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# Dietary prospects of coconut oil for the prevention and treatment of Alzheimer's disease (AD): A review of recent evidences

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ABSTRACT

*Background:* Alzheimer's disease (AD) is a devastating neurodegenerative ailment having pathological hallmarks of plaques due to amyloid β (Aβ) peptides and neurofibrillary tangles in brain. These cerebral plaques and neurofibrillary tangles potentially affect the neuronal synaptic transmission and ultimately cause cognitive decline. In the absence of an effective treatment module for AD, alternative therapeutic strategies are being explored.

*Scope and approach:* Given the fact that dysregulation of brain glucose metabolism is an early detectable trait of AD, coconut oil and its variants/derivatives have generated considerable interests as an invaluable therapeutic agent for AD. The role of coconut oil-derived medium chain fatty acids (MCFAs) which are rapidly metabolized into ketone bodies to serve as an alternate source of energy for the cerebral tissue is well recognized. Recently, evidences underlying the mode of action of coconut oil in alleviating the symptoms of AD have started emerging. In this review, a comprehensive snapshot of the recent developments and biochemical basis of coconut oil-induced amelioration of AD symptoms including its dietary role in suppression of neuro-inflammation, reversing the process of neurodegeneration, enhancement of cell survival pathways and inhibition of secretion of Aβ peptides are presented. Investigations in animal models and clinical trials in humans using coconut oil and its derivatives aimed at reversing the AD-induced cognitive decline are also discussed. To conclude the knowledge gaps in the treatment of AD using coconut oil and way forward are presented.

*Key findings and conclusion:* Scientific evidences point toward the immense therapeutic value of coconut oil in the prevention or treatment of AD through its multi-pronged biochemical effects. Nevertheless, identification of bioactive components, besides MCFAs, responsible for the neuroprotective effects, clinical trials to fix the dosage and consolidation of information flow are warranted.

## 1. Introduction

Age-related neurodegeneration and cognitive deficits are the hall marks of Alzheimer's disease (AD). AD is recognized as the most common cause of dementia, morbidity and mortality in aging population (Bischof & Park, 2015; Brookmeyer et al., 2007; Swerdlow, 2011). Scientific reports have divulged that at the ages >65 one in eight develops AD and the probability of developing the disease is still high (45–50%) at the age of 85 (Evans et al., 1989; Hebert et al., 2004). AD is characterized by the gradual accumulation of cerebral extracellular amyloid-β (Aβ) peptides (Chetelat et al., 2010) causing senile plaques and intracellular neurofibrillary tangles formed due to phosphorylation of tau, an essential microtubule associated protein involved in the process of microtubule assembly of the mature neurons (Asih et al., 2014; Iqbal et al., 2010). Tau, in its hyperphosphorylated state-as observed during AD-gets polymerized into paired helical filaments and amalgamates with straight filaments to form the characteristic neurofibrillary tangles (Iqbal et al., 2010). In a normal, healthy brain expression and clearance of amyloid precursor protein (APP)- proteolytic cleavage of which yields Aβ peptides-happen constitutively. However, with the age APP turnover activity is affected, causing the pathological condition of accumulation of Aβ (Kanekiyo et al., 2013). Another view states that AD is a protein mis-folding disease because of the accumulation of abnormally folded Aβ peptides, glial fibrillary acidic protein (GFAP) and tau proteins in the brain and their translocation to mitochondrial membrane perpetuates neurodegeneration (Hashimoto et al., 2003). In addition, the increased

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