss and atrophy mainly in the temporoparietal and frontal cortices, with accompanying responses to the inflammatory response of amyloid plaques. By the release of protein fragments and agg regates and abnormalities of neurofibrillary tangles. This destructive process spreads to a nearby structure called the hip pocampus, which is important for memory formation.

There are many factors thought to cause disease, including genetics, environment and lifestyle. The importance of these factors in preventing or delaying AD varies from person to person due to differences in genetics and lifestyle. These pl aques can now be seen when the brains of living people are examined. The results of these studies will help doctors und erstand the cause of the disease. Additionally, in people with Alzheimer's disease, the presence of monocytes/macropha ges in blood vessels in the brain and the presence of reactive or active microglia in the adjacent parenchyma are increas ed. The main protein of amyloid in AD is 39-42 amino acids (β-amyloid peptide).

One of the mysteries of AD is why it usually affects older people. Research on how the brain naturally changes with ag e is a glimpse into these questions. For example, scientists are studying how age-
related changes in the brain damage neurons and lead to the demise of Alzheimer's disease. These age-
related changes include atrophy of parts of the brain, inflammation and oxidative stress, and the production of harmful a nd unstable molecules called free radicals due to cellular respiration

Genetics

A small number of people in their 30s, 40s and 50s have been reported to have Alzheimer's (type 1). Many of these peo ple have mutations or mutations in one of our genes that they inherited from their parents. We know that mutations in th ese genes cause AD in early familial cases. Not all early symptoms are caused by mutations.

AD patients often suffer from late Alzheimer's disease, usually after age 60 (type 2). Several studies have linked a gene called APOE to lateonset Alzheimer's disease. There are many types of this disease, one of them is APOE e4, which inc reases the risk of the disease. Approximately 40% of people with late-
onset AD carry this gene. However, carrying the APOE e4 form of the gene does not necessarily mean that the person will develop AD, and people who do not carry the APOE e4 form may also develop the disease. There are reports from experts in this field that additional genes may be involved in the development of late-onset AD.

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Environmental factors

One of the most controversial theories about lead is that lead is suspected to cause AD after scientists found traces of thi s metal in the brains of AD patients. Many studies since then have either not confirmed this finding or raised questionab le questions. Aluminum has been shown to be higher than normal in some mortality studies of AD patients, but not in al l. 6

Aluminum is found in small amounts in many household items and many foods. Therefore, there is concern that alumin um consumed in foods or absorbed from other sources may be an important factor in AD. Excess zinc and some toxins i n the diet are thought to damage the brain and contribute to early dementia

Diagnosis

Early diagnosis with CT and MRI is beneficial for many reasons. Although the underlying disease process cannot be ch anged, getting an early diagnosis and starting treatment early in the course of the disease can help preserve function for months to years. Early diagnosis also helps families plan for the future, organize their lives, manage finances and legal affairs, and build support. Additionally, early diagnosis may provide more time for individuals to participate in clinical t rials. In clinical studies, researchers test medications or treatments to see what works best for whom.

FH research has advanced to the point where researchers can move beyond treating symptoms to address the underlying disease. . In ongoing clinical trials, scientists are investigating a variety of interventions, including heart disease and dia betes treatment, antioxidants, immunity, intelligence, and physical activity.

Treatment of Alzheimer's disease

Alzheimer's disease is a complex disease and there is no "magic bullet" that can prevent or cure it. That's why current tr eatments focus on many different things, including helping people manage mental disorders and managing behavioral s ymptoms to prevent, slow, or prevent disease.

Available Drugs

Four drugs have been approved by the FDA to treat Alzheimer's disease. Donepezil (Aricept), rivastigmine (Exelon), an d galantamine (Razadyne) are used to treat mild to moderate AD (donepezil is also used to treat AD). This medication p revents the breakdown of acetylcholine by inhibiting the production of acetylcholinesterase. This causes an increase in t he concentration of acetylcholine available for synaptic transmission in the central nervous system, thus improving cog nitive deficits.

The fourth drug, memantine (Namenda), is an NmethylDaspartate receptor antagonist used in the moderate treatment of AD. These drugs work by controlling neurotransmitters (chemicals that carry messages between neurons). They can he lp control thinking, memory, and speech, and can help with some behavior problems. But all these drugs do not change the underlying disease and can only be used for a few months to several years. It also has its own set of side effects and side effect:

About rivastigmine

Rivastigmine transdermal patch appears to be effective in treating patients with mild to moderate Alzheimer's disease, it is also better tolerated due to the effectiveness of rivastigmine compared to other drugs and compared to Latin capsules regional differences have been discovered

Rivastigmine is a second generation acetylcholinesterase inhibitor that was commercialized after its success. It is curre ntly sold in more than 60 countries worldwide, including Europe, South America and the United Kingdom. This is a cen tral choice that means less environmental downside. These are nausea, vomiting, abdominal pain and anorexia . Alzhei mer's patients are susceptible and improvement is seen at doses up to 12 mg per day

: Rivastigmine, another AChE inhibitor, is an effective drug used in the treatment of many neurological diseases. Due t o its AChE inhibitory activity, this compound may be useful in the treatment of AD. It is a so-

called irreversible competitive inhibitor Therefore, the pharmacokinetic properties of rivastigmine differ significantly f rom those of DonepezilRivastigmine inhibits AChE activity in the cerebrospinal fluid of AD patients. Rivastigmine is more selective for AChE inhibition in the central nervous system than the peripheral system. Therefore, Alzheimer's pa

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tients whose disease worsens to a selective AChE inhibitor or who cannot tolerate donepezil may benefit from switchin g to rivastigmine. Rivastigmine inhibits AChE by binding to its active site for a long time (up to 10 hours) and also inh ibits butyrylcholinesterase (BuChE)

Rivastigmine was recommended by the FDA in 2000 Noncompetitive pseudoirreversible carbamate AChEI was chosen for study due to its high affinity for AChE in the brain compared to peripheral forms of the enzyme.

About Curcumin

Curcumin structurally belongs to curcuminoids and is a group of polyphenolic compounds with strong antioxidant prop erties. The botanical name of the turmeric plant is Curcuma longa and it belongs to the Curcuma family. Turmeric is a s terile plant and does not produce seeds. The plant is 3 to 5 meters tall and has yellow flowers. The underground rhizom e or root of the plant is used in medicine and food preparation. The rhizome is boiled, then dried and ground to produce the bright yellow spice turmeric.

Preliminary research published in the July 2009 issue of the Journal of Alzheimer's Disease may lead to new discoverie s about ways to prevent and treat AD with curcumin. Natural or synthetic curcumin, alone or in combination with vitam in D3, has been reported to strengthen the body and protect the brain from beta-

amyloid. The process of this process is reported by activating the immune system such as MGAT III and TLR-

3. Bisdemethoxycurcumin has been found to play a role in this process.

Natural curcumin is not easily absorbed and is easily digested in the intestine before use. It seems to be better absorbed with food and other spices like black pepper. Adding 20 mg of piperine (derived from black pepper) increases the bioav ailability of curcumin by 2,000%. 3,9 Synthetic curcuminoids have been shown to be more stable and effective than nat ural products. After absorption, curcumin enters the intestines and liver to form curcumin glucuronide.

Since vitamin D and curcumin have different effects on the immune system, curcumin or a combination of the two (dep ending on the patient) may be more effective. Taking vitamin D or curcumin is not recommended at this time. Larger st udies of vitamin D and curcumin are being conducted in more patients to determine the dosage of both.

#Mode of action

Curcumin is a promising drug because it fights against AD in many ways. Curcumin has been reported to strengthen the immune system and remove beta-

amyloid from the brain. It inhibits the growth of microglia (glial cells). Prolonged activation of microglia releases cytok ines and reactive substances that exacerbate β-

amyloid pathology. 11 It has been reported that curcumin has an inhibitory effect on proinflammatory cytokines and ca n treat AD with these different effects.

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Curcumin has been shown to have strong antioxidant effects by inhibiting the formation and conduction of free radicals. They reduce LDL oxidation and free radicals, which cause neuronal deterioration not only in AD but also in other neur odegenerative diseases such as Huntington's disease and Parkinson's disease. Curcumin increases the level of glutathion e, an important endogenous antioxidant and an important cofactor for antioxidant enzymes that protect mitochondria fro m oxygen-free radicals.

The lipophilicity of curcumin allows it to cross the blood-brain barrier. At high concentrations, curcumin binds to beta-amyloid and prevents it from self-assembling. Researchers found that curcuminoids improved the binding of beta-amyloid to macrophages, while vitamin D3 promoted the uptake and absorption of beta-amyloid by macrophages in most patients.

Finally, curcumin can interact with heavy metals such as cadmium and lead and prevent the neurotoxicity caused by the se metal

Curcumin and Neurological Disease: Focusing on Mechanism

The possibility of using curcumin as a treatment for neurological diseases is a relatively recent Development. Although the specific effects may differ for each disease, we can identify common Pathways by which curcumin exerts its effects. For instance, inflammation is a common factor in Many neurological diseases. In most cases, NFκB plays a pivotal role in the inflammatory response by inducing TNF-α, which in turn mediates ROS activation. Curcumin has an inhibitory effect onNFκB, thereby reducing inflammation [45]. Neurological diseases, such as multiple sclerosis (MS),spinal cord injury (SCI), and AD [46], show immune cell invasion and another mechanism that can be modulated by curcumin is T-lymphocyte activation and monocyte chemotaxis. Curcumin negativelyregulates pathways that modulate chemokines responsible for monocyte recruitment and activation ofT-lymphocytes [47,48].

Alzheimer's Disease

The presence of amyloid or plaques comprising β-amyloid (Aβ) peptide is one the majorcharacteristics of AD. Curcumin acts on Aβ levels, deposits, and aggregation in a multitude of ways. In vitro studies demonstrated that compounds derived from curcumin protect PC12 cells againstAβ(25–35) and Aβ(1–42) insult [49]. When used as a retreatment, curcumin prevents Aβ-inducedtoxicity in the human SH-SY5Y neuroblastoma cell line by increasing mRNA and protein levels ofmitochondrial genes, such as nuclear respiratory factor 1 (Nrf1) and mitochondrial transcriptionfactor A (TFAM) [50]. Bisdemethoxycurcumin (BDC) pre-treatment in an ex vivo study on peripheral blood mononuclear cells (PBMCs) of AD patients showed that curcumin decreases mRNA levels

NFκB, BACE1 and Toll-like receptor and upregulates mRNA levels of mannosyl-glycoprotein4-β-N-acetylglucosaminyltransferase (MGAT3) and vitamin D receptor (VDR), leading to diminished Aβ aggregates [51]. This protective effect of curcumin has been confirmed also in vivo studies. In amouse model of AD, curcumin decreased the levels of inflammatory molecules, such as IL1β, TNF-α, cyclooxygenase 2 (COX 2) and nitric oxide (NO), while also decreasing the number of glial fibrillaryacidic protein-(GFAP) and Iba-1-positive cells, thus suppressing the neuroinflammatory response Moreover, Curcumin was associated with a protective effect on some forms of memory: althoughno effects were noted on spatial memory in the object location test and Y maze in a rat model of ADtreated with 50 or 100 mg/kg curcumin, it did prevent deficits in recognition memory in the objectrecognition test [53].Finally, curcumin can also protect against Tau-induced neurotoxicity by inhibiting glycogensynthase kinase-3 (GSK-3), which regulates the phosphorylation of Tau protein

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Marketed product of curcumin

II. CONCLUSION

Over the past 30 years our understanding of AD has been very limited. Since then, scientists have made many important advances. Research supported by the National Institutes of Health, the Alzheimer's Foundation, and other organizations is expanding our understanding of brain chemistry and function in healthy adults. Drugs have found ways to slow the a gerelated decline in cognitive function and have improved our understanding of Alzheimer's. As part of ongoing interna tional efforts, scientists are now working together to discover the genetic, biological and environmental factors that cont ribute to AD worldwide. This effort brings us closer to preventing, slowing, and ultimately curing this devastating disea se.

These new results will open the door to further research and development to find more effective curcumin to treat AD. New reports also support some of the abov-
mentioned claims of curcumin in AD; However, larger human studies are needed to determine the preventive and thera peutic effects of curcumin. 9 Other brain-
boosting compounds, such as resveratrol (an antioxidant) and ginkgo biloba, have also been studied for their effects on dementia.

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