Format: Abstract

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Long-term cannabidiol treatment prevents the development of social recognition memory deficits in Alzheimer's disease transgenic mice.

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Abstract

Impairments in cognitive ability and widespread pathophysiological changes caused by neurotoxicity, neuroinflammation, oxidative damage, and altered cholesterol homeostasis are associated with Alzheimer's disease (AD). **Cannabidiol** (CBD) has been shown to reverse cognitive deficits of AD transgenic mice and to exert neuroprotective, anti-oxidative, and anti-inflammatory properties in vitro and in vivo. Here we evaluate the preventative properties of long-term CBD treatment in male AβPPSwe/PS1ΔE9 (AβPP × PS1) mice, a transgenic model of AD. Control and AD transgenic mice were treated orally from 2.5 months of age with CBD (20 mg/kg) daily for 8 months. Mice were then assessed in the social preference test, elevated plus maze, and fear conditioning paradigms, before cortical and hippocampal tissues were analyzed for amyloid load, oxidative damage, cholesterol, phytosterols, and inflammation. We found that AβPP × PS1 mice developed a social recognition deficit, which was prevented by CBD treatment. CBD had no impact on anxiety or associative learning. The prevention of the social recognition deficit was not associated with any changes in amyloid load or oxidative damage. However, the study revealed a subtle impact of CBD on neuroinflammation, cholesterol, and dietary phytosterol retention, which deserves further investigation. This study is the first to demonstrate CBD's ability to prevent the development of a social recognition deficit in AD transgenic mice. Our findings provide the first evidence that CBD may have potential as a preventative treatment for AD with a particular relevance for symptoms of social withdrawal and facial recognition.

KEYWORDS: Alzheimer's disease; amyloid load; behavior; **cannabidiol**; cholesterol; neuroinflammation; oxidative stress; phytosterol; social recognition memory; transgenic $A\beta PP_{Swe}$/PS1ΔE9 mice

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