Is Green Tea a Protective Agent Against Alzheimer's Disease?

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Abstract

Alzheimer's Disease (AD) is characterized by the progressive deterioration of cognitive function. This fatal illness, which results in memory loss, is the most common cause of dementia.[1] In 2017, there were approximately 76,000 new Dementia cases in Canada annually, accounting for an AD prevalence of 7.1%.[2] It is anticipated that these numbers will increase in the near future due to the growth and aging of the Canadian population.[2]

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The accumulation of β-amyloid peptide (Aβ) in the brain is a primary characteristic of AD.[1] Interestingly, recent studies have shown that green tea is an effective therapeutic agent in both treating and preventing AD by minimizing Aβ levels.[1] Green tea contains an ester group named epigallocatechin-3-gallate (EGCG), which operates as a bioactive polyphenol.[1] Contrary to fully fermented tea, green tea preserves its original polyphenolic compositions, therefore having important antioxidant, anti-inflammatory, antidiabetic, anticarcinogenic and antineurodegenerative properties.[1] This review focuses on the latter property of neuroprotection as a result of EGCG in green tea.[1]

In a study conducted by Youn et al., EGCG treatment was administered in amyloid precursor protein (APP) transgenic mouse models for 3 months. It was found that only 40% of the initial Aβ buildup remained in the frontal cortex, and 48% remained in the hippocampus.[1] These results are consistent with another study conducted by Rezai-Zadeh et al., where it was found that when EGCG was injected intraperitoneally, it reduced Aβ deposition in transgenic APP mouse models.[3] Similar effects were perceived by Rezai-Zadeh et al. in these mouse models when EGCG was administered orally in drinking water.[3]

Moreover, EGCG has also been shown to reduce the onset of Aβ-generated mitochondrial impairment and oxidative stress.[1] This was observed in both cellular and mouse models, where EGCG decreased lipid peroxidation in hippocampal neurons, thereby inhibiting Aβ-caused impairment.[1] For instance, oral administration of green tea extract over a 26-week period depressed reactive oxygen species concentrations in the hippocampus and lipid peroxides in the plasma of rats, in addition to regenerating mitochondrial function and ATP levels in mice.[1] This reduction in Aβ accumulation results in a lower risk of AD onset.

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EGCG especially holds promise for the prevention of AD given its permeability of the blood-brain barrier (BBB).[1] The BBB prevents certain compounds from entering the brain tissue from the blood.[1] In order for neuroprotective agents to be effective, they must have the ability to cross the BBB.[1] Following consumption, a portion of EGCG, although the exact percentage is not known, appeared to enter the bloodstream in humans and rats, rather than being excreted entirely in the bile.[1]

In summary, the neuroprotective role that green tea provides through increased levels of EGCG occurs in the inhibition of Aβ accumulation via controlling amyloid precursor protein processing, as well as the attenuation of Aβ-induced oxidative stress and neuroinflammatory response.[1] Although the properties of EGCG act as a therapeutic agent and its BBB permeability are promising in preventing AD, additional research and human clinical trials are required to substantiate the potency of EGCG as a neuroprotectant.[1]

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