

Suicide/ Schizophrenia

***Consequences of Acute and
Chronic Cannabis Use***

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**Presented to
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INTRODUCTION

It was with grave concern that I learned that the suicide rate in Australia is now one of the highest. It was also with grave concern that I read a paper in the *Drug and alcohol review 1994*, by MacDonald J. Christie and Gregory B. Chesher, "The Human toxicity of marijuana : a critique of a review by Nahas and Latour" (pp. 209-216).

Two statements by Christie and Chesher has compelled us to update our paper *Suicide/ schizophrenia : consequences of acute and chronic cannabis use* (Baker, 1988) presented to a number of committees in 1988, 1989 and 1990 with eighty-six references being cited and in the words of Dr J. McGrath, Director of Postgraduate Studies, Queensland Department of Health, "I agree with the main thrust of the paper, that cannabis can worsen schizophrenia." (Letter from Dr J. McGrath..., 1989).

This is an illness with a very high suicide rate (10%) and because of this every effort should be taken to keep people compliant with treatment. Abstinence from illicit drugs such as marijuana is **crucial** in maintaining people with the illness in remission.

Also, a statement by M. Bolton, Director of the Queensland Department of Health, "I certainly agree that the cannabis group of drugs have potentially serious and wide-ranging side effects and that there is no room for complacency by the community in this area." (Letter from M. Bolton..., 1989)

This data was supplied to Dr P. J. Tucker (Letter from Under Secretary [of Dr P. J. Tucker]..., [1989?]), chairman of an investigation committee into youth suicides in Queensland. This paper was also widely circulated to libraries which specialised in the drug and alcohol field.

Christie and Chesher (1994, p. 209) made the statement that Nahas and Latour "claimed that previous knowledge on the subject should now be revised in the light of 'new evidence'."

In the A.P.F.D.F.Y.¹ review paper (2) in 1988, the most critical studies were animal studies only. It was not until 1991 that actual human studies confirmed what the previous studies on animals were telling us. Christie and Chesher (1994, p. 214) recognised that " '...Andréasson et. al.'s suggestion to the effect that cannabis use is a stressor capable of triggering the psychotic breakdown, is an aspect that requires considerably more exploration' " (1994, p. 214).

In light of the critical need for the mental health services of Australia to have access to this important data to reduce the appalling suicide rate here in Australia, we are reviewing additional studies to correct inaccurate citations of scientific data that may have confused clear thinking and concerned people.

¹ Australian Parents for Drug-Free Youth.

THE YOUNGER AGE OF THE FIRST EXPERIENCE

Our 1988 paper stated "In the eighties the means age was 14." (Baker, 1988, p. 1) for marijuana use. Today, in 1996, the average age for first time drugs use has reached the frightening level of 12.5 years for alcohol and 11.8 years for marijuana (Commonwealth Department of Community Services and Health, 1990, p. 38; *Drugs, crime and society...*, 1989, p.10; Schlosser, 1984, p. 230-250; Commonwealth Department of Health, 1984, p. 230-250; Criminal Justice Commission, 1984, p. A 39).

It is known that the ability of children and teenagers to avoid indiscriminate use and abuse of drugs appears to be related to a number of social, economic, physiological, and psychological factors (Kozicki, 1986). Data from many national and state surveys (Rollin, 1995; Queensland School Drug Education Programs Evaluation Committee, 1988; Carlson, 1994; Schwartz, 1987) shows that primary school is the place to begin the attack on cannabis and alcohol abuse. If we wait until secondary school you're really only talking to the drug, not the person.

Marijuana has been described by schizophrenic patients as both anxiolytic and activating, alcohol as relaxing. These powerful drugs, combining in the young, undeveloped body, could very well have disastrous consequences in later life.

INCREASED POTENCY OF CANNABIS SINCE THE EARLY SIXTIES

The very important factor that must be considered is the increase in potency of dimethylheptyl analogs of Δ^9 -THC (Martin, 1995, p. 231 & 237; Järbe, et. al., 1989; Little, et. al., 1988), which is consistent with previous studies.

"Incorporation of a hydroxy at C11, along with this branched side chain resulted in an extremely potent cannabinoid with ED₅₀s of 0.01, 0.04, 0.16 and 0.04 μ mol/kg in depression of spontaneous activity..." (Martin, 1995, p. 231)

This is more than one hundredfold in several pharmacological measures.

The side chain plays a predominate role in the pharmacological potency of Δ^9 -THC. The addition of a dimethylheptyl side chain enhanced potency as much as fifty-fold. This potency is in addition to the existing hybrid varieties of cannabis which are continuing to gain popularity in Australia, such as skunk, with a THC content of up to 30% (Australian Bureau of Criminal Intelligence, 1993, p. 22; *Handbook for medical practitioners...*, 1993, p. 49).

Over thirty drug users have been admitted to the psychiatric ward of Sutherland Hospital, Sydney N.S.W., alone after using the potent, heavy-resin-type cannabis (*More potent drug in use*, 1995). This new strain might cause psychosis after several joints and could induce psychosis after just one or two joints. Sufferers might hear voices or have severe paranoia. This strain has more than one active ingredient of THC.

In light of the increased potency of dimethylheptyl analogs of Δ^9 -THC (Schuchard, 1992, p. 1; Wu, 1988; Stamler, 1985) it would be very unwise to think that cannabis does not play an

important part in some suicide and schizophrenia (U.S. Congress, House Committee on Government Operations, 1983; Russell, 1977; Gruber, 1994).

CANNABINOIDS BEING FAT SOLUBLE, THUS ALLOWING THC MOLECULES TO BE ABSORBED BY LIPIDS IN THE CELL MEMBRANE OF THE BRAIN WHICH CAN CAUSE MEMORY LOSS

"Higher doses of Δ^9 -THC can induce frank hallucinations, delusions, and paranoid feelings. Thinking becomes confused and disorganized; depersonalisation and altered time sense are accentuated. Anxiety reaching panic proportion may replace euphoria, often as a result of feeling that the drug-induced state will never end... Use of marihuana can also cause an acute exacerbation of symptomatology in stabilized schizophrenics, and is an independent risk factor for the development of schizophrenia." (*Extract from Goodman & Gilman, 1991, pp. 2-3*)

The effects of cannabis on memory has been well documented (Deahl, 1991, p. 249) and the conclusion reached is that persistently heavy marijuana use induces significant and surprisingly long-lasting deficits of short-term memory (Schwartz, 1990; Nahas, 1991, pp. 16-17).

"One may presume that individuals with learning disabilities who struggle to concentrate and to learn and remember important information in high school and college might be even more susceptible to marijuana-induced short-term memory deficits." (Schwartz, 1990)

In fact, a study in Alaska of the reading abilities of drug users has shown "that the average reading level for the subjects in this study is lower than 76 percent of [that of] the U.S. population." and "it is surprising that the correlations were of such a low magnitude. Indeed, these correlations indicate [that] level of schooling only accounts for 4.9 to 11.2 percent of the variance in reading level measures." and "...would suggest that current prevention and treatment materials are most likely written at reading levels that exceed that of the target population." (Johnson, 1995, pp. 77-79).

We must remember that it is impossible to learn without memory.

CANNABIS PSYCHOTIC DISORDER

Further research is urgently needed to help validate the diagnosis of cannabis psychosis by health professionals (Greyner, Luborsky and Solowij, 1995; Dews, 1972; Gruber and Pope, 1994), especially since up-to-date research has not been done on the new strain with very high potency.

In 1994, (Ashwood, 1995) a number of young people, aged between the late teens to late twenties, were affected by florid psychotic episodes.

"There was, and as is [sic] now, no known evidence for the cause of their problems, other than their heavy cannabis use, whether it [is] chronic or acute. Laboratory tests and histories failed to reveal any use of the psycho stimulants one would expect to see involved. Nor was there any pre existing latent or

prodromal psychotic disorder as the alternative explanation." (p. 49)

As Goodman and Gilman explain, "Because of the high prevalence of marijuana use, dysphoric reactions and psychiatric emergencies as a result of smoking marijuana... can also cause an acute exacerbation of symptomatology in stabilized schizophrenics, and is an independent risk factor for the development of schizophrenia." (p. 3).

In a study in Sweden, it was shown that

"...acute cannabis psychosis is characterized by

- (1) An increasing cannabis consumption just before the onset of symptoms...
- (3) The onset of the psychotic symptoms with rapid changing mood is characteristic. Most often the patient is hypomanic or manic and has megalomaniac ideas. At the same time he suffers from concentration difficulties and/or confusion. Paranoid symptoms are frequent.
- (4) The course of the illness is as a rule self-limiting. In the majority of cases, the patient has fully recovered within weeks but, on rare occasions, the process may last as long as 1 year.
- (5) Relapse into psychosis in connection with continuous abuse is common." (Tunving, 1987, p.87)

With the large increase in suicides and marijuana use in Australia, there is clearly an urgent need to research new varieties.

HEALTH CONSEQUENCES OF CHRONIC CANNABIS USE

It does appear

"that the bronchodilator properties of THC do not protect against the development of AHR² in smokers of both tobacco and marijuana. The results also suggest that respiratory irritants within marijuana smoke (including THC and other cannabinoids) augment, or possibly potentiate, the effects of tobacco in increasing airways reactivity. These findings are consistent with ... cheobronchial epithelial injury in marijuana-only smokers or with concomitant tobacco use ... and with more recent evidence of an additive effect of marijuana plus tobacco on bronchial pathology." (Tashkin, 1988, p.24)

²

AHR - Nonspecific airways hyperreactivity

Also, "Smoking marijuana may impair the body's immune system by preventing complete development of certain white blood cells. This may cause the immune system to function less effectively, making marijuana users more susceptible to disease." (Huberman³, 1989, p. 1)

It is evident from

"The increased number of inflammatory cells found in lung-washing (also called lung lavage) specimens of tobacco smokers ... may play a role in the pathogenesis of lung disease, such as emphysema Histopathologic changes in the airways ... and increased numbers of cells in the lung lavage of marijuana smokers have also been reported ... although the occurrence and/or development of significant lung disease from habitual marijuana smoking is not yet clear.". However, "...the increased numbers of macrophages in lung-lavage specimens obtained from marijuana smokers would suggest that there may be an enhanced recruitment of monocytes from the circulation...". (Barbers, 1989, p. 15)

"THC is almost 100 per cent protein bound. It is highly lipid soluble and plasma levels decline rapidly after inhalation followed by a slow elimination phase." (Levy, 1990, p. 644) We know Δ^9 -THC crosses the placenta and also "In heavy users breast milk analysis of Δ^9 -THC [reveals] an eightfold accumulation compared with maternal plasma. The nursing infant can absorb Δ^9 -THC from mother's milk." (p. 645).

"Using the Brazelton Neonatal Behavioral Assessment Scale, smoking cannabinoids during pregnancy correlated with a decrease in the likelihood of the offspring to respond to light directed at their eyes (46% did not respond ... in contrast to 16% of babies born to matched non-users)... Marked tremor, mainly around day 9 of life, and startle reflex are other characteristic features described in newborns of heavy cannabinoid users." (p. 646)

If we only compare marijuana with tobacco smoking, the "...total burden of tar to the lungs and absorption of carbon monoxide ... are four times greater when smoking marijuana." (Wu, 1988, pp. 30-31).

It would appear that after reviewing the data on cannabis over the last fifteen years we can reach the conclusion that there appears to be some scientists in Australia who have adopted an attitude which among clear thinking scientists *would be generally regarded as unacceptable*.

SUICIDE/SCHIZOPHRENIA

Among adolescent substance abusers who had attempted suicide the drugs of choice were marijuana and/or hashish (61%) (Berman, 1990, p. 310; Reynolds, 1988, p. 256).

In a study in Stockholm, the hypothesis is that cannabis does play an aetiological role in

³Eliezer Huberman is director of Argonne National Laboratory's division of biological and medical research.

schizophrenia (Andréasson, 1989, p. 505).

We find a study of U.S. Army personnel in Europe showed "... the odds of developing schizophrenia increase in a linear fashion along frequency of use; both among subjects with a psychiatric diagnosis *and* in those without detectable disorders." "...community samples have found the lifetime prevalence of cannabis use to be significantly higher among schizophrenics than in comparison groups, including individuals suffering from other psychiatric disorders." (Negrete, 1989, p. 350).

A pilot study by Dr. I. L. Fowler of N.S.W. (1992) explains the difficulty of distinguishing "between substance use and abuse in schizophrenic subjects as their everyday functioning is impaired by their illness. Furthermore, the relative contributions of substance abuse and the schizophrenic illness to levels of functioning are unclear." (p. 14).

"Drug urine screens may potentially improve the reliability of self report." (p. 19) It is important to be aware that "Marijuana using schizophrenic patients are reported to have significantly more delusional and hallucinatory activity" (p. 24) and that marijuana was the most preferred drug (p. 26). Another problem in the study of an association between cannabis and schizophrenia is the possibility that the use of other drugs might compound the association or enhance the drug, as is the case with alcohol and tobacco which, when combined, increase the danger of certain chemicals. The same may be the case with marijuana.

Schizophrenic patients "must be alerted to the special hazard of marijuana [like] the patient with porphyria to the special hazard of barbiturates. It is the physician's responsibility to alert all high-risk patients about the possibility of...interactions between their illness and the substances they use." (Treffert, 1978, p. 1215).

The monograph series no. 25 (Hall, 1994) states

"Cannabis is a psychoactive drug that is probably psychotomimetic in high doses, and its use seems to be relatively common among schizophrenic patients...There is also anecdotal clinical evidence that schizophrenic patients who use cannabis and other drugs experience exacerbations of symptoms, and have a worse clinical course, with more frequent psychotic episodes, than those who do not." (p.177)

Andréasson et. al.'s study provides strong evidence of an association between cannabis use and schizophrenia which is not completely explained by prior psychiatric history. According to Gabriel Nahas (Nahas, Latour & Hardy, [1992]), "Results of standard in vitro and in vivo toxicological tests performed on animal preparations administered marijuana extracts were good predictors of the long term physiopathologic manifestations observed twenty years later on in chronic marijuana smokers." (p. [12]).

"The property of cannabis to induce long-lasting mental disturbances in Western man now epidemiologically documented, would confirm older anecdotal reports from mediaeval Islam (1396), India (1878-1972), Egypt (1843-1925), Brazil (1955), Bahamas (1970) and Jamaica (1976). Cannabis-induced psychosis would provide evidence that the repetitive disturbance of brain neuro-transmission carries the most serious risk of impairing lastingly

the basic biochemical neural mechanisms which control coherent behaviour."
(pp. [11-12])

CONCLUSION

Suicides in Australia may be much greater than 27 per 100,000 because many causes of death are not listed as suicide e.g. fatalities where drivers run head on into oncoming traffic or run off the road and into trees could be suicide. It is well known that drugs and/or alcohol reduce normal inhibitions against suicide and marijuana is the illegal drug most used by those under twenty-five.

We must note the younger age of the first experience, increased potency of Δ^9 -THC and cannabis analogs of Δ^9 -THC, that cannabinoids are fat soluble thus allowing THC molecules to be absorbed by lipids in the cell membranes.

The 63 cannabinoids in cannabis are of no chemical use to the cell. They are not eliminated quickly, but remain embedded for months at a time. The other 363 chemicals which are only found in the cannabis plant will block the transmission of sodium, potassium, calcium, chlorides and the complex messenger chemicals called neurotransmitters that must all go in and out through membrane channels. This disturbance in chemical message transport affects thought, behaviour, feelings, memory, motor co-ordination and glandular activity.

Juan C. Negrete (1989) quotes that

"A recent review of the evidence arrived at the conclusion that it is quite certain that cannabis is capable of causing a toxic psychosis with confusion, delusions, hallucinations and affective symptoms; that the reports about a 'functional' cannabis psychosis which persists after cessation of use are less convincing; and that a chronic course is likely to be seen only in individuals who continue using the drug. Such an appraisal would appear fairly accurate and should provide clinicians with helpful guidelines to conduct their work." (p. 368).

Andréasson et. al.'s study provides strong evidence of an association between cannabis use and schizophrenia which is not completely explained by prior psychiatric history. In Australia we must begin a sharp learning curve about this dangerous drug. Our recommendation will take a multi-component approach to give us more chance of success in regards to this dangerous drug.

RECOMMENDATIONS

1. That we implement, with extreme urgency, drug education on cannabis starting in grade four and going through to university, using the latest scientific data on the harmful effects of marijuana as supplied by the chairman of the Australian Medical Association Queensland Branch's Working Party on Cannabis, Dr Dana Wainwright, or other credible organisations such as Drug Arm in Queensland, PRYDE⁴ in Australia Inc., RAIN⁵ in Victoria, APFDFY⁶ in Queensland, APADA⁷ in NSW, and

⁴Parents Reaching Youth Through Drug Education

⁵Relatives Against Injecting Narcotics

Teen Challenge NSW Inc.

2. That we accept the recommendation on pages 7 and 8 in the Report of the International Narcotics Control Board for 1995, especially (d) which recommends the implementation of the 40 recommendations formulated by the Financial Action Task Force.

This is not about putting people in prison. This is about taking the ill-gotten gains from people who receive monies from making, financing, or selling drugs. We must realise that our generation is faced with a more subtle, organised, internal enemy to democracy. These people are using the power and position which their large sums of money give them in the community. Once we take that away they are required to take chances to acquire wealth because they are unable to hire young people to do their dirty work.

3. That we urgently implement a media campaign, like the "Bloody Idiot" alcohol advertisements on T.V., which inform the community of the harmful effects of marijuana use.
4. We should regard the large number of fat loving cannabinoids found in cannabis, the dramatic increase of Δ^9 -THC potency, "... that the 11-hydroxylation in conjunction with the substituted heptyl side chain increased potency more than a hundredfold in several pharmacological measures." (Martin, et. al., 1995, p. 237), and that a comparison of the pharmacological properties of Δ^9 -THC to Δ^8 -THC-DMH clearly demonstrated an enhancement of potency by approximately 50-fold when a dimethylheptyl is substituted for a pentyl side chain (pp. 237-238).

As we observed earlier, Δ^9 -THC has increased in potency from .5 percent in the sixties to thirty percent in the nineties. Research into the effects of cannabis with high potency on the young developing body must be implemented wherever any research on high potency alone is being done at present. We must gather support and evaluate this data for the safety of future generations.

5. There is a need to re-evaluate current definitions of use, abuse and dependence in schizophrenia and to re-evaluate current measures used in assessing substance abuse disorders in schizophrenia. Drug urine screens must be used to improve the reliability of self reporting. Single drug urine screens are less useful with short, half life drugs or where there is infrequent or sporadic drug use.

⁶ Australian Parents for Drug-Free Youth

⁷ Australian Pharmacists Against Drug Abuse

6. Clearly with the large increase in suicides in Australia further research is urgently needed to help validate the diagnosis of cannabis psychosis by the health profession, especially since up-to-date research has not been done on the new strains with very high potency.
7. The government should obtain the rights to place on CD-ROM and market to Asia and the Oceania region Marijuana: an annotated bibliography, Volumes I and II (see below).

A significant amount of this data is already available on diskette and it would be an easy matter to load it on to a CD-ROM. The CD-ROM should be donated to every library in Australia and offered to the overseas regions already mentioned. This would become an extremely useful educational and research tool.

This report has not touched on the many other health consequences of acute and chronic cannabis use. The community must recognise that cannabis affects all systems of the body. We cannot ignore the many scientific studies done on cannabis, not one of which gives it a clean bill of health.

We would be neglecting our duty of care to the community if we continue to dismiss the mounting evidence about this dangerous drug.

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HOW TO LOCATE SCIENTIFIC MARIJUANA RESEARCH INFORMATION

Marijuana: An Annotated Bibliography, Volumes I and II, and its eight supplements containing over 11,600 citations (supplements from 1985 on are also available on disk in ASCII format) is published by the Macmillan Publishing Co., 200 Brown St., Riverside, NJ, 609-461-6500. The listings are alphabetical by author and there is an extensive subject and key-word index.

A computer search service is available to the public for a minimal fee and includes materials published from 1975 to 1992. If you are interested, contact:

Beverley Urbanek, The University of Mississippi
School of Pharmacy/RIPS, university, MS 38677
Phone: 601-232-5914 Fax: 601-232-5118⁸

⁸How to Locate Scientific Marijuana Research Information, Danvers, MA : Committees of Correspondence, Inc.

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