THE CANNABIS CURE FOR CANCER

by Dr. Mark Sircus

There should be no more confusion about whether or not marijuana is effective for cancer patients. Medical marijuana is chemotherapy, natural style, for all cancer patients. The two *forms of hemp oil*, one with THC and CBD and the other CBD alone, provide the body with chemotherapeutics without danger and staggering side effects. We present here a quick overview of the science that backs up the assertion that every cancer patient and every oncologist should put medical marijuana on their treatment maps.

What you will see in this article is reference to many scientific studies that are all viewable on government sites. The United States government is pathetic in its dishonesty about medical marijuana, both believing in it and holding patents for its medical use, and claiming at the same time that it has no medical use. The US government and still many states would rather throw innocent people in jail for using medical marijuana than be honest about how much it can help people recover from cancer and other diseases.

Below are summaries of just some of the scientific research out there that confirms that medical marijuana will help people cure cancer.

One of the most exciting areas of current research in the cannabinoid field is the study of the potential application of these compounds as antitumor drugs. 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. [1],[2] The CBD concentrations effective at inhibiting Id-1 expression correlated with those used to inhibit the proliferative and invasive phenotype of breast cancer cells. Of the five cannabinoids tested: cannabidiol, cannabigerol, cannnabichromene; cannabidiol-acid and THC-acid, it was found that cannabidiol is the most potent inhibitor of cancer cell growth. Taken together, these data might set the bases for a cannabinoid therapy for the management of breast cancer. [3]

Results show that Δ9-tetrahydrocannabinol reduces tumor growth, tumor number, and the amount/severity of lung metastases in MMTV-neu mice.[4] Cannabinoids induce ICAM-1, thereby conferring TIMP-1 induction and subsequent decreased cancer cell invasiveness and inhibits lung cancer invasion and metastasis.[5]

Non-small cell lung cancer (NSCLC) is the leading cause of cancer deaths worldwide. Researchers have observed expression of CB1 (24%) and CB2 (55%) in NSCLC patients. They have also shown that the treatment of NSCLC cell lines (A549 and SW-1573) with CB1/CB2- and CB2-specific agonists Win55,212-2 and JWH-015, respectively, significantly attenuated random as well as growth factor-directed in vitro chemotaxis and chemoinvasion in these cells. [6]

Researchers in lung cancers also reported that they observed significant reduction in focal adhesion complex, which plays an important role in cancer migration. Medical marijuana significantly inhibited in vivo tumor growth and lung metastasis (□50%). [7]

In research on pancreatic cancer it was found that cannabinoids lead to apoptosis of pancreatic tumor cells via a CB2 receptor and *de novo* synthesized ceramide-dependent up-regulation of p8 and the endoplasmic reticulum stress–related genes *ATF-4 and TRB3*. These findings may set the basis for a new therapeutic approach for the treatment of pancreatic cancer, as reported by the National Cancer Institute.

Prostate cancer cells possess increased expression of both cannabinoid 1 and 2 receptors, and stimulation of these results in decrease in cell viability, increased apoptosis, and decreased androgen receptor expression and prostate-specific antigen excretion. [8]

In colorectal carcinoma cell lines, cannabidiol protected DNA from oxidative damage, increased endocannabinoid levels and reduced cell proliferation in a CB(1)-, TRPV1- and PPARγ-antagonists sensitive manner. It is concluded that cannabidiol exerts chemopreventive effect in vivo and reduces cell proliferation through multiple mechanisms. [9]

Ovarian cancer represents one of the leading causes of cancer-related deaths for women and is the most common gynecologic malignancy. Results with medical marijuana support a new therapeutic approach for the treatment of ovarian cancer. It is also conceivable that with available cannabinoids as lead compounds, non-habit forming agents that have higher biological effects could be developed. [10]

Examination of a number of human leukaemia and lymphoma cell lines 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. [11]Plant-derived cannabinoids, including Delta9-tetrahydrocannabinol (THC), induce apoptosis in leukemic cells. [12]

Cannabinoid-treated tumors showed an increased number of apoptotic cells. This was accompanied by impairment of tumor vascularization, as determined by altered blood vessel morphology and decreased expression of proangiogenic factors (VEGF, placental growth factor, and angiopoietin. Abrogation of EGF-R function was also observed in cannabinoid-treated tumors. [13] These results support a new therapeutic approach for the treatment of skin tumors.

Hepatocellular carcinoma (HCC) is the third cause of cancer-related death worldwide. When these tumors are in advanced stages, few therapeutic options are available. In this study, the effects of cannabinoids–a novel family of potential anticancer agents–on the growth of HCC was investigated. It was found that Δ(9)-tetrahydrocannabinol (Δ(9)-THC, the main active component of Cannabis sativa) and JWH-015 (a cannabinoid receptor 2 (CB(2)) cannabinoid receptor-selective agonist) reduced the viability of the human HCC cell lines Cannabinoids were able to inhibit tumor growth and ascites in an orthotopic model of HCC xenograft. [14] These findings may contribute to the design of new therapeutic strategies for the management of HCC.

Both cholangiocarcinoma cell lines and surgical specimens from cholangiocarcinoma patients expressed cannabinoid receptors. THC inhibited cell proliferation, migration and

invasion, and induced cell apoptosis. THC also decreased actin polymerization and reduced tumor cell survival in anoikis assay. pMEK1/2 and pAkt demonstrated the lower extent than untreated cells. Consequently, THC is potentially used to retard cholangiocarcinoma cell growth and metastasis.[15]

THC is a potent inducer of apoptosis, even at 1 x IC(50) (inhibitory concentration 50%) concentrations and as early as 6 hours after exposure to the drug. These effects were seen in leukemic cell lines (CEM, HEL-92, and HL60) as well as in peripheral blood mononuclear cells.[16]Cannabinoids represent a novel class of drugs active in increasing the lifespan in mice carrying Lewis lung tumors and decreasing primary tumor size. [17]

Research has also found a cannabidiol-driven impaired invasion of human cervical cancer (HeLa, C33A) and human lung cancer cells (A549) that was reversed by antagonists to both CB(1) and CB(2) receptors as well as to transient receptor potential vanilloid 1 (TRPV1). The decrease of invasion by cannabidiol appeared concomitantly with up-regulation of tissue inhibitor of matrix metalloproteinases-1 (TIMP). The 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. [18]

A new anticancer quinone (HU-331) was synthesized from cannabidiol. It shows high efficacy against human cancer cell lines in vitro and against in vivo tumor grafts in nude mice. Two non-psychotropic cannabinoids, cannabidiol (CBD) and cannabidiol-dimethylheptyl (CBD-DMH),induced apoptosis in a human acute myeloid leukemia (AML) HL-60 cell line. [19]

Other studies show a synthetic and potent cannabinoid receptor agonist, investigated in hepatoma HepG2 cells and a possible signal transduction pathway that is proposed, indicates a potential positive role in liver cancer. [20] Cannabinoids have been found to counteract intestinal inflammation and colon cancer. [21]

The control of cellular proliferation has become a focus of attention as opening new therapeutic possibilities for the use of cannabinoids as potential antitumor agents. [22]Cannabinoid treatment inhibits angiogenesis of gliomas in vivo. [23] Remarkably,

cannabinoids kill glioma cells selectively and can protect non-transformed glial cells from death. These and other findings reviewed here might set the basis for a potential use of cannabinoids in the management of gliomas. Other confirming studies may provide the basis for a new therapeutic approach for the treatment of malignant gliomas. [24]

In summary:

Cannabinoids are found to exert anti-cancer effects in a number of ways and in a variety of tissues.

- Triggering cell death, through a mechanism called apoptosis
- Stopping cells from dividing
- Preventing new blood vessels from growing into tumours
- Reducing the chances of cancer cells spreading through the body, by stopping cells from moving or invading neighbouring tissue
- Speeding up the cell's internal 'waste disposal machine' – a process known as autophagy – which can lead to cell death

All these effects are thought to be caused by cannabinoids locking onto the CB1 and CB2 cannabinoid receptors. Almost daily we are seeing new or evidence that cannibinoids can be used to great benefit in cancer treatment of many types.

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PATIENTS SUFFER INVASIVE TREATMENTS FOR HARMLESS CANCERS

Australians are increasingly being diagnosed with cancers that will do them no harm if left undetected or untreated, exposing them to unnecessary surgeries and chemotherapy, says a new study published in the *Medical Journal of Australia*.

The research, led by Professor Paul Glasziou, the Director of the Institute for Evidence-Based Healthcare at Bond University, drew on data from the Australian Institute of Health and Welfare to compare how the lifetime risk of five cancers had changed between 1982 and 2012.

The study shows compared to 30 years ago, Australians are much more likely to experience a cancer diagnosis in their lifetime.

The figures suggest that in 2012 24 percent of cancers or carcinomas in men were overdiagnosed. These included 42 percent of prostate cancers, 42 percent of renal cancers, 73 percent of thyroid cancers and 58 percent of melanomas.

For women, 18 percent of cancers or carcinomas were overdiagnosed, including 22 percent of breast cancers, 58 percent of renal cancers, 73 percent of thyroid cancers and 58 percent of melanomas.

The figures are significant because of the harm that can occur from cancer treatment of patients who would never have had symptoms in their lifetime.

"Cancer treatments such as surgery, radiotherapy, endocrine and chemotherapy carry risks of physical harms," the authors of the study reported.

"In the absence of overdiagnosis, these harms are generally considered acceptable.

"In the context of overdiagnosed cancers, however, affected individuals cannot benefit but can only be harmed by these treatments."

The authors also refer to separate studies showing overdiagnosis could be linked to psychological problems.

"For example, men's risk of suicide appears to increase in the year after receiving a prostate cancer diagnosis."

The new study, which was led by Professor Glasziou in conjunction with co-authors Professor Alexandra Barratt and Associate Professor Katy Bell of University of Sydney, Associate Professor Mark Jones of Bond University, and Dr. Thanya Pathirana of Griffith University, calls for urgent policy changes to address overdiagnosis.

Professor Glasziou said increasing rates of diagnosis were a result of improvements and wider use of testing and screening.

"The problem is that some screening identifies abnormal cells that look like cancer but don't behave like cancer. However, reducing that problem is not easy, as some types of screening are important".

"While much of the overdiagnosis is due to screening, many overdiagnosed cancer cases are incidental findings, that is, the patient is being tested for something else when the cancer is detected," Professor Glasziou said.

"Getting the balance right between too little and too much screening and testing will not be easy, but this is an important step.

It is the first time that the risk of overdiagnosis has been quantified across five cancers, anywhere in the world."