

Cannabinoid Receptor Type 1 Agonist ACEA Improves Cognitive Deficit on STZ-Induced Neurotoxicity Through Apoptosis Pathway and NO Modulation

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Original Article

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Abstract

The cannabinoid system has the ability to modulate cellular and molecular mechanisms, including excitotoxicity, oxidative stress, apoptosis, and inflammation, acting as a neuroprotective agent, by its relationship with signaling pathways associated to the control of cell proliferation, differentiation, and survival. Recent reports have raised new perspectives on the possible role of cannabinoid system in neurodegenerative diseases like Alzheimer disease's (AD). AD is a neurodegenerative disorder characterized by the presence of amyloid plaques, neurofibrillary tangles, neuronal death, and progressive cognitive loss, which could be caused by energy metabolism impairment, changes in insulin signaling, chronic oxidative stress, neuroinflammation, Tau hyperphosphorylation, and A β deposition in the brain. Thus, we investigated the presumptive protective effect of the cannabinoid type 1 (CB1)-selective

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occurs by positive regulation of the anti-apoptotic proteins, suggesting the participation of this system in neurodegenerative processes. Our data suggest that the cannabinoid system is an interesting therapeutic target for the treatment of neurodegenerative diseases.

Keywords

Agonist CB1 ACEA Neuroprotection Streptozotocin Alzheimer's disease
Neurodegeneration

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Notes

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Author Contributions

F.C. designed the experiments, collected, and analyzed the data and wrote the paper; T.A.V. and A.P.C. collected, analyzed, and discussed the data; A.S.T. designed the experiments, discussed the data, wrote, edited, and commented on the manuscript. All authors approved the final version of the manuscript.

Compliance with Ethical Standards

The experimental protocol was evaluated and approved by the “Ethics Committee for Animal use” of the Institute of Biomedical Sciences, University of São Paulo (Protocol no. 33/55/02) following the Brazilian Federal Law (no. 11794; 10/08/2008). All applicable international,

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