ALS
(Amyotrophic Lateral Sclerosis)

Excerpt from www.als.ca:

ALS is also known as Charcot's Disease, Lou Gehrig's Disease, and the most common form of Motor Neuron Disease (MND). In Canada, two to three people with ALS die each day.

ALS destroys motor neurons which are an important link in the nervous system. It is through motor neurons that the brain sends messages to the voluntary muscles throughout the body (muscles whose movement you can control as opposed to those you cannot, like the heart). Leg and foot muscles are controlled by motor neurons in the lower spinal cord. Arm, hand and finger muscles are controlled by motor neurons in the upper spinal cord. Speaking, swallowing and chewing are con- trolled by motor neurons in the brain stem. Respiratory muscles are controlled by motor neurons in the upper and thoracic levels (mid section) of the spinal cord. ALS does not affect the five senses of sight, hearing, taste, smell and touch, nor does it normally affect the eye muscles, heart, bladder, bowel, or sexual muscles.

There is no possibility that ALS is contagious.

Amyotroph Lateral Scler Other Motor Neuron Disord. 2004 Mar;5(1):33-9.

Amyotrophic lateral sclerosis: delayed disease progression in mice by treatment with a cannabinoid.

Raman C[1], McAllister SD, Rizvi G, Patel SG, Moore DH, Abood ME.

Author information

Abstract

Effective treatment for amyotrophic lateral sclerosis (ALS) remains elusive. Two of the primary hypotheses underlying motor neuron vulnerability are susceptibility to excitotoxicity and oxidative damage. There is rapidly emerging evidence that the cannabinoid receptor system has the potential to reduce both excitotoxic and oxidative cell damage. Here we report that treatment with Delta(9)-tetrahydrocannabinol (Delta(9)-THC) was effective if administered either before or after onset of signs in the ALS mouse model (hSOD(G93A) transgenic mice). Administration at the onset of tremors delayed motor impairment and prolonged survival in Delta(9)-THC treated mice when compared to vehicle controls. In addition, we present an improved method for the analysis of disease progression in the ALS mouse model. This logistic model provides an estimate of the age at which muscle endurance has declined by 50% with much greater accuracy than could be attained for any other measure of decline. In vitro, Delta(9)-THC was extremely effective at reducing oxidative damage in spinal cord cultures. Additionally, Delta(9)-THC is anti-excitotoxic in vitro. These cellular mechanisms may underlie the presumed neuroprotective effect in ALS. As Delta(9)-THC is well tolerated, it and other cannabinoids may prove to be novel therapeutic targets for the treatment of ALS.

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Increasing cannabinoid levels by pharmacological and genetic manipulation delay disease progression in SOD1 mice

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Abstract

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disorder characterized by the selective loss of motoneurons in the spinal cord, brain stem, and motor cortex. However, despite intensive research, an effective treatment for this disease remains elusive. In this study we show that treatment of postsymptomatic, 90-day-old $SOD1^{G93A}$ mice with a synthetic cannabinoid, WIN55,212–2, significantly delays disease progression. Furthermore, genetic ablation of the Faah enzyme, which results in raised levels of the endocannabinoid anandamide, prevented the appearance of disease signs in 90-day-old $SOD1^{G93A}$ mice. Surprisingly, elevation of cannabinoid levels with either WIN55,212–2 or Faah ablation had no effect on life span. Ablation of the CB_1 receptor, in contrast, had no effect on disease onset in $SOD1^{G93A}$ mice but significantly extended life span. Together these results show that cannabinoids have significant neuroprotective effects in this model of ALS and suggest that these beneficial effects may be mediated by non-CB_1 receptor mechanisms.— Bilsland, L. G., Dick, J. R. T., Pryce, G., Petrosino, S., Di Marzo, V., Baker, D., Greensmith, L. Increasing cannabinoid levels by pharmacological and genetic manipulation delay disease progression in SOD1 mice.

http://www.fasebj.org/content/20/7/1003.long

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Cannabinol delays symptom onset in SOD1 (G93A) transgenic mice without affecting survival.

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Author information

Abstract

Therapeutic options for amyotrophic lateral sclerosis (ALS), the most common adult-onset motor neuron disorder, remain limited. Emerging evidence from clinical studies and transgenic mouse models of ALS suggests that cannabinoids, the bioactive ingredients of marijuana (Cannabis sativa) might have some therapeutic benefit in this disease. However, Delta(9)-tetrahydrocannabinol (Delta(9)-THC), the predominant cannabinoid in marijuana, induces mind-altering effects and is partially addictive, compromising its clinical usefulness. We therefore tested whether cannabinol (CBN), a non-psychotropic cannabinoid, influences disease progression and survival in the SOD1 (G93A) mouse model of ALS. CBN was delivered via subcutaneously implanted osmotic mini-pumps (5 mg/kg/day) over a period of up to 12 weeks. We found that this treatment significantly delays disease onset by more than two weeks while survival was not affected. Further research is necessary to determine whether non-psychotropic cannabinoids might be useful in ameliorating symptoms in ALS.

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Cannabis and amyotrophic lateral sclerosis: hypothetical and practical applications, and a call for clinical trials.

Carter GT[1], Abood ME, Aggarwal SK, Weiss MD.

Author information

Abstract

Significant advances have increased our understanding of the molecular mechanisms of amyotrophic lateral sclerosis (ALS), yet this has not translated into any greatly effective therapies. It appears that a number of abnormal physiological processes occur simultaneously in this devastating disease. Ideally, a multidrug regimen, including glutamate antagonists, antioxidants, a centrally acting anti-inflammatory agent, microglial cell modulators (including tumor necrosis factor alpha [TNF-alpha] inhibitors), an antiapoptotic agent, 1 or more neurotrophic growth factors, and a mitochondrial function-enhancing agent would be required to comprehensively address the known pathophysiology of ALS. Remarkably, cannabis appears to have activity in all of those areas. Preclinical data indicate that cannabis has powerful antioxidative, anti-inflammatory, and neuroprotective effects. In the G93A-SOD1 ALS mouse, this has translated to prolonged neuronal cell survival, delayed onset, and slower progression of the disease. Cannabis also has properties applicable to symptom management of ALS, including analgesia, muscle relaxation, bronchodilation, saliva reduction, appetite stimulation, and sleep induction. With respect to the treatment of ALS, from both a disease modifying and symptom management viewpoint, clinical trials with cannabis are the next logical step. Based on the currently available scientific data, it is reasonable to think that cannabis might significantly slow the progression of ALS, potentially extending life expectancy and substantially reducing the overall burden of the disease.

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