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Alzheimer's disease; taking the edge off with Cannabinoids

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Alzheimer desease (AD) is the most common age-related neurological disorder, featured by select neuronal loss, neuroinflammation and progressive memory and cognitive impairment. The molecular pathogenesis of the disease encompasses extracellular accumulation of the beta-amyloid peptide (Aβ) as amyloid deposits in different regions of the brain, especially in the hippocampus. Aβ can interact with various cellular components to trigger signal transduction cascades that prompt inflammatory response, caspase activation, free-radical generation and Ca^{2+} deregulation. Cannabinoids including plant derived phytocannabinoids, synthetic cannabinoids and endocannabinoids have been shown to exert neuroprotective effects in a plethora of conditions with the common feature of neuroinflammation including Alzheimer's disease (AD) (1-2). In recent years, attempts have been made toward unraveling mechanisms underlying cannabinoid-induced neuroprotection *in vitro* and *in vivo*. The majority of cannabinoid ligand effects are thought to be mediated via cell surface receptors. Among them are cannabinoid types 1 and 2 receptors (CB_1 and CB_2, respectively), the classical cannabinoid receptors that are coupled to inhibitory G-proteins (Gi/o). This presentation will focus on the findings on neuroprotective aspects of cannabinoids in AD.