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Effects of cannabinoids on neurodegenerative diseases

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ABSTRACT

Cannabinoids, a group of chemical compounds naturally occurring in cannabis, are attracting growing interest in the medical world due to their therapeutic potential. Although more than 120 cannabinoids, both exogenous and endogenous, have been identified, today's work will focus on two. Although both compounds interact with the human endocannabinoid system, their mechanisms of action and effects on the body differ significantly, which is crucial for their clinical applications. Neurodegenerative diseases are a broad and complex group of diseases, the most common of which are Alzheimer's, amyotrophic lateral sclerosis (ALS), and Parkinson's, which pose a serious problem for modern medicine due to their progressive nature and resistance to treatment. Despite extensive research efforts, discovering successful treatments for these conditions remains a challenge. Recently, there has been increasing interest in cannabinoids—compounds that naturally occur in cannabis—due to their neuroprotective, anti-inflammatory, and endocannabinoid system-modulating properties. These qualities are opening new therapeutic avenues for treatment.

Keywords: THC, CBD, neurodegenerative, Alzheimer's, Parkinson's

1. INTRODUCTION

Neurodegenerative disorders are increasingly becoming a significant public health concern around the globe. The researchers estimate that more than 50 million people worldwide suffer from these diseases, and the number of new cases doubles every 20 years due to increasing life expectancy (Hasbi and George, 2025). The most clinically significant neurodegenerative diseases include Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), amyotrophic lateral sclerosis (ALS), and multiple sclerosis (MS). A common feature of these diseases is chronic inflammation affecting the nervous system. An additional factor significant for pathogenesis is oxidative stress. Neurons, or cells of the nervous system, are particularly susceptible to damage associated with oxidative stress due to their high metabolism.

In 1964, a group of Israeli scientists isolated tetrahydrocannabinol (THC), and they devoted the next 24 years to identifying the physiology of THC, which

coincided with the discovery of cannabinoid receptors - CB1 and CB2, in 1988 and 1993, respectively. These receptors are part of the endocannabinoid system, a complex regulatory system within the central nervous system (CNS). Recent decades have seen cannabinoids undergo numerous studies. The scientists discovered the enormous therapeutic potential of these substances, and compared to other therapeutically active compounds, they are very safe in terms of toxic effects (Woroń and Dobrogowski, 2017).

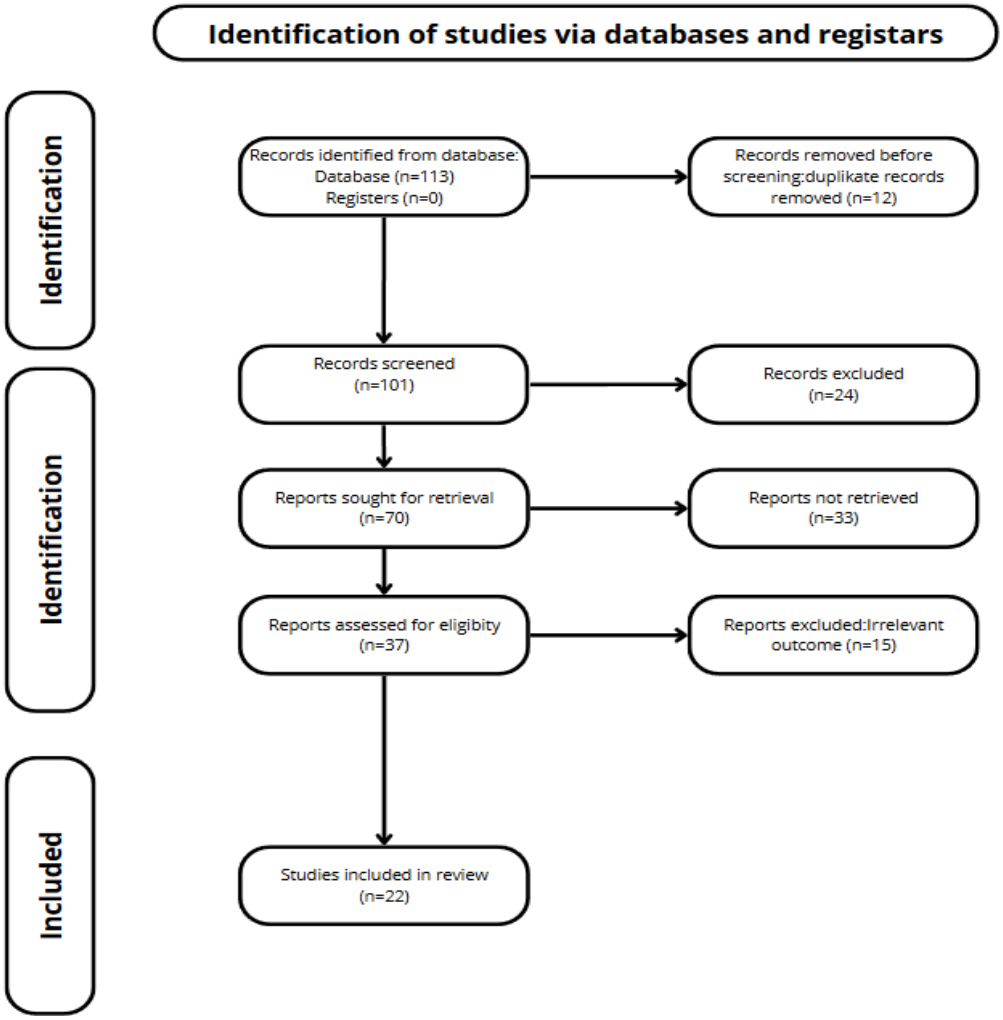

Figure 1. PRISMA consort chart of selected studies

2. REVIEW METHODS

To determine the current role and future potential of cannabinoids in the treatment of neurodegenerative diseases, focusing mainly on Parkinson's and Alzheimer's. The study focuses on the analysis of research and scientific papers on the role of cannabinoids in neurodegenerative diseases. The terms cannabinoids, THC, CBD, Parkinson's, and Alzheimer's were used to search for information. We used scientific publications as sources of information, with the oldest dating back to 2002 and the most recent to March 2025 (Fig.1). Previous studies, both preclinical and preliminary clinical, suggest that cannabinoids may alleviate the symptoms of certain neurodegenerative diseases and have neuroprotective effects. Among other things, they reduce inflammation, oxidative stress, and excitotoxicity, which play a key role in neurodegeneration. Although the results are promising, further well-designed clinical studies are key to confirming the safety and efficacy of cannabinoid therapy in patients with neurodegenerative diseases. Cannabinoids show potential as an adjunctive therapy in neurodegenerative diseases, primarily due to their neuroprotective, anti-inflammatory, and modulating properties of the endocannabinoid system. Promising preclinical results suggest the potential to slow disease progression and alleviate symptoms. However, due to the limited number of certified clinical studies and their varying quality, further large-scale, controlled research is needed to determine the actual effectiveness, optimal doses, and safety profile of cannabinoids in this group of diseases.

3. RESULTS & DISCUSSION

Historical Note

Hemp (cannabis) is one of the first plants that people used for medicinal purposes. The first recorded references to the medical use of these plants date back approximately 5,000 years. However, it took much longer to discover that cannabis is a source of dozens of compounds called cannabinoids. Scientists from around the world have already isolated a total of over 100 cannabinoids from cannabis, the most essential of which are CBD and THC. Roger Adams and his team of researchers in 1940 were the first scientists to isolate Cannabidiol (CBD). The structure of CBD was thoroughly researched and presented 23 years later by Raphael Mechoulam. However, since CBD is not a psychoactive substance, it lost the interest of research teams in favor of another cannabinoid - (−)-trans-Δ9-tetrahydrocannabinol, more commonly known as THC (Crocq, 2020). The year 1964 was crucial for the current state of knowledge about this compound, when a group of scientists coordinated by the Israeli organic chemist Raphael Mechoulam managed to isolate the (−)-trans-Δ9-tetrahydrocannabinol molecule (Rodriguez de Fonseca and Schneider, 2008).

Mechoulam tested THC on monkeys and volunteers, confirming its psychoactive properties

At the turn of the 1980s and 1990s, another revolutionary discovery took place, which significantly drove further exploration of the therapeutic potential of cannabis. American researchers, neuropharmacologist Allyn Howlett and her protégé William Devane at St. Louis University Medical School, discovered the existence of the CB1 receptor, and within the next few years, the CB2 receptor (Pertwee, 2006). In 1992, a team led by Raphael Mechloum, the "father" of cannabinoid research, discovered anandamide (AEA), the first endocannabinoid produced by the human body. In the following years, researchers found a second very significant endocannabinoid: 2-arachidonoylglycerol (2-AG). These two compounds were the first to be identified and remain the best-studied endocannabinoids, which are derivatives of the fatty acid arachidonic acid. These discoveries ultimately led to the confirmation of the existence of an independent regulatory system known as the endocannabinoid system (ECS) (Zou et al., 2018).

Endocannabinoid System

There is a neuromodulatory system that is crucial for the development of the central nervous system (CNS), synaptic plasticity, and responding to both endogenous and environmental stimuli. This system comprises endocannabinoid receptors (CB1 and CB2), endogenous cannabinoids (AEA and 2-AG), and enzymes responsible for the synthesis and degradation of these endocannabinoids. Exogenous cannabinoids such as THC and CBD also interact with CB1 and CB2 receptors. A characteristic feature of the ECS is retrograde transmission, which means that endocannabinoids are released from the postsynaptic neuron and act on the presynaptic neuron, thereby modulating its activity. This mechanism enables precise control of synaptic transmission and plays a key role in the ability of synapses to strengthen or weaken. Scientists speculate that CB1 receptors are mainly located in the central nervous system and peripherally (peripheral nerve endings, spleen, heart, lungs). Until recently, scientists believed that CB2 receptors were found only in immune system cells, but recent studies indicate that they are also present in astrocytes and the lower urinary tract (Lu & Mackie, 2016). CB1 and CB2 receptors are a part of the GPCR 4 family and are G protein-coupled receptors. Activation of the agonist triggers the inhibition of adenylate cyclase and calcium channels, together with the activation of potassium channels, mitogen-activated protein kinase (MAPK), and phosphoinositide 3-kinase (PI3K)/Akt signaling pathways. Initially, scientists believed that CB1 and CB2 receptors, like other GPCRs, functioned as single signaling receptors; however, studies over the past decade have convincingly shown that some GPCRs in different tissues can also form homodimers and even heteromers. In the case of cannabinoid receptors, heteromers have been shown to form between CB1 and dopamine, adenosine, angiotensin (AT1), opioid μ1, and orexin OX1 receptors. However, to date, no studies have been conducted on possible interactions between CB1 and CB2 receptors, despite their overlapping expression and both receptors demonstrated influence on similar cellular processes (Callén et al., 2012)

The Therapeutic Potential of Endocannabinoids in Alzheimer's Disease

Alzheimer's disease (AD) is a neurodegenerative progressive disease characterized by the slow deterioration of the central nervous system, resulting in substantial memory impairment and cognitive decline. Alzheimer's disease (AD) is the primary factor behind dementia in older individuals. Although the specific causes are still under investigation, researchers have traditionally claimed that genetics and environmental factors are most crucial in pathogenesis. Gradual deterioration in memory, personality shifts, and difficulties with daily activities happen to people with Alzheimer's every day, which can lead to complete dependence on caregivers as the condition progresses.

Despite many years of intensive research, a causal treatment for Alzheimer's disease remains beyond the reach of modern medicine. Currently available pharmacological therapies are symptomatic and only slow the progression of the disease. With the growing number of people affected by this condition, especially in aging societies, Alzheimer's disease poses a serious health, social, and economic challenge.

A characteristic feature of patients with Alzheimer's disease (AD) is the presence of cognitive deficits. After a thorough examination of brain tissue from patients with Alzheimer's disease, investigators discovered that the density of CB1 receptors is reduced, especially in the frontal cortex. In experimental animal models, a decrease in CB1 receptor expression results in reduced levels of PSD-95 protein, resulting in memory impairment in APP/PS1 mice, which suggests that these receptors protect against pathological changes in Alzheimer's disease (AD) and play a crucial role in the progression of the condition (Aso et al., 2018). The two primary endocannabinoids in brain tissue (anandamide and 2-arachidonylglycerol) are agonists of CB1 and CB2 receptors. Their neuroprotective effects may result from interference with several cellular and molecular mechanisms, including inflammation and apoptosis. The progression of Alzheimer's disease is closely associated with changes in the endocannabinoid system. Both cannabinoid receptor agonists and endocannabinoids, such as anandamide, can reduce Aβ-induced neurotoxicity in the mitogen-activated protein kinase (MAPK) pathway in a CB1 receptor-dependent manner, thereby protecting human NTERA-2/cl-D1 teratocarcinoma cells. cl-D1 (Milton, 2002).

Scientific studies have shown that 8-month-old Aβ APP/PS1 mice have lower levels of 2-arachidonylglycerol than wild-type mice. Treatment with this endocannabinoid may prevent inflammatory features in astrocytes and neuronal damage caused by cytotoxic glutamate release in Aβ-treated hippocampal slices (Gajardo-Gómez et al., 2017). Furthermore, early administration of a 2-AG reuptake inhibitor prevents hippocampal damage and memory loss in rats (van der Stelt et al., 2006). Increasing 2-AG by inhibiting monoacylglycerol lipase (MAGL) may prevent APP/PS1 prostaglandin (PGE2) production in mice, as well as neuroinflammation-related Aβ42 accumulation and neurodegeneration. To avoid neuroinflammation and to reduce neurodegeneration, MAGL inhibition may also reduce the expression of β-amyloid precursor protein 1 (BACE1), inhibit Aβ production and accumulation, maintain the integrity of hippocampal synaptic structure and function, improve long-term synaptic plasticity, spatial learning, and memory, and inhibit microglia and astrocyte activation (Chen et al., 2012).

Additionally, AEA has an impact on the regulation of Notch-1 signaling in cultured neurons. When those neurons are exposed to Aβ peptide, there is an increase in the expression of the endogenous Notch-1 inhibitor known as numb (Nb), which leads to impaired Notch-1 signaling. Nevertheless, the addition of anandamide can prevent the expression of numb and enhance Notch-1 signaling. The stimulating effect of AEA on Notch-1 transmission persists in the presence of Aβ. Through Notch-1 signaling, anandamide can restore neurogenesis and cognitive function in Alzheimer's disease (Tanveer et al., 2012).

Therapeutic Potential of Thc and CBD in Alzheimer's Disease

At present, there is no drug capable of eliminating Alzheimer's disease, and current treatment methods focus mainly on alleviating the symptoms of the disease. However, there is growing scientific evidence that exocannabinoids: THC and CBD have neuroprotective effects in animal models of AD. The impact of vaporizing or smoking marijuana is reversible, causing short-term memory impairment, which is deficient in THC. In addition, recent research reports indicate improved neurological function in aged animals when they are getting THC in low doses. For example, a recent study showed that the N2a version of the amyloid precursor protein (APP) treated with low doses of THC protected cells. Lowering Aβ levels in the cells studied is one of the potential effects of THC. THC inhibits aggregation by directly interacting with the Aβ peptide.

Additionally, it inhibits the enzyme acetylcholinesterase, enhances mitochondrial function, and reduces glycogen synthase kinase-3β (GSK3β) and related signaling pathways (Cao et al., 2014). Since THC has multiple actions and pathways, these data suggest that it may be an alternative treatment for Alzheimer's disease. In addition, recent studies show that THC stimulates hippocampal neurogenesis, inhibits neurodegenerative processes in animal models of AD, and improves cognitive function and memory in aged mice (Cooray et al., 2020).

Cannabidiol's (CBD) distinguishing feature is its low toxicity and poor systemic absorption. Studies clearly show that CBD promotes neurogenesis and reverses as well as prevents the development of cognitive deficits in rat and mouse models of Alzheimer's disease. An additional function of CBD is protection against neurotoxicity and improvement of cell viability. The above-mentioned characteristics of both exocannabinoids indicate that they are suitable for the prevention and treatment of Alzheimer's disease's

pathophysiology. However, further scientific research is needed to allow us to precisely determine the doses and routes of administration of these substances.

Cannabinoids in Parkinson's Disease

Parkinson's disease (PD) is a progressive disorder of the nervous system that affects the central nervous system, particularly the regions of the brain involved in regulating movement. Mechanisms of this condition include the degeneration of neurons that generate dopamine, a neurotransmitter and hormone. The most common symptoms include resting tremors, slow movement (bradykinesia), and muscle stiffness. The etiology of this condition is not fully understood, and PD is incurable and progressive.

The researchers generate animal models of Parkinson's disease by reproducing the pathophysiological conditions of the disease, specifically the degeneration of dopaminergic neurons in the substantia nigra, using neurotoxins or by manipulating genes that encode proteins related to PD, such as parkin or α-synuclein (Cristino et al., 2020). Observational studies suggest that CB may improve some symptoms of Parkinson's disease, such as motor and non-motor symptoms. Published surveys of Parkinson's disease patients have shown that smoking marijuana improves patients' motor and non-motor symptoms. However, these studies had several limitations that may have affected their results (Venderová et al., 2004).

The article "Cannabinoids in Parkinson Disease" (Stampanoni Bassi et al., 2017) presents the results of a study on the effects of THC and CBD on motor and non-motor functions in Parkinson disease - from a group of 84 patients who smoked cannabis Forty-six percent of patients described some benefit; 31% reported improvement of rest tremor, 45% of bradykinesia and 14% of LID, 78% reported improvement of mood and sleep.

Data from the other three scientific studies showing the response of patients with Parkinson's disease after smoking cannabis:

1. Balash et al., 2017- in a group of 48 patients; Improvement (r2) for falls (0.89), pain relief (0.73), depression (0.64), tremor (0.64), muscle stiffness (0.62), sleep (0.60)
2. Yenilmez et al., 2021 - in a group of 113 patients; the survey indicated that 54% of participants experienced positive effects. Specifically, improvements that patients noted in several areas: The symptoms include pain (43.9%), muscle cramps (41.4%), depression (28.1%), stiffness, and immobility., or akinesia (27.3%), sleep issues (27.1%), tremors (25%), fear (24%), and restless legs syndrome (21.4%).
3. Lotan et al., 2014 - in a group of 261 patients; Improvement in severity of anxiety (78.0%, 71/91), pain (71.6%, 63/88), sleep disorders (76.1%, 67/88), stiffness (64.0%, 55/86), and tremor (63.1%, 53/84)

Therapeutic prospects for cannabinoids in neurodegenerative diseases seem to be promising, indicating real benefits, particularly in neuroprotection, symptomatic relief of pain, cognitive impairment, and spasticity, as well as the modulation of oxidative and inflammatory reactions. According to insights gained from preclinical and initial clinical studies, cannabinoids may provide neuroprotective advantages and improve the quality of life for people living with multiple sclerosis, Parkinson's disease, and Alzheimer's disease. This effect is associated with anti-inflammatory and antioxidant activity, as well as the potential to modulate the endocannabinoid system, providing neuroprotection and improving neurological function.

Nevertheless, the degree of clinical research remains low. Most studies have small sample sizes, short durations, and lack standardization in the dosage of cannabinoid intake by patients. The preparations used. The variety of research approaches, the scarcity of well-controlled large-scale clinical trials, and the heterogeneity of findings make the development of specific therapeutic guidelines challenging. Furthermore, it is necessary to precisely determine the optimal dose and the initiation time of therapy. It is also crucial to monitor for potential side effects and interactions between medications to avoid them in future therapies.

4. CONCLUSION

In summary, cannabinoids show great promise in the treatment of neurodegenerative diseases; academics are gradually discovering, and the subject of cannabinoids has been generating much excitement among researchers in recent years. One need only note how many new publications are appearing on the therapeutic effects of THC or CBD. However, better-designed clinical trials with larger patient numbers and longer durations are needed to more accurately assess their efficacy, safety, and potential for inclusion in standard treatment regimens. Time and further refined research will allow us to maximize the therapeutic potential of cannabinoids in the treatment of these difficult-to-treat diseases.

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Conflict of interest
The authors declare that there is no conflict of interest.

Data and materials availability
All data sets collected during this study are available upon reasonable request from the corresponding author.

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