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Contents lists available at ScienceDirect

Asian Pacific Journal of Tropical Biomedicine

journal homepage: www.elsevier.com/locate/apjtb

Document heading doi: 10.12980/APJTB.4.2014APJTB-2014-0412

A review study on medicinal plants used in the treatment of learning and memory impairments

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Peer Review

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Comments

This is an interesting review article on the tropical available plants that can be used in the treatment of learning and memory impairments (such as Alzheimer). The article can fulfill the present scattering knowledge in medicinal plants. The topic is interesting and can be further referenced. The review data can be the source for used in further study in tropical ethnopharmacology study.
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ABSTRACT

Alzheimer's disease (AD) is a progressive brain disorder that gradually impairs the person's memory and ability to learn, reasoning, judgment, communication and daily activities. AD is characterized clinically by cognitive impairment and pathologically by the deposition of β amyloid plaques and neurofibrillary tangles, and the degeneration of the cholinergic basal forebrain. During the progression of AD patients may produce changes in personality and behavior, such as anxiety, paranoia, confusion, hallucinations and also to experience delusions and fantasies. The first neurotransmitter defect discovered in AD involved acetylcholine as cholinergic function is required for short-term memory. Oxidative stress may underlie the progressive neurodegeneration characteristic of AD. Brain structures supporting memory are uniquely sensitive to oxidative stress due to their elevated demand for oxygen. The neurodegenerative process in AD may involve β amyloid toxicity. Neurotoxicity of β amyloid appears to involve oxidative stress. Currently, there is no cure for this disease but in new treatments, reveals a new horizon on the biology of this disease. This paper reviews the effects of a number of commonly used types of herbal medicines for the treatment of AD. The objective of this article was to review evidences from controlled studies in order to determine whether herbs can be useful in the treatment of cognitive disorders in the elderly.

KEYWORDS
Alzheimer's disease, Medicinal plants, Oxidative stress, Cholinergic function

1. Introduction

Alzheimer's disease (AD) is a progressive, irreversible neurological disorder that occurs gradually and results in memory loss, unusual behavior, personality changes, and loss of the ability to thinking[1]. It is estimated to affect 15 million people worldwide. AD is the cause of dementia in the elderly. AD is a progressive neurological disorder with duration of around 8.5 years between onset of clinical symptoms and death[2].

AD starts with loss of short term memory, forgetting names and addresses, as this condition progresses, the change become more marked and even individuals forget the home way. Unfortunately, AD has not any cure but can be prevented from progressing. Seventy percent of causes for AD is genetic and 21% is environmental. Most cases of

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Foundation Project: Supported by Research and Technology Deputy of Shahrekord University of Medical Sciences.

Article history:
Received 7 Aug 2014
Received in revised form 19 Aug, 2nd revised form 25 Aug, 3rd revised form 10 Sep 2014
Accepted 15 Jun 2014
Available online 22 Sep 2014

Alzheimer's, approximately 95%, are the late-onset form, which develops after age 60[3].

The causes for disease progress in American and European countries are feeding, and reduced physical and mental activity. Unfortunately, the number of people with AD is expected to triple in the next 50 years. The average cost per patient is estimated 150 thousand dollars that expected future increases to 450 thousand dollars[3].

Brain areas associated with cognitive functions, particularly the neocortex and hippocampus, are the regions that mostly affected by the pathology which is characteristic of AD[4].

The main cure for AD is pharmacological treatment. Better understanding of the disease process and designed clinical trial are step forward and have improved related treatments for cognitive and noncognitive symptoms. Pharmacological treatment strategies in AD include three categories of drug: 1) their mechanism is based on disease-modifying therapies such as vitamin E; 2) their mechanism is based on compensation of neurotransmitter such as a cholinesterase inhibitor; 3) psychotherapy factors that are prescribed for symptoms of conduct disorder[5].

At present, the most accepted AD treatment strategy is cholinesterase inhibitors that can inactivate the acetylcholinesterase (AChE) enzyme in order to increase acetylcholine levels in the brain. Acetylcholinesterase inhibitors include rivastigmine, tacrine, donepezil, and galantamine whereas methyl-D-aspartate receptor antagonist (memantine) has recently been prescribed. However, there is no cure for AD, except to relieve symptoms of the disease[6].

These results lead us to factor that increase levels of the acetylcholine in the brain.

In this study, medicinal plants that have shown the early promising signs of clinical efficacy in the treatment of AD have been investigated. One common feature of these plants is their ability to exert neuroprotective effects through inhibition of AChE or inhibition of oxidative stress.

Although recently several synthetic drugs have been introduced to treat learning and memory disorder, but their therapeutic effects is low and most of them have undesirable side effects. Today we can see the increasing tendency of people towards traditional medicine[7].

Although the mechanism of anti-dementia effect of most herbal extracts and their compounds is not yet fully understood, one or more of medicinal plants and their constituents that are discussed in this study act through inhibition of AChE and activation of the synthesis of acetylcholine. While cholinesterase inhibitors which have been recently introduced such as tacrine and donepezil reduced the number of AD patients and relieve their symptoms, most of Alzheimer's patients have not still benefited considerably from major financial investments in research and development programs[8].

Recent studies have shown promising results of the effectiveness of herbal medicines for the treatment of various diseases include memory problems[9-13], stroke [14-19], gastrointestinal problems[20], and many others disease. Although these effects can related to their specific compounds, but most of them have been related to their antioxidant properties.

2. Pathogenesis of AD

Impairment of learning and memory, the most characteristic manifestation of dementia can be chemically induced in experimental animals by scopolamine. Scopolamine is a known cholinergic antagonist that involved in the transmission of acetylcholine in the central nervous system[21]. Cholinergic transmission is mainly terminated by acetylcholine hydrolysis by the enzyme AChE, which is responsible for degradation of acetylcholine to acetate and choline in the synaptic cleft[22].

Scopolamine-induced amnesia in animal models is widely used to screen for compounds with potential therapeutic value in treatment of dementia[23].

Decrease of acetylcholinetransferase enzyme activity and loss of cholinergic neurons were observed in the basal part of the frontal brain of AD patient that associated with cognitive impairment[24].

Lesion patterns of the nucleus basalis of Meynert (NBM) is used to study the role of cortical cholinergic system in awareness and understanding, also to indicate cognitive deficits that caused in AD[25]. Destruction of NBM in animal models showed reduction in cholinergic markers include levels of acetylcholine, acetylcholine release and turnover, uptake of acetylcholine, AChE activity and number of cholinergic muscarinic receptors in the frontal cortex[26]. Because the cholinergic ramifications are sent from Maynert nuclei into the cortex and septal area, electrical destruction of NBM causes death of the cholinergic cells in this nucleus and reduces the amount of acetylcholine in the cortex[27].

Hippocampus plays a critical role in learning and memory, which is a complex biological process including the acquisition, consolidation and retrieval of information[28]. Neurogenesis in the hippocampus, defined as the generation of new nerve cells, is involved in memory formation. Increased neurogenesis is improved spatial memory while impaired neurogenesis indicates poor cognitive function[29]. Important neuropathological features of AD include deposition of amyloid plaques in brain tissue and meningeal blood vessels as well as presence of neurofibrillary tangles in the hippocampus and the cerebral cortex of the brain[30]. Recent studies have demonstrated that AD is associated with inflammatory processes. Reactive oxidative species can damage cellular components and function as a second messenger in the inflammation. Utilization of antioxidants may be useful in prevention and treatment of AD[31].

One factor that plays an important role in the pathogenesis of AD is oxidative stress that is an imbalance between free radicals and antioxidant systems. Oxygen free radicals