



Marijuana Research Findings

There seems to be a growing consensus on the medical and mental health concerns associated with smoking marijuana. The first report on the association between marijuana and lung cancer appeared as a news brief on the Anselm Ministries web site in 2006. Beginning in the summer of 2007, a series of research studies into the health risks of smoking marijuana began to appear in various medical and research journals. They are gathered here for your convenience. I will periodically update this article as new research studies on marijuana become known to me.

Brain Scans and Pot

A 2012 study by British researchers suggests that marijuana can mean different things to different people. Some people mellow out, while others become paranoid and anxious. A unique brain scan study suggests that two ingredients of marijuana tetrahydrocannabinol (THC) and cannabidiol (CBD) may work independently to achieve these effects.

The new study used functional MRI (fMRI) scans, which track brain activity in real time. It found that ingesting THC prompted a significant increase in paranoid and delusional thinking by boosting the brain's responses to otherwise insignificant stimuli, while reducing response to what would typically be seen as significant. The more that "normal" brain responses were set off-kilter, the more severe the paranoid or even psychotic the reaction.

The effect of CBD was nearly opposite. Ingesting CBD appeared to prompt brain activity linked to appropriate responses to significant stimuli in the environment.

According to these findings, Dr. Sagnik Bhattacharyya suggested that marijuana played both a good and a bad role in the context of psychosis. CBD may have potential use for the treatment of psychosis, while THC raises the risk for developing psychotic complications.

Dr. Joseph Coyle, a professor of psychiatry and neuroscience at Harvard Medical School, said the current study helps to “connect the dots” in understanding the effects of marijuana.

What we're talking about here is the kind of perception, in this case prompted by marijuana, that leads a person to think that other people who are just talking in the subway are all actually talking about him," he noted. "Or people who are just tipping their hat for no reason are actually doing so specifically about him. And so this paper strikes me as important, because it actually looks at this kind of increased anxiety and increased hyper-alertness which are major factors in psychosis -- and then finds out what's going on in the brain among people who experience them.

To read the original article this was taken from go to:

<http://www.philly.com/philly/health/136832408.html?cmpid=15585797>

Marijuana and Brain Shrinkage

Australian researchers reported in the June 2008 edition of the *Archives of General Psychiatry* that long-term use of marijuana may cause two areas of the brain, the hippocampus and amygdala, to shrink in size. Brain scans of 15 men (an average age of 39.8 years) who had smoked at least five joints of marijuana daily for over 10 years (an average duration of 19.7 years) showed that their hippocampus was 12 percent smaller in volume, while their amygdala was 7 percent smaller, when compared to 16 men who were not marijuana users. The study also found the heavy cannabis users earned lower scores than the nonusers in a verbal learning task; when trying to recall a list of 15 words.

The hippocampus regulates memory and emotion, while the amygdala plays a critical role in fear responses (such as immobilization, rapid heart beat or increased respiration). It also modulates emotional arousal surrounding a memory, and therefore our recollection of the original event.

The marijuana users were more likely than nonusers to exhibit mild signs of psychotic disorders, but not enough to be formally diagnosed with any such disorder, the researchers said. Additionally, about half of the marijuana users reported experiencing some form of paranoia and social withdrawal, while only one of the nonusers reported such symptoms. The heavy marijuana users reported that they had used other illicit drugs less than 10 times, according to the researchers.

Murat Yucel of ORYGEN Research Centre and the University of Melbourne, who led the study, said that: “These findings challenge the widespread perception of cannabis as having limited or no harmful effects on (the) brain and behavior.” He added that everyone is vulnerable to the potential problems, namely memory problems and psychiatric symptoms “if they use heavily enough and for long enough.”

Bruce Mirken, a spokesman for Marijuana Policy Project (a group supporting legal sales and regulation of marijuana) challenged the study’s findings, particularly because they were based on men who were such heavy, long-term users. “This study says nothing about moderate or occasional users, who are the vast majority -- and the (study) even acknowledges this.”

For more information on this topic, see the abstract for “Regional Brain Abnormalities Associated With Long-term Heavy Cannabis Use” at: <http://archpsyc.ama-assn.org/cgi/content/short/65/6/694>. The above information was also based upon a June 2nd 2008 report from Reuters, found at: <http://www.reuters.com/article/newsOne/idUSN0227147420080602?sp=true>.

Marijuana Withdrawal

Cannabis (marijuana) is the most widely used illicit drug in the U.S. and world. Since the early 1990s, drug and alcohol treatment admissions for marijuana have increased to the point that they are now comparable to admissions for cocaine or heroin. Although there is clear evidence of a cannabis withdrawal syndrome, its diagnosis is excluded from the DSM IV reportedly because its “clinical significance is uncertain.”

A number of findings have challenged that assessment. A majority adults and adolescents seeking outpatient treatment for cannabis dependence have difficulty achieving initial abstinence. Many of them complain that withdrawal contributes to their problems quitting; and they report using marijuana and other substances to alleviate the withdrawal symptoms. These withdrawal symptoms are observable by others, and comments by these observers suggest that the symptoms can be disruptive of daily living.

Most symptoms of marijuana withdrawal begin within the first 24 hours of cessation, peak within the first week and last between one and two weeks. The symptoms generally include: increased anger and aggression, anxiety, depressed mood, irritability, restlessness, sleep difficulty and strange dreams, decreased appetite, and weight. Headaches, physical tension, sweating, stomach pain, and general physical discomfort have also been reported, but are less common.

A research study by Vandrey et al. (2008), “A within-subject comparison of withdrawal symptoms during abstinence from cannabis, tobacco, and both substances”, investigated the withdrawal syndrome of individuals who were simultaneously smokers of tobacco and marijuana. Participants had to have a pattern of regular, heavy marijuana use (at least 25 days per month); smoke at least 10 cigarettes daily; and report maintaining this usage pattern for at least six months prior to the study.

The study compared the abstinence effects associated with cessation from cannabis only, tobacco only, and both cannabis and tobacco combined over a 5-day period of abstinence. Participants attended 30 minute sessions each weekday to obtain self-reported affective and behavioral measures, physiological measures, and staff-observed urine and breathe samples.

Results found that the following items from a Withdrawal Symptom Checklist were rated significantly higher by all the study’s participants during the abstinence period: anxiety/nervousness, decreased appetite, depressed mood, difficulty concentrating, feverish, increased anger, irritability, physical discomfort, restlessness, shakiness, sleep difficulty, stomach pain, strange dreams, sweating, and tension. Significant withdrawal effects were observed for each of these symptoms except decreased appetite in the dual

abstinence condition. However, clear differences in withdrawal symptoms were evident between the cannabis only and tobacco only groups.

Withdrawal symptoms reported by the cannabis abstinence group, but not the tobacco abstinence group included: decreased appetite, difficulty concentrating and strange dreams. Withdrawal symptoms reported by the tobacco abstinence group, but not the cannabis abstinence group included: increased anger, physical discomfort, restlessness, shakiness and tension. Withdrawal symptoms common to both groups were: anxiety/nervousness, irritability and sleep difficulty.

Significant condition by day interactions were observed for ratings of difficulty concentrating, increased aggression, increased anger, irritability and sleep difficulty. Post hoc analyses indicated that ratings of difficulty concentrating were greater on days 4 and 5 in the tobacco and dual abstinence conditions compared with the cannabis abstinence condition. Increased aggression, increased anger, and irritability were greater in the dual abstinence condition compared with the cannabis and tobacco abstinence conditions on day 2. Increased aggression was also greater in the dual abstinence condition compared with the cannabis abstinence condition on day 4. Sleep difficulty was greater in the dual abstinence condition compared with the tobacco abstinence condition on days 2 and 4.

Overall withdrawal discomfort and individual symptom severity during cannabis abstinence was similar to that observed during tobacco abstinence in the present study. The differences observed between the cannabis and tobacco abstinence conditions were mostly for symptoms expected to differ based on previous studies. Exceptions to this were that ratings of anger and craving appeared to be higher during tobacco abstinence compared with cannabis abstinence.

The authors concluded that the cannabis and tobacco withdrawal syndromes are of comparable severity. Their results were similar in magnitude to that observed in prior studies using similar measures; and consistent with their previously reported research in 2005, “A cross–study comparison of cannabis and tobacco withdrawal,” published in *The American Journal on Addictions*.

At the 2008 annual meeting of the American Psychiatric Association, National Institute on Drug Abuse (NIDA) researcher David Gorelick reported that in a study of nearly 500 marijuana smokers, a total of 42.4 percent of the studies participants

experienced at least one symptom of withdrawal when they tried to quit; and 78.4 percent of those who reported withdrawal symptoms said they started smoking marijuana again to reduce them. This equaled about 33.3 percent of the total participants in the study.

The study involved 469 marijuana smokers, ages 18 to 64, none of whom suffered from recognized psychiatric disorders. About one in four reported smoking marijuana more than 10,000 times; approximating 27 years of daily use. More than half included in the study smoked more than 2,000 times. "These were heavy users," Gorelick says. He added that heavy marijuana users should be prepared to experience an uncomfortable withdrawal syndrome when they try to quit. Gorelick predicted that cannabis withdrawal syndrome will be recognized as a psychiatric disorder in the next edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM), which is due out in 2012.

Reports on the Gorelick study can be found at CBS News:

<http://www.cbsnews.com/stories/2008/05/08/health/webmd/main4080584.shtml>

and Join Together:

<http://www.jointogether.org/news/research/summaries/2008/heavy-marijuana-users.html>

To review the original article by Vandrey, go to:

[http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T63-4P83HJN-1&_user=10&_coverDate=01%2F01%2F2008&_rdoc=8&_fmt=summary&_orig=browse&_srch=doc-info\(%23toc%235019%232008%23999079998%23676094%23FLA%23display%23Volume\)&_cdi=5019&_sort=d&_docanchor=&_ct=43&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=ad092ca7aaa2ac816e89dfd4b110d738](http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T63-4P83HJN-1&_user=10&_coverDate=01%2F01%2F2008&_rdoc=8&_fmt=summary&_orig=browse&_srch=doc-info(%23toc%235019%232008%23999079998%23676094%23FLA%23display%23Volume)&_cdi=5019&_sort=d&_docanchor=&_ct=43&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=ad092ca7aaa2ac816e89dfd4b110d738)

Marijuana and Lung Cancer

An investigation done by Case Western Reserve University researchers into the association between marijuana smoking and lung cancer found that marijuana may cause precancerous changes in the lungs.

Various research studies since the 1960s have investigated whether smoking marijuana causes lung cancer, but the overall conclusion of these studies is unclear. Given that marijuana use is the most commonly used drug worldwide (according to the 2006 *World Drug Report*), and that over the last decade cannabis use is outpacing increases for opiates and cocaine, further efforts to investigate this potentially fatal association need to occur.

The Case Western researchers reviewed 19 previous studies from 1965 through October of 2005 that examined premalignant or cancerous changes in the lungs of persons who smoked marijuana. Within studies that examined these lung cancer risk factors, they found a link between marijuana use and certain changes in lung tissue that promote cancer. These changes in lung tissue included oxidative stress, dysfunction of tumor-fighting cells, changes in tissue structure and DNA alterations. The changes among marijuana smokers were also greater than those among tobacco smoking or nonsmoking control subjects. However, after adjusting for concurrent tobacco use among the research participants of other observationally-based studies, the researchers failed to demonstrate a significant association between marijuana smokers and a diagnosis of lung cancer.

They noted several methodological problems with these observational studies that challenged the validity of generalizing their failure to show an association between marijuana smoking and lung cancer to others. One glaring error was the overall youth of the participants, which precluded sufficient lag time for lung cancer to have developed in the marijuana smoking participants. Additional concerns included a selection bias, small sample sizes, an inconsistent measurement of marijuana exposure, and the lack of a standardized method for diagnosing the presence of lung cancer among participants of the various studies. The researchers concluded:

Although observational studies have not shown a substantive marijuana smoking–lung cancer association, these studies are fraught with serious methodologic limitations. Therefore, the combination of the widespread use of marijuana, potential marijuana-related health implications outlined in this review, and studies evaluating lung premalignant alterations supporting a biologically plausible association between marijuana smoking–lung cancer association. . . . Given the prevalence of marijuana smoking and studies

predominantly supporting biological plausibility of an association of marijuana smoking with lung cancer on the basis of molecular, cellular, and histopathologic findings, physicians should advise patients regarding potential adverse health outcomes until further rigorous studies are performed that permit definitive conclusions.

A pdf copy of this study, “The Association Between Marijuana Smoking and Lung Cancer,” can be found in the July 10, 2006 issue of the *Archives of Internal Medicine* (vol. 166, no. 13) or at: <http://archinte.ama-assn.org/cgi/reprint/166/13/1359>.

Marijuana and Lung Damage

Researchers at the Medical Research Institute in New Zealand reported that smoking one joint caused lung damage equivalent to smoking 2.5 to five cigarettes in rapid succession.

The study originally sought to investigate whether smoking marijuana put smokers at a greater risk of developing emphysema. Participants were placed in one of four different groups: marijuana smokers, tobacco smokers, smokers of both marijuana and tobacco and nonsmokers. All volunteers were given tests and x-ray scans to assess the health status of their lungs and airways. All smokers in the study complained of coughs and wheezing, but only the tobacco smokers showed any signs of emphysema. Smoking marijuana was found to be associated with the impairment of large airways function leading to hyperinflation and airflow obstruction. The extent of lung damage found was directly related to the number of joints smoked; and the researchers calculated that “one joint of cannabis was similar to 2.5 to five tobacco cigarettes in terms of causing airflow obstruction.” They speculated the difference was due to how cannabis was smoked: usually without a filter and with its smoke at a higher temperature.

Further research at Medical Research Institute in New Zealand reported in January of 2008 that smoking one joint had the equivalent risk of smoking 20 cigarettes in

terms of lung cancer risk. Cannabis smoke contains twice the level of carcinogens found in tobacco smoke. “Cannabis smokers end up with five times more carbon monoxide in their bloodstream (than tobacco smokers),” said Richard Beasley of the Medical Research Institute of New Zealand. Within a high–exposure group for lung cancer, individuals were interviewed in an attempt to find the primary risk factors for the disease. After adjusting for variables including cigarette smoking, the risk of lung cancer in this high–exposure group rose by 5.7 times for patients for patients who smoked more than a joint a day for 10 years, or two joints a day for 5 years. Beasley concluded that long–term cannabis smoking increases the risk of contracting lung cancer; and he predicted that in the near future we could see an “epidemic” of lung cancers in many countries associated with the carcinogens in marijuana because of the increasing use of marijuana among young adults worldwide.

See Aldington, S., et al. (2007) “The Effects of Cannabis on Pulmonary Structure, Function and Symptoms.” *Thorax*, published online July 31, 2007; doi: 10.1136/thx.2006.077081. Also look at an article published online July 31, 2007 in *The Guardian*, www.guardian.co.uk/drugs/Story/0,,2138260,00.html.

“Cannabis bigger cancer risk than cigarettes: study:”

http://news.yahoo.com/s/nm/20080129/hl_nm/cancer_cannabis_dc;_ylt=A0WTcU2lJ59HJzkBOQWsONUE

Marijuana smokers are also at greater risk of developing bullous lung disease, and exhibit symptoms at a younger age (approximately 20 years earlier) than tobacco smokers. Bullous lung disease is a rare type of respiratory distress usually found with advanced cases of emphysema. It functionally impairs typical pulmonary activity and leads to diminished exercise capacity and even acute respiratory distress. Most patients with bullae have a significant cigarette smoking history, although cocaine smoking, marijuana smoking and inhaled fiberglass exposure have been shown to be associated with emphysematous lung bullae.

“Bullous Lung Disease Due to Marijuana,”

<http://www.blackwell-synergy.com/doi/abs/10.1111/j.1440-1843.2007.01186.x>

“Marijuana Smokers Face Breathing Problems at Young Age,”

<http://www.jointogether.org/news/research/summaries/2008/marijuana-smokers-face.html>

Marijuana and Psychosis

A June 2007 article published in the British medical journal *Lancet* indicated a relationship between marijuana use and psychosis. The authors reported that their analysis showed the risk of psychosis increased by 40% in people who have used marijuana even one time. An even greater risk was evident with the most frequent users, where the risk of psychosis was 50 to 200% greater than normal.

Theresa Moore and six other researchers completed what was called “the most comprehensive meta-analysis to date of a possible causal relation between cannabis use and psychotic and affective illness.” Moore et al said that “We believe that there is now enough evidence to inform people that using cannabis could increase their risk of developing a psychotic illness later in life.” They also noted some evidence for an association between affective disorders (i.e., depression and anxiety) and marijuana use. “The evidence that cannabis use leads to affective outcomes is less strong than for psychosis but is still of concern.”

The research evidence falls short of concluding a direct causal link between smoking marijuana and psychosis. But Moore states the relationship is strong enough to justify public policy changes, such as educational campaigns to alert people to the possible risks associated with cannabis. “The question of whether cannabis causes psychotic or affective disorders is perhaps the wrong one to be asking, because it will be difficult to answer with any degree of certainty.” The ultimate proof of a causal relationship would require a large-scale placebo-controlled randomized trial of cannabis exposure in healthy young people, with long term follow up. Such a trial cannot be done because of the practical and ethical reasons involved. But there is some evidence in short term studies that cannabis is responsible temporarily for more severe psychotic symptoms, which is suggestive of a longer term causal effect of marijuana use on psychosis.

The evidence is strong enough that the editorial staff of *Lancet* acknowledged how in a 1995 editorial, they said, “The smoking of cannabis, even long term, is not harmful to health.” However, research published since 1995, including Moore’s study, “leads us now to conclude that cannabis use could increase the risk of psychotic illness.”

The *2006 World Drug Report* estimated that 162 million people used cannabis in 2004, equivalent to some 4 per cent of the global population between the ages of 15 and 64. Since the late 1990s, cannabis use has increased by more than 10 per cent at the global level. All available global indicators suggest that the expansion of cannabis use over the last decade was stronger than increases for opiates or cocaine. Increased use of marijuana in early adolescence, when developing brains are susceptible to environmental influences, are a particular concern. About 20% of young people report using marijuana at least once per week.

For the original article: “Cannabis use and risk of psychotic or affective mental health outcomes: a systematic review” by Theresa H M Moore et al. *Lancet* 2007 vol. 370 319-28.

Marijuana and Mental Health

In July of 2007, the Office of National Drug Control Policy released a survey of recent research into the association between marijuana and mental illness. It contained brief summaries of research studies noting evidence for an association between marijuana use and depression as well as schizophrenia.

With regards to depression and suicide, a 16-year study published in 2001 showed that individuals who initially were not depressed and then smoked marijuana after the study began, were four times more likely to be depressed at the time of the follow up assessment. Two separate studies published in 2002 showed evidence of an association between marijuana and depression. One study looked at changes over a 14-year period of time and found that marijuana use was a predictor of individuals developing a major depressive disorder later on in life. Another longitudinal study over 21 years found that marijuana use was associated with depression, suicidal thoughts and suicide attempts.

In 2007, an Australian study of young adults under the age of 21 found a relationship between early initiation and frequency of marijuana use and the symptoms of anxiety or depression. This was regardless of whether there was a personal or family history of mental illness.

Previously I noted the findings of Theresa H. M. Moore et al. in a *Lancet* 2007 article that concluded there was enough evidence “to warn young people that using marijuana could increase their risk of developing a psychotic illness later in life.” Several studies in addition to Moore’s were reviewed in the Office of National Drug Control Policy survey. A 2007 study that compared the brain scans of heavy or long term marijuana users and those of schizophrenics found similarities in the areas of the brain that show cognitive dysfunction, problems with thinking and reasoning. Another brain-scanning study found similar abnormalities in the brains of frequent adolescent marijuana users and adolescents with schizophrenia. These defects were in areas of the brain that continued to develop during adolescence; parts of the brain associated with emotion and higher cognitive functioning such as language, perception, creativity, and problem-solving.

There is also some evidence for genetic predisposition or vulnerability to the effects of marijuana on mental health. “A study published in *Biological Psychiatry* found that as many as one in four people may have a genetic profile that makes marijuana five times more likely to trigger psychotic disorders.” . . . “A 2006 review of six longitudinal studies in five countries found that cannabis use precipitates schizophrenia in individuals who are vulnerable because of a personal or family history of schizophrenia.”

For a copy of the research survey, including an annotated bibliography of the recent research cited in the survey, go to: “The Link Between Marijuana & Mental Illness: A Survey of Recent Research” at:

http://www.theantidrug.com/pdfs/MARIJUANA_AND_MENTAL.pdf

There have been some studies that demonstrate potential health benefits of marijuana use. The following are a few of those. However, the accumulated evidence

noted above suggests that while marijuana may give some pain and depression relief (among other positive outcomes), its risks far outweigh its benefits.

Marijuana and Pain Relief

Marijuana was widely used for pain relief in the 1800s; and several studies have suggested that marijuana can help decrease pain. Research also suggests that marijuana can enhance the effects of opiate pain medications, meaning that effective pain relief can be provided at lower opiate doses if the patient uses marijuana in conjunction with his pain medication.

A study reported in the November issue of the medical journal *Anesthesiology* investigated the pain relief potential of three dosage levels of THC (tetrahydrocannabinol), the psychoactive ingredient in marijuana. Fifteen healthy volunteers were given marijuana to smoke after they had capsaicin (the chemical found in chili peppers) injected under the skin of their arms to induce pain. Some of the volunteers were given a placebo.

Five minutes after smoking the drug, none of the subjects reported any effect on their felt pain. However, 45 minutes later, those subjects who smoked the moderate dose of marijuana reported their pain was much better, while those who used the high dose said their pain was worse. But in partial consolation for their greater discomfort, they did feel “higher” than the moderately dosed volunteers. The low dose of marijuana had no delayed effects on pain relief.

The researchers cautioned that the results of their study should not be taken as supporting the analgesic efficacy of smoking marijuana. They had a small number of subjects, who were all healthy, and who were all experienced marijuana users able to tolerate the highest study dose of cannabis. “It is possible that clinically ill samples, especially cannabis-naive subjects, would have a different analgesic response and incidence of side effects when exposed to the effective dose found in this study.” For an html or pdf version of the article, “Dose-dependent Effects of Smoked Cannabis on Capsaicin-induced Pain and Hyperalgesia in Healthy Volunteers,” go to:

<http://www.anesthesiology.org/pt/re/anes/home.htm;jsessionid=Hvsb5WccgnB9NXCDz3JNqvBwV9JyWkfSP9tTfVZtlXCpk9yFnq8k11821113646!181195629!8091!-1>.

THC and Serotonin Levels

A study published in *The Journal of Neuroscience* found that a synthetic form of THC, the active ingredient in marijuana, was an effective antidepressant at low doses, but worsens symptoms at high doses. It has been known for many years that depletion of the neurotransmitter serotonin in the brain leads to depression, so SSRI antidepressants (Prozac, Celexa and others) work by increasing the levels of serotonin in the brain. This study offers the first evidence that THC, when given in lower doses, will increase serotonin.

The antidepressant and intoxicating effects of cannabis are due to its chemical similarity to natural substances in the brain known as “endo-cannabinoids,” which are released under conditions of high stress or pain, explained Dr. Gabriella Gobbi, the lead researcher in the study. They interact with the brain through structures called cannabinoid CB1 receptors. This study demonstrates for the first time that these receptors have a direct effect on the cells producing serotonin.

Laboratory animals were injected with a synthetic cannabinoid and then given the Forced Swim Test—a test to measure “depression” in animals. The researchers observed that an antidepressant effect of cannabinoids was paralleled by an increased activity in the neurons that produce serotonin. However, increasing the cannabinoid dose beyond a certain point reversed the effects. “Low doses had a potent anti-depressant effect, but when we increased the dose, the serotonin in the rats' brains actually dropped below the level of those in the control group. So we actually demonstrated a double effect: At low doses it increases serotonin, but at higher doses the effect is devastating, completely reversed.”

Dr. Gobbi was prompted to investigate the potential of marijuana as an antidepressant through anecdotal clinical evidence. She noticed that several of her patients suffering from depression used to smoke marijuana. Conversely, there was some evidence that people suffering with multiple sclerosis and AIDS showed an improvement in their mood disorder when they were treated with cannabis. “But there

were no laboratory studies demonstrating the anti-depressant mechanism of action of cannabis.”

The new study doesn't unequivocally show the antidepressant benefits of THC. Controlling the dosage of THC in natural cannabis, marijuana, is difficult. Other studies have shown the potential risk of developing psychosis from excessive use of marijuana (see the “Marijuana and Mental Health” section of this article). But it does suggest a new line of research into drugs that enhance the effects of the brain's natural endo-cannabinoids. “We know that it's entirely possible to produce drugs which will enhance endo-cannabinoids for the treatment of pain, depression and anxiety.”

For further information on this study, see the press release from McGill University at: <http://www.mcgill.ca/newsroom/news/?ItemID=27677>.

Also see the original article by Francis Rodriguez Bambico, Noam Katz, Guy Debonnel, and Gabriella Gobbi in *The Journal of Neuroscience*, Oct. 2007; 27: 11700 – 11711: “Cannabinoids Elicit Antidepressant-Like Behavior and Activate Serotonergic Neurons through the Medial Prefrontal Cortex.”