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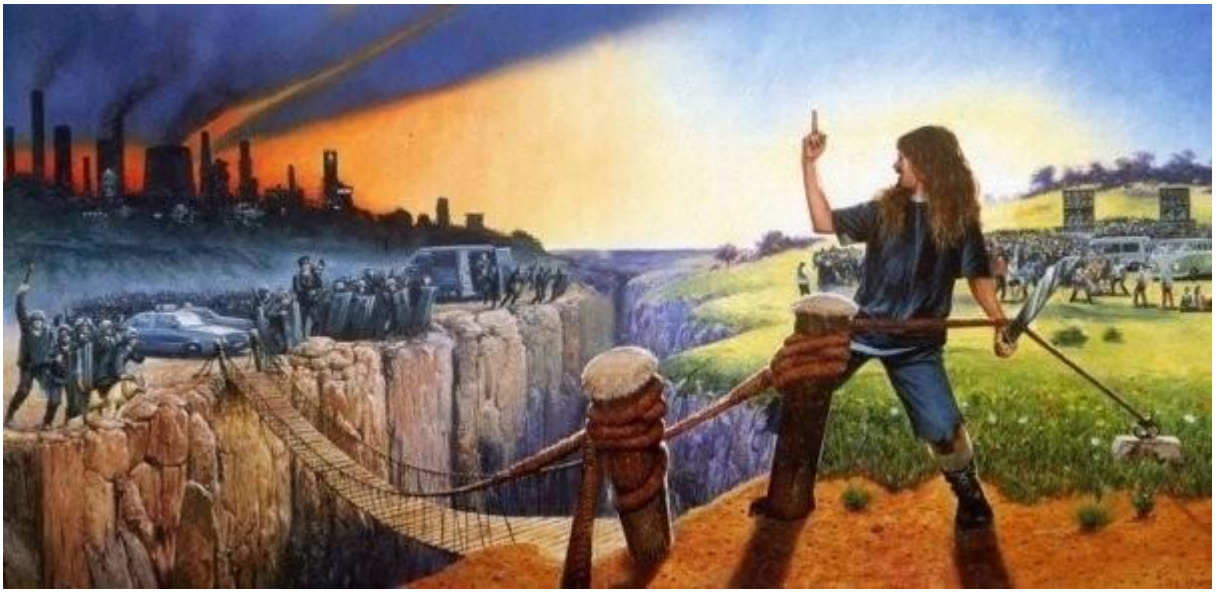

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http://www.equalrights4all.org/religious/bible.htm

http://ccguide.org/bible.php

(the verses can be found here: http://www.biblegateway.com/versions/King-James-Version-KJV-Bible/)
100 published studies on the anti-cancer effects of cannabinoids, sorted by country: UNITED

KINGDOM http://www.ncbi.nlm.nih.gov/pubmed/15454482
http://www.ncbi.nlm.nih.gov/pubmed/17583570 http://www.ncbi.nlm.nih.gov/pubmed/17931597
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http://www.ncbi.nlm.nih.gov/pubmed/18438336

37 Medical Studies about the Anti-Cancer Effects of Cannabinoids

on Brain cancer, Breast cancer, Cancer (all types), Cervical cancer, Cholangiocarcinoma, Colorectal cancer, Leukemia, Lung cancer, Lymphoma, Prostate cancer, Rhabdomyosarcoma, Stomach cancer

Research and summaries by CANNABIS CURES CANCERS!

BRAIN CANCER:

Journal of Neuropathology & Experimental Neurology
September 2004

Arachidonylethanolamide Induces Apoptosis of Human Glioma Cells through Vanilloid Receptor-1

Abstract

The anti-tumor properties of cannabinoids have recently been evidenced, mainly with Δ9-tetrahydrocannabinol (THC). However, the clinical application of this drug is limited by possible undesirable side effects due to a broad expression of cannabinoid receptors (CB1 and CB2). An attractive field of research therefore is to identify molecules with more selective tumor targeting. This is particularly important for malignant gliomas, considering their poor prognosis and their location in the brain. Here we investigated whether the most potent endogenous cannabinoid, arachidonylethanolamide (AEA), could be a candidate. We observed that AEA induced apoptosis in long-term and recently established glioma cell lines via aberrantly expressed vanilloid receptor-1 (VR1). In contrast with their role in THC-mediated death, both CB1 and CB2 partially protected glioma against AEA-induced apoptosis. These data show that the selective targeting of VR1 by AEA or more stable analogues is an attractive research area for the treatment of glioma.

http://journals.lww.com/jneuropath/pages/articleviewer.aspx?year=2004&issue=09000&article=00006&type=abstract

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Molecular Pharmacology
December 2004

Up-Regulation of Cyclooxygenase-2 Expression Is Involved in R(+)-Methanandamide-Induced Apoptotic Death of Human Neuroglioma Cells

Abstract

Cannabinoids have been implicated in the reduction of glioma growth. The present study investigated a possible relationship between the recently shown induction of cyclooxygenase (COX)-2 expression by the endocannabinoid analog R(+)methanandamide [R(+)-MA] and its effect on the viability of H4 human neuroglioma cells. Incubation with R(+)-MA for up to 72 h decreased the cellular viability and enhanced accumulation of cytoplasmic DNA fragments in a time-dependent manner. ... As a whole, this study defines COX-2 as a hitherto unknown target by which a cannabinoid induces apoptotic death of glioma cells. Furthermore, our data show that pharmacological concentrations of celecoxib may interfere with the proapoptotic action of R(+)-MA and anandamide, suggesting that cotreatment with COX-2 inhibitors could diminish glioma regression induced by these compounds.

http://molpharm.aspetjournals.org/content/66/6/1643.long

Cellular Signalling
January 2005

Abstract

Cannabinoids were shown to induce apoptosis of glioma cells in vitro and tumor regression in vivo, but mechanisms of their antiproliferative action remain elusive. In the present studies, C6 cells were exposed to a synthetic cannabinoid ...which produced down-regulation of the Akt and Erk signalling pathways prior to appearance of any sign of apoptosis. We hypothesized that cannabinoid-induced cell death may be mediated by a Bcl-2 family member—Bad ... treatment further resulted in mitochondrial depolarization and activation of caspase cascade. Thus, we suggest that the increase of proapoptotic Bad activity is an important link between the inhibition of survival pathways and an onset of execution phase of cannabinoid-induced glioma cell death.

http://www.sciencedirect.com/science/article/pii/S0898656804001007

Journal of Neurochemistry
August 2006

Cannabinoid receptors in human astroglial tumors

Abstract

In animal models, cannabinoids are reported to inhibit the growth of tumors, including gliomas. These effects have been claimed to be mediated via cannabinoid receptors 1 and 2 (CB1, CB2). To elucidate a possible relevance for treatment of human gliomas, we investigated receptor subtype expression in surgical material of solid human astrocytomas, gliomas and cultivated glioma cells ... In normal brain, cultivated glioma cells and solid tumors, CB1 ... was expressed to a much greater extent than CB2, which in some samples was even undetectable. Expression of both receptor subtypes was unrelated to malignancy, varied between patients, and was not significantly increased in relation to normal brain tissues. In normal brain, CB1 protein was localized on astroglial and other cell types; in gliomas, it was found on astroglial/glioma cells. CB2 protein was detected on microglial cells/macrophages

but rarely on astroglial cells. Functionally, CB1 receptor agonists ... slightly reduced proliferation of glioma cells in vitro, but did not induce apoptosis. We conclude that cannabinoid therapy of human gliomas targets not only receptors on tumor, but also on other cell types. Therefore, complex and potential side-effects should be considered carefully.

http://onlinelibrary.wiley.com/doi/10.1111/j.1471-4159.2006.03911.x/abstract

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Journal of Neuroscience Research
November 2008

High concentrations of cannabinoids activate apoptosis in human U373MG glioma cells

Abstract

Cannabinoids bind to two G-protein-coupled receptors, CB1 and CB2, expressed by neurons and cells of the immune system, respectively. Glioma cells (astrocyte-derived brain tumor cells) express cannabinoid receptors, and numerous in vitro and in vivo studies performed in rodents have concluded that apoptosis could be induced by cannabinoids in these cells. Whether this also applies to human cells is controversial; we, therefore, assessed the effect of cannabinoids on human glioma cell viability with the human astrocytoma cell line U373MG. We report here that U373MG human glioma cells are sensitive only to high concentrations of cannabinoids (>5 μg/ml for Δ9-THC). ... suggesting that cannabinoid receptors are functional in U373MG cells. ... CB1 is expressed in U373MG cells and is involved in cannabinoid-induced cell death ... In addition, as already reported, some cannabinoids may have modest proproliferative properties in U373MG cells. Human U373MG glioma cells are sensitive only to very high, pharmacologically irrelevant concentrations of cannabinoids, so it seems unlikely that cannabinoids would constitute promising molecules for treating malignant astrocytoma; they do not induce glioma cell death at doses that could be applied safely to humans.

http://onlinelibrary.wiley.com/doi/10.1002/jnr.21757/abstract

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Acta Oncologica
2008

Δ9-Tetrahydrocannabinol inhibits cell cycle progression by downregulation of E2F1 in human glioblastoma multiforme cells

Background:
The active components of Cannabis sativa L., Cannabinoids, traditionally used in the field of cancer for alleviation of pain, nausea, wasting and improvement of well-being have received renewed interest in recent years due to their diverse pharmacologic activities such as cell growth inhibition, anti-inflammatory activity and induction of tumor regression.
Here we used several experimental approaches, which identified delta-9-tetrahydrocannabinol (Δ9-THC) as an essential mediator of cannabinoid antitumoral action.

Methods and results:
Administration of Δ9-THC to glioblastoma multiforme (GBM) cell lines results in a significant decrease in cell viability.
Cell cycle analysis showed G0/1 arrest ... analyses revealed a THC altered cellular content of proteins that regulate cell progression through the cell cycle. The cell content of ... two proteins that promote cell cycle progression, were suppressed in both U251-MG and U87-MG human glioblastoma cell lines ...

Conclusions:
Δ9-THC is shown to significantly affect viability of GBM cells via a mechanism that appears to elicit G1 arrest ... Hence, it is suggested that Δ9-THC and other cannabinoids be implemented in future clinical evaluation as a therapeutic modality for brain tumors.

http://informahealthcare.com/doi/abs/10.1080/02841860701678787

Brain Research Bulletin
June 2009

Predominant CB2 receptor expression in endothelial cells of glioblastoma in humans

Abstract

Background and objectives:

The most abundant malignant brain tumor in human is glioblastoma and patients with this type of tumor have a poor prognosis with high mortality. Glioblastoma are characterized particularly

by fast growth and a dependence on blood vessel formation for survival. Cannabinoids (CBs) inhibit tumor growth by inducing apoptosis of tumor cells and impairing tumor angiogenesis. The distribution of CB1 and CB2 receptors in glioblastoma and associated endothelial vessels is still unknown.

Methods:

Tissue samples were collected consecutively after neurosurgery of 19 patients suspected glioblastoma and examined immunohistochemically for CB1 and CB2 receptor expression. ...

Results:

In endothelia of control tissue, about 24% and 45% of the cells were positive for CB1 and CB2 receptors. In glioblastoma endothelial cells, CB1 and CB2 receptors were present in about 38% and 54% of the cells respectively. In comparison to CB1, an elevated CB2 receptor expression was identified in glioblastoma.

Conclusions:

The abundant expression and distribution of CB2 receptors in glioblastoma and particularly endothelial cells of glioblastoma indicate that impaired tumor growth in presence of CB may be associated with CB2 activation. Selective CB2 agonists might become important targets attenuating vascular endothelial growth factor (VEGF) signalling and thereby diminishing neoangiogenesis and glioblastoma growth.

http://www.sciencedirect.com/science/article/pii/S0361923009000598

BREAST CANCER:

Molecular Cancer Therapeutics
November 2007

Cannabidiol as a novel inhibitor of Id-1 gene expression in aggressive breast cancer cells

Abstract

Invasion and metastasis of aggressive breast cancer cells is the final and fatal step during

cancer progression, and is the least understood genetically. Clinically, there are still limited therapeutic interventions for aggressive and metastatic breast cancers available. Clearly, effective and nontoxic therapies are urgently required. Id-1, an inhibitor of basic helix-loop-helix transcription factors, has recently been shown to be a key regulator of the metastatic potential of breast and additional cancers. Using a mouse model, we previously determined that metastatic breast cancer cells became significantly less invasive in vitro and less metastatic in vivo when Id-1 was down-regulated ... Here, we report that cannabidiol (CBD), a cannabinoid with a low-toxicity profile, could down-regulate Id-1 expression in aggressive human breast cancer cells. The CBD concentrations effective at inhibiting Id-1 expression correlated with those used to inhibit the proliferative and invasive phenotype of breast cancer cells. CBD was able to inhibit Id-1 expression at the mRNA and protein level in a concentration-dependent fashion. These effects seemed to occur as the result of an inhibition of the Id-1 gene ... CBD represents the first nontoxic exogenous agent that can significantly decrease Id-1 expression in metastatic breast cancer cells leading to the down-regulation of tumor aggressiveness.

http://mct.aacrjournals.org/content/6/11/2921.long

Molecular Cancer Therapeutics
November 2009

Synthetic cannabinoid receptor agonists inhibit tumor growth and metastasis of breast cancer

Cannabinoids have been reported to possess antitumorogenic activity. Not much is known, however, about the effects and mechanism of action of synthetic nonpsychotic cannabinoids on breast cancer growth and metastasis. We have shown that the cannabinoid receptors CB1 and CB2 are overexpressed in primary human breast tumors compared with normal breast tissue. We have also observed that the breast cancer cell lines ... express CB1 and CB2 receptors. Furthermore, we have shown that the CB2 synthetic agonist JWH-133 and the CB1 and CB2 agonist WIN-55,212-2 inhibit cell proliferation and migration under in vitro conditions. These results were confirmed in vivo in various mouse model systems. Mice treated with JWH-133 or WIN-55,212-2 showed a 40% to 50% reduction in tumor growth and a 65% to 80% reduction in lung metastasis. ... In addition, the CB2 agonist JWH-133 was shown to delay and reduce mammary gland tumors in the ... transgenic mouse model system. ... These results indicate that CB1 and CB2 receptors could be used to develop novel therapeutic strategies against breast cancer growth and metastasis.

http://mct.aacrjournals.org/content/8/11/3117.long

CANCER, ALL TYPES:

Clinical Pharamacokinetics
April 2003

Abstract

Δ9-Tetrahydrocannabinol (THC) is the main source of the pharmacological effects caused by the consumption of cannabis, both the marijuana-like action and the medicinal benefits of the plant. However, its acid metabolite THC-COOH, the non-psychotropic cannabidiol (CBD), several cannabinoid analogues and newly discovered modulators of the endogenous cannabinoid system are also promising candidates for clinical research and therapeutic uses. Cannabinoids exert many effects through activation of G-protein-coupled cannabinoid receptors in the brain and peripheral tissues. Additionally, there is evidence for nonreceptor-dependent mechanisms.

Natural cannabis products and single cannabinoids are usually inhaled or taken orally; the rectal route, sublingual administration, transdermal delivery, eye drops and aerosols have only been used in a few studies and are of little relevance in practice today. The pharmacokinetics of THC vary as a function of its route of administration. Pulmonary assimilation of inhaled THC causes a maximum plasma concentration within minutes, psychotropic effects start within seconds to a few minutes, reach a maximum after 15–30 minutes, and taper off within 2–3 hours. Following oral ingestion, psychotropic effects set in with a delay of 30–90 minutes, reach their maximum after 2–3 hours and last for about 4–12 hours, depending on dose and specific effect.

At doses exceeding the psychotropic threshold, ingestion of cannabis usually causes enhanced well-being and relaxation with an intensification of ordinary sensory experiences. The most important acute adverse effects caused by overdosing are anxiety and panic attacks, and with regard to somatic effects increased heart rate and changes in blood pressure. Regular use of cannabis may lead to dependency and to a mild withdrawal syndrome. The existence and the intensity of possible long-term adverse effects on psyche and cognition, immune system, fertility and pregnancy remain controversial. They are reported to be low in humans and do not preclude legitimate therapeutic use of cannabis-based drugs. Properties of cannabis that might be of therapeutic use include analgesia, muscle relaxation,

immunosuppression, sedation, improvement of mood, stimulation of appetite, antiemesis, lowering of intraocular pressure, bronchodilation, neuroprotection and induction of apoptosis in cancer cells.

http://link.springer.com/article/10.2165%2F00003088-200342040-00003

Expert Opinion on Therapeutic Targets
December 2003

Cannabinoid receptor systems: therapeutic targets for tumour intervention

The past decade has witnessed a rapid expansion of our understanding of the biological roles of cannabinoids and their cognate receptors. It is now certain that Δ9-tetrahydrocannabinol, the principle psychoactive component of the Cannabis sativa plant, binds and activates membrane receptors ... Synthesis of numerous cannabinomimetics has also greatly expanded the repertoire of cannabinoid receptor ligands with the pharmacodynamic properties of agonists, antagonists and inverse agonists. Collectively, these ligands have proven to be powerful tools both for the molecular characterisation of cannabinoid receptors and the delineation of their intrinsic signalling pathways. Much of our understanding of the signalling mechanisms activated by cannabinoids is derived from studies of receptors expressed by tumour cells; hence, this review provides a succinct summary of the molecular pharmacology of cannabinoid receptors and their roles in tumour cell biology. Moreover, there is now a genuine expectation that the manipulation of cannabinoid receptor systems may have therapeutic potential for a diverse range of human diseases. Thus, this review also summarises the demonstrated antitumour actions of cannabinoids and indicates possible avenues for the future development of cannabinoids as antitumour agents.

http://informahealthcare.com/doi/abs/10.1517/14728222.7.6.749%20

Mini-Reviews in Medicinal Chemistry
October 2005

Cannabinoids and Cancer

Abstract:
Marijuana has been used in medicine for millennia, but it was not until 1964 that Δ9-tetrahydrocannabinol (Δ9-THC), its major psychoactive component, was isolated in pure form and its structure was elucidated. Shortly thereafter it was synthesized and became readily available. However, it took another decade until the first report on its antineoplastic activity appeared. In 1975, Munson discovered that cannabinoids suppress Lewis lung carcinoma cell growth. The mechanism of this action was shown to be inhibition of DNA synthesis. Antiproliferative action on some other cancer cells was also found. In spite of the promising results from these early studies, further investigations in this area were not reported until a few years ago, when almost simultaneously two groups initiated research on the antiproliferative effects of cannabinoids on cancer cells: Di Marzos group found that cannabinoids inhibit breast cancer cell proliferation, and Guzmans group found that cannabinoids inhibit the growth of C6 glioma cell. Other groups also started work in this field, and today, a wide array of cancer cell lines that are affected is known, and some mechanisms involved have been elucidated.

http://www.eurekaselect.com/78957/article

Molecular Pharmacology
March 2006

A Cannabinoid Quinone Inhibits Angiogenesis by Targeting Vascular Endothelial Cells

... We have reported that a new cannabinoid anticancer quinone, cannabidiol hydroxyquinone (HU-331), is highly effective against tumor xenografts in nude mice. ...
The ability of cannabinoids to cause endothelial cell apoptosis was assayed ...
Immunostaining with anti-CD31 of tumors grown in nude mice served to indicate inhibition of tumor angiogenesis. HU-331 was found to be strongly antiangiogenic, significantly inhibiting angiogenesis at concentrations as low as 300 nM. HU-331 inhibited angiogenesis by directly inducing apoptosis of vascular endothelial cells without changing the expression of pro- and antiangiogenic cytokines and their receptors. A significant decrease in the total area occupied by vessels in HU-331-treated tumors was also observed. These data lead us to consider HU-331 to have high potential as a new antiangiogenic and anticancer drug.

http://molpharm.aspetjournals.org/content/70/1/51.long

Molecular Cancer Therapeutics
January 2007

HU-331, a novel cannabinoid-based anticancer topoisomerase II inhibitor

Abstract

Anthracyclines, a large group of quinonoid compounds, are used to treat some forms of cancer. Although highly effective in cancer therapy, the mechanism of action of these compounds is not specific; they act on cancer and other cells by numerous mechanisms. A new anticancer quinone (HU-331) was synthesized from cannabidiol. It shows significant high efficacy against human cancer cell lines in vitro and against in vivo tumor grafts in nude mice. In this study, we investigated its mode of action and present evidence on its unique mechanism. ... HU-331–caused cell death of human cancer cell lines is not mediated by reactive oxygen intermediates/species ... The cannabinoid quinone HU-331 is a highly specific inhibitor of topoisomerase II, compared with most known anticancer quinones. It might represent a new potent anticancer drug.

http://mct.aacrjournals.org/content/6/1/173.long

Dialogues in Clinical Neuroscience
2007

Cannabinoids in health and disease

Abstract

Cannabis sativa L. preparations have been used in medicine for millenia. ... Only recently, marijuana and individual natural and synthetic cannabinoid receptor agonists and antagonists, as well as chemically related compounds, whose mechanism of action is still obscure, have come back to being considered of therapeutic value. However, their use is highly restricted. ... the therapeutic value of cannabinoids is too high to be put aside. Numerous diseases, such as anorexia, emesis, pain, inflammation, multiple sclerosis, neurodegenerative disorders (Parkinson's disease, Huntington's disease, Tourette's syndrome, Alzheimer's disease), epilepsy, glaucoma, osteoporosis, schizophrenia, cardiovascular disorders, cancer, obesity, and metabolic syndrome-related disorders, to name just a few, are being treated or have the

potential to be treated by cannabinoid agonists/antagonists/cannabinoid-related compounds. In view of the very low toxicity and the generally benign side effects of this group of compounds, neglecting or denying their clinical potential is unacceptable - instead, we need to work on the development of more selective cannabinoid receptor agonists/antagonists and related compounds, as well as on novel drugs of this family with better selectivity, distribution patterns, and pharmacokinetics, and - in cases where it is impossible to separate the desired clinical action and the psychoactivity - just to monitor these side effects carefully.

http://www.dialogues-cns.com/publication/cannabinoids-in-health-and-disease/

Cancer Research
January 2008

Cannabinoids for Cancer Treatment: Progress and Promise

Abstract

Cannabinoids are a class of pharmacologic compounds that offer potential applications as antitumor drugs, based on the ability of some members of this class to limit inflammation, cell proliferation, and cell survival. In particular, emerging evidence suggests that agonists of cannabinoid receptors expressed by tumor cells may offer a novel strategy to treat cancer. Here, we review recent work that raises interest in the development and exploration of potent, nontoxic, and nonhabit forming cannabinoids for cancer therapy.

http://cancerres.aacrjournals.org/content/68/2/339.long

Experimental Oncology
March 2008

Antineoplastic and apoptotic effects of cannabinoids. N-acylethanolamines: protectors or killers?

Abstract

The proapoptotic and antineoplastic properties of cannabinoids ... were analyzed. Cannabinoids enhanced apoptotic and necrotic processes in many types of tumour cells and tissues. Involvement of different types of receptors and signaling pathways in mediating the proapoptotic effects of cannabinoids are discussed. The evidences in favour of both proapoptotic, pronecrotic and protective, antiapoptotic effects of cannabinoids ... are evaluated. ... The conclusion is made on promising of cannabinoids as potential anticancer agents.

http://www.ncbi.nlm.nih.gov/pubmed/18438336

The Journal of Pharmacy and Pharmacology
July 2009

Cannabinoid receptor ligands as potential anticancer agents--high hopes for new therapies?

Abstract

OBJECTIVES:
The endocannabinoid system is an endogenous lipid signalling network comprising arachidonic-acid-derived ligands, cannabinoid (CB) receptors, transporters and endocannabinoid degrading enzymes. The CB(1) receptor is predominantly expressed in neurons but is also co-expressed with the CB(2) receptor in peripheral tissues. In recent years, CB receptor ligands, including Delta(9)-tetrahydrocannabinol, have been proposed as potential anticancer agents.

KEY FINDINGS:
This review critically discusses the pharmacology of CB receptor activation as a novel therapeutic anticancer strategy in terms of ligand selectivity, tissue specificity and potency. Intriguingly, antitumour effects mediated by cannabinoids are not confined to inhibition of cancer cell proliferation; cannabinoids also reduce angiogenesis, cell migration and metastasis, inhibit carcinogenesis and attenuate inflammatory processes. ...

SUMMARY:
The role of the endocannabinoid system in tumourigenesis is still poorly understood and the molecular mechanisms of cannabinoid anticancer action need to be elucidated. The development of CB(2)-selective anticancer agents could be advantageous in light of the unwanted central effects exerted by CB(1) receptor ligands. Probably the most interesting

question is whether cannabinoids could be useful in chemoprevention or in combination with established chemotherapeutic agents.

http://www.ncbi.nlm.nih.gov/pubmed/19589225

Trends in Pharmacological Sciences
August 2009

The endocannabinoid system of the skin in health and disease: novel perspectives and therapeutic opportunities

The newly discovered endocannabinoid system (ECS; comprising the endogenous lipid mediators endocannabinoids present in virtually all tissues, their G-protein-coupled cannabinoid receptors, biosynthetic pathways and metabolizing enzymes) has been implicated in multiple regulatory functions both in health and disease. Recent studies have intriguingly suggested the existence of a functional ECS in the skin and implicated it in various biological processes (e.g. proliferation, growth, differentiation, apoptosis and cytokine, mediator or hormone production of various cell types of the skin and appendages, such as the hair follicle and sebaceous gland). It seems that the main physiological function of the cutaneous ECS is to constitutively control the proper and well-balanced proliferation, differentiation and survival, as well as immune competence and/or tolerance, of skin cells. The disruption of this delicate balance might facilitate the development of multiple pathological conditions and diseases of the skin (e.g. acne, seborrhea, allergic dermatitis, itch and pain, psoriasis, hair growth disorders, systemic sclerosis and cancer).

http://www.sciencedirect.com/science/article/pii/S0165614709001072

Cancer Letters
November 2009

Cannabinoids in the treatment of cancer

Abstract
Cannabinoids, the active components of the hemp plant Cannabis sativa, along with their endogenous counterparts and synthetic derivatives, have elicited anti-cancer effects in many

different in vitro and in vivo models of cancer. While the various cannabinoids have been examined in a variety of cancer models, recent studies have focused on the role of cannabinoid receptor agonists (both CB1 and CB2) in the treatment of estrogen receptor-negative breast cancer. This review will summarize the anti-cancer properties of the cannabinoids, discuss their potential mechanisms of action, as well as explore controversies surrounding the results.

http://www.cancerletters.info/article/S0304-3835(09)00252-3/abstract

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The Journal of Pharmacology and Experimental Therapeutics
February 2010

Antitumorigenic Effects of Cannabinoids beyond Apoptosis

Abstract

According to the World Health Organization, the cases of death caused by cancer will have been doubled until the year 2030. By 2010, cancer is expected to be the number one cause of death. Therefore, it is necessary to explore novel approaches for the treatment of cancer. Over past years, the antitumorigenic effects of cannabinoids have emerged as an exciting field in cancer research. Apart from their proapoptotic and antiproliferative action, recent research has shown that cannabinoids may likewise affect tumor cell angiogenesis, migration, invasion, adhesion, and metastasization. This review will summarize the data concerning the influence of cannabinoids on these locomotive processes beyond modulation of cancer cell apoptosis and proliferation. The findings discussed here provide a new perspective on the antitumorigenic potential of cannabinoids. ...

Apart from regulating tumor cell growth and apoptosis, other antitumorigenic mechanisms of cannabinoids are currently emerging as a focus of research work. Therefore, the present review focuses on the impact of cannabinoids on tumor neovascularization, tumor cell migration, adhesion, invasion, and metastasization.

http://jpet.aspetjournals.org/content/332/2/336.long

Biochemical Pharmacology

April 2010

Cannabidiol inhibits cancer cell invasion via upregulation of tissue inhibitor of matrix metalloproteinases-1

Abstract

Although cannabinoids exhibit a broad variety of anticarcinogenic effects, their potential use in cancer therapy is limited by their psychoactive effects. Here we evaluated the impact of cannabidiol, a plant-derived non-psychoactive cannabinoid, on cancer cell invasion. ... we found a cannabidiol-driven impaired invasion of human cervical cancer ... and human lung cancer cells ... Additionally, in vivo studies in thymic-aplastic nude mice revealed a significant inhibition of ... lung metastasis in cannabidiol-treated animals as compared to vehicle-treated controls. Altogether, these findings provide a novel mechanism underlying the anti-invasive action of cannabidiol and imply its use as a therapeutic option for the treatment of highly invasive cancers.

http://www.sciencedirect.com/science/article/pii/S000629520900971X

CERVICAL CANCER:

Gynecologic Oncology
April 2004

Abstract

Objective:
Δ9-Tetrahydrocannabinol, the active agent of Cannabis sativa, exhibits well-documented antitumor properties, but little is known about the possible effects mediated by endogenous cannabinoids on human tumors. In the present study, we analyzed the effect of arachidonyl ethanolamide (AEA) on cervical carcinoma (CxCa) cell lines.

Methods:
To assess the sensitivity of CxCa cells to AEA, we selected three cell lines that were exposed to increasing doses of AEA ... The expression of receptors to AEA were analyzed in CxCa cell lines as well as CxCa biopsies.

Results:
The major finding was that AEA induced apoptosis of CxCa cell lines ... whereas AEA binding to the classical CB1 and CB2 cannabinoid receptors mediated a protective effect. Furthermore, unexpectedly, a strong expression of the three forms of AEA receptors was observed in ex vivo CxCa biopsies.

Conclusion:
Overall, these data suggest that the specific targeting of VR1 by endogenous cannabinoids or synthetic molecules offers attractive opportunities for the development of novel potent anticancer drugs.

http://www.sciencedirect.com/science/article/pii/S0090825803009521

CHOLANGIOCARCINOMA (Bile duct cancer):

Cancer Investigation
May 2010

Abstract
Currently, only gemcitabine plus platinum demonstrates the considerable activity for cholangiocarcinoma. The anticancer effect of Delta (9)-tetrahydrocannabinol (THC), the principal active component of cannabinoids has been demonstrated in various kinds of cancers. We therefore evaluate the antitumor effects of THC on cholangiocarcinoma cells. Both cholangiocarcinoma cell lines and surgical specimens from cholangiocarcinoma patients expressed cannabinoid receptors. THC inhibited cell proliferation, migration and invasion, and induced cell apoptosis. ... Consequently, THC is potentially used to retard cholangiocarcinoma cell growth and metastasis.

http://www.ncbi.nlm.nih.gov/pubmed/19916793

COLORECTAL CANCER:

International Journal of Cancer
November 2007

The cannabinoid δ9-tetrahydrocannabinol inhibits RAS-MAPK and PI3K-AKT survival signalling and induces BAD-mediated apoptosis in colorectal cancer cells

Abstract

Deregulation of cell survival pathways and resistance to apoptosis are widely accepted to be fundamental aspects of tumorigenesis. As in many tumours, the aberrant growth and survival of colorectal tumour cells is dependent upon a small number of highly activated signalling pathways, the inhibition of which elicits potent growth inhibitory or apoptotic responses in tumour cells. Accordingly, there is considerable interest in therapeutics that can modulate survival signalling pathways and target cancer cells for death. There is emerging evidence that cannabinoids, especially Δ9-tetrahydrocannabinol (THC), may represent novel anticancer agents, due to their ability to regulate signalling pathways critical for cell growth and survival. Here, we report that CB1 and CB2 cannabinoid receptors are expressed in human colorectal adenoma and carcinoma cells, and show for the first time that THC induces apoptosis in colorectal cancer cells. ... These data suggest an important role for CB1 receptors and BAD in the regulation of apoptosis in colorectal cancer cells. The use of THC, or selective targeting of the CB1 receptor, may represent a novel strategy for colorectal cancer therapy.

http://onlinelibrary.wiley.com/doi/10.1002/ijc.22917/abstract;jsessionid=D671A9D0BD2DC948D6BE316EDEB65865.f02t03

LEUKEMIA:

Blood: Journal of the American Society of Hematology
July 2002

Targeting CB2 cannabinoid receptors as a novel therapy to treat malignant lymphoblastic disease

Abstract

In the current study, we examined whether ligation of CB2 receptors would lead to induction of apoptosis in tumors of immune origin and whether CB2 agonist could be used to treat such cancers.
Exposure of murine [mouse] tumors ... to delta-9-tetrahydrocannabinol (THC) in vitro led to a significant reduction in cell viability and an increase in apoptosis.

Exposure of EL-4 tumor cells to the synthetic cannabinoid HU-210 and the endogenous cannabinoid anandamide led to significant induction of apoptosis ...
Treatment of EL-4 tumor-bearing mice with THC in vivo led to a significant reduction in tumor load, increase in tumor-cell apoptosis, and increase in survival of tumor-bearing mice.
Examination of a number of human leukemia and lymphoma cell lines ... revealed that they expressed CB2 receptors but not CB1.
These human tumor cells were also susceptible to apoptosis induced by THC ...
Culture of primary acute lymphoblastic leukemia cells with THC in vitro reduced cell viability and induced apoptosis. Together, the current data demonstrate that CB2 cannabinoid receptors expressed on malignancies of the immune system may serve as potential targets for the induction of apoptosis. Also, because CB2 agonists lack psychotropic effects, they may serve as novel anticancer agents to selectively target and kill tumors of immune origin.

Introduction

... Cells of the immune system express high levels of CB2 receptors. ... If CB2 receptor agonists can induce apoptosis in transformed immune cells, it could lead to development of a novel class of anticancer agents. In the current study, we investigated this possibility by using both murine and human leukemia and lymphoma lines as well as primary acute lymphoblastic leukemia (ALL) cells. ...
We demonstrate that ligation of CB2 receptors can induce apoptosis in a wide range of cancers of immune-cell origin. Furthermore, we demonstrate that THC can inhibit the growth of murine lymphoma cells in vivo by inducing apoptosis and cure approximately 25% of the mice bearing the tumor.
Together, the current data suggest that CB2 agonists that are devoid of psychotropic effects may constitute a novel and effective modality to treat malignancies of the immune system.

http://bloodjournal.hematologylibrary.org/content/100/2/627.long

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Leukemia & Lymphoma
October 2003

γ-Irradiation Enhances Apoptosis Induced by Cannabidiol, a Non-psychotropic Cannabinoid, in Cultured HL-60 Myeloblastic Leukemia Cells

Two non-psychotropic cannabinoids, cannabidiol (CBD) and cannabidiol-dimethylheptyl (CBD-DMH), induced apoptosis in a human acute myeloid leukemia (AML) HL-60 cell line. ...

A dose dependent increase of apoptosis was noted, reaching 61 and 43% with 8 μg/ml CBD and 15 μg/ml CBD-DMH, respectively, after a 24 h treatment. Prior exposure of the cells to γ-irradiation (800 cGy) markedly enhanced apoptosis, reaching values of 93 and 95%, respectively. Human monocytes from normal individuals were resistant to either cannabinoids or γ-irradiation. Caspase-3 activation was observed after the cannabinoid treatment, and may represent a mechanism for the apoptosis. Our data suggest a possible new approach to treatment of AML.

http://informahealthcare.com/doi/abs/10.1080/10428190310001039l7

Blood: Journal of the American Society of Hematology
February 2005

Cannabis-induced cytotoxicity in leukemic cell lines: the role of the cannabinoid receptors and the MAPK pathway

Abstract

Δ9-Tetrahydrocannabinol (THC) is the active metabolite of cannabis. THC causes cell death in vitro through the activation of complex signal transduction pathways. However, the role that the cannabinoid 1 and 2 receptors (CB1-R and CB2-R) play in this process is less clear. We therefore investigated the role of the CB-Rs in mediating apoptosis in 3 leukemic cell lines ... to establish further the mechanism of cell death. ... We have shown that THC is a potent inducer of apoptosis, even at 1 × IC50 (inhibitory concentration 50%) concentrations and as early as 6 hours after exposure to the drug. These effects were seen in leukemic cell lines ... as well as in peripheral blood mononuclear cells. Additionally, THC did not appear to act synergistically with cytotoxic agents such as cisplatin. One of the most intriguing findings was that THC-induced cell death was preceded by significant changes in the expression of genes ... Both apoptosis and gene expression changes were altered independent of p53 and the CB-Rs.

http://bloodjournal.hematologylibrary.org/content/105/3/1214.long

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LUNG CANCER:

Biochemical and Biophysical Research Communications
December 7, 2007

Cannabinoid receptor agonists are mitochondrial inhibitors: A unified hypothesis of how cannabinoids modulate mitochondrial function and induce cell death

Abstract

Time-lapse microscopy of human lung cancer (H460) cells showed that the endogenous cannabinoid anandamide (AEA), the phyto-cannabinoid Δ-9-tetrahydrocannabinol (THC) and a synthetic cannabinoid HU 210 all caused morphological changes characteristic of apoptosis. ... In rat heart mitochondria, all three ligands caused significant decreases in oxygen consumption and mitochondrial membrane potential. THC and HU 210 caused significant increases in mitochondrial hydrogen peroxide production, whereas AEA was without significant effect. All three ligands induced biphasic changes in either mitochondrial complex I activity and/or mitochondrial complex II–III activity. These data demonstrate that AEA, THC, and HU 210 are all able to cause changes in integrated mitochondrial function, directly, in the absence of cannabinoid receptors.

http://www.sciencedirect.com/science/article/pii/S0006291X07021079

LYMPHOMA:

FEBS Letters (Federation of European Biochemical Societies)
December 2005

Cannabinoid receptor ligands mediate growth inhibition and cell death in mantle cell lymphoma

Abstract

We have earlier reported overexpression of the central and peripheral cannabinoid receptors CB1 and CB2 in mantle cell lymphoma (MCL), a B cell non-Hodgkin lymphoma. In this study, treatment with cannabinoid receptor ligands caused a decrease in viability of MCL cells, while control cells lacking CB1 were not affected. Interestingly, equipotent doses of the CB1 antagonist ... and the CB1/CB2 agonist anandamide inflicted additive negative effects on

viability. Moreover, treatment with the CB1/CB2 agonist ... caused a decrease in long-term growth of MCL cells in culture. Induction of apoptosis ... contributed to the growth suppressive effect ... Our data suggest that cannabinoid receptors may be considered as potential therapeutic targets in MCL.

http://www.ncbi.nlm.nih.gov/pubmed/16337199

Molecular Pharmacology
November 2006

Cannabinoid Receptor-Mediated Apoptosis Induced by R(+)-Methanandamide and Win55,212-2 Is Associated with Ceramide Accumulation and p38 Activation in Mantle Cell Lymphoma

Abstract

We have recently shown that cannabinoids induce growth inhibition and apoptosis in mantle cell lymphoma (MCL), a malignant B-cell lymphoma that expresses high levels of cannabinoid receptor types 1 and 2 (CB1 and CB2). In the current study, the role of each receptor and the signal transduction triggered by receptor ligation were investigated. Induction of apoptosis after treatment with the synthetic agonists ... was dependent on both cannabinoid receptors ... Taken together, these results suggest that concurrent ligation of CB1 and CB2 ... induces apoptosis via a sequence of events in MCL cells: accumulation of ceramide, phosphorylation of p38, depolarization of the mitochondrial membrane, and caspase activation. Although induction of apoptosis was observed in both MCL cell lines and primary MCL, normal B cells remained unaffected. The present data suggest that targeting CB1/CB2 may have therapeutic potential for the treatment of mantle cell lymphoma.

http://molpharm.aspetjournals.org/content/70/5/1612.long

International Journal of Cancer
September 2008

Expression of cannabinoid receptors type 1 and type 2 in non-Hodgkin lymphoma: Growth inhibition by receptor activation

Abstract

Endogenous and synthetic cannabinoids exert antiproliferative and proapoptotic effects in various types of cancer and in mantle cell lymphoma (MCL). In this study, we evaluated the expression of cannabinoid receptors type 1 and type 2 (CB1 and CB2) in non-Hodgkin lymphomas of B cell type ... A majority of the lymphomas expressed higher mRNA levels of CB1 and/or CB2 as compared to reactive lymphoid tissue. With the exception of MCL, which uniformly overexpresses both CB1 and CB2, the levels of cannabinoid receptors within other lymphoma entities were highly variable, ranging from 0.1 to 224 times the expression in reactive lymph nodes. ... In functional studies using MCL ... chronic lymphatic leukemia (CLL) and plasma cell leukemia cell lines, the stable anandamide analog ... induced cell death only in MCL and CLL cells, which overexpressed both cannabinoid receptors, but not in BL. In vivo treatment ... caused a significant reduction of tumor size ... in mice xenografted with human MCL. Together, our results suggest that therapies using cannabinoid receptor ligands will have efficiency in reducing tumor burden in malignant lymphoma overexpressing CB1 and CB2.

http://onlinelibrary.wiley.com/doi/10.1002/ijc.23584/abstract

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Molecular Cancer Research
July 2009

Potentiation of Cannabinoid-Induced Cytotoxicity in Mantle Cell Lymphoma through Modulation of Ceramide Metabolism

Abstract

Ceramide levels are elevated in mantle cell lymphoma (MCL) cells following treatment with cannabinoids. Here, we investigated the pathways of ceramide accumulation in the MCL cell line ... Furthermore, this is the first study were the cytotoxic effect of a cannabinoid is enhanced by modulation of ceramide metabolism.

http://mcr.aacrjournals.org/content/7/7/1086.long

PROSTATE CANCER:

Cancer Research
March 2005

Abstract

Cannabinoids, the active components of Cannabis sativa Linnaeus (marijuana) and their derivatives have received renewed interest in recent years due to their diverse pharmacologic activities such as cell growth inhibition, anti-inflammatory effects and tumor regression. Here we show that expression levels of both cannabinoid receptors, CB1 and CB2, are significantly higher in CA-human papillomavirus-10 (virally transformed cells derived from adenocarcinoma of human prostate tissue) ... than in human prostate epithelial ... (virally transformed cells derived from normal human prostate tissue) cells. ... Our results suggest that WIN-55,212-2 or other non–habit-forming cannabinoid receptor agonists could be developed as novel therapeutic agents for the treatment of prostate cancer.

http://cancerres.aacrjournals.org/content/65/5/1635.long

RHABDOMYOSARCOMA:

Molecular Cancer Therapeutics
July 2009

Cannabinoid receptor 1 is a potential drug target for treatment of translocation-positive rhabdomyosarcoma

Abstract

Gene expression profiling has revealed that the gene coding for cannabinoid receptor 1 (CB1) is highly up-regulated in rhabdomyosarcoma biopsies ... Because cannabinoid receptor agonists are capable of reducing proliferation and inducing apoptosis in diverse cancer cells such as glioma, breast cancer, and melanoma, we evaluated whether CB1 is a potential drug target in rhabdomyosarcoma. Our study shows that treatment with the cannabinoid receptor agonists HU210 and Δ9-tetrahydrocannabinol lowers the viability of translocation-positive rhabdomyosarcoma cells through the induction of apoptosis. ... Finally, treatment of xenografts with HU210 led to a significant suppression of tumor growth in vivo. These results support the notion that cannabinoid receptor agonists could represent a novel targeted approach for

treatment of translocation-positive rhabdomyosarcoma.

http://mct.aacrjournals.org/content/8/7/1838.long

STOMACH CANCER:

Journal of Surgical Research
July 2009

Abstract

Orally applicable Δ9-tetrahydrocannabinol and its synthetic derivatives have been used as antiemetic drugs during chemotherapy in cancer patients. However, it is not well known how cannabinoids influence the effects of chemotherapeutic agents on malignant tumors. In this study, we investigated how the endogenous cannabinoid anandamide (AEA) changes the effect of paclitaxel on gastric cancer cell lines. In the human gastric cancer cell line, HGC-27, which express cannabinoid receptor 1 (CB1), AEA stimulated proliferation at concentrations under 1 μM, while it strongly suppressed proliferation through the induction of apoptosis at 10 μM. ... When AEA was used with paclitaxel, AEA at 10 μM synergistically enhanced the cytotoxic effect of paclitaxel, whereas it showed no significant effect at lower concentrations. ... Our results suggest that cannabinoids could be a good palliative agent for cancer patients receiving paclitaxel.

http://www.journalofsurgicalresearch.com/article/S0022-4804(08)00452-6/abstract

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Journal of Cellular Biology
May 2010

Effect of a synthetic cannabinoid agonist on the proliferation and invasion of gastric cancer cells

Abstract

Although cannabinoids are associated with antineoplastic activity in a number of cancer cell

types, the effect in gastric cancer cells has not been clarified. In the present study, we investigated the effects of a cannabinoid agonist on gastric cancer cell proliferation and invasion. The cannabinoid agonist ... inhibited the proliferation of human gastric cancer cells in a dose-dependent manner and that this effect was mediated partially by the CB1 receptor. ... Our results open the possibilities in using cannabinoids as a new gastric cancer therapy

http://onlinelibrary.wiley.com/doi/10.1002/jcb.22540/abstract