

Cannabidiol Reduces A β -Induced Neuroinflammation and Promotes Hippocampal Neurogenesis through PPAR γ Involvement

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Abstract

Peroxisome proliferator-activated receptor- γ (PPAR γ) has been reported to be involved in the etiology of pathological features of Alzheimer's disease (AD). Cannabidiol (CBD), a Cannabis derivative devoid of psychomimetic effects, has attracted much attention because of its promising neuroprotective properties in rat AD models, even though the mechanism responsible for such actions remains unknown. This study was aimed at exploring whether CBD effects could be subordinate to its activity at PPAR γ , which has been recently indicated as its putative binding site. CBD actions on β -amyloid-induced neurotoxicity in rat AD models, either in presence or absence of PPAR antagonists were investigated. Results showed that the blockade of PPAR γ was able to significantly blunt CBD effects on reactive gliosis and subsequently on neuronal damage. Moreover, due to its interaction at PPAR γ , CBD was observed to stimulate hippocampal neurogenesis. All these findings report the inescapable role of this receptor in mediating CBD actions, here reported.