How is Herpes Normally Treated?

Sometimes for your first outbreak you may be prescribed antiviral drugs from a sexual health clinic. These can shorten the length of the outbreak for 1-2 days if you start taking them as soon as symptoms appear. However, outbreaks do tend to settle by themselves. Recurrences tend to be milder, and some people never experience them.

Since there is no cure for herpes, you can only take measures to prevent passing the virus on and to help make things easier for yourself. This includes:

- Keeping the area clean and preventing blisters from becoming infected
- Pouring water over your genitals while you pee to ease the pain
- Avoiding tight clothing that may irritate blisters or sores
- Not touching the blisters or sores unless applying cream
- Avoiding sex until the sores have gone
- Always wearing a condom to prevent spreading the virus.

It is very unfortunate that there is no cure, but you can at least take measures to feel better. So, where could CBD come into this?

Could CBD Help to Cure Herpes?

Cannabidiol is one of the active compounds from the cannabis plant, known as a cannabinoid. This particular cannabinoid has been praised for several potential health benefits. It is able to interact positively with the endocannabinoid system (ECS), an innate system in our body that controls a number of functions from mood all the way to our immune system. By helping the ECS to work properly, CBD supplements could help us to stay fit and healthy.

When a herpes outbreak occurs, the virus is attacking cell tissues in the mucous membranes, causing pain, blisters and inflammation. This is caused in part by white blood cells rushing to the area to combat the attack. White blood cells are part of our immune system, and if CBD can really interact with our immune system through the ECS, then it could help this process to work more effectively.

What's more, CBD is actually a patented anti-inflammatory in the United States. This means that it could take down the inflammation in the area and lessen any pain caused as a result.

A few studies are being carried out into CBD as an anti-viral. While nothing is yet set in stone, some conclusions have been positive. For example, the Department of Biology at the NYU Cancer Institute published a study in 2010 which suggested phytocannabinoids (those found in plants) were able to help in the fight against persistent infections – like herpes. The study was done on in vitro and in vivo subjects,

though, meaning it might not be fully effective on adults; we will have to await more studies.

Whether it is able to “cure” herpes or not, it might be able to support your immune system and make you feel much better.

How Should I Take CBD for Herpes?

There are many different ways to take CBD. New products are popping up all the time, but which way is the best if you have herpes?

Well, topical treatments can be applied to the skin to soothe and ease discomfort. This is ideal during outbreaks, so grab a soothing CBD-infused cream to keep in your cupboard in case of emergency. During the rest of the time, however, you might choose to take CBD oils or capsules. As these are broken down in the blood and digestive system, you can allow the CBD to influence the whole body.

Final Thoughts on CBD and Herpes

While none of the studies so far are conclusive about CBD for herpes specifically, it is generally accepted nowadays that CBD is able to help you and your general health. As a result, there’s really no harm in giving CBD supplements a shot.

Cannabinoids and Viral Infections

Abstract

Exogenous cannabinoids or receptor antagonists may influence many cellular and systemic host responses. The anti-inflammatory activity of cannabinoids may compromise host inflammatory responses to acute viral infections, but may be beneficial in persistent infections. In neurons, where innate antiviral/pro-resolution responses include the activation of NOS-1, inhibition of Ca^{2+} activity by cannabinoids, increased viral replication and disease. This review examines the effect(s) of cannabinoids and their antagonists in viral infections.

Keywords: pathogens, virus infection, immunomodulation, inflammation

1. Introduction

Both endogenous and exogenous cannabinoids can influence the course of infections *in vitro* and *in vivo*. This review will focus on viral infections of mammals, but will also describe what is known about other infections. Readers are directed to the excellent accompanying reviews in this issue which expertly discuss the clinical trials, cell biology, mechanisms of action, impact on inflammation, clinical applications, and so forth.

Cannabinoids may act either through the CB_1 or the CB_2 receptor, which are found on distinct cell types. The CB_1 receptor is found on neurons as well as some astrocytes and skeletal muscle cells; neurons are frequently the target of viral infection. Engagement of the CB_1 receptor by its endogenous or exogenous agonists may inhibit the release of Ca^{2+} from intracellular or extracellular stores. Since many important intracellular proteins are Ca^{2+}-dependent for activation, signal transduction through the CB_1 receptor may impair these secondary pathways and have a profound influence on the ability of viruses to replicate in neurons.

In contrast, the response of cells expressing the CB_2 receptor may influence not only the responses in that cell, but may alter the course of the host innate and adaptive immune response to the pathogen, suppressing inflammation and the development of virus-specific cellular and humoral responses. The outcome on the viral infection will depend on whether inflammation is beneficial or pathogenic in the specific case.

2. Discussion

When a host is infected with a virus, there is a dynamic competition between the ability of the host to first marshal innate (hours to days) and then adaptive immunity (>7 days post infection) *vs.* the replication and spread of the virus first within the host and then to additional susceptible individuals. When a virus is able to out-pace the containment efforts, the host may succumb. Pathology may result from damage to tissues by viral-induced cellular apoptosis or necrosis, or alternatively, host immune responses may result in immunopathology or the perceived symptoms of the infection. If, however, innate and adaptive immunity successfully suppress viral replication, specific life-long immunity may result.

In order to understand the influences on the host response which may be the result of cannabinoids, it is important to examine some of the cellular pathways which are dependent on Ca^{2+}-dependent enzymes. Table 1 indicates some of the well characterized pathways involved and their potential impact on viral infections.

The common recurring impact of Ca^{2+}-dependent enzymes is a role in inflammation. This ranges from regulation of many signal transduction pathways, production of pro-inflammatory and pro-resolving lipid mediators downstream of arachidonic acid, to activation of Nitric Oxide Synthase and the production of reactive nitrogen intermediates, to proteolytic enzymes which remodel the cytoskeleton or extracellular matrix, and apoptosis.

Inflammation is essential for recruitment of both innate and adaptive immune cells to the site of infection to control virus production and limit spread, and then to promote recovery. Inflammation is comprised not only of non-specific cells (sequentially these are polymorphonuclear leukocytes, natural killer cells, macrophages) and then pathogen-specific T lymphocytes recruited from circulation, and activation of antibody-secreting B lymphocytes, but also induction of production and secretion of cytokines, chemokines, interferons, complement components, acute phase reactants, reactive oxygen and nitrogen intermediates, and other mediators [24,25,26]. Readers are referred to the accompanying review by Bani, Mannaioni, Passani, and Masini [27]. Thus, many of these critical pathways may be impaired or compromised when endogenous or exogenous cannabinoids are present during an infection [28].

Cannabinoids have been used both recreationally by groups of people who have viral infections, and experimentally by scientists investigating their impact *in vitro* or in animal models. Table

2 presents what has been published about these populations in peer reviewed journals. In most of the infections studied (Table 2), it is apparent that cannabinoid treatment, whether *in vitro* or *in vivo*, had profound impact on the virus-host (cell) interactions. For HSV-2, HIV-1, KSHV, influenza and VSV viral replication, or surrogate measures of infection, were found to be substantially increased upon cannabinoid treatment [30,34,39,50,52,63]. In HIV-1 infection, syncytia formation was enhanced, and monocytes were stickier on endothelial cells [57,58]. In one study, KHSV was more likely to exit latency and enter lytic infection when transformed cells were treated with THC [39], however, another study found the opposite result in several herpesvirus infections [38].

Disease was more severe in HSV-2-infected guinea pigs which were treated with THC [29,30,32]. In HCV infections, clinical studies have shown a profound co-morbidity of recreational cannabinoid use, for disease progression [54,56]. One case report of Cowpox infection, a very rare human pathogen, indicated that recreational use of cannabinoids was associated with generalized infection and very poor immune responses to the virus [40].

In contrast, in those infections where host inflammatory responses are often associated with pathology, and not with clearance and recovery, cannabinoid treatment of hosts was beneficial. These included one mouse model of multiple sclerosis, the Theiler's murine encephalomyelocarditis virus (TMEV)-induced demyelinating disease (IDD), where progression towards the paralysis and disability were ameliorated [44,45,48] and in Borna disease virus (BDV) where neural progenitors were protected from proinflammatory cytokine-mediated damage [53] infections. TMEV-IDD is characterized by microglial activation in the spinal cord of mice and a T cell-mediated autoimmune demyelinating disease, triggered by the viral infection [42,67,68,69]. Persistent BDV infection of the central nervous system is associated with immunopathology associate with inflammation and production of pro-inflammatory cytokines, induction of NOS-2 in microglia, and breakdown of the blood-brain barrier [70,71,72,73]. In both BVD and TMEV-IDD, the targets for the anti-inflammatory effects of the cannabinoid treatment are lymphocytes and mononuclear cells.

Two excellent reviews of the impact of cannabinoids on bacterial, yeast, and protozoan infections were published in the same issue of *Journal of Neuroimmunology* [26,74]. These infections included *Treponema pallidum* (Syphilis), *Legonella pneumophila* (Legionnaires' disease), *Staphylococci aureus* and *S. albus*, *Listeria monocytogenes, Candida albicans* (Thrush), and *Naegleria fowleri*. Both reviews concluded that THC significantly reduced host resistance to infection of experimental animals, and speculated that similar host compromise would be found in man. In the more than 12 years since those reviews were published, additional findings have extended the serious consequences of cannabinoids on host responses to pathogens and opportunistic infections. Marijuana use is a risk factor for *Mycobacterium tuberculosis* (TB) infections [75,76,77]; this author speculates the suppression of host innate immune responses by THC contributes to the increased severity of TB in users. Similarly, more serious exacerbations central nervous system infection by *Acanthamoeba* among HIV-infected patients has been attributed to marijuana consumption [78], possibly by inhibiting macrophage chemotaxis [79]. However, the antiinflammatory effects of cannabinoids have been found to be beneficial in attenuating fever induced by bacterial endotoxin [65,80], inhibiting cytokine responses to *Corynebacterium parvum* endotoxin [81]. These drugs may also offer therapeutic efficacy in meningitis caused by *Streptococcus pneumoniae* [82] and in irritable bowel syndrome [83,84].

Cannabinoids may relieve pain and may induce hyperphagia, which could be beneficial in cancer [85,86]. However, these physiological characteristics are not relevant to most viral, bacterial fungal or parasitic infections, where the regulation of inflammation is central to controlling pathogen replication and immunopathology. However, the same anti-inflammatory properties of cannabinoids just described are detrimental to the host in handling the other infections. In most cases, a rapid and robust inflammatory response, associated with production of proinflammatory cytokines and effect T lymphocytes capable of eliminating infected cells is essential to recovery and survival.

Go to:

3. Conclusions

Cannabinoids are profoundly anti-inflammatory and impair many Ca^{2+}-dependent enzyme systems which are central to inflammatory and cell-autonomous antiviral responses. When viral-induced host responses lead to immunopathology, as is seen in a rodent model of multiple sclerosis, TMEV-IDD, or in a persistent infection of the central nervous system caused by a non-lytic virus, BDV, cannabinoid treatment was beneficial.

In all other virus infections, both *in vitro* and *in vivo*, cannabinoid treatment led to disease progression, increased pathology, and sometimes to host death. Therefore, in many clinical settings, including latent infections caused by HIV-1 or HSV-1, and persistent infection of the liver caused by HCV, cannabinoids lead to worsened disease outcome.

Go to:

Acknowledgements

The generous support of the U.S. National Institutes of Health, both grants DC039746 and NS039746, enabled my lab to perform these studies, and to summarize the work of many other labs. Two former students, Ramon Antonio Herrera and Joseph H. Oved, contributed published [52] and unpublished data, provided stimulating discussions and critically read this manuscript.