

2017-39



Δ^9 -tetrahydrocannabinol (THC, CBN) Cannabidiol (CBD)

● Carbon
○ Hydrogen
● Oxygen

Saturday, September 16th 2017

New Mexico State Department of Health
Medical Cannabis Advisory Board
Medical Cannabis Program
PO Box 26110
Santa Fe, NM, 87502-6110

Petition: Requesting The Inclusion Of A New Medical Condition: Degenerative Neurological Disorder And Neuroprotective Applications

All Petitions Can Be Viewed Online At

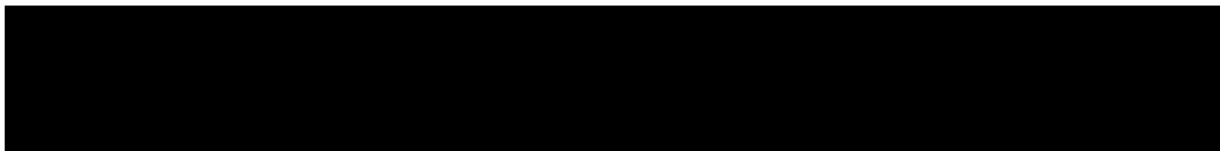
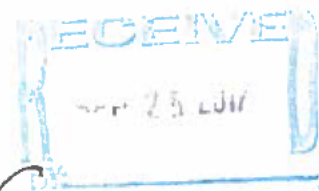
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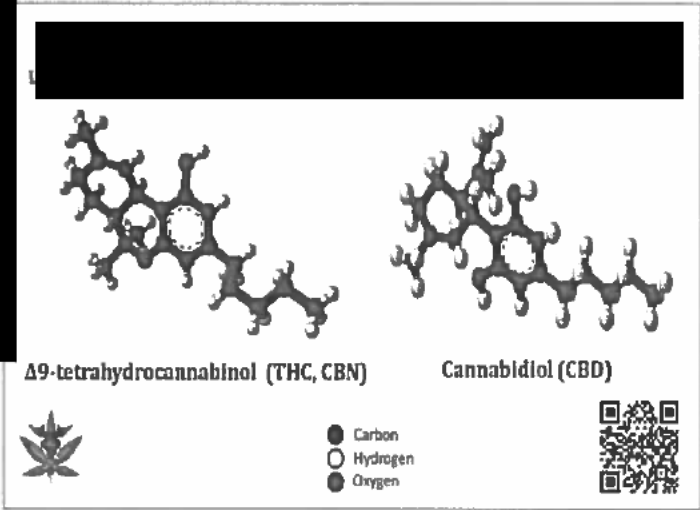
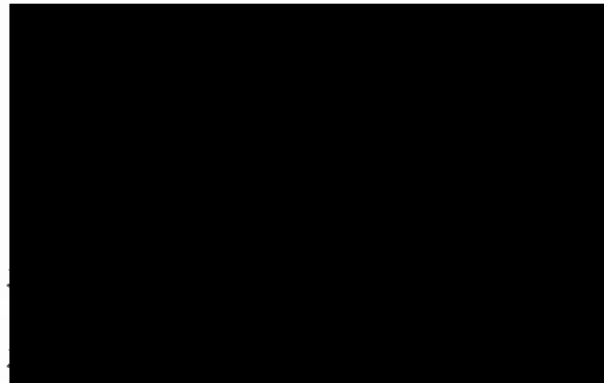
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Petitions Information Page

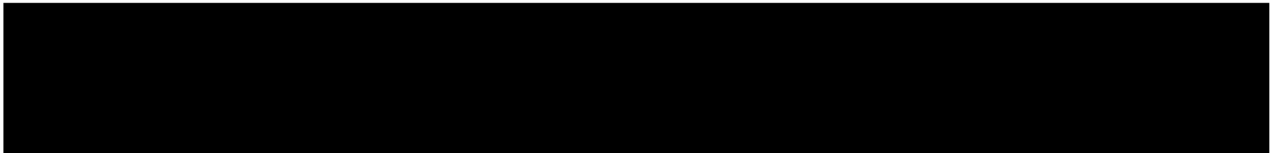
All Petitions Can Be Viewed Online At

This is the best way to accurately review them. Everything that is provided in the printed format is exactly the same on the Petitions website as well. If there are any questions during the review process, please do not hesitate to contact me at anytime of the day or night and my contact information is provided below this purpose.

The website was created to provide; the Medical Cannabis Advisory Board members, Secretary Gallagher, Medical Cannabis Program office officials, and the community- the easiest access at viewing all the Petitions. As all the petitions have resources within them, as embedded web links going to additions research and science for the petition it is in, along with the references cited.

About The Author:

I am very proactively involved with New Mexico's Medical Cannabis Community through my advocacy and activism for all in our community. At the Spring 2017, Medical Cannabis Advisory Board meeting, I organized and authored over 20 different petition in hopes of expanding current patient rights, producer rights and adding new health condition into the program. I have also launched a positive inclusive grassroots movement for all in our medical cannabis community, the in which the advocacy is guided by my advocate training from my membership with Americans For Safe Access. In addition to promoting awareness for the



benefits of cannabis for the public at large; I am also a member of American Cannabis Nurses Association and currently looking into Nursing programs with intentions to enroll and the pursue the American Cannabis Nurses Associations course curriculum to complement my Exercise Science background. I also recently attended the International Cannabis Research Conference 2017 that was held at Colorado State University - Pueblo's new ICR Facility. Later in the summer I will also be attending the Cannabis Law Institute in Denver, Colorado.

The [REDACTED] is the only patient led group in the State with a primary focus on medical cannabis, that operates in full compliance of the Act, providing patient-community advocacy, and the only group that has no outside financial influences from program producers or ancillary businesses in the medical cannabis program.

I am also an accomplished, well- organized, person with 10+ years of customer service experience. I am also a Patient in the medical cannabis program, with [REDACTED], [REDACTED] and [REDACTED] which provides me with great understanding for what other patients are going through. Medical Cannabis has been part of my life and treatment for over 20 years. Throughout my career I consistently established integrity, quality and professionalism to provide organizations/clients with broad array of services, skills and vast knowledge I have. My strong background from my First Responder/EMT/Beach Patrol experience, Exercise Science, Nutrition & Psychology studies at Ohio State, and a strong mechanical/ problem solving ability from my years in the cycling industry as a mechanic.

I also use my very strong health & wellness background from my exercise science studies, and I am writing some articles and submitting them to medical cannabis publications, as [Cannabis News Journal](#) with a central theme of most all my articles pertaining to the fitness, health, diet and wellness of the patient in relation to use of medical cannabis and how individuals can improve health with the benefits of cannabis & other positive lifestyle adaptations. My goals when I write are to always provide something that will Unite, Network, Grow, Inform and Educate the reader with hopes each one is able to take one good thing from the article to improve their own life.

[REDACTED]
Establishing the [REDACTED] is a great way to formally bring together like-minded activists in OUR medical cannabis community to work together toward ensuring safe access to medical cannabis. [REDACTED] will provide a state network of activists and goals to provide a national coalition network and staff; who work together on a regular basis to achieve shared goals. A great grassroots movement brings together vibrant activists in an environment of mutual respect, shared responsibility, and constructive political activism and creates a space for new advocates to plug into the movement. The ideal grassroots movement is networked into the larger community and is a constructive voice for patients, future patients and providers of medical cannabis in the state of New Mexico.

[REDACTED]

The best grassroots movements combine the art of conversation with skilled activism. They are considered by elected officials to be a principled voice and smart resource for community leaders who are interested in addressing the question of medical cannabis.

The primary focus is on Medical Cannabis. [REDACTED] is solely focused on expanding safe access to medical cannabis in New Mexico.

This means that [REDACTED] position does support legalization of cannabis for nonmedical therapeutic purposes or on related issues, such as incarceration or sentencing standards for recreational drug use; this support will be to provide advocacy for policy writing that first & foremost protects and improves the spirit and intent of the LECUA, 2007, protects and improves the Medical Cannabis Program in said legislation, and improves the State Department of Health Medical Cannabis Rules & Regulations.

Petitions Authored & Organized By [REDACTED]

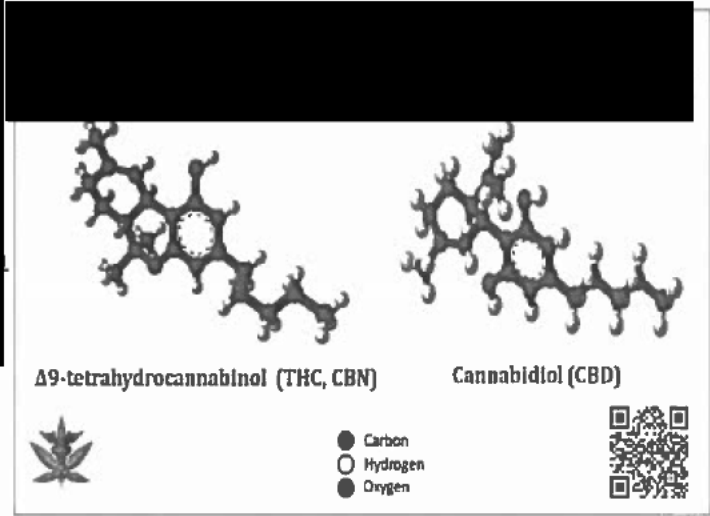
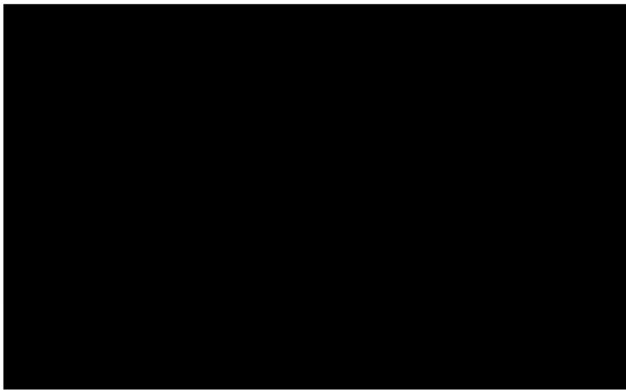
Americans For Safe Access - Member
American Cannabis Nurses Association - Member
LECUA Patient's Coalition Of New Mexico - Founder/Organizer
Medical Cannabis Patient in New Mexico

"The American Medical Association has no objection to any reasonable regulation of the medicinal use of cannabis and its preparations and derivatives. It does pretest, however, against being called upon to pay a special tax, to use special order forms in order to procure the drug, to keep special records concerning its professional use and to make special returns to the Treasury Department officials, as a condition precedent to the use of cannabis in the practice of medicine."

~Wm. C. Woodward, Legislative Counsel - 11:37 AM Monday, July 12, 1937

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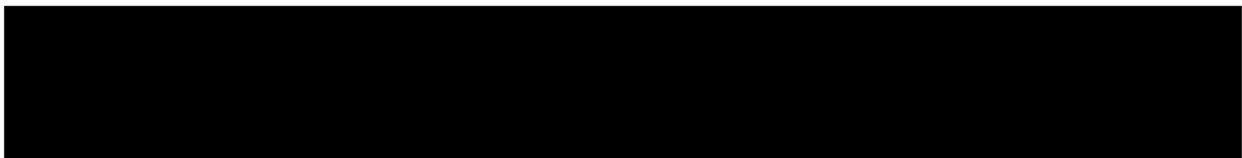
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Preface

The Governor's Office has highlighted the importance of such priorities like; Ensuring Transparency and Ethics in Government, and Keeping all New Mexicans Safe. In the Roundhouse, one of the state legislators' primary functions is to represent the people who elect them, but it is by no means their only function. They help to solve the numerous problems of their constituents, they serve on interim committees and they continually study new ideas for legislation. The Department of Health's mission is to promote health and wellness, improve health outcomes, and assure safety net services for all people in New Mexico. And the purpose of the Lynn and Erin Compassionate Use Act is to allow the beneficial use of medical cannabis in a regulated system for alleviating symptoms caused by debilitating medical conditions and their medical treatments.

These Petitions are being provided to the State Department of Health Medical Cannabis Program so the advisory board can review and recommend to the department for approval additional debilitating medical conditions that would benefit from the medical use of cannabis with the Lynn and Erin Compassionate Use Act.

In maintaining what Governor Martinez said, about the 'important responsibility' of the Medical Cannabis Advisory Board, these are the Petitions being providing for review; as all the Petitions meet the five items as criteria stated in (Part B) for the Duties and responsibilities of the Medical Cannabis Advisory Board:



November 2017 MCAB Petitions

Health Conditions To Add Petitions:

1. ADD/ADHD And Tourette's Syndrome
2. All Forms of Arthritis
3. Cystic Fibrosis
4. Degenerative Neurological Disorder / Neuroprotective Applications
5. Diabetes
6. Dysmenorrhea
7. Eczema / Psoriasis
8. Muscular Dystrophy
9. Polymyalgia Rheumatica
10. Post-Concussion Syndrome And TBI
11. All Types Seizures (such as: psychogenic neurologic disorders; Motor Disorders / Motor Development Disorders)
12. Substance Abuse Disorder(s)

Medical Treatment Petitions:

13. Medical Treatment; Pediatric Oncology & Medical Cannabis Use for Antiemetic in State Hospitals
14. Medical Treatment; Medical Cannabis Program Research & Education Established
15. Medical Treatment; ADA language for Section 8 of LECUA; Medical cannabis registry
16. Medical Treatment; Medical Cannabis 3 yr registry identification cards.
17. Medical Treatment; Recognition of nonresident medical cards.
18. Medical Treatment; Adequate Supply: LNPP Plant Count Increase
19. Medical Treatment; Increase MCAB membership

As these Petitions are being reviewed, I wanted to point out that many of the Petitions for new health conditions to add into the program are health conditions that also include chronic pain as part of them. These health conditions are ones where chronic pain is a symptom or underlying symptom. The exact causes of chronic pain without injury aren't well understood. The pain may sometimes result from an underlying health condition, such as: chronic fatigue syndrome: characterized by extreme, prolonged weariness that's often accompanied by the chronic pain. And for the beneficial use of medical cannabis in the LECUA, it's important to keep updating the Rules and Regulations for Health Conditions as not all providers or potential program participants may recognize or realize this. Thus a person, for example, suffering from Migraines may not realize how their health condition qualifies if the current qualifying health condition of Chronic Pain become an umbrella for other health conditions that are not stated.

As it states in the MCP Rules and Regulations, Section 7.34.2.9 Part A Petition Requirements, " The advisory board may accept and review petitions from any individual or association of individuals requesting the addition of a new medical condition, medical treatment or disease for the purpose of participating in the medical cannabis program and all lawful privileges under the act." All these Petitions fulfil this requirement and all fall under, requesting the addition of a new medical



condition, medical treatment or disease. In regard to “Scope of Work” for the medical cannabis advisory board and how the LECUA states;

7.34.2.2 STATUTORY AUTHORITY: The requirements set forth herein are promulgated by the secretary of the department of health pursuant to the authority granted under Section 9-7-6 (E) NMSA 1978, and the Lynn and Erin Compassionate Use Act, 26-2B-1 et seq. NMSA 1978.
[7.34.2.2 NMAC - Rp, 7.34.2.2 NMAC, 2/27/2015]

7.34.2.3 SCOPE: This part governs the membership, duties, responsibilities and public hearing proceedings of the medical cannabis advisory board.
[7.34.2.3 NMAC - Rp, 7.34.2.3 NMAC, 2/27/2015]

And then continues to further outline the MCAB Duties providing the following;

7.34.2.8 ADVISORY BOARD MEMBERSHIP REQUIREMENTS AND RESPONSIBILITIES:

B. Duties and responsibilities: The advisory board shall convene at least twice per year to:

- (1) review and recommend to the department for approval additional debilitating medical conditions that would benefit from the medical use of cannabis;
- (2) recommend quantities of cannabis that are necessary to constitute an adequate supply for qualified patients and primary caregivers;
- (3) accept and review petitions to add medical conditions, medical treatments or diseases to the list of debilitating medical conditions that qualify for the medical use of cannabis and all lawful privileges under the act and implementing rules;
- (4) issue recommendations concerning rules to be promulgated for the issuance of registry identification cards; and
- (5) review conditions previously reviewed by the board and approved by the secretary for the purpose of determining whether to recommend the revision of eligibility criteria for persons applying under those conditions or to review new medical and scientific evidence pertaining to currently approved conditions.

And in regards to Petitions for April 2017;

During the 2017 Regular Legislative Session, state lawmakers made efforts to legislate a number of changes to the state’s Medical Cannabis Program with over 25 different pieces of legislation, one of the bills vetoed by Gov. Susana Martinez, was House Bill-527, on Friday-April 7th 2017 (same day as the medical cannabis advisory board meeting), saying she did so in part because she didn’t want to “eliminate an important responsibility” of the Medical Cannabis Advisory Board. Health Secretary Lynn Gallagher, who has the final word on changes to the state Medical Cannabis Program, hasn’t decided whether to accept new conditions and petitions the board recommended yet.

That same Friday morning, on April 7th 2017, the Department of Health’s Medical Cannabis Advisory Board held a meeting exercising that important responsibility the Governor spoke of, that resulted in some of the following: The Medical Cannabis Advisory Board voted in favor of the following Petitions and recommended to add them into the program;



- 2017-022 Patient Run Collectives- Recommended to add to MCP 4-0

The addition of Patient Run Collectives would help relieve the medical cannabis plant count shortage.

- 2017-005 Change/increase possession limit to 16 oz- Recommended to add to MCP 4-0
- 2017-009 Removal of Max THC Content- Recommended Removal of Cap 4-0

Per the Department of Health's legal counsel's input I was told that, the following petitions numbered 3 and 11 were said that they would require statutory changes or are not covered under the duties of the MCAB and were not discussed at the MCAB meeting. Yet on that same day of this Medical Cannabis Advisory Board Meeting, the Governor of New Mexico said she didn't want to take away this important responsibility of the MCAB...so according to the Governor these Petitions should have been heard?

- 2017-003 Change LECUA to give MCAB more authority (increase membership)

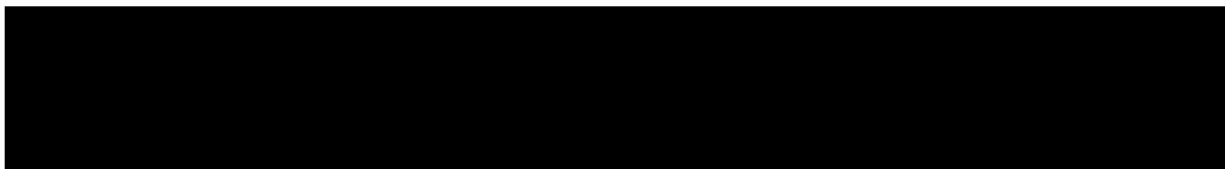
The addition of this Petition would allow the MCAB to better exercise that important responsibility the Governor spoke of thru increased membership, thus leading to relieving the medical cannabis plant count shortage.

- 2017-011 Add definition of Medical Treatment definition to LECUA and add Adequate Supply

Petitions 8,10 and 23 concern the licensed producers and would require statutory changes and are not covered under the duties of the MCAB and will not be discussed. Once again, on that same day of this Medical Cannabis Advisory Board Meeting, the Governor of New Mexico said she didn't want to take away this important responsibility of the MCAB...so according to the Governor these Petitions should have been heard.

Why was there a denial of hearing these petitions that are the "important responsibility" of the Medical Cannabis Advisory Board? Once a patient has that medical cannabis card the DoH MCP & MCAB set and regulate the patient's doctor-recommended treatment use of medicine (medical cannabis) by setting usage & dosage limits like; potency, quantity, availability and time of beneficial use.

Therefore, in order for the Department of Health Medical Cannabis Program to allow for the medical treatment of cannabis, the Department must properly have "adequate supply" and have it properly defined. And for the Department to have "adequate supply" they would need to know the different amounts of plant material that goes into all the different types of medicine being produced. Dried cannabis flower (bud), pre-rolls, edibles, tinctures, topicals/salves, and concentrated forms of cannabis all require different amounts of cannabis plant material to produce. Adequate Supply can not have a set definition in the rules and regulations and needs to be reviewed to coincide with MCP growth and patient/caregiver needs. Adequate Supply should be reviewed quarterly (4 times per



year) with a current census completed of qualified patients, caregivers and licensed non-profit producers.

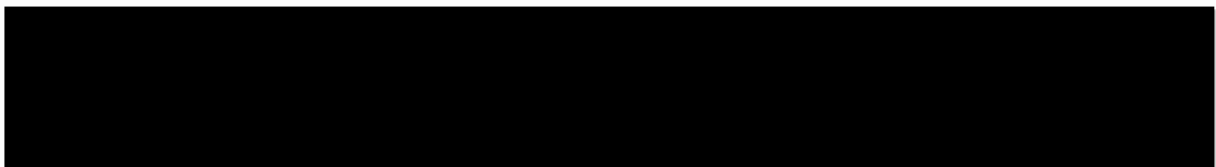
(Ad·e·quate: (ˈadəkweɪt/) adjective; satisfactory or acceptable in quality or quantity.

Sup·ply (səˈplɪ/) verb; 1. make (something needed or wanted) available to someone; provide.

"the farm supplies apples to cider makers" or a noun; 1. a stock of a resource from which a person or place can be provided with the necessary amount of that resource.)

This is empirical data that has not been collected within the state's medical cannabis program by the Department of Health. Therefore "adequate supply" can not be properly defined by the department by using unknown variables it has not collected. This further prevents the Department of Health from being able to set a proper plant count for each kind of licensed producer in the program for the means of achieving adequate supply within the medical cannabis program as required by law.

This is empirical data has been studied and researched by the state of Colorado by the Colorado Department of Revenue: "An assessment of physical and pharmacokinetic relationships in marijuana production and consumption in Colorado".



Petition: Requesting The Inclusion Of A New Medical Condition: Degenerative Neurological Disorder And Neuroprotective Applications

New Mexico's medical cannabis history started in 1978, after public hearings the legislature enacted H.B. 329, the nation's first law recognizing the medical value of cannabis. The New Mexico's medical cannabis program (MCP) is the only program in the U.S. that places sole responsibility for regulation on the state's Department of Health. Doctors must comply with state requirements for patients to be considered for applying to the medical cannabis program.

In the Lynn and Erin Compassionate Use Act, (2007) the law states; The Secretary of Health shall establish an advisory board consisting of eight practitioners representing the fields of neurology, pain management, medical oncology, psychiatry, infectious disease, family medicine and gynecology. The practitioners shall be nationally board-certified in their area of specialty and knowledgeable about the medical use of cannabis. The members shall be chosen for appointment by the Secretary from a list proposed by the New Mexico Medical Society. A quorum of the advisory board shall consist of three members. The advisory board shall:

- A. review and recommend to the department for approval additional debilitating medical conditions that would benefit from the medical use of cannabis;
- B. accept and review petitions to add medical conditions, medical treatments or diseases to the list of debilitating medical conditions that qualify for the medical use of cannabis;
- C. convene at least twice per year to conduct public hearings and to evaluate petitions, which shall be maintained as confidential personal health information, to **add medical conditions**, medical treatments or diseases to the list of debilitating medical conditions that qualify for the medical use of cannabis;
- D. issue recommendations concerning rules to be promulgated for the issuance of the registry identification cards; and
- E. recommend quantities of cannabis that are necessary to constitute an adequate supply for qualified patients and primary caregivers.

First, do no harm. As an important step in becoming a doctor, medical students must take the Hippocratic Oath. And one of the promises within that oath is "first, do no harm".

We have a sound law in the Lynn and Erin Compassionate Use Act, as Section 2 reads; **PURPOSE OF ACT.--**The purpose of the Lynn and Erin Compassionate Use Act is to allow the beneficial use of medical cannabis in a regulated system for alleviating symptoms caused by debilitating medical conditions and their medical treatments.

**"ARTICLE 2B. LYNN AND ERIN COMPASSIONATE USE ACT
N.M. Stat. Ann. § 26-2B-2 (2009)**



§ 26-2B-2. Purpose of act

The purpose of the Lynn and Erin Compassionate Use Act [26-2B-1 NMSA 1978] is to allow the beneficial use of medical cannabis in a regulated system for alleviating symptoms caused by debilitating medical conditions and their medical treatments.

HISTORY: Laws 2007, ch. 210, § 2.

EFFECTIVE DATES. --Laws 2007, ch. 210, § 12 makes the act effective July 1, 2007."

Mosby's Medical Dictionary states that "medical treatment" means; the management and care of a patient to combat disease or disorder. Medical treatment includes: Using prescription medications, or use of a non-prescription drug at prescription strength; and or treatment of disease by hygienic and pharmacologic remedies, as distinguished from invasive surgical procedures. Treatment may be pharmacologic, using drugs; surgical, involving operative procedures; or supportive, building the patient's strength. It may be specific for the disorder, or symptomatic to relieve symptoms without effecting a cure.(Mosby's Medical Dictionary, 9th edition.)

What is a chronic medical condition?

A chronic disease is one lasting 3 months or more, by the definition of the U.S. National Center for Health Statistics. Chronic diseases generally cannot be prevented by vaccines or cured by medication, nor do they just disappear. Harvard Medical Dictionary defines chronic as: Any condition that lasts a long time or recurs over time; chronic pain as: Pain that persists after an injury has healed or a disease is over; and chronic pain syndrome as : Long-term, severe pain that doesn't spring from an injury or illness, that interferes with daily life, and is often accompanied by other problems, such as depression, irritability, and anxiety.

What is the meaning of debilitating?

Something that's debilitating seriously affects someone or something's strength or ability to carry on with regular activities, like a debilitating illness. Debilitating comes from the Latin word debilis, meaning "weak." That's why you'll often see the adjective used to describe illness, despite the negative reference.

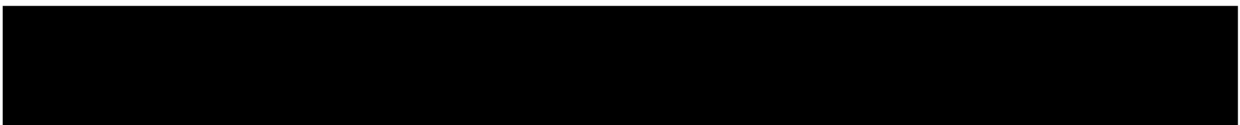
Petition Purpose and Background

The purpose of this Petition: Requesting The Inclusion Of A New Medical Condition: Degenerative Neurological Disorder And Neuroprotective Applications

This Petition: Requesting The Inclusion Of A New Medical Condition: Degenerative Neurological Disorder And Neuroprotective Applications is being provided to the state Department of Health Medical Cannabis Program so the advisory board can review and recommend to the department for approval additional debilitating medical conditions that would benefit from the medical use of cannabis with the Lynn and Erin Compassionate Use Act.

What is a neurological disorder?

Neurological disorders are diseases of the brain, spine and the nerves that connect them. There are



more than 600 diseases of the nervous system, such as brain tumors, epilepsy, Parkinson's disease and stroke as well as less familiar ones such as frontotemporal dementia.

What is a neurodegenerative disorder?

Neurodegenerative disease is an umbrella term for a range of conditions which primarily affect the neurons in the human brain. Neurons are the building blocks of the nervous system which includes the brain and spinal cord. ... Parkinson's disease (PD) and PD-related disorders. Prion disease.

Degenerative nerve diseases affect many of your body's activities, such as balance, movement, talking, breathing, and heart function. Many of these diseases are genetic. Sometimes the cause is a medical condition such as alcoholism, a tumor, or a stroke. Other causes may include toxins, chemicals, and viruses. Sometimes the cause is not known.

Degenerative nerve diseases include

- Alzheimer's disease
- Amyotrophic lateral sclerosis
- Friedreich's ataxia
- Huntington's disease
- Lewy body disease
- Parkinson's disease
- Spinal muscular atrophy

Degenerative nerve diseases can be serious or life-threatening. It depends on the type. Most of them have no cure. Treatments may help improve symptoms, relieve pain, and increase mobility. (<https://medlineplus.gov/degenerativenervediseases.html>)

Who Should Qualify for Medical Cannabis Use?

According to Americans For Safe Access Policy Studies & Research:

Background: The most fundamental aspect of medical cannabis laws is the relationship between a patient and their physician. It is often only the physician and the patient that possess information about a patient's health condition. However, many public officials and others who oppose medical cannabis laws often make assumptions about people's health. The media have even fomented such inappropriate assumptions by naming a category of patients "Young Able Bodied Males," condemning certain patients by visual assessment alone.

Findings: The health care information discussed between a patient and physician is considered private and protected under federal HIPAA laws. It is typically the purview of state medical boards to assess whether a physician has inappropriately recommended cannabis to someone who should not be qualified. Studies have shown in some medical cannabis states that the majority of patients suffer from chronic pain, an ailment that is not obviously detectable by another person.



Nevertheless, police will often harass and arrest patients based on the assumption that someone is faking their illness.

Position: Medical professionals should have an unrestricted ability to recommend cannabis therapeutics and that should not be impacted by law enforcement's perceptions.

Americans For Safe Access policy further states:

"Qualifying medical condition" shall mean any condition for which treatment with medical cannabis would be beneficial, *as determined by a patient's qualified medical professional, including but not limited to* cancer, glaucoma, positive status for human immunodeficiency virus, acquired immune deficiency syndrome (AIDS), hepatitis C, amyotrophic lateral sclerosis (ALS), Crohn's disease, Parkinson's disease, post-traumatic stress disorder, arthritis, chronic pain, neuropathic and other intractable chronic pain, and multiple sclerosis.

"Qualifying patient" shall mean a person who has a written recommendation from a qualified medical professional for the medical use of cannabis.

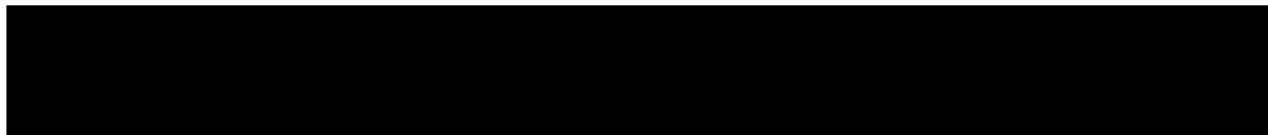
Neurodegenerative Disorder And Neuroprotective Applications With Medical Cannabis

Neurodegenerative diseases are those which result mainly from dysfunction of the central nervous system (the CNS, made up of the brain and spinal cord) as a result of damage to neurons, the primary cells of this system that communicate with each other to send signals throughout the brain and body. Damage to neurons of the CNS can result in a decreased ability to send signals to the peripheral, autonomic, and enteric nervous systems, which make it possible for us to move, touch, digest, breathe, react to and sense our environment, and in general, to live.

When cells of the central nervous system are destroyed and/or not able to communicate with each other efficiently and effectively, symptoms such as cognition and memory impairment, muscle incoordination, weakness, spasticity [i.e. tight muscles and exaggerated reflexes], paralysis [i.e. an inability to move], rigidity [i.e. tight muscles], and more can occur. These symptoms can cause substantial decreases in quality of life for patients, and even death when involving reduction in function of important physiological processes like breathing and heart function. Neurodegenerative disorders are so debilitating partially because neurons are one of the few cell types with a very limited ability to regenerate (along with heart cells and skeletal muscle cells). In most cases, once neurons have been destroyed, they cannot grow back.

"If left unchecked 30 years from now, more than 12 million Americans will suffer from neurodegenerative diseases." – Harvard Neurodiscovery Center

Examples of neurodegenerative diseases include Alzheimer's, Parkinson's, Huntington's, amyotrophic lateral sclerosis(ALS, also known as "Lou Gehrig's Disease"), spinal muscular atrophy, prion disease, and others. While multiple sclerosis (MS) has been thought to stem primarily from an



autoimmune response (one in which the body starts attacking itself), there is mounting evidence that it is a disease caused by a mixture of an autoimmune and primary neurodegeneration process.

A significant amount of research on cannabis has been conducted on the plant's potential harms in relation to brain function. However, the evidence suggests that not only are long-term, clinically significant cognitive deficits unlikely if use begins in adulthood, especially in the absence of chronic and excessive use, but cannabinoid medicine may actually prove effective in halting or reversing debilitating neurodegenerative disorders. *Note: Cognitive declines as a result of use have generally been conducted using recreational users who self-report frequency of use and smoke marijuana of unknown potency and quality; studying marijuana use with alternative delivery methods (e.g. vaporization, ingestion) and controlled frequency of use with whole-plant cannabis of known ratio/concentration/potency in an adult patient population may yield vastly different results than those discovered to date which have indicated harm.*

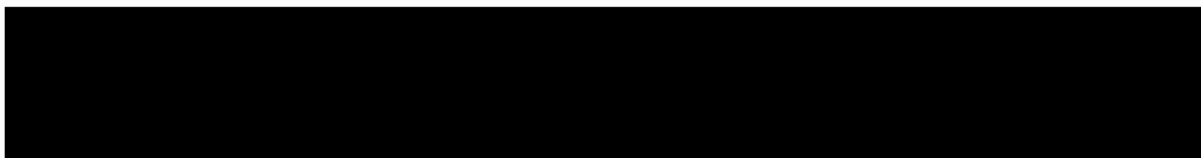
According to an article published by the Harvard Neurodiscovery Center, "If left unchecked 30 years from now, more than 12 million Americans will suffer from neurodegenerative diseases." It is therefore imperative that the medical and scientific communities continue to extensively research any and all potentially successful therapies for these disease processes.

Cannabinoid Therapy for Neurodegenerative Diseases

Extensive research on the impact of endocannabinoid system modulation and its effects on neurodegenerative disorders has occurred in the past several years. In 2014, British Journal of Pharmacology published a review titled "The influence of cannabinoids on generic traits of neurodegeneration", in which the authors concluded the following:

"Signalling from the CB1 and CB2 [i.e. cannabinoid] receptors are known to be involved in the regulation of Ca²⁺ [calcium] homeostasis [i.e. the mechanism by which systems are kept balanced], mitochondrial function [i.e. function of components of cells that produce energy], trophic [i.e. growth] support and inflammatory status... while other receptors gated [i.e. modulated/controlled] by cannabinoids... are gaining interest in their anti-inflammatory properties. Through multiple lines of evidence, this evolutionarily conserved neurosignalling system has shown neuroprotective capabilities and is therefore a potential target for neurodegenerative disorders." *While the current article briefly touches on the evidence that exists for the potential of cannabinoid therapy as treatment for neurodegenerative disorders, the BJP article will provide a more extensive overview.*

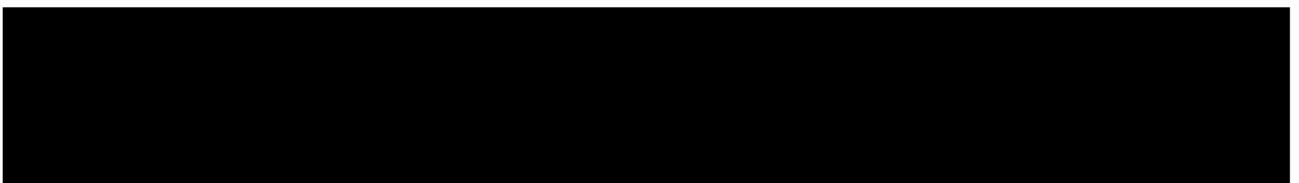
"Elevation of cannabinoid receptor activity either by pharmacological blockade of the degradation of cannabinoids or by receptor agonists could be a promising strategy for slowing down the progression of brain ageing and for alleviating the symptoms of neurodegenerative disorders." — Dr. Andras Bilkei-Gorzo

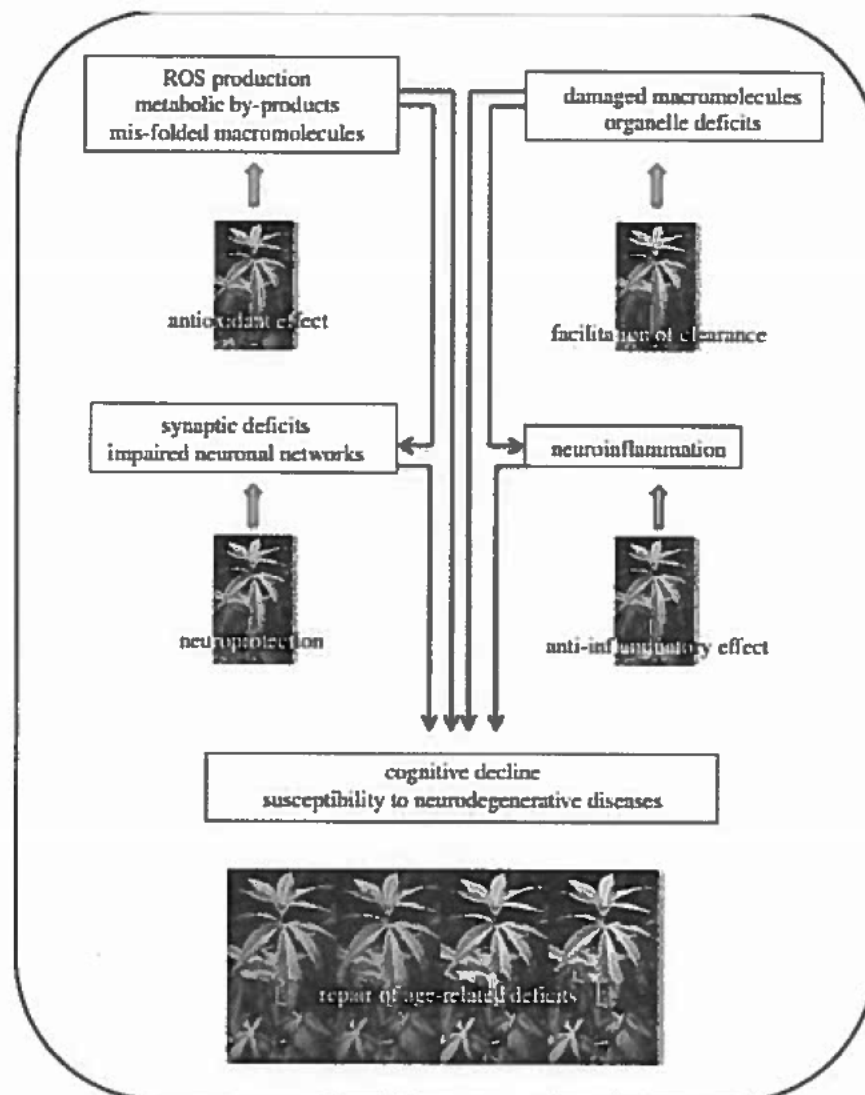


Another review published in 2012 in *Philosophical Transactions of the Royal Society* (source of the graphic below) discusses that cannabis may exert neuroprotective effects through mitochondrial regulation, anti-inflammatory and antioxidant (i.e. agents that prevent free radical damage) properties, and clearance of damaged cells and molecules in the brain. The author also noted that signaling of the endocannabinoid system (ECS) may decrease as people age, and therefore decreased function of the ECS may be a partial cause for age-related cognitive decline. According to the author, Dr. Andras Bilkei-Gorzo, “[E]levation of cannabinoid receptor activity either by pharmacological blockade of the degradation of cannabinoids [i.e. keeping cannabinoids active and in the brain for a longer amount of time] or by receptor agonists [i.e. receptor activation] could be a promising strategy for slowing down the progression of brain ageing and for alleviating the symptoms of neurodegenerative disorders.”

Additionally, a study published in July 2014 in the *Journal of Neuroscience Research* found more evidence to support the potential of cannabinoids to act as anti-inflammatory and neuroprotective agents, showing that ultralow (non-psychoactive) doses of THC were protective against “neuroinflammation-induced cognitive damage” (this study and was reviewed previously on *Medical Jane*).

In the image below, ROS stands for “reactive oxygen species”, which are created as intermediate products in natural physiological processes, but can cause damage to cells and tissues. “Synapses” are the junction between two neurons where signals are passed/communicated, and “organelles” are components of cells that have specific functions (e.g. mitochondria are organelles).





Conclusion

Given the highly favorable safety profile of whole-plant cannabis, and the severely debilitating symptoms caused by certain neurodegenerative diseases which could potentially be alleviated by its use, whole-plant cannabis medicine may be a safe and useful additional therapy for patients with certain neurodegenerative diseases who are finding it difficult to control their symptoms with standard therapy. Increased research on cannabinoid medicine and modulation of the endocannabinoid system in relation to neurodegeneration has the potential to lead to novel therapies which may help to prevent progression, and potentially initiation, of these diseases.

For information on how you can advocate, expectations, and safety in considering whole-plant medical cannabis use, click [here](#).



(<https://www.medicaljane.com/2014/11/11/cannabis-classroom-neurodegenerative-disease-and-medical-marijuana/>)

Beneficial Cannabinoids and Terpenoids Useful for Treating Degenerative Neurological Disorder And Neuroprotective Applications

The cannabis plant offers a plethora of therapeutic benefits and contains cannabinoids and terpenoid compounds that are useful for treating the symptoms of Degenerative Neurological Disorder And Neuroprotective Applications. While most of the ongoing research focuses on CBD and THC, the following list also denotes which cannabinoids and terpenoids work synergistically with each other for possible therapeutic benefit. It may be beneficial to seek out strains that contain these cannabinoids and terpenoids.

<p>ANALGESIC</p> <p>Cannabinoids</p> <p>THC CBG CBD CBC</p>	<p>Terpenoids</p> <p>CINEOL LIMONENE LINALOOL</p>
<p>ANTI-ANXIETY</p> <p>Cannabinoids</p> <p>Δ-8 THC CBD</p>	<p>Terpenoids</p> <p>LIMONENE LINALOOL</p>
<p>ANTI-CONVULSIVE</p> <p>Cannabinoids</p> <p>THCV CBD</p> <p>CBN</p>	<p>Terpenoids</p> <p>LINALOOL</p>
<p>ANTI-DEPRESSANT</p> <p>Cannabinoids</p> <p>THC CBGA CBD CBC</p> <p>CBN CBG</p>	<p>Terpenoids</p> <p>BORNEOL MYRCENE</p>
<p>ANTI-EMETIC</p> <p>Cannabinoids</p> <p>Δ-8 THC CBD</p>	
<p>ANTI-INSOMNIA</p> <p>Cannabinoids</p> <p>CBN CBD CBC</p>	<p>Terpenoids</p> <p>BORNEOL CITRONELLOL LINALOOL MYRCENE NEROLIDOL PHYTOL TERPINOLENE</p>
<p>ANTI-PSYCHOTIC</p> <p>Cannabinoids</p> <p>CBD</p>	
<p>ANTI-SPASMOTIC</p> <p>Cannabinoids</p> <p>THCA CBD</p> <p>THC</p>	<p>Terpenoids</p> <p>CITRONELLOL MYRCENE</p>
<p>NEUROPROTECTIVE</p> <p>Cannabinoids</p> <p>THC CBD</p> <p>THCV</p>	

(See References Section For Sources Cited Listed As: References)



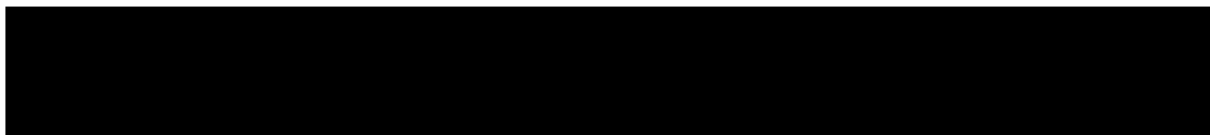
FINDINGS: EFFECTS OF CANNABIS ON NEUROLOGICAL DISORDERS

Research has shown that cannabis possesses neuroprotective effects, which in turn support the health of the brain, spinal cord and nerves, and help in preventing and limiting the progression of various neurological disorders. The major cannabinoids found in cannabis, including cannabidiol (CBD) and tetrahydrocannabinol (THC), have both shown they can help protect neurons, modulate the inflammatory response and encourage neuroregeneration (Lafuente, et al., 2011) (Kubajewska & Constantinescu, 2010) (Croxford, et al., 2008). Amyotrophic Lateral Sclerosis (ALS) The cannabinoids in cannabis have shown they are capable of delaying the onset of ALS, prolonging neuron survival and slowing the progression of the disease (Bilsland, et al., 2006) (Carter, Abood, Aggarwal & Weiss, 2010) (Raman, et al., 2004). CBD specifically has been found to significantly slow the onset of ALS (Weydt, et al., 2005). Cannabis can also help with managing the pain, appetite loss, depression, sleeping problems, spasticity and drooling associated with ALS (Amtmann, et al., 2004) (Carter, Abood, Aggarwal & Weiss, 2010). Epilepsy In numerous studies, CBE has demonstrated the ability to reduce or even eliminate seizures (Blair, Deshpande & DeLorenzo, 2015) (Rosenberg, Tsien, Whalley & Devinsky, 2015) (Szafarski & Bebin, 2014) (Devinsky, et al., 2014). Migraine Through their activation of the CB1 and CB2, cannabinoids effectively inhibits the pain response caused by migraines (Akerman, Holland, Lasalandra & Goardsby, 2013) (Baron, 2015) (Greco, et al., 2014). Multiple Sclerosis (MS) Cannabis' cannabinoids slow the neurodegenerative process of multiple sclerosis by helping to regulate the body's immune system, modulating its inflammatory response and encouraging neuroregeneration (Kubajewska & Constantinescu, 2010) (Croxford, et al., 2008). One study showed that cannabinoids reduced the damage to myelin caused from inflammation, thereby offering neuroprotection (Pryce, et al., 2003). Another found that cannabinoids reduced neurological disability, improved motor coordination and limited the progression of the MS in animals with a model of multiple sclerosis (de Lago, et al., 2012). Parkinson's Disease Studies show that cannabis' neuroprotective effects can slow the progression of Parkinson's. Its cannabinoids suppress excitotoxicity, glial activation and oxidative injury that lead to neuron degeneration. They improve the mitochondria function and the clearance of cellular debris, which also supports neuron health (More & Choi, 2015) (Garcia-Arencibia, Garcia & Fernandez-Ruiz, 2009) (Lastres-Becker & Fernandez-Ruiz, 2006). CBD has also shown to support the health of neural cells mitochondria (da Silva, et al., 2014) (Zuardi, 2008). Peripheral Neuropathy Cannabis effectively reduces neuropathic pain (Jensen, Chen, Furnish & Wallace, 2015) (Baron, 2015) (McDonough, McKenna, McCreary & Downer, 2014). Cannabis-based medicines have even shown they can reduce chronic neuropathic pain that had previously proven refractory to other treatments (Boychuk, Goddard, Mauro & Orellana, 2015). Prion Diseases CBD has shown to protect neurons against prion toxicity and therefore reduced the risk of prion diseases, a group of rare degenerative brain disorders (Dirikoc, et al., 2007). Spinal Cord Injury Cannabis' cannabinoids limit neurological damage caused by a spinal cord injury if administered shortly after the traumatic event. The cannabinoids reduce the proinflammatory cytokines and delay the atrophy and degeneration of neurons and thereby protect the white matter and myelin sheath surrounding the cord and nerves (Arevalo-Martin, Garcia-Ovejero & Molina-Holgado, 2010) (Latini, et al., 2014) (Arevalo-Martin, Garcia-Ovejero & Molina-Holgado, 2010) (Arevalo-Martin, et al., 2012). An animal trial have found the administration of cannabinoids shortly after a spinal cord injury caused an improvement in locomotor functional recovery (Kwiatkoski, Guimaraes & Del-Bel, 2012). In addition, cannabis has

found to be among the most effective pain relief treatments for people with spinal cord injuries (Wilsey, et al., 2013) (Heutink, Post, Wollaars & van Asbeck, 2011). Stroke Cannabinoids reduce infarct volume and improving functional outcome following strokes (England, Hind, Rasid & O'Sullivan, 2015). When administered shortly after a stroke, CBD specifically protects neurons and astrocytes from damage, and therefore leads to improved functional, histological, biochemical, and neurobehavior recovery (Lafuente, et al., 2011). Tourette Syndrome Cannabis effectively suppresses tics and improves behavioral problems associated with Tourette syndrome (Muller-Vahl, 2013) (Muller-Vahl, et al., 2002). Tumors of the Brain and Spinal Cord CBD has shown it has anti-tumor properties, with one study showing it significantly inhibited the growth of cancer cells (Massi, et al., 2004).

STATES THAT HAVE APPROVED MEDICAL CANNABIS FOR NEUROLOGICAL DISORDERS

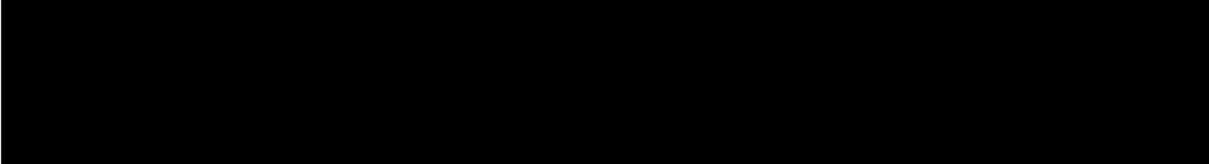
No states include “neurological disorders” on their list of approved conditions for medical cannabis, despite the Federal Government owning the Patent providing it should be use for it. Pennsylvania and West Virginia allow medicinal cannabis for “damage to the nervous tissue of the spinal cord with objective neurological indication of intractable spasticity.” Additionally, many other states allow medical marijuana for the treatment of specific neurological disorders. For example, Arizona, Arkansas, Connecticut, Delaware, Florida, Georgia, Maine, Massachusetts, Michigan, Minnesota, New Hampshire, New Jersey, New Mexico, New York, North Dakota, Ohio, Pennsylvania and West Virginia have approved medical marijuana for the treatment of ALS. Alabama, Connecticut, Delaware, Florida, Georgia, Iowa, Louisiana, Maine, Mississippi, Missouri, New Hampshire, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Pennsylvania, South Carolina, Texas, Utah, Virginia, West Virginia, Wisconsin, and Wyoming have approved medical marijuana for the treatment of either epilepsy or seizure disorders. California and Illinois have specifically approved medical marijuana for the treatment of migraines. Arkansas, Montana, New Mexico, New York, Pennsylvania and West Virginia have approved medical marijuana for the treatment of neuropathy. New Hampshire, New Mexico, New York, North Dakota, Ohio and Pennsylvania have approved medical marijuana specifically for the treatment of spinal cord injuries. Arkansas, Illinois, Minnesota and Ohio have approved medical marijuana specifically for the treatment of Tourette syndrome. Connecticut, Florida, Georgia, Illinois, Maine, Massachusetts, New Hampshire, New Mexico, New York, Ohio, Pennsylvania and West Virginia have approved medical marijuana for the treatment of Parkinson’s disease. Alaska, Connecticut, Florida, Georgia, Illinois, Maine, Massachusetts, New Hampshire, New Jersey, New Mexico, New York, Ohio, Pennsylvania, Vermont and West Virginia allow medical marijuana for the treatment of multiple sclerosis. Alaska, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, Florida, Georgia, Hawaii, Illinois, Louisiana, Maine, Massachusetts, Michigan, Minnesota, Montana, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Dakota, Ohio, Oregon, Pennsylvania, Rhode Island, Vermont, Washington and West Virginia legally allow medical marijuana for the treatment of cancer, including tumors. Currently, no states have approved medical marijuana specifically for the treatment of stroke. However, in Washington D.C., any condition can be approved for medical marijuana as long as a DC-licensed physician recommends the treatment. Plus, various other states will consider



allowing medical marijuana to be used for the treatment of neurological disorders with the recommendation from a physician. These states include: California (any debilitating illness where the medical use of marijuana has been recommended by a physician), Connecticut (other medical conditions may be approved by the Department of Consumer Protection), Massachusetts (other conditions as determined in writing by a qualifying patient's physician), Nevada (other conditions subject to approval), Oregon (other conditions subject to approval), Rhode Island (other conditions subject to approval), and Washington (any "terminal or debilitating condition"). In addition, various states have approved medical marijuana for symptoms commonly associated with neurological disorders. Many states have approved medical marijuana specifically to treat chronic pain. These states include: Alaska, Arizona, California, Colorado, Delaware, Hawaii, Maine, Maryland, Michigan, Montana, New Mexico, Ohio, Oregon, Pennsylvania, Rhode Island and Vermont. The states of Nevada, New Hampshire, North Dakota, Montana, Ohio, Vermont and West Virginia allow medical marijuana to treat "severe pain." The states of Arkansas, Minnesota, Ohio, Pennsylvania and Washington have approved cannabis for the treatment of "intractable pain." Alaska, Arizona, Arkansas, California, Colorado, Delaware, Hawaii, Louisiana, Maryland, Michigan, Minnesota, Montana, Nevada, New Hampshire, North Dakota, Ohio, Oregon, Pennsylvania (intractable seizures), Rhode Island, Tennessee(intractable seizures), Vermont, Washington and West Virginia have approved medical marijuana to treat seizures. Arizona, Arkansas, California, Colorado, Delaware, Florida, Hawaii, Maryland, Michigan, Minnesota, Montana, Nevada, New Hampshire, Oregon, Rhode Island and Washington have approved medical marijuana for the treatment of spasms.

RECENT STUDIES ON CANNABIS' EFFECT ON NEUROLOGICAL DISORDERS

<p>Cannabis delays the onset of ALS and slow the progression of the disease. <i>Cannabis and amyotrophic lateral sclerosis: hypothetical and practical applications, and a call for clinical trials.</i>(http://journals.sagepub.com/doi/pdf/10.1177/1049909110369531)</p>
<p>CBD-enriched cannabis reduced seizure frequency in 85% of children and caused complete seizure freedom in 14% of children. <i>Perceived efficacy of cannabidiol-enriched cannabis extracts for treatment of pediatric epilepsy: A potential role for infantile spasms and Lennox-Gastaut syndrome.</i> (http://www.epilepsybehavior.com/article/S1525-5050(15)00157-2/fulltext)</p>
<p>Cannabinoids administered shortly following spinal cord injury limits damage. <i>Early endogenous activation of CB1 and CB2 receptors after spinal cord injury is a protective response involved in spontaneous recovery.</i> (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3496738/)</p>
<p>Smoking cannabis significantly improved tremors, rigidity and bradykinesia in Parkinson's disease patients. <i>Cannabis (medical marijuana) treatment for motor and non-motor symptoms of Parkinson disease: an open-label observational</i></p>



autoimmune diseases. The cannabinoids are found to have particular application as neuroprotectants, for example in limiting neurological damage following ischemic insults, such as stroke and trauma, or in the treatment of neurodegenerative diseases, such as Alzheimer's disease, Parkinson's disease and HIV dementia. Nonpsychoactive cannabinoids, such as cannabidiol, are particularly advantageous to use because they avoid toxicity that is encountered with psychoactive cannabinoids at high doses useful in the method of the present invention. A particular disclosed class of cannabinoids useful as neuroprotective antioxidants is formula (I) wherein the R group is independently selected from the group consisting of H, CH.sub.3, and COCH.sub.3. ##STR1##



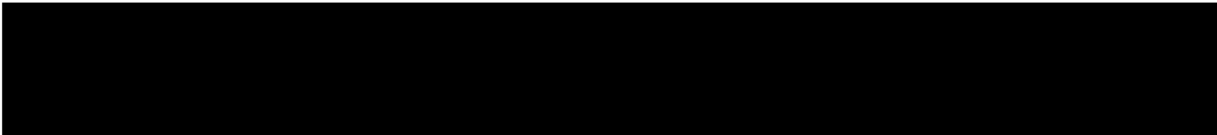
<p>(12) United States Patent Hampson et al.</p>	<p>119 Patent No.: US 6,630,507 B1 115 Date of Patent: Oct. 7, 2003</p>
<p>(54) CANNABINOIDS AS ANTIOXIDANTS AND NEUROPROTECTANTS</p> <p>(71) Inventor: Alden E. Hampson, Irvine, CA (US); Julius Anthony, Bethesda, MD (US); Slavomir Cizemicki, Bethesda, MD (US)</p> <p>(73) Assignor: The United States of America as represented by the Department of Health and Human Services, Washington, DC (US)</p> <p>* * * Notice: Subject to any disclaimer, the term of this</p>	<p>OTHER PUBLICATIONS</p> <p>Winkler et al., <i>The Mandel Index</i>, 19th Edition (1983) p. 261, abstract No. 1723 *</p> <p>Michoniere et al., "A Total Synthesis of Δ^9-Tetrahydrocannabinol: The Active Constituent of Hashish," <i>Journal of the American Chemical Society</i>, 87:14 3273-3275 (1965).</p> <p>Michoniere et al., "Chemical State of Hashish Acetyl," <i>Science</i>, 146:11-12 (1970).</p> <p>Chenon et al., "The Crystal and Molecular Structure of Cannabidiol," <i>Acta Chem Scand B</i> 11, 9685-612 (1977)</p> <p>Chenon et al., "Chronic Administration of Cannabidiol to Healthy Volunteers and Lipoic Patients," <i>Pharmacology</i>, 71, 47-56 (2000).</p>

United States Patent **6,630,507**
Hampson, et al. **October 7, 2003**

The Patent Research Show That:

1. A method of treating diseases caused by oxidative stress, comprising administering a therapeutically effective amount of a cannabinoid that has substantially no binding to the NMDA receptor to a subject who has a disease caused by oxidative stress.
2. The method of claim 1, wherein the cannabinoid is nonpsychoactive.
3. The method of claim 2, wherein the cannabinoid has a volume of distribution of 10 L/kg or more.
4. The method of claim 1, wherein the cannabinoid is not an antagonist at the NMDA receptor.
5. The method of claim 1, wherein the cannabinoid is: ##STR22##
6. The method of claim 5, wherein R is H, substituted or unsubstituted alkyl, carboxyl or alkoxy.
7. The method of claim 2, wherein the cannabinoid is: ##STR23##

where A is cyclohexyl, substituted or unsubstituted aryl, or ##STR24## but not a pinene; R.sub.1 is H, substituted or unsubstituted alkyl, or substituted or unsubstituted carboxyl; R.sub.2 is H, lower substituted or unsubstituted alkyl, or alkoxy; R.sub.3 is of H, lower substituted or unsubstituted



alkyl, or substituted or unsubstituted carboxyl; R.sub.4 is H, hydroxyl, or lower substituted or unsubstituted alkyl; and R.sub.5 is H, hydroxyl, or lower substituted or unsubstituted alkyl.

8. The method of claim 7, wherein R.sub.1 is lower alkyl, COOH or COCH.sub.3 ; R.sub.2 is unsubstituted C.sub.1 -C.sub.5 alkyl, hydroxyl, methoxy or ethoxy; R.sub.3 is H, unsubstituted C.sub.1 -C.sub.3 alkyl, or COCH.sub.3 ; R.sub.4 is hydroxyl, pentyl, heptyl, or diethylheptyl; and R.sub.5 is hydroxyl or methyl.

9. The method of claim 1, wherein the cannabinoid is: ##STR25##

where R.sub.1, R.sub.2 and R.sub.3 are independently H, CH.sub.3, or COCH.sub.3.

10. The method of claim 9, wherein the cannabinoid is: ##STR26##

where: a) R.sub.1 =R.sub.2 =R.sub.3 =H; b) R.sub.1 =R.sub.3 =H, R.sub.2 =CH.sub.3 ; c) R.sub.1 =R.sub.2 =CH.sub.3, R.sub.3 =H; d) R.sub.1 =R.sub.2 =COCH.sub.3, R.sub.3 =H; or e) R.sub.1 =H, R.sub.2 =R.sub.3 =COCH.sub.3.

11. The method of claim 2, wherein the cannabinoid is: ##STR27##

where R.sub.19 is H, lower alkyl, lower alcohol, or carboxyl; R.sub.20 is H or OH; and R.sub.21 -R.sub.25 are independently H or OH.

12. The method of claim 11, wherein R.sub.19 is H, CH.sub.3, CH.sub.2 OH, or COOH, and R.sub.20 -R.sub.24 are independently H or OH.

13. The method of claim 2, wherein the cannabinoid is: ##STR28##

where R.sub.19 and R.sub.20 are H, and R.sub.26 is alkyl.

14. The method of claim 10, wherein the cannabinoid is cannabidiol.

15. A method of treating an ischemic or neurodegenerative disease in the central nervous system of a subject, comprising administering to the subject a therapeutically effective amount of a cannabinoid, where the cannabinoid is ##STR29##

where R is H, substituted or unsubstituted alkyl, carboxyl, alkoxy, aryl, aryloxy, arylalkyl, halo or amino.

16. The method of claim 15, wherein the cannabinoid is not a psychoactive cannabinoid.

17. The method of claim 15 where the ischemic or neurodegenerative disease is an ischemic infarct, Alzheimer's disease, Parkinson's disease, and human immunodeficiency virus dementia, Down's syndrome, or heart disease.

18. A method of treating a disease with a cannabinoid that has substantially no binding to the NMDA receptor, comprising determining whether the disease is caused by oxidative stress, and if the



disease is caused by oxidative stress, administering the cannabinoid in a therapeutically effective antioxidant amount.

19. The method of claim 18, wherein the cannabinoid has a volume of distribution of at least 1.5 L/kg and substantially no activity at the cannabinoid receptor.

20. The method of claim 19, wherein the cannabinoid has a volume of distribution of at least 10 L/kg.

21. The method of claim 1, wherein the cannabinoid selectively inhibits an enzyme activity of 5- and 15-lipoxygenase more than an enzyme activity of 12-lipoxygenase.

22. A method of treating a neurodegenerative or ischemic disease in the central nervous system of a subject, comprising administering to the subject a therapeutically effective amount of a compound selected from any of the compounds of claims 9 through 13.

23. The method of claim 22 where the compound is cannabidiol.

24. The method of claim 22, wherein the ischemic or neurodegenerative disease is an ischemic infarct, Alzheimer's disease, Parkinson's disease, and human immunodeficiency virus dementia, Down's syndrome, or heart disease.

25. The method of claim 24 wherein the disease is an ischemic infarct.

26. The method of claim 1, wherein the cannabinoid is not an antagonist at the AMPA receptor.

U.S. Patent Documents Pertaining to This Patent

<u>2304669</u>	December 1942	Adams
<u>4876276</u>	October 1989	Mechoulam et al.
<u>5227537</u>	July 1993	Stoss et al.
<u>5284867</u>	February 1994	Kloog et al.
<u>5434295</u>	July 1995	Mechoulam et al.
<u>5462946</u>	October 1995	Mitchell et al.
<u>5512270</u>	April 1996	Ghio et al.
<u>5521215</u>	May 1996	Mechoulam et al.
<u>5538993</u>	July 1996	Mechoulam et al.
<u>5635530</u>	June 1997	Mechoulam et al.
<u>5696109</u>	December 1997	Malfroy-Camine et al.
<u>6410588</u>	June 2002	Feldmann et al.

Foreign Patent Documents

427518	May 1991	EP
576357	Dec 1993	EP
656354	Jun 1995	EP
658546	Jun 1995	EP
WO9305031	Mar 1993	WO
WO9412667	Jun 1994	WO
WO9612485	May 1996	WO
WO9618600	Jun 1996	WO
WO9719063	May 1997	WO
99/53917	Oct 1999	WO

Other References For This Patent

- Windholz et al., *The Merck Index, Tenth Edition (1983)* p. 241, abstract No. 1723.* .
- Mechoulam et al., "A Total Synthesis of Δ^1 -Tetrahydrocannabinol, the Active Constituent of Hashish," *Journal of the American Chemical Society*, 87:14:3273-3275 (1965) .
- Mechoulam et al., "Chemical Basis of Hashish Activity," *Science*, 18:611-612 (1970) .
- Ottersen et al., "The Crystal and Molecular Structure of Cannabidiol," *Acta Chem. Scand. B* 31, 9:807-812 (1977) .
- Cunha et al., "Chronic Administration of Cannabidiol to Healthy Volunteers and Epileptic Patients," *Pharmacology*, 21:175-185 (1980) .
- Consroe et al., "Acute and Chronic Antiepileptic Drug Effects in Audiogenic Seizure-Susceptible Rats," *Experimental Neurology*, Academic Press Inc., 70:626-637 (1980) .
- Turkanis et al., "Electrophysiologic Properties of the Cannabinoids," *J. Clin. Pharmacol.*, 21:449S-463S (1981) .
- Carlini et al., "Hypnotic and Antiepileptic Effects of Cannabidiol," *J. Clin. Pharmacol.*, 21:417S-427S (1981) .
- Karler et al., "The Cannabinoids as Potential Antiepileptics," *J. Clin. Pharmacol.*, 21:437S-448S (1981) .
- Consroe et al., "Antiepileptic Potential of Cannabidiol Analogs," *J. Clin. Pharmacol.*, 21:428S-436S (1981) .
- Colasanti et al., "Ocular Hypotension, Ocular Toxicity, and Neurotoxicity in Response to Marijuana Extract and Cannabidiol," *Gen Pharm.*, Pergamon Press Ltd., 15(6):479-484 (1984) .
- Colasanti et al., "Intraocular Pressure, Ocular Toxicity and Neurotoxicity after Administration of Cannabinol or Cannabigerol," *Exp. Eye Res.*, Academic Press Inc., 39:251-259 (1984) .
- Volfe et al., "Cannabinoids Block Release of Serotonin from Platelets Induced by Plasma from Migraine Patients," *Int. J. Clin. Pharm. Res.*, Bioscience Ediprint Inc., 4:243-246 (1985) .
- Agurell et al., "Pharmacokinetics and Metabolism of Δ^1 -Tetrahydrocannabinol and Other Cannabinoids with Emphasis on Man*," *Pharmacological Reviews*, 38(1):21-43 (1986) .
- Karler et al., "Different Cannabinoids Exhibit Different Pharmacological and Toxicological Properties," *NIDA Res. Monogr.*, 79:96-107 (1987) .
- Samara et al., "Pharmacokinetics of Cannabidiol in Dogs," *Drug Metabolism and Disposition*, 16(3):469-472 (1988) .
- Choi, "Glutamate Neurotoxicity and Diseases of the Nervous System," *Neuron*, Cell Press, 1:623-634 (1988) .
- Eshhar et al., "Neuroprotective and Antioxidant Activities of HU-211, A Novel NMDA Receptor Antagonist," *European Journal of Pharmacology*, 283:19-29 (1995) .
- Skaper et al., "The ALIamide Palmitoylethanolamide and Cannabinoids, but not Anandamide, are Protective in a Delayed Postglutamate Paradigm of Excitotoxic Death in Cerebellar Granule Neurons," *Neurobiology*, Proc. Natl. Acad. Sci. USA, 93:3984-3989 (1996) .
- Alonso et al., "Simple Synthesis of 5-Substituted Resorcinols: A Revisited Family of

Interesting Bioactive Molecules," J. Org. Chem., American Chemical Society, 62(2):417-421 (1997). .

Combes et al. "A Simple Synthesis of the Natural 2,5-Dialkylresorcinol Free Radical Scavenger Antioxidant: Resorstatin," Synthetic Communications, Marcel Dekker, Inc., 27(21):3769-3778 (1997). .

Shohami et al., "Oxidative Stress in Closed-Head Injury: Brain Antioxidant Capacity as an Indicator of Functional Outcome," Journal of Cerebral Blood Flow and Metabolism, Lippincott-Raven Publishers, 17(10):1007-1019 (1997). .

Zurier et al., "Dimethylheptyl-THC-11 OIC Acid," Arthritis & Rheumatism, 41(1):163-170 (1998). .

Hampson et al., "Dual Effects of Anandamide on NMDA Receptor-Mediated Responses and Neurotransmission," Journal of Neurochemistry, Lippincott-Raven Publishers, 70(2):671-676 (1998). .

Hampson et al., "Cannabidiol and (-)-DELTA⁹-tetrahydrocannabinol are Neuroprotective Antioxidants," Medical Sciences, Proc. Natl. Acad. Sci. USA, 8268-8273 (1998)..

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Attorney, Agent or Firm: Klarquist Sparkman, LLP

A number of experts, including CNN's chief medical correspondent Dr. Sanjay Gupta, have noted the contradiction between federal marijuana law and the government's patent.

"The United States government owns a patent on cannabis as a medical application... So we have a patent through our Department of HHS on cannabis as a therapeutic and we also schedule it as a Schedule I."

Rules, Regulations, & Policy Solution For Petition: Requesting The Inclusion Of A New Medical Condition: Degenerative Neurological Disorder And Neuroprotective Applications

The approval of this Petition: Requesting The Inclusion Of A New Medical Condition: Degenerative Neurological Disorder And Neuroprotective Applications, that is being provided to the state Department of Health Medical Cannabis Program so the advisory board can review and recommend to the department for approval additional debilitating medical conditions that would benefit from the medical use of cannabis with the Lynn and Erin Compassionate Use Act.

The approval of this petition would bring the Department of Health in compliance with the intent of the law and uphold the spirit of the Lynn and Erin Compassionate Use Act, 2007. Fulfilling both;" Section 2. PURPOSE OF ACT.--The purpose of the Lynn and Erin Compassionate Use Act is to allow the beneficial use of medical cannabis in a regulated system for alleviating symptoms caused by debilitating medical conditions and their medical treatments" And Section 6. ADVISORY BOARD CREATED--DUTIES: The advisory board shall: A. review and recommend to the department for approval additional debilitating medical conditions that would benefit from the medical use of

cannabis." New Mexico's medical cannabis history started in 1978. After public hearings the legislature enacted H.B. 329, the nation's first law recognizing the medical value of cannabis...the first law.

References

Understanding medical cannabis. Elemental Wellness Center, 2014 Jul.

References 1:

Akerman, S., Holland, P.R., Lasalandra, M.P. and Goadsby, P.J. (2013, September). Endocannabinoids in the brainstem modulate dural trigeminovascular nociceptive traffic via CB1 and "triptan" receptors: implications in migraine. *Journal of Neuroscience*, 33(37), 14869-77. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3771033/>. Alvarez, F.J., Lafuente, H., Rey-Santano, M.C., Mielgo, V.E., Gastiasoro, E., Rueda, M., Pertwee, R.G., Castillo, A.I., Romero, J., and Martinez-Orgado, J. (2008). Neuroprotective effects of the nonpsychoactive cannabinoid cannabidiol in hypoxic-ischemic newborn piglets. *Pediatric Research*, 64, 653-648. Retrieved from <http://www.nature.com/pr/journal/v64/n6/full/pr2008260a.html>. Amtmann, D., Weydt, P., Johnson, K.L., Jensen, M.P., and Carter, G.T. (2004). Survey of cannabis use in patients with amyotrophic lateral sclerosis. *The American Journal of Hospice and Palliative Care*, 21(2), 94-104. Retrieved from <http://journals.sagepub.com/doi/pdf/10.1177/104990910402100206>. Arevalo-Martin, A., Garcia-Ovejero, D., and Molina-Holgado, E. (2010, May). The endocannabinoid 2-arachidonoylglycerol reduces lesion expansion and white matter damage after spinal cord injury. *Neurobiology of Disease*, 38(2), 304-12. Retrieved from <http://www.sciencedirect.com/science/article/pii/S0969996110000409>. Arevalo-Martin, A., Garcia-Ovejero, D., Sierra, Palomares, Y., Paniagua-Torija, B., Gonzalez-Gil, I., Oretaga-Gutierrez, S., and Molina-Holgado, E. (2012). Early endogenous activation of CB1 and CB2 receptors after spinal cord injury is a protective response involved in spontaneous recovery. *PLOS One*, 7(11), e49057. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3496738/>. Baron, E.P. (2015, June). Comprehensive Review of Medicinal Marijuana, Cannabinoids, and Therapeutic Implications in Medicine and Headache: What a Long Strange Trip It's Been... *Headache*, 55(6), 885-916. Retrieved from <http://onlinelibrary.wiley.com/wo1/doi/10.1111/head.12570/full>. Bilsland, L.G., Dick, J.R., Pryce, G., Petrosino, S., Di Marzo, V., Baker, D., and Greensmith, L. (2006). Increasing cannabinoid levels by pharmacological and genetic manipulation delay disease progression in SOD1 mice. *The FASEB Journal*, 20(7), 1003-1005. Retrieved from <http://www.fasebj.org/content/20/7/1003.long>. Blair, R.E., Deshpande, L.S., and DeLorenzo, R.J. (2015, September). Cannabinoids: is there a potential treatment role in epilepsy? *Expert Opinion on Pharmacology*, 16(13), 1911-4. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4845642/>. Blázquez, C., Chiarlone, A., Bellocchio, L., Resel, E., Pruunsild, P., García-Rincón, D., Sendtner, M., Timmusk, T., Lutz, B., Galve-Roperh, I., and Guzmán, M. (2015). The CB1 cannabinoid receptor signals striatal neuroprotection via a PI3K/Akt/mTORC1/BDNF pathway. *Cell Death and Differentiation*, 22(10), 1618-1629. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4563779/>. Boychuk, D.G., Goddard, G., Mauro, G., and Orellana, M.R. (2015, Winter). The effectiveness of cannabinoids in the management

of chronic nonmalignant neuropathic pain: a systematic review. *Journal of Oral & Facial Pain and Headache*, 29(1), 7-14. Retrieved from <https://goo.gl/R28LWD>. Carter, G.T., Abood, M.E., Aggarwal, S.K and Weiss, M.D. (2010). Cannabis and amyotrophic lateral sclerosis: hypothetical and practical applications, and a call for clinical trials. *American Journal of Hospice & Palliative Medicine*, 27(5), 347-356. Retrieved from <http://journals.sagepub.com/doi/pdf/10.1177/1049909110369531>. Castelli, M.P., Madeddu, C., Casti, A., Casu, A., Casti, P., Scherma, M., Fattore, L., Fadda, P., and Ennas, M.G. (2014). Δ 9-Tetrahydrocannabinol Prevents Methamphetamine-Induced Neurotoxicity. *PLoS ONE*, 9(5), e98079. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4028295/>. Chen, J., Lee, C.T., Errico, S., Deng, X., Cadet, J.L., and Freed, W.J. (2005). Protective effects of Δ 9-tetrahydrocannabinol against N-methyl-D-aspartate-induced AF5 cell death. *Brain Research. Molecular Brain Research*, 134(2), 215–225. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1824211/>. Croxford, J.L., Pryce, G., Jackson, S.J., Ledent, C., Giovannoni, G., Pertwee, R.G., Yamamura, T., and Baker, D. (2008, January). Cannabinoid-mediated neuroprotection, not immunosuppression, may be more relevant to multiple sclerosis. *Journal of Neuroimmunology*, 193(1-2), 120-9. Retrieved from [http://www.jni-journal.com/article/S0165-5728\(07\)00396-7/fulltext](http://www.jni-journal.com/article/S0165-5728(07)00396-7/fulltext). da Silva, V.K., de Freitas, B.S., da Silva Dornelles, A., Nery, L.R., Falavigna, L., Ferreira, R.D., Bogo, M.R., Hallak, J.E., Zuardi, A.W., Crippa, J.A., and Schroder, N. (2014, February). Cannabidiol normalizes caspase 3, synaptophysin, and mitochondrial fission protein DNMI1 expression levels in rats with brain iron overload: implications for neuroprotection. *Molecular Neurobiology*, 49(1), 222-33. Retrieved from <http://link.springer.com/article/10.1007%2Fs12035-013-8514-7>. de Lago, E., Moreno-Martet, M., Cabranes, A., Ramos, J.A., and Fernandez-Ruiz, J. (2012, June). Cannabinoids ameliorate disease progression in a model of multiple sclerosis in mice, acting preferentially through CB1 receptor-mediated anti-inflammatory effects. *Neuropharmacology*, 62(7), 2299-308. Retrieved from <http://www.sciencedirect.com/science/article/pii/S0028390812000500>. Devinsky, O., Cilio, M.R., Cross, H., Fernandez-Ruiz, J., French, J., Hill, C., Katz, R., Di Marzo, V., Jutras-Aswad, D., Notcutt, W.G., Martinez-Orgado, J., Robson, P.J., Rohrback, B.G., Thiele, E., Whalley, B., and Friedman, D. (2014, June). Cannabidiol: pharmacology and potential therapeutic role in epilepsy and other neuropsychiatric disorders. *Epilepsia*, 55(6), 791-802. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4707667/>. Dirikoc, S., Priola, S.A., Marella, M., Zsurger, N., and Chabry, J. (2007, September 5). Nonpsychoactive cannabidiol prevents prion accumulation and protects neurons against prion toxicity. *Journal of Neuroscience*, 27(36), 9537-44. Retrieved from <http://www.jneurosci.org/content/27/36/9537.long>. Disorder Index. (n.d.). *National Institute of Neurological Disorders and Stroke*. Retrieved from http://www.ninds.nih.gov/disorders/disorder_index.htm#A. England, T.J., Hind, W.H., Rasid, N.A., and O'Sullivan, S.E. (2015, March). Cannabinoids in experimental stroke: a systematic review and meta-analysis. *Journal of Cerebral Blood Flow and Metabolism*, 35(3), 348-58. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4348386/>. Fernandez-Ruiz, J., Sagredo, O., Pazos, M.R., Garcia, C., Pertwee, R., Mechoulam, R., and Martinez-Orgado, J. (2013, February). Cannabidiol for neurodegenerative disorders: important new clinical applications for this phytocannabinoid? *British Journal of Clinical Pharmacology*, 75(2), 323-33. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3579248/>. Fernández-Ruiz, J., Romero, J., Velasco, G., Tolon, R.M., Ramos, J.A., and Guzman, M. (2007, January). Cannabinoid CB2 receptor: a new

target for controlling neural cell survival. *Trends in Pharmaceutical Sciences*, 28(1), 39-45.

Retrieved from

[http://www.cell.com/trends/pharmacological-sciences/fulltext/S0165-6147\(06\)00267-7](http://www.cell.com/trends/pharmacological-sciences/fulltext/S0165-6147(06)00267-7).

Fernández-Ruiz, J., Moro, M. A., & Martínez-Orgado, J. (2015). Cannabinoids in Neurodegenerative Disorders and Stroke/Brain Trauma: From Preclinical Models to Clinical Applications.

Neurotherapeutics, 12(4), 793–806. Retrieved from

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4604192/>. Fernández-Ruiz, J., Moreno-Martet, M.,

Rodríguez-Cueto, C., Palomo-Garo, C., Gómez-Cañas, M., Valdeolivas, S., Guaza, C., Romero, J.,

Guzman, M., Mechoulam, R., and Ramos, J. A. (2011). Prospects for cannabinoid therapies in basal

ganglia disorders. *British Journal of Pharmacology*, 163(7), 1365–1378. Retrieved from

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3165947/>. Fishbein, M., Gov, S., Assaf, F., Gafni,

M., Keren, O., and Sarne, Y. (2012, September). Long-term behavioral and biochemical effects of an

ultra-low dose of Δ 9-tetrahydrocannabinol (THC): neuroprotection and ERK signaling.

Experimental Brain Research, 221(4), 437-48. Retrieved from

<http://link.springer.com/article/10.1007%2Fs00221-012-3186-5>. Garcia-Arencibia, M., Garcia, C., and

Fernandez-Ruiz, J. (2009, December). Cannabinoids and Parkinson's disease. *CNS & Neurological*

Disorders Drug Targets, 8(6), 432-9. Retrieved from <http://www.eurekaselect.com/93569/article>.

Greco, R., Mangione, A.S., Sandrini, G., Nappi, G. and Tassorelli, C. (2014, March). Activation of CB2

receptors as a potential therapeutic target for migraine: evaluation in an animal model. *The Journal*

of Headache and Pain, 15, 14. Retrieved from

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3995520/>. Hamelink, C., Hampson, A., Wink, D.A.,

Eiden, L.E., and Eskay, R.L. (2005). Comparison of Cannabidiol, Antioxidants, and Diuretics in

Reversing Binge Ethanol-Induced Neurotoxicity. *The Journal of Pharmacology and Experimental*

Therapeutics, 314(2), 780–788. Retrieved from

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4183207/>. Hampson, A.J., Grimaldi, M., Lolic, M.,

Wink, D., Rosenthal, R., and Axelrod, J. (2000). Neuroprotective antioxidants from marijuana. *Annals*

of the New York Academy of Sciences, 899,274-82. Retrieved from

<http://onlinelibrary.wiley.com/doi/10.1111/j.1749-6632.2000.tb06193.x/full>. Heutink, M., Post,

M.W., Wollaars, M.M., and van Asbeck, F.W. (2011). Chronic spinal cord injury pain: pharmacological

and non-pharmacological treatments and treatment effectiveness. *Disability and Rehabilitation*,

33(5), 433-40. Retrieved from

<http://www.tandfonline.com/doi/full/10.3109/09638288.2010.498557?needAccess=true>. Hussain,

S.A., Zhou, R., Jacobson, C., Weng, J., Cheng, E., Lay, J., Hung, P., Lerner, J.T., and Sankar, R. (2015,

June). Perceived efficacy of cannabidiol-enriched cannabis extracts for treatment of pediatric

epilepsy: A potential role for infantile spasms and Lennox-Gastaut syndrome. *Epilepsy & Behavior*,

47, 138-41. Retrieved from

[http://www.epilepsybehavior.com/article/S1525-5050\(15\)00157-2/fulltext](http://www.epilepsybehavior.com/article/S1525-5050(15)00157-2/fulltext). Iuvone, T., Esposito, G.,

Esposito, R., Santamaria, R., Di Rosa, M., and Izzo, A.A. (2004, April). Neuroprotective effect of

cannabidiol, a non-psychoactive component from Cannabis sativa, on beta-amyloid-induced toxicity

in PC12 cells. *Journal of Neurochemistry*, 89(1), 134-41. Retrieved from

<http://onlinelibrary.wiley.com/doi/10.1111/j.1471-4159.2003.02327.x/full>. Jensen, B., Chen, J., Furnish,

T., and Wallace, M. (2015, October). Medical Marijuana and Chronic Pain: a Review of Basic Science

and Clinical Evidence. *Current Pain and Headache Reports*, 19(10), 524. Retrieved from

<http://link.springer.com/article/10.1007%2Fs11916-015-0524-x>. Jiang, W., Zhang, Y., Xiao, L., Van Cleemput, J., Ji, S.P., Bai, G., and Zhang, X. (2005). Cannabinoids promote embryonic and adult hippocampus neurogenesis and produce anxiolytic- and antidepressant-like effects. *Journal of Clinical Investigation*, 115(11), 3104–3116. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1253627/>. Kim, S.H., Won, S.J., Mao, X.O., Jin, K., and Greenberg, D.A. (2006, March). Molecular mechanisms of cannabinoid protection from neuronal excitotoxicity. *Molecular Pharmacology*, 69(30), 691-6. Retrieved from <http://molpharm.aspetjournals.org/content/69/3/691.long>. Kubajewska, I., and Constantinescu, C.S. (2010, August). Cannabinoids and experimental models of multiple sclerosis. *Immunobiology*, 215(8), 647-57. Retrieved from <http://www.sciencedirect.com/science/article/pii/S0171298509001442>. Kwiatkoski, M., Guimaraes, F.S., and Del-Bel, E. (2012, April). Cannabidiol-treated rats exhibited higher motor score after cryogenic spinal cord injury. *Neurotoxicity Research*, 21(3), 271-80. Retrieved from <http://link.springer.com/article/10.1007%2Fs12640-011-9273-8>. Lafuente, H., Alvarez, F.J., Pazos, M.R., Alvarez, A., Rey-Santano, M.C., Mielgo, V., Murgia-Esteve, X., Hilario, E., and Martinez-Orgado, J. (2011, September). Cannabidiol reduces brain damage and improves functional recovery after acute hypoxia-ischemia in newborn pigs. *Pediatric Research*, 70(3), 272-7. Retrieved from <http://www.nature.com/pr/journal/v70/n3/full/pr2011171a.html>. Lastres-Becker, I., and Fernandez-Ruiz, J. (2006). An overview of Parkinson's disease and the cannabinoid system and possible benefits of cannabinoid-based treatments. *Current Medicinal Chemistry*, 13(30) 3705-18. Retrieved from <http://www.eurekaselect.com/58342/article>. Latini, L., Bisicchia, E., Sasso, V., Chiurciu, V., Cavallucci, V., Molinari, M., Maccarrone, M., and Viscomi, M.T. (2014, September 4). Cannabinoid CB2 receptor (CB2R) stimulation delays rubrospinal mitochondrial-dependent degeneration and improves functional recovery after spinal cord hemisection by ERK1/2 inactivation. *Cell Death & Disease*, e1404. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4540196/>. López Rodríguez, A.B., Siopi, E., Finn, D.P., Marchand-Leroux, C., Garcia-Segura, L.M., Jafarian-Tehrani, M.H., and Viveros, M.P. (2013). CB1 and CB2 cannabinoid receptor antagonists prevent minocycline-induced neuroprotection following traumatic brain injury in mice. *Cerebral Cortex*. Retrieved from <http://cercor.oxfordjournals.org/content/early/2013/08/19/cercor.bht202.abstract>. Lotan, I., Treves, T., Roditi, Y., and Djaldetti, R. (2014, March/April). Cannabis (medical marijuana) treatment for motor and nonmotor symptoms of Parkinson disease: an open-label observational study. *Clinical Neuropharmacology*, 37(2), 41-44. Retrieved from <http://journals.lww.com/clinicalneuropharm/pages/articleviewer.aspx?year=2014&issue=03000&article=00001&type=abstract>. Marsicano, G., Goodenough, S., Monory, K., Hermann, H., Eder, M., Cannich, A., Azad, S.C., Cascio, M.G., Gutiérrez, S.O., van der Stelt, M., López-Rodríguez, M.L., Casanova, E., Schütz, G., Zieglängsberger, W., Di Marzo, V., Behl, C., and Lutz, B. (2003, October 3). CB1 Cannabinoid Receptors and On-Demand Defense Against Excitotoxicity. *Science*, 302(5642), 84-8. Retrieved from <http://science.sciencemag.org/content/302/5642/84/tab-pdf>. Massi, P., Vaccani, A., Ceruti, S., Colombo, A., Abbracchio, M.P., and Parolaro, D. (2004, March). Antitumor effects of cannabidiol, a nonpsychoactive cannabinoid, on human glioma cell lines. *The Journal of Pharmacology and Experimental Therapeutics*, 308(3), 838-45. Retrieved from <http://jpet.aspetjournals.org/content/308/3/838.long>. McDonough, P., McKenna, J.P., McCreary, C., and Downer, E.J. (2014, October). Neuropathic orofacial pain: cannabinoids as a therapeutic avenue.

The International Journal of Biochemistry & Cell Biology, 55, 72-8. Retrieved from <http://www.sciencedirect.com/science/article/pii/S1357272514002581>. Mechoulam, R., and Hanus, L. (2001). The cannabinoids: An overview. Therapeutic implications in vomiting and nausea after cancer chemotherapy, in appetite promotion, in multiple sclerosis and in neuroprotection. *Pain Research and Management*, 6(2), 67-73. Retrieved from <http://downloads.hindawi.com/journals/prm/2001/183057.pdf>. More, S.V., and Choi, D.K. (2015, April). Promising cannabinoid-based therapies for Parkinson's disease: motor symptoms to neuroprotection. *Molecular Neurodegeneration*, 10, 17. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4404240/>. Muller-Vahl, K.R., Schneider, U., Koblenz, A., Jobges, M., Kolbe, H., Daldrup, T., and Emrich, H.M. (2002, March). Treatment of Tourette's syndrome with Delta 9-tetrahydrocannabinol (THC): a randomized crossover trial. *Pharmacopsychiatry*, 35(2), 57-61. Retrieved from <https://www.thieme-connect.com/DOI/DOI?10.1055/s-2002-25028>. Muller-Vahl, K.R. (2003). Cannabinoids reduce symptoms of Tourette's syndrome. *Expert Opinion on Pharmacotherapy*, 4(10), 1717-1725. Retrieved from <http://www.tandfonline.com/doi/pdf/10.1517/14656566.4.10.1717?needAccess=true>. Muller-Vahl, K.R. (2013). Treatment of Tourette syndrome with cannabinoids. *Behavioral Neurology*, 27(1), 119-24. Retrieved from <http://downloads.hindawi.com/journals/bn/2013/294264.pdf>. Neurological Disorders. (n.d.). *UCSF Medical Center*. Retrieved from http://www.ucsfhealth.org/conditions/neurological_disorders/. Novotna, A., Mares, J., Ratcliffe, S., Novakova, I., Vachova, M., Zapletalova, O., Gasperini, C., Pozzilli, C., Cefaro, L., Comi, G., Rossi, P., Ambler, Z., Stelmasiak, Z., Erdmann, A., Montalban, X., Klimek, A., Davies, P. (2011, September). A randomized double-blind-placebo-controlled, parallel-group, enriched-design study of nabiximols* (Sativex®), as add-on therapy, in subjects with refractory spasticity caused by multiple sclerosis. *European Journal of Neurology*, 18(9), 1122-31. Retrieved from <http://onlinelibrary.wiley.com/wo/jdoi/10.1111/j.1468-1331.2010.03328.x/full>. Pope, C., Mechoulam, R., and Parsons, L. (2010). Endocannabinoid Signalling in Neurotoxicity and Neuroprotection. *Neurotoxicology*, 31(5), 562-571. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2891218/>. Pryce, G., Ahmed, Z., Hankey, D.J., Jackson, S.J., Croxford, J.L. Pocock, J.M., Ledent, C., Petzold, A., Thompson, A.J., Giovannoni, G., Cuzner, M.L., and Baker, D. (2003, October). Cannabinoids inhibit neurodegeneration in models of multiple sclerosis. *Brain*, 126(Pt 10), 2191-202. Retrieved from <https://academic.oup.com/brain/article-lookup/doi/10.1093/brain/awg224>. Raman, C., McAllister, S.D., Rizvi, G., Patel, S.G., Moore, D.H., and Abood, M.E. (2004). Amyotrophic lateral sclerosis: delayed disease progression in mice by treatment with a cannabinoid. *Amyotrophic Lateral Sclerosis & Other Motor Neuron Disorders*, 5(1), 33-30. Retrieved from <http://www.tandfonline.com/doi/abs/10.1080/14660820310016813>. Rosenberg, E.C., Tsien, R.W., Whalley, B.J., and Devinsky, O. (2015, August 18). Cannabinoids and Epilepsy. *Neurotherapeutics*, Epub ahead of print. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/26282273>. Sagredo, O., Garcia-Arencibia, M., de Lago, E., Finetti, S., Decio, A., and Fernandez-Ruiz, J. (2007, August). Cannabinoids and Neuroprotection in Basal Ganglia Disorders. *Molecular Neurobiology*, 36(1), 82-91. Retrieved from <http://link.springer.com/article/10.1007%2Fs12035-007-0004-3>. Scotter, E.L., Abood, M.E., and Glass, M. (2010). The endocannabinoid system as a target for the treatment of

neurodegenerative disease. *British Journal of Pharmacology*, 160(3), 480–498. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2931550/>. Szaflarski, J.P., and Bebin, E.M. (2014, December). Cannabis, cannabidiol, and epilepsy—from receptors to clinical response. *Epilepsy & Behavior*, 41, 277-82. Retrieved from [http://www.epilepsybehavior.com/article/S1525-5050\(14\)00413-2/fulltext](http://www.epilepsybehavior.com/article/S1525-5050(14)00413-2/fulltext). van der Stelt, M., Veldhuis, W.B., Bar, P.R., Veldink, G.A., Vliegthart, J.F., and Nicolay, K. (2001, September 1). Neuroprotection by Δ 9-Tetrahydrocannabinol, the Main Active Compound in Marijuana, against Ouabain-Induced In Vivo Excitotoxicity. *The Journal of Neuroscience*, 21(17), 6475-9. Retrieved from <http://www.jneurosci.org/content/21/17/6475.long>. Weydt, P., Hong, S., Witting, A., Moller, T., Stella, N., and Kliot, M. (2005). Cannabinol delays symptom onset in SOD1 transgenic mice without affecting survival. *Amyotrophic Lateral Sclerosis & Other Motor Neuron Disorders*, 6(3), 182-184. Retrieved from <http://www.tandfonline.com/doi/abs/10.1080/14660820510030149?journalCode=iafd19>. Wilsey, B., Marcotte, T.D., Deutsch, R., Gouaux, B., Sakai, S., and Donaghe, H. (2003, February). Low dose vaporized cannabis significantly improves neuropathic pain. *Journal of Pain*, 14(2), 136-148. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3566631/>. Witting, A., Chen, L., Cudaback, E., Straiker, A., Walter, L., Rickman, B., Moller, T., Brosnan, C., and Stella, N. (2006, April 18). Experimental autoimmune encephalomyelitis disrupts endocannabinoid-mediated neuroprotection. *PNAS*, 103(16), 6362-7. Retrieved from <http://www.pnas.org/content/103/16/6362.full>. Wolf, S.A., Bick-Sander, A., Fabel, K., Leal-Galicia, P., Tauber, S., Ramirez-Rodriguez, G., Muller, A., Melnik, A., Waltinger, T.P., Ullrich, O., and Kempermann, G. (2010). Cannabinoid receptor CBR1 mediates baseline and activity-induced survival of new neurons in adult hippocampal neurogenesis. *Cell Communication and Signaling : CCS*, 8, 12. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898685/>. Zogopoulos, P., Vasileiou, I., Patsouris, E., and Theocharis, S. (2013, April). The neuroprotective role of endocannabinoids against chemical-induced injury and other adverse effects. *Journal of Applied Toxicology*, 33(4), 246-64. Retrieved from <http://onlinelibrary.wiley.com/doi/10.1002/jat.2828/full>. Zuardi, A.W. (2008, September). Cannabidiol: from an inactive cannabinoid to a drug with wide spectrum of action. *Revista Brasileira De Psiquiatria*, 30(3), 271-80. Retrieved from [http://www.scielo.br/scielo.php?script=sci_arttext&pid=S1516-44462008000300015&lng=en&nrm=iso&tlng=en.\]\]>](http://www.scielo.br/scielo.php?script=sci_arttext&pid=S1516-44462008000300015&lng=en&nrm=iso&tlng=en.]]>)

Appendix A:

WHEREAS cannabis (marijuana) has been used as a medicine for at least 5,000 years and can be effective for serious medical conditions for which conventional medications fail to provide relief;

WHEREAS modern medical research has shown that cannabis can slow the progression of such serious diseases as Alzheimer's and Parkinson's and stop HIV and cancer cells from spreading; has both anti-inflammatory and pain-relieving properties; can alleviate the symptoms of epilepsy, PTSD and multiple sclerosis; is useful in the treatment of depression, anxiety and other mental disorders; and can help reverse neurological damage from brain injuries and stroke;

WHEREAS the World Health Organization has acknowledged the therapeutic effects of cannabinoids, the primary active compounds found in cannabis, including as an anti-depressant, appetite stimulant, anticonvulsant and anti-spasmodic, and identified cannabinoids as beneficial in the treatment of asthma, glaucoma, and nausea and vomiting related to illnesses such as cancer and AIDS;

WHEREAS the American Medical Association has called for the review of the classification of cannabis as a Schedule I controlled substance to allow for clinical research and the development of cannabinoid-based medicines;

WHEREAS the National Cancer Institute has concluded that cannabis has antiemetic effects and is beneficial for appetite stimulation, pain relief, and improved sleep among cancer patients;

WHEREAS the American Herbal Pharmacopoeia and the American Herbal Products Association have developed qualitative standards for the use of cannabis as a botanical medicine;

WHEREAS the U.S. Supreme Court has long noted that states may operate as "laboratories of democracy" in the development of innovative public policies;

WHEREAS twenty-eight states and the District of Columbia have enacted laws that allow for the medical use of cannabis;

WHEREAS seventeen additional states have enacted laws authorizing the medical use of therapeutic compounds extracted from the cannabis plant;

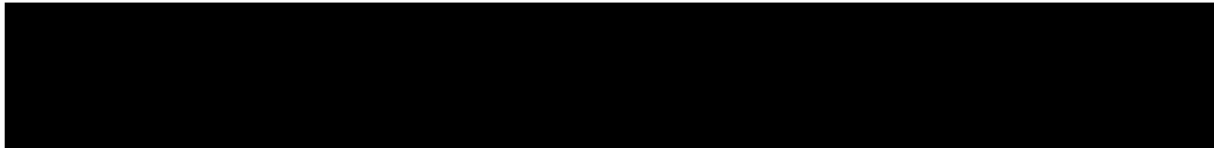
WHEREAS more than 17 years of state-level experimentation provides a guide for state and federal law and policy related to the medical use of cannabis;

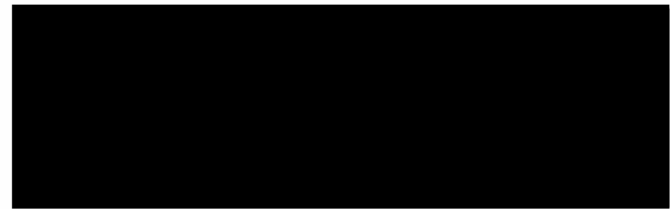
WHEREAS accredited educational curricula concerning the medical use of cannabis have been established that meets Continuing Medical Education requirements for practicing physicians;

WHEREAS Congress has prohibited the federal Department of Justice from using funds to interfere with and prosecute those acting in compliance with their state medical cannabis laws, and the Department of Justice has issued guidance to U.S. Attorneys indicating that enforcement of the



Controlled Substances Act is not a priority when individual patients and their care providers are in compliance with state law, and that federal prosecutors should defer to state and local enforcement so long as a viable state regulatory scheme is in place;





Nystagmus (congenital or acquired)

Petition to add Nystagmus (Congenital or Acquired) as a qualifying medical condition for the use of medical cannabis in the state of New Mexico

Introduction/Personal Narrative:

My name is [REDACTED] and I am writing this petition to the New Mexico Medical Cannabis Advisory Board as a concerned citizen and a Nystagmus patient. I have Congenital Nystagmus (often referred to as Infantile Nystagmus Syndrome). I also have Retinopathy of Prematurity as my underlining condition with Nystagmus. These are a result of my premature birth. I had cryogenic surgery to freeze my retinas and was on oxygen until the age of two. This halt in the normal development of my visual system is what's thought to have caused my Nystagmus. I, like many others with this condition have poor vision. I have had 4 eye muscle surgeries to try to ease the repetitive eye oscillations.

My first surgery in 2011 was to move my eyes up to center my focal point where my eyes shake the least. My second eye muscle surgery the following year was a "Horizontal Tenotomy". This surgery is done by detaching and reattaching the muscles on both sides of the eyes. The goal is that it should "reset" the brain and ease the muscle spasms in the eyes. Unfortunately this surgery didn't improve my ocular muscle spasms. My third and fourth eye surgeries were to revise a 15 degree tilt in my right eye. All of these surgeries resulted in little success. I am no longer eligible for any more eye muscle surgeries due to the risk of blood restriction to my eyes.

My eye conditions make it difficult for me to focus on anything. I can't drive a car due to my eye conditions. I have trouble waking down stairs. It's sometimes difficult for me to have my own independence. Even helping my son with his homework is a challenge for me because I struggle to read. Something as simple as reading a sentence can be difficult because of my eyes constantly jerking and refocusing. Even writing this paper has been a struggle as I continuously lose my place, fix typing errors and try to proof-read. Constant strain to keep my eyes steady causes frequent headaches. This is quite common in the Nystagmus community. Nystagmus also effects my balance and depth perception. As you can imagine, I'm very accident prone.

I, with the help of Dr. Louis Dell'Osso have written a detailed explanation of Nystagmus, it's effects and how medical marijuana/cannabis could be beneficial. Dr. Dell'Osso (Cleveland, OH) has spent the past 40 plus years researching nystagmus. Many of his research papers can be found on: <http://omlab.org/OMLAB.html>

Dr. Dell'Osso also emailed me one of his published research papers supporting the benefits of cannabis in a patient with Nystagmus. A printed copy of his research publication can be found within this petition. An additional supporting research paper from a separate source is also attached.

Understanding Nystagmus (congenital or acquired):

Nystagmus is a neurological eye movement disorder that causes reduced or limited vision. Nystagmus causes rapid involuntary movements in one or both eyes. These movements can be horizontal, vertical, diagonal/circular/elliptical, rotary, see-saw or a combination of those movements. These oscillating movements make it difficult to see detail due to the image not being able to remain on the fovea of the eye for an interval of time long enough for good vision. Other associated conditions resulting from nystagmus are light sensitivity, reduced depth perception, impaired balance, and coordination issues.

A person with nystagmus may hold their head in an unusual position to place their eyes at a lateral gaze angle. This is called a "null point". A null point is the area in the visual field of the eye in which the shaking is damped and foveation is better achieved. Head turns can cause strain and neck pain due to the abnormal head posture. Nystagmus oscillations have been known to increase due to nervousness, anxiety, fatigue or sickness. Excessive strain to focus can cause headaches and fatigue as well.

Infantile Nystagmus Syndrome (INS, aka, congenital nystagmus) is present at or around the time of birth. It can be a stand alone condition or be accompanied by other conditions such as (but not limited to), macular degeneration, ocular albinism, or other retinal diseases/disorders.

The initial cause of INS is hypothesized to be oscillation in the smooth pursuit portion of the ocular motor system and there is currently no cure. In recent years, eye muscle surgeries, Botox injections into the extraocular eye muscles and medicines like baclofen have been prescribed with limited success.

Acquired nystagmus is nystagmus that is acquired later in life as a result of a stroke, inner ear disorder, brain tumor, brain injury or spinal injury. Acquired nystagmus can cause severe dizziness and vertigo in the patient along with the aforementioned depth perception, balance and coordination issues.

Why I believe medical marijuana/cannabis would benefit a nystagmus patient:

Medical marijuana/cannabis has the potential to significantly improve a nystagmus patient's quality of life by slowing down the involuntary oscillating eye movements. Slowed down movements allow better fixation between oscillation cycles. The image is able to stay on the fovea longer in order to maintain better focus. Slowing down these repetitive involuntary movements could improve overall visual acuity. This could also reduce neck strain, fatigue and headaches. Headaches and fatigue associated with nystagmus are caused by the constant strain in an effort to reduce eye movements. In nystagmus patients with anxiety, it can greatly improve quality of life by reducing the eye movements that are brought on by nervousness. In addition, medical marijuana/cannabis could help patients who have acquired nystagmus by easing their nausea symptoms from vertigo.

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Suppression of pendular nystagmus by smoking cannabis in a patient with multiple sclerosis

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Outline

References

To the Editor:

I read with interest the article by Schon et al. 1 In the early 1960s, I observed an individual with congenital nystagmus whose nystagmus damped after smoking cannabis; the damping was obvious and it was evident to others. Unfortunately, the setting precluded ocular motor recording and the date preceded the development of accurate techniques to accomplish such recording. However, the subject was able to read small print on a poster across the room on the wall opposite to where he was seated, which was not possible before smoking the cannabis. Over the ensuing years, that observation has been supported by unsolicited, first-hand reports of similar effects by several patients with congenital nystagmus referred to our laboratory for ocular motor recordings; the most recent report came from a participant of the inaugural meeting of the American Nystagmus Network, held in Cleveland, OH, July 30-31, 1999.

Research into the therapeutic benefits of cannabis is discouraged in the United States so I have not properly recorded or studied these effects in a controlled setting. Nevertheless, the damping effect was great enough to be observed visually and to increase visual acuity. To a lesser extent, alcohol has a similar damping effect on congenital nystagmus (personal observation).

Schon et al. noted that only by smoking the cannabis was the pendular nystagmus of MS damped; that, itself, is worthy of further study. Even more interesting is that cannabis damped an acquired form of nystagmus. Although the beneficial effects of cannabis on congenital nystagmus have been recognized for many years by a small number of people with congenital nystagmus, this latter observation raises the possibility that this drug can be used in different types of acquired nystagmus (horizontal and vertical) where damping the oscillations can have the beneficial effect of reducing or eliminating the debilitating effects of oscillopsia. I encourage these authors to continue their work in this area.

Louis F. Dell'Osso PhD

References

1. Schon F, Hart PE, Hodgson TL, et al. Suppression of pendular nystagmus by smoking cannabis in a patient with multiple sclerosis. *Neurology* 1999; 53:2209-2210. [Ovid Full Text](#)
2. Boecker H, Wills AJ, Ceballos-Baumann A, et al. The effect of ethanol on alcohol-responsive essential tremor: a positron emission tomography study. *Ann Neurol* 1996; 39:650-658.
3. Mossman SS, Bronstein AM, Rudge P, Gretsya MA. Acquired pendular nystagmus suppressed by alcohol. *Neuro-ophthalmology* 1993; 13:99-106.



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↓ Full text

Reduction of congenital nystagmus in a patient after smoking cannabis.

Pradeep A, et al. *Strabismus*. 2008 Jan-Mar.

Authors

Pradeep A¹, Thomas S, Roberts EO, Proudlock FA, Gottlob I.

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Citation

Strabismus. 2008 Jan-Mar;16(1):29-32. doi: 10.1080/09273970701821063.

Abstract

INTRODUCTION: Smoking cannabis has been described to reduce acquired pendular nystagmus in MS, but its effect on congenital nystagmus is not known.

PURPOSE: To report the effect of smoking cannabis in a case of congenital nystagmus.

METHODS: A 19-year-old male with congenital horizontal nystagmus presented to the clinic after smoking 10 mg of cannabis. He claimed that the main reason for smoking cannabis was to improve his vision. At the next clinic appointment, he had not smoked cannabis for 3 weeks. Full ophthalmologic examination and eye movement recordings were performed at each visit.

RESULTS: Visual acuity improved by 3 logMar lines in the left eye and by 2 logMar lines in the right eye after smoking cannabis. The nystagmus intensities were reduced by 30% in primary position and 44%, 11%, 10% and 40% at 20-degree eccentricity to the right, left, elevation and depression, respectively, after smoking cannabis.

CONCLUSION: Cannabis may be beneficial in the treatment of congenital idiopathic nystagmus (CIN). Further research to clarify the safety and efficacy of cannabis in patients with CIN, administered for example by capsules or spray, would be important.

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Full text

 Full text at journal site



Prepared for: [REDACTED] / CM6031507

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Nystagmus

Nystagmus is abnormal movement of one or both eyes. This sheet tells you about this condition. It also tells you about diagnosis and treatment options.

Eye Movements with Nystagmus

With nystagmus, eye movements are quick and repetitive. They can be continuous (all the time) or paroxysmal (happening "on and off"). They are always involuntary (not controlled by the person). The direction of eye movements can be:

- Horizontal (side to side)
- Vertical (up and down)
- Rotary (around in a circle)
- See-saw (one eye goes up while the other goes down)
- Any combination of the above

Because of these abnormal movements, the eyes can't focus well. This impairs vision. To see better, a person may hold the head in an unusual position. This is done to access the "null point." This is an angle of vision that helps limit abnormal movement of the eyes. A person may also nod the head to reduce eye movement.



A person with nystagmus may hold his or her head in an unusual position to access the "null point." This helps reduce abnormal eye movement and improve vision.

Types and Causes of Nystagmus

There are two main types of nystagmus. Each type can have one or more causes. But the exact cause is often unknown. Most cases of nystagmus are permanent.

- **Congenital (or infantile) nystagmus.** This is the most common type. The child is born with it. Symptoms appear soon after birth. This type has two main causes: sensory problems and neurologic problems. It can also be linked to other health conditions.
 - **Sensory problems.** Vision problems such as cataracts (a clouded lens of the eye) or glaucoma (increased pressure within the eye) may be a cause of nystagmus. Another possible cause is a problem with the retina (the light-sensing layer of nerves that lines the back of the eye) or the optic nerve (the nerve that connects the eye to the brain).
 - **Neurologic problems.** Problems in the brain can cause nystagmus. This is because the brain and the eyes must work closely together to make good vision possible.
 - **Other conditions.** Albinism (lack of pigment in the skin and eyes) and aniridia (absence of the iris of the eye) are linked to nystagmus.
- **Acquired nystagmus.** This type is not present at birth. It has several possible causes. These include:
 - Alcohol use
 - Use of certain medications (most often anti-seizure medications)
 - Inner ear disorders, such as Meniere's disease
 - Disease in the brain, such as multiple sclerosis or a brain tumor
 - Stroke

- Injury to the head

Symptoms of Nystagmus

Symptoms depend on the type of nystagmus. With the congenital type, impaired vision may occur. But the eye movement may not be bothersome. With the acquired type, the eye movement may be more pronounced and bothersome. Blurry vision may occur. Also, depth perception may be affected. This can impair balance and coordination. It can also cause dizziness.

Diagnosis of Nystagmus

The doctor or eye care provider will take a health history. A physical exam, including an eye exam, will be done. And certain tests may be done. These include:

- **CT (computed tomography) or MRI (magnetic resonance imaging) scan of the head.** These imaging tests create pictures of the brain. They can show any swelling, stroke, or tumors that may be causing nystagmus.
- **Electro-oculography (EOG).** This tests the function of the retina. It does this by recording movement of the eyes.
- **Vestibular testing.** This checks for problems in part of your inner ear (the vestibule). This part of the ear helps control eye movement.
- **Lab tests.** These check for infections, an imbalance of chemicals in the body, and tumors.

Treatment of Nystagmus

The American Academy of Pediatrics suggests that children with nystagmus should preferably be managed by a pediatric ophthalmologist. This is a doctor with special training in the diagnosis and treatment of children with eye disorders. In most cases, treatment helps reduce symptoms but does not eliminate them. Treatment depends on the cause of the nystagmus. There are five main treatment options:

- **Treatment of the underlying cause.** If there seems to be a clear cause, it is addressed. For instance, a cataract is treated or a tumor may be removed. If a medication is the cause, it may be stopped or replaced. Unlike with other causes, when these underlying causes are treated, nystagmus may be cured.
- **Treatment of related vision problems.** For instance, contact lenses or eyeglasses can help correct problems such as near- or farsightedness. Special prisms may be used to adjust the gaze. This helps reduce symptoms.
- **Medications.** These can help reduce symptoms, such as eye movement and vision loss.
- **Botulinum injections.** These weaken certain muscles within the eye. This helps reduce symptoms.
- **Surgery.** This procedure alters the position of the muscles that move the eyes. It does not cure nystagmus, but may reduce the amount the head needs to turn to see best.

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A Petition to Add Alzheimer's Disease to the List of Qualifying Conditions in New Mexico



A Petition to Add Alzheimer's Disease to the List of Qualifying Conditions in New Mexico

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“Penalties against possession of a drug should not be more damaging to an individual than the use of the drug itself, and where they are, they should be changed.” – Jimmy Carter

If this is true, following to the next logical step; isn't the reverse also true? When prohibition is more harmful to a person than the actual substance, then it should be changed. There are two major principles that guide practitioners: beneficence and non-maleficence. Broken down into the simplest terms, this means that as practitioners we have a moral obligation to do what is right and an imperative to do no harm. In the case of medical cannabis and degenerative neurological disorders, preventing these patients from being able to use a medication that is shown by research to improve their quality of life is violating our guiding principles. We have a moral obligation to allow these people access to their medication. By preventing access to this medication, we cause harm not only to the patient, but also to the values that we as New Mexicans hold dear.

My name is [REDACTED]. As a registered nurse I have spent 8 years of my career in the cardiovascular intensive care unit and am currently enrolled in a nurse practitioner program through Simmons University in Boston. My interest in this issue is both professional and personal. As a professional I have seen the ravages of Alzheimer's disease on my patients. Personally, I have seen friends and family members struggle with not only Alzheimer's, but other various neurodegenerative diseases. Unfortunately, the side effects of the treatments can be as devastating for these patients as the disease states themselves. I am petitioning the advisory board to either A) Add Alzheimer's disease as a qualifying condition in New Mexico or B) Add a sub-category of neurodegenerative diseases (an umbrella covering Alzheimer's disease, ALS, Parkinson's disease, MS, and more) to the list of qualifying conditions. This petition is also

supported by the findings of the Senate Memorial 105 Task force recommendations from October 2018.

Alzheimer's disease is a neurodegenerative disorder characterized by cognitive impairment caused by amyloid plaques and tangles that develop in the brain. It is a debilitating condition that negatively impacts not only the lives of the patients who are affected by it, but the lives of their families and friends as well. On top of disrupting their day to day lives, this disease also produces agitation, depression, and anxiety.

The CDC (2018) noted:

An estimated 5 million Americans aged 65 years or older had Alzheimer's disease. This number may triple to as high as 13.8 million people by 2050. In 2010, the costs of treating Alzheimer's disease were projected to fall between \$159 and \$215 billion.

(<https://www.cdc.gov/aging/aginginfo/alzheimers.htm>)

In New Mexico there are more than 38,000 patients who are affected by this disease.

There is no currently accepted treatment to stop the progression of Alzheimer's disease; rather therapies focus on merely holding symptoms at bay while having the potential to cause side effects as debilitating as the disease. These medication side effects range from relatively benign (fatigue, nausea, vomiting) to debilitating (confusion, aggression, and hallucinations).

Alzheimer's disease is one of the few diseases that fall under the umbrella of neurodegenerative diseases that is not accepted as a qualifying condition for medical cannabis in New Mexico. A summary of the benefits medical cannabis could provide for patients suffering from Alzheimer's disease include:

- Blocking of the development of beta-amyloid plaques delaying the progression of the disease (Currais et al., 2016).

- Regulating a long list of physiologic processes including stress and emotions, digestion, nociception (i.e. pain) through the endocannabinoid systems distribution throughout the brain and spinal cord (Sinclair, 2016, p. 108).
- Neural protection and can help with sleep disturbance, pain, and depression associated with both Parkinson's disease and Alzheimer's disease (Babayeva et al., 2016).
- "Early intervention via the reduction of intraneuronal A β proteotoxicity may reduce AD disease initiation or progression" (Currais et al., 2016, p. 6-7).
- Protection "against long-term cognitive damage that is caused by neuroinflammation" (Fishbein-Kaminietsky et al., 2014, p. 1670).
- Preservation of memory and decreased learning impairment through combinations of THC and CBD.
- Helping control the agitation, depression, and anxiety that accompany the disease.
- Enhanced memory associated with the terpenoid pinene, while a combination of THC and CBD have been associated with decreased learning impairment and preserved memory in rats with Alzheimer's disease (Goldstein, 2016, p. 132-133).

These are just some of the benefits that medical cannabis can provide for these patients, with more therapeutic effects being discovered regularly.

As previously stated, the treatment for Alzheimer's disease does not stop or reverse progression of the disease but is rather focused on symptom management. Cannabis can help these patients to manage their symptoms with fewer side effects, leading to a better overall

quality of life. I am respectfully requesting the Medical Cannabis Advisory Board to approve Alzheimer's disease or a degenerative neurological disease category that is an umbrella containing Alzheimer's disease, Parkinson's disease, and Huntington's.



References

- Babayeva, M., Assefa, H., Basu, P., Chumki, S., & Loewy, Z. (2016). Marijuana Compounds: A Nonconventional Approach to *Parkinson's Disease* Therapy. *Parkinson's Disease*, 2016(1), 1-19. <https://doi.org/10.1155/2016/1279042>
- Currais, A., Quehenberger, O., Armando, A. M., Daugherty, D., Maher, P., & Schubert, D. (2016, June 23). Amyloid proteotoxicity initiates an inflammatory response blocked by cannabinoids. *npj Aging and Mechanisms of Disease*, 2(0), 1-8. <http://dx.doi.org/10.1038/npjamd.2016.12>
- Goldstein, B. (2016). Medical symptoms and conditions: Alzheimer's disease. In *Cannabis revealed: How the world's most misunderstood plant is healing everything from chronic pain to epilepsy* (pp. 115-240). San Bernadino, CA: Author.