ACKNOWLEDGED

Seamons, Colleen

From: Anne Bevan <smithandbevan@westnet.net.au>
Sent: Wednesday, 27 April 2011 4:55 PM
To: submissions
Subject: submission for the cannabis enquiry
Attachments: FSANZ.doc

Hello
please accept the attachment as a 'submission'
thank you
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ENTERED IN SMS / CDS
...../...../.....

Questions for submitters

1. Are you aware of any evidence that consumers believe low THC hemp foods have psychoactive effects?
Once the person ingests & doesn't get a 'high' they will then believe the packaging & labeling, and may decide not to buy / use the product.

2. Are you aware of any evidence that representations on low THC hemp foods (including labelling and advertising) mislead consumers by leading them to believe that low THC hemp foods have psychoactive effects when consumed?
& 7. Do you consider that trade practices legislation in Australia and New Zealand is sufficient to mitigate the potential risk that representations (including labelling and advertising) of hemp foods could suggest psychoactive properties relating to consumption of those foods?

I fail to see how this can in any way be a problem or a risk (of what?). This may explain why there hasn't been any research done by other countries on this; they realize, unlike us, that it is simply not a problem!

6 Do you agree that there are adequate controls currently in place, or that would be achieved by imposing maximum limits for THC, to mitigate any risk of high THC *Cannabis* varieties entering the food supply?

Surely if a seller wanted to pass off THC cannabis as a fibre hemp food, he would inflate the price! What drug dealer in his right mind (!) is going to pass off THC hemp as fibre hemp at current market rates for food! Hence very little risk of the THC variety being sold as food.

Medicinal Use of fibre hemp
An issue NOT raised by the Consultation Paper is the medicinal, use of fibre hemp (Low THC).
Consumers particularly those with anxiety or psychotic disorders stand to miss out on the anxiolytic and anti-psychotic properties of fibre hemp. By allowing fibre hemp consumption, many social and mental health problems could be radically ameliorated and it is bound to also save the government many health and welfare dollars at a time of decreasing funds for these areas.

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Antonio Waldo Zuardi. Cannabidiol: from an inactive cannabinoid to a drug with wide spectrum of action Rev Bras Psiquiatr. 2008

Mechanism of action

The way in which cannabinoids such as THC exert their effects on the human body is known as their "mechanism of action". This has recently become clearer with the discovery of two cannabinoid receptors CB1 and CB2 together with that of a chemical called "anandamide". Anandamide is an endogenous ligand, which literally means that it occurs naturally within the body (endogenous) and is a binding agent or "ligand". The full name of anandamide is arachidonoyl ethanolamide but it was nicknamed anandamide after the Sanskrit word for bliss "ananda" (1) . Anandamide has its effect by inhibiting cyclic AMP (part of the cellular energy gerneration process), through G-protein coupling in target cells, which cluster in areas of the central nervous system that mediate pain (2) , memory (3) , and other key functions.

Preliminary tests of pharmacology and behavioural activity support the similarity of anandamide to THC (4) . Both anandamide and THC bind weakly to the cannabinoid type one (CB1) receptors, which are found in the brain and are called partial agonists (5, 6). In contrast, cannabidiol (CBD) has little activity at CB1 but greater activity at the cannabinoid type 2 receptors (CB2) that are mostly located in the periphery, in lymphoid tissues (5). Both THC and CBD are neuroprotective antioxidants that have been shown to inhibit NMDA-mediated excitotoxicity under conditions of traumatic head injury, stroke and degenerative brain diseases (7).

The discovery of the endocannabinoid system (8) has provided new insights into a neuromodulatory scheme that may provide better explanations of, and treatments for, a wide variety of previously poorly treatable, often painful disorders (reviewed in (6, 9)).

It has recently been demonstrated that CBD also stimulates vanilloid pain receptors (VR1), inhibits uptake of the anandamide, and weakly inhibits its breakdown (10). These new findings have important implications in elucidating the pain-relieving, anti-inflammatory, and immunodulatory effects of CBD.

The combination of THC, CBD and essential oils in cannabis-based medicinal extracts may produce a therapeutic preparation whose benefits are greater than the sum of its parts (11).

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Delayed treatment with **cannabidiol** has a cerebro-protective action via a cannabinoid receptor-independent myeloperoxidase-inhibiting mechanism

Authors: Hayakawa, Kazuhide1; Mishima, Kenichi1; Nozako, Masanori1; Hazekawa, Mai1; Irie, Keiichi1; Fujioka, Masayuki1; Orito, Kensuke2; Abe, Kohji3; Hasebe, Nobuyoshi3; Egashira, Nobuaki1; Iwasaki, Katsunori; Fujiwara, Michihiro

Source: Journal of Neurochemistry, Volume 102, Number 5, September 2007 , pp. 1488-1496(9).

Unlike Δ9-THC, cannabidiol did not affect the excess release of glutamate in the cortex after occlusion.

Cannabidiol suppressed the decrease in cerebral blood flow by the failure of cerebral microcirculation after reperfusion and inhibited MPO activity in neutrophils. Furthermore, the number of MPO-immunopositive cells was reduced in the ipsilateral hemisphere in cannabidiol-treated group. **Cannbidiol provides potent and long-lasting neuroprotection through an anti-inflammatory CB1 receptor**-independent mechanism, suggesting that cannabidiol will have a palliative action and open new therapeutic possibilities for treating cerebrovascular disorders

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Distinct Effects of {Delta}9-Tetrahydrocannabinol and Cannabidiol on Neural Activation During Emotional Processing

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Arch Gen Psychiatry. 2009;66(1):95-105.

Context Cannabis use can both increase and reduce anxiety in humans. The neurophysiological substrates of these effects are unknown.

Objective To investigate the effects of 2 main psychoactive constituents of Cannabis sativa ({Delta}9-tetrahydrocannabinol [{Delta}9-THC] and cannabidiol [CBD]) on regional brain function during emotional processing.

Design Subjects were studied on 3 separate occasions using an event-related functional magnetic resonance imaging paradigm while viewing faces that implicitly elicited different levels of anxiety. Each scanning session was preceded by the ingestion of either 10 mg of {Delta}9-THC, 600 mg of CBD, or a placebo in a double-blind, randomized, placebo-controlled design.

Participants Fifteen healthy, English-native, right-handed men who had used cannabis 15 times or less in their life.

Main Outcome Measures Regional brain activation (blood oxygenation level–dependent response), electrodermal activity (skin conductance response [SCR]), and objective and subjective ratings of anxiety.

Results {Delta}9-Tetrahydrocannabinol increased anxiety, as well as levels of intoxication, sedation, and psychotic symptoms, whereas there was a trend for a reduction

in anxiety following administration of CBD. The number of SCR fluctuations during the processing of intensely fearful faces increased following administration of {Delta}9-THC but decreased following administration of CBD. Cannabidiol attenuated the blood oxygenation level–dependent signal in the amygdala and the anterior and posterior cingulate cortex while subjects were processing intensely fearful faces, and its suppression of the amygdalar and anterior cingulate responses was correlated with the concurrent reduction in SCR fluctuations. {Delta}9-Tetrahydrocannabinol mainly modulated activation in frontal and parietal areas.

Conclusions {Delta}9-Tetrahydrocannabinol and CBD had clearly distinct effects on the neural, electrodermal, and symptomatic response to fearful faces. The effects of CBD on activation in limbic and paralimbic regions may contribute to its ability to reduce autonomic arousal and subjective anxiety, whereas the anxiogenic effects of {Delta}9-THC may be related to effects in other brain regions.